GUIDELINES FOR THE MANAGEMENT OF COMMON MEDICAL EMERGENCIES AND FOR THE USE OF ANTIMICROBIAL DRUGS
St George's Hospital
August 2019
70th edition

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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 references used to support the advice are on the Intranet version, which can be found at the St George’s NHS Trust Intranet website. If you have any comments or questions please send them to the Link Consultant named at the beginning of the section concerned. The 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, Twm Davies
Pharmacy liaison: Wendy Pullinger
Arrhythmias associated with cardiac arrest are divided into (1) shockable (VF/VT) and (2) non-shockable (asystole and PEA) rhythms. Other than need for defibrillation in VF/VT, subsequent management is identical. The ALS algorithm provides a standardised approach to manage cardiac arrest in adults:

- Confirm cardiac arrest – check for signs of breathing and pulse simultaneously.
- Call resuscitation team (2222).
- 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. Plan actions before pausing CPR for rhythm analysis and communicate these to the team.
- Stop chest compressions to confirm rhythm from the ECG.

**Shockable rhythms (VF/VT)**

VF/VT is the first monitored rhythm in ~25% of all cardiac arrests and in ~25% at some stage during resuscitation of cardiac arrests with initial documented rhythm of asystole or PEA. Once VF/VT is confirmed:

1. Resume chest compressions immediately. Simultaneously, the designated person should select the appropriate energy on the defibrillator (150-200J biphasic for the 1st shock and 150-360J biphasic for subsequent shocks – ENERGY LEVEL SPECIFIED BY MANUFACTURER – and then press the charge button.
2. As the defibrillator is charged, warn all rescuers other than the individual doing chest compressions to “stand clear”. Remove any oxygen delivery device as appropriate. Ensure rescuer giving compressions is the only person touching the patient.
3. Once the defibrillator is charged, tell the rescuer performing chest compressions to “stand clear”. When clear, give the shock.
4. Without reassessing the rhythm or feeling for a pulse, restart CPR using a ratio of 30:2, starting with chest compressions. Continue CPR for 2 min. The team leader prepares the team for the next pause in CPR.
5. Pause briefly to check the monitor: if VF/VT, repeat steps 1-5 above and deliver a 2nd shock. If VF/VT persists repeat steps 1-3 above and deliver a 3rd shock. Resume chest compressions immediately and then give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR.
6. Repeat 2 min CPR – rhythm/pulse check – defibrillation sequence if VF/VT persists.
7. Give further adrenaline 1 mg IV after alternate shocks (i.e. ~ every 3-5 min).

**If organised electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of return of spontaneous circulation (ROSC):**

- Check a central pulse and end-tidal carbon dioxide [ET CO2] trace if available?
- If there is evidence of ROSC, start post-resuscitation care (induced hypothermia and primary percutaneous coronary intervention (PPCI) should be considered).
- If no signs of ROSC, continue CPR and switch to the non-shockable algorithm.

**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 and not exceed a few seconds (ideally <5s). Longer interruptions to chest compressions reduce the chance of a shock restoring spontaneous circulation. If an organised rhythm is seen during a 2-minute period of CPR, do not interrupt compressions to palpate a pulse unless the patient shows signs suggesting ROSC (this may include a sudden increase in [ET CO2]). If there is doubt about the existence of a pulse with an organised rhythm, resume CPR. If the patient has ROSC, begin post-resuscitation care.

**Precordial thump:** A precordial thump has very low success rate for cardioversion of a shockable rhythm and is only likely to succeed if given within few seconds of the onset of a shockable rhythm. There is more success with pulseless VT than VF.
Delivery of a thump must not delay calling for help or accessing a defibrillator. It is therefore appropriate only when several clinicians are present at a witnessed, monitored arrest, and when a defibrillator is not immediately to hand. In practice, this is likely to be in a monitored environment eg A&E resus room, ICU, CCU, cardiac catheter lab or pacemaker room. A precordial thump should be undertaken immediately after confirmation of cardiac arrest and only by healthcare professionals trained in the technique. Using the ulnar edge of a tightly clenched fist, deliver a sharp impact to the lower half of the sternum from a height of ~20 cm, then retract immediately to create an impulse-like stimulus. There are very few reports of a precordial thump converting a perfusing rhythm to a non-perfusing rhythm.


Unresponsive?
Not breathing or only occasional gasps

CPR 30:2
Attach defibrillator/monitor. Minimise interruptions

Assess rhythm

Shockable
VF/Pulseless VT

1 Shock
Immediately resume CPR for 2 mins
Minimal interruptions

Non-shockable
PEA/Asystole

Return of spontaneous circulation

Immediately resume CPR for 2 mins
Minimal interruptions

Immediate Post-arrest Care
• Use ABCDE approach
• Controlled oxygenation & ventilation
• 12-lead ECG
• Treat precipitation cause
• Temperature control / therapeutic hypothermia

During CPR:
• Ensure high quality CPR (rate, depth, recoil);
• Plan actions before interrupting CPR;
• Give oxygen;
• Consider advanced airway & capnography;
• Continuous chest compressions when advanced airway in place;
• Vascular access - iv or intraosseous;
• Give Adrenaline every 3-5mins;
• Correct irreversible causes

Reversible Causes: Hypoxia, Hypovolaemia, Hypokalaemia, Hyperkalaemia; Toxins, Thrombosis (cardiac or pulmonary), Tamponade (cardiac), Tension pneumothorax

Non-shockable rhythms (PEA and asystole) Pulseless electrical activity (PEA) is defined as the absence of any palpable pulse in the presence of cardiac electrical activity expected to produce cardiac output. These patients often have some mechanical
myocardial contractions that are too weak to produce a detectable pulse or blood pressure – sometimes described as ‘pseudo-PEA’.

PEA may be caused by reversible conditions that can be treated if identified and corrected. Survival following cardiac arrest with asystole or PEA is unlikely unless a reversible cause can be found and treated effectively.

**Sequence of actions for PEA**
- Start CPR 30:2. Give adrenaline 1 mg as soon IV access is achieved.
- Continue CPR 30:2 until the airway is secured, then continue chest compressions without pausing during ventilation. Consider and correct reversible causes of PEA
- Recheck the patient after 2 min:
  - If there is still no pulse and no change in the ECG appearance:
    - Continue CPR; Recheck the patient after 2 min and proceed accordingly.
    - Give further adrenaline 1 mg every 3-5 min (alternate loops).
  - If VF/VT, change to the shockable rhythm algorithm. If a pulse is present, start post-resuscitation care.

**Sequence of actions for asystole**
- Start CPR 30:2. Without stopping CPR, check that the leads are attached correctly.
- Give adrenaline 1 mg as soon as IV access is achieved.
- Continue CPR 30:2 until the airway is secured, then continue chest compression without pausing during ventilation.
- Consider possible reversible causes of PEA and correct any that are identified.
- Recheck the rhythm after 2 min and proceed accordingly.
- If VF/VT, change to the shockable rhythm algorithm.
- Give adrenaline 1 mg IV every 3-5 min (alternate loops).

Whenever a diagnosis of asystole is made, check the ECG carefully for P waves as the patient may respond to cardiac pacing when there is ventricular standstill with continuing P waves. There is no value attempting pacing in true asystole.
Patients require admission and urgent treatment when blood pressure is known to have risen rapidly or is severely raised, such that the systolic pressure is equal to or above 220mmHg and/or diastolic pressure equal to or above 120mmHg. Urgent treatment is also needed for lower blood pressure levels if there is evidence of severe or life-threatening end-organ damage. Patients with severe hypertension and retinopathy (haemorrhages, exudates or papilloedema) have malignant hypertension, which is a very serious and potentially lethal condition if not properly recognised and treated. Patients with malignant hypertension should be treated in a high dependency unit (HDU) or intensive therapy unit (ITU) and urgent advice from the Blood Pressure Unit should be sought.

**WHEN THERE IS ACUTE, LIFE-THREATENING ORGAN DAMAGE.** The situation is a true hypertension emergency when there is acute and life-threatening organ damage, such as hypertensive encephalopathy (headache, lethargy, confusion, seizures, coma), intra-cranial haemorrhage, aortic dissection, acute coronary syndromes (unstable angina/acute myocardial infarction), acute left ventricular failure with pulmonary oedema, or pre-eclampsia/eclampsia. The initial aim of treatment is to lower blood pressure in a rapid (within 2-4 hours), controlled but not overzealous way, to safe (not normal) levels – about 160mmHg systolic and 100mmHg diastolic, with the maximum initial fall in blood pressure not exceeding 25% of the presenting value. Too rapid a fall in pressure may precipitate cerebral or myocardial infarction, or acute renal failure. Always seek advice from the Blood Pressure Unit.

- **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. In many instances, patients will be salt and water deplete and will require fluid replacement with normal saline in addition to antihypertensive agents.

- **Sodium nitroprusside** (SNP) is the parenteral drug of choice for most hypertensive emergencies. SNP should only be given in HDU or ITU with continuous intra-arterial blood pressure 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,** a combined α- and β-blocker, can be used in most hypertensive emergencies and urgencies and is a logical option for patients with ischaemic heart disease, aortic dissection or stroke; it is also safe 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.

- **Glyceryl trinitrate** (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, blood pressure 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**, an arteriolar dilator, 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**, 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.

**Malignant Hypertension** Malignant (accelerated) hypertension is a syndrome characterised by severely elevated blood pressure accompanied by retinopathy (retinal haemorrhages, exudates or papilloedema), visual impairment, nephropathy AKI (malignant nephrosclerosis) with or without hypertensive encephalopathy (confusion, lethargy, or coma) and microangiopathic haemolytic anaemia. Malignant hypertension is a very serious condition with very poor prognosis if not properly diagnosed and treated. It is usually a consequence of untreated essential or secondary hypertension. 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.

**Aortic Dissection** Aortic dissection must be excluded in any patient presenting with severe hypertension and chest, back, or abdominal pain. It is life-threatening with very poor prognosis if not treated. The initial treatment is a combination of IV 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.

**WHEN THERE IS NO LIFE-THREATENING ORGAN DAMAGE**, the situation becomes Hypertensive Urgency rather than an emergency. **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). ACE inhibitors are best given only after advice from the Blood Pressure Unit.
• 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, phaeochromocytoma, primary hyperaldosteronism, other adrenal pathology or underlying renal disease).

Advice on the investigation and treatment of all types of hypertension can be obtained during weekdays (08.30-17.00) from the Blood Pressure Unit at St George’s (ext 4461).
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 (NSTE-ACS).

**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, fax the ECG to CCU (0208 725 0294) and immediately bleep the Cardiology SpR (bleep 6002).

- **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 NSTE-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 NSTE-ACS.

Repeat the ECG if the patient’s symptoms change or if the initial ECG is non-diagnostic but clinical suspicion remains high.

**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).
The rapid 0/3 hour ‘rule-out’ algorithm

- Acute chest pain

  hs-cTn < 14
  - Pain > 6h
  - Pain < 6h

  Re-test hs-cTn 3h
  - hs-cTn no change
  - hs-cTn > 14 or >20% increase

  - Pain-free, GRACE < 140, differential diagnoses excluded
  - Discharge to GP/RACPC
  - Work-up differential diagnosis
  - Refer to medical team if pain-free and GRACE < 140. If high-risk features discuss with cardiology

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**GRACE Risk Score 2.0**

**Calculator**

1. **INPUT DATA**
2. **DEATH / DEATH MI RESULTS**

<table>
<thead>
<tr>
<th>Death</th>
<th>Time</th>
<th>% Risk (Score)</th>
<th>Histograms</th>
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<tr>
<td></td>
<td>In hospital</td>
<td>0.4</td>
<td>Not available</td>
</tr>
<tr>
<td></td>
<td>6 months</td>
<td>1.6</td>
<td>(70) Not available</td>
</tr>
<tr>
<td></td>
<td>1 year</td>
<td>1.6</td>
<td></td>
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<tr>
<td></td>
<td>3 years</td>
<td>7.5</td>
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**Number of patients by risk group for 1-year death**

Area plot: distribution (log scale) of risk based on the entire GRACE population of 102,345 patients.

- Line: risk of death or death/MI
- Vertical bar: individual risk of death or death/MI
  - green = low, yellow = intermediate, red = high

Disclaimer

This risk scoring tool is intended for use by clinicians, in conjunction with individual patient assessment. We assume no responsibility for how you use or interpret the GRACE 2.0 ACS Risk Calculator app or any other information provided on this website. Read more.
Refer the patient immediately to Cardiology for Primary Percutaneous Intervention (1° PCI); the target door-balloon time is within 60mins. Establish an IV line. Take blood samples for full blood count, U&Es, glucose, markers of cardiac damage (see Appendix 1) and lipids. A chest x-ray should be requested but should not delay therapy.

**Aspirin**  As soon as possible give soluble Aspirin 300 mg to be chewed. This should be followed by Aspirin 75mg daily. 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.

**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.

**Oxygen**  In patients at no risk of hypercapnic respiratory failure, controlled oxygen should be administered if oxygen saturation (SpO₂) is <94%. 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% until blood gas analysis is available.

**Anticoagulation after 1° PCI**
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).


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. 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 \(\leq 40\%\), consider an aldosterone antagonist such as Eplerenone 25 mg od (contraindicated if eGFR is \(< 30\text{mL/min}/1.73\text{m2}\), or potassium \(> 5.0 \text{mmol/L}\) at initiation).

Nitrates  Give IV Glyceryl Trinitrate at a dose of 1-10mg per hour for continuing chest pain or pulmonary oedema if the systolic blood pressure is \(> 90\text{mmHg}\) 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.

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:  Please be aware that certain sub-groups of patients may be on Ticagrelor 60mg bd for up to 3 years following their PCI (STEMI / NSTE-ACS).

Please DO NOT change this regimen without discussion with the responsible consultant interventional cardiologist for that patient.
Non ST-segment elevation acute coronary syndromes (NSTE-ACS) is an umbrella term which includes patients presenting with unstable angina (UA) and non ST-segment elevation myocardial infarction (NSTEMI). Patients with NSTE-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. 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 NSTE-ACS. Cardiology use the GRACE 2 calculator to score such patients, which helps guide the timing of angiography [http://gracescore.org/WebSite/WebVersion.aspx](http://gracescore.org/WebSite/WebVersion.aspx). If the GRACE 2 score is > 140, or there are other features of High Risk ACS (persistent ST depression > 1mm and on-going chest pain, T wave inversion or biphasic T waves in leads V1-V4 – Wellen’s syndrome) you should contact the Cardiology SpR directly (bleep 6002). They will then review the patient and transfer 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 (M-F 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 or a history of intracranial haemorrhage). This should be followed by Ticagrelor 90 mg twice daily. Patients established on warfarin or direct oral anticoagulants should be discussed with the cardiology team first.

- 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).

- In patients at no risk of hypercapnic respiratory failure controlled oxygen should be administered if oxygen saturation (SpO₂) is <94%. 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% until blood gas analysis is available. **Do not give supplementary oxygen routinely in patients who are achieving target SpO₂ of 94-99% on room air.**

- Give Fondaparinux 2.5 mg SC od for 48–72 hours or until the patient has undergone angiography (maximum 8 days) except when patient is taking a Direct Oral
Anticoagulant or Warfarin and has 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-ACS (see STEMI-ACS section) the following are also recommended in NSTE-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 >11 mmol/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 sections for dose and contraindications). The cardiology SpR should be informed if signs of ongoing ischaemic sounding chest pain requiring a GTN infusion.

**Further Risk Assessment**

- Cardiac biomarkers (Troponin T – see Appendix 1) should be taken on admission and at 3 and 6 hours from admission.

Patients should be risk assessed using the GRACE Score (http://www.outcomes.umassmed.org/GRACE/acs_risk/acs_risk_content.html) and cardiac catherisation undertaken accordingly:

1) **Very high risk cases who should have angiography within hours (<2 hours by ESC guidelines) are those with at least one of the following:**

   - Haemodynamic instability or cardiogenic shock thought to be due to ischaemia
   - Recurrent or ongoing cardiac type chest pain refractory to medical treatment
   - Life-threatening arrhythmias or cardiac arrest
   - Mechanical complications of MI
   - Acute heart failure (due to ECG or symptomatic evidence of ischaemia)
   - Recurrent dynamic ST-T wave changes, particularly with intermittent ST-elevation

2) **High risk cases who should have urgent angiography (<24 hours by ESC guidelines) are those with at least one of the following:**

   - GRACE Score > 140
   - Isolated (i.e. no other high risk features) Troponin T value > 250 with a significant change (delta) over 2 measurements and presentation compatible with MI
Dynamic ST-depression or deep T-wave inversion (symptomatic or silent), especially precordial leads (Wellen’s pattern)

- For all other NSTEMI, the current NICE recommendation is for angiography within 72 hours.
- Low risk patients or patients unsuitable for early angiography should be discussed with the cardiology registrar or ACS practitioner, to determine management strategy (invasive versus conservative) and whether this is to be performed as an inpatient or outpatient.

**Normal or equivocal ECG**

For patients presenting with ischaemic sounding chest pain and a normal or equivocal ECG, they should have cardiac biomarkers performed on admission and at 3 and 6 hours.

- If the initial troponin T concentration is > 14 ng/L and the 3-hour troponin increases by over 7 ng/L, the patient should be treated for NSTE-ACS.
- If the initial troponin T concentration is ≤ 14 ng/L and the 3-hour troponin > increases by over 7 ng/L and is > 14 ng/L, the patient should be treated for NSTE-ACS.
- If the initial troponin is > 14 ng/L with no significant increase at 3-hours, then review the clinical history and consider other clinical conditions (e.g. trauma, heart failure, pulmonary embolus, aortic valve disease, hypertrophic cardiomyopathy, renal failure, hypotension, sepsis) and investigate and manage as appropriate.
- If the initial 3 and 6-hour troponins are ≤ 14 ng/L then the patient should be assessed with the GRACE Score. Patients with a high GRACE Score or unstable symptoms should be considered for in-patient assessment (review by ACS practitioner or Cardiologist). Patients with a low-intermediate GRACE Score (<140) and stable symptoms should be suitable for out-patient assessment (e.g. Rapid Access Chest Pain Clinic RACPC).

**ADDENDUM:**

Please be aware that certain sub-groups of patients may be on Ticagrelor 60mg bd for up to 3 years following their PCI (STEMI / NSTE-ACS).

Please DO NOT change this regimen without discussion with the responsible consultant interventional cardiologist for that patient.
MANAGEMENT OF ACUTE HEART FAILURE
Link Consultants: Dr Robin Ray & Dr Lisa Anderson*

Acute decompensated heart failure is a life-threatening condition with 30-day mortality of 15% in those with NT-proBNP > 5000ng/L and 5% in those with NT-proBNP < 5000ng/L.\textsuperscript{1} Patients with heart failure should generally be discharged from hospital only when their clinical condition is stable and the management plan is optimised.\textsuperscript{1}

Community heart failure nurse follow-up reduces the 3-month risk of re-admission by 35%. 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**
Acute heart failure is the leading cause of hospital admission in people 65 years or older in the UK and one in seven people > 85 years of age has heart failure. Therefore it should be in the differential of all elderly patients presenting with breathlessness. If heart failure is suspected, request serum NT-proBNP with the U+E sample.

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>&lt;50</th>
<th>50-75</th>
<th>&gt;75</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Heart Failure likely if NT-proBNP (ng/L) is</td>
<td>&gt;450</td>
<td>&gt;900</td>
<td>&gt;1800</td>
</tr>
</tbody>
</table>

If the NT-proBNP is normal (< 300ng/L), search for an alternative diagnosis. If the NT-proBNP is significantly elevated (see above) acute heart failure is likely and should be confirmed by echocardiography if not already documented. All patients admitted with a new diagnosis of heart failure (with raised NT-proBNP) should have an in-patient echocardiogram prior to discharge (ideally within 48 hours of admission). If the NT-proBNP concentration is intermediate (above 300 ng/L but below acute heart failure levels), reconsider the diagnosis. If after full reassessment, heart failure is likely, request an echocardiogram.

**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\textsuperscript{2}**

**Acute pulmonary oedema:**
- **Call the cardiology SpR (Bleep 6002) to arrange admission to CCU**
- \( O_2 \) to maintain \( SaO_2 \) (95-98%)
- IV furosemide 40-80mg bolus followed by an infusion at 5-20 mg/hr if required
- Consider IV GTN infusion (10-200 micrograms/min) for patients with concomitant myocardial ischaemia, severe hypertension or regurgitant aortic or mitral valve disease.
- 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)

Please note that pulmonary oedema is an unpredictable and potentially fatal cardiac emergency. Although the condition reverses rapidly with diuretics, it will recur with precipitous onset if the underlying cause is not addressed. An audit of the pulmonary
oedema patients admitted to CCU in 2017 showed 29% had critical valve disease requiring inpatient surgery/TAVI and 6% had critical ischaemia requiring CABGs.

**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, plasma U&E’s, creatinine, NT-proBNP TFTs, LFTs, troponin, glucose and lipids; arterial blood gases if oxygen saturation is low or oxygen is required to maintain saturation.
- Review medication: stop Ca\(^{2+}\) channel blockers and NSAIDs where possible.
- In unstable patients with diabetes, switch to insulin sliding scale.
- Patients already on ACE and/or Beta-Blockers: efforts should be made to maintain usual medication doses even if the first dose(s) need to be omitted due to hypotension. Withdrawal of beta-blockers in acute heart failure patients has been shown to be associated with increased mortality risk.
- If patient presents in fast atrial fibrillation and pulmonary oedema, consider digoxin initially until beta-blockers can be initiated and up-titrated.

**Management of Chronic Heart Failure with Left Ventricular Systolic Dysfunction**¹

Diuretics are used for the relief of congestive symptoms and fluid retention in patients. They should be titrated (up and down) according to need, following the initiation of heart failure therapies:
1. Start ACE inhibitor (e.g. ramipril) and titrate upwards. If not tolerated (e.g. due to persistent cough) try an angiotensin II receptor antagonist (e.g. candesartan).
2. Start a beta-blocker, unless contra-indicated, (e.g. bisoprolol) and titrate upwards
3. Add a Mineralocorticoid Receptor Antagonist (Spironolactone or Eplerenone 12.5–25mg od).
4. Referral to heart failure team for consideration of additional therapies including ivabradine, sacubitril valsartan, device therapy and percutaneous procedures.

For those with heart failure with preserved LV function (HFpEF), fluid balance and management of blood pressure is all that is required.

**DISCHARGE AND FOLLOW-UP**

All acute heart failure admissions need community heart failure nurse follow-up after discharge. This can be arranged 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.
- If a new diagnosis of heart failure, document key echocardiographic findings in discharge summary.
- 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 Diagnosing and Managing Acute Heart Failure in Adults. NICE Clinical Guideline 187, October 2014
DISORDERS OF CARDIAC RHYTHM
Link Consultant: Dr Elijah Behr

SINUS BRADYCARDIA
This requires no treatment unless it is causing symptoms. If treatment is deemed necessary, give atropine 600-1200 micrograms 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.

### Indications for Temporary Pacing – Emergency/Acute

<table>
<thead>
<tr>
<th>Acute MI with:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asystole</td>
</tr>
<tr>
<td>Symptomatic bradycardia not responsive to atropine</td>
</tr>
<tr>
<td>Bilateral bundle branch block (alternating BBB or RBBB with alternating LAHB/LPHB</td>
</tr>
<tr>
<td>New or indeterminate age bifascicular block with 1st degree AV block</td>
</tr>
<tr>
<td>2nd or 3rd degree AV block after an acute anterior MI</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bradycardia not associated with acute MI:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asystole</td>
</tr>
<tr>
<td>Any symptomatic bradycardia resistant to medication</td>
</tr>
<tr>
<td>2nd or 3rd degree AV block with haemodynamic compromise or syncope at rest</td>
</tr>
<tr>
<td>Asymptomatic AV block with severe bradychardia (&lt;30bpm) +/- QRS&gt;120ms +/- QTc prolongation &gt;500ms</td>
</tr>
</tbody>
</table>

| VT secondary to bradycardia eg Torsades de Pointes |
| Suppression of drug-resistant VT or SVT |
| Drug overdose, eg. digoxin, beta blockers, verapamil |

<table>
<thead>
<tr>
<th>Indications for Temporary Pacing – Elective</th>
</tr>
</thead>
<tbody>
<tr>
<td>Support for procedures that may promote bradycardia</td>
</tr>
<tr>
<td>General anaesthesia with: 2nd or 3rd degree AV block; intermittent AV block</td>
</tr>
<tr>
<td>Cardiac surgery when epicardial pacing has failed</td>
</tr>
<tr>
<td>Rarely considered for coronary angioplasty</td>
</tr>
</tbody>
</table>

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 bifasicular 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.
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 request form completed electronically and the Ultrasound (US) Department should be contacted – the scan can then be performed on the next inpatient list.
- Outpatients suspected of DVT are assessed by ED or Urgent Care Centre according to the the Emergency Department Clinical Guideline: Assessment and management of patients with suspected DVT guidelines. If an ultrasound scan is indicated, a time is arranged by ringing ext 1473. Complete an electronic request form and arrange for the patient to be sent to the US department. The radiology request should include the pre-test probability score (PTP) score. When DVT is confirmed by US Doppler, the thrombosis clinical nurse specialist should be contacted on bleep 7380 for further plan of care. If the diagnosis is received out of hours the patient should be provided with a prescription for treatment which can be obtained via the on call pharmacist in Lanesborough wing. The patient should be told to return to the ED review clinic the next working day when the thrombosis CNS should be contacted and asked to come and see the patient.
- Those presenting to A&E will be re-assessed by the Thrombosis clinical nurse specialist (bleep 7380; x2826) with the results of the ultrasound; ambulatory management will be commenced if appropriate. Patients at enhanced risk of bleeding (eg. those with liver disease, peptic ulcer, alcohol abuse, uncontrolled hypertension, heart failure, recent major trauma, or on drugs that interfere with the anticoagulant’s effect), need to be assessed before treatment is started and may require admission to hospital. Admission may also be required if the patient is an IV drug user, has dementia, has a co-existing diagnosis of pulmonary embolus (see separate protocol), if the DVT is bilateral or extending to the IVC, or if the patient is pregnant.
- 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:
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, using the Warfarin Dosing Chart.

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 on fax 0839 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’ 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:

<table>
<thead>
<tr>
<th>Body Weight</th>
<th>Daily Dalteparin Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 46kg</td>
<td>7,500 units od</td>
</tr>
<tr>
<td>46-56kg</td>
<td>10,000 units od</td>
</tr>
<tr>
<td>57-68kg</td>
<td>12,500 units od</td>
</tr>
<tr>
<td>69-82kg</td>
<td>15,000 units od</td>
</tr>
<tr>
<td>83-110kg</td>
<td>18,000 units od</td>
</tr>
<tr>
<td>over 110kg</td>
<td>10,000 units bd</td>
</tr>
</tbody>
</table>

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 \( \leq 30 \text{ ml/min} \) use unfractionated heparin.
For patients needing ambulatory management of DVT, dalteparin injection can be self-administered. However, patients should be referred to the district nurse team if they are unable to self-administer the injections. 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, arrange for a Yellow Anticoagulant booklet to be issued by the anticoagulant clinic, and a follow-up appointment made with the clinic before the patient is discharged. For patients who fall outside of the St George’s catchment area for warfarin monitoring, the ward team will need to make an appointment 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, refer to the out-patient DVT guidelines for information on further management for the patient. 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.
Pulmonary embolism (PE) should be considered in anyone presenting with:
Breathlessness, chest pain, cough/haemoptysis and hypotension (this occurs if
embolism sufficient to compromise cardiac output, in this instance assume a high
risk pulmonary embolism (see later).
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.
The following clinical characteristics are more frequent in those with PE (than
without): pleuritic chest pain, haemoptysis, signs of DVT.

In suspected PE (not pregnant) decide if patient has a low risk of death or a high risk
of death as evidenced by presence of shock i.e. hypotensive with a systolic BP
<90mmHg (in a previously normotensive individual) or a drop in systolic BP of
40mmHg for 15 minutes (not caused by new onset arrhythmia, hypovolaemia or
sepsis).
If low risk of death (majority of patients) assess clinical probability of PE according
to Wells score and follow algorithm below.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical signs and symptoms of DVT (objectively measured leg swelling and pain with palpation in the deep-vein region)</td>
<td>3.0</td>
</tr>
<tr>
<td>Heart rate &gt;100 beats/min.</td>
<td>1.5</td>
</tr>
<tr>
<td>Immobilisation (bed rest, except access to bathroom, for 3 or more days; or surgery in previous 4 weeks)</td>
<td>1.5</td>
</tr>
<tr>
<td>Haemoptysis</td>
<td>1.0</td>
</tr>
<tr>
<td>Previously objectively diagnosed DVT or PE</td>
<td>1.5</td>
</tr>
<tr>
<td>Malignancy (patients with cancer receiving treatment or treatment stopped within previous 6 months or receiving palliative care)</td>
<td>1.0</td>
</tr>
<tr>
<td>Alternative diagnosis less likely than PE</td>
<td>3.0</td>
</tr>
</tbody>
</table>

Assess clinical probability of PE

Low clinical probability of PE (4 points or less)
- D-dimer
  - Negative: CTPA
    - No PE: no treatment
    - PE: treatment
  - Positive: CTPA

High clinical probability of PE (more than 4 points)
- CTPA
  - No PE: investigate further
  - PE: treatment
If patient is high risk of death PE arrange ECHO and/or CTPA – if PE confirmed, patient requires primary reperfusion with thrombolysis in a critical care setting. Unfractionated IV heparin should be administered and a thrombolytic should only be given if patient is haemodynamically unstable, 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). Alteplase has a lower incidence of hypotension than does streptokinase. For streptokinase, give 250,000 units over 30 minutes, then 100,000 units every hour for 12-24 hours.

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), active cancer or is pregnant or breastfeeding (use LMW heparin). 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 confirmed, please refer patient to thrombosis team ext.1332 or blp 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 contra-indicated or not tolerated. If DOACs are contra-indicated or not tolerated use warfarin.

PESI score

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Age in years</td>
</tr>
<tr>
<td>Male Sex</td>
<td>+10 points</td>
</tr>
<tr>
<td>Cancer</td>
<td>+30 points</td>
</tr>
<tr>
<td>Chronic heart failure</td>
<td>+10 points</td>
</tr>
<tr>
<td>Chronic pulmonary disease</td>
<td>+10 points</td>
</tr>
<tr>
<td>Heart rate &gt;110 beats/min.</td>
<td>+20 points</td>
</tr>
<tr>
<td>Systolic BP &lt; 100mm Hg</td>
<td>+30 points</td>
</tr>
<tr>
<td>Respiratory rate &gt; 30 / min</td>
<td>+20 points</td>
</tr>
<tr>
<td>Temperature &lt; 36 degreesC</td>
<td>+20 points</td>
</tr>
<tr>
<td>Altered mental status</td>
<td>+60 points</td>
</tr>
<tr>
<td>Arterial saturation &lt; 90%</td>
<td>+20 points</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Class</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I: &lt;= 65 points</td>
<td>Very low 0-1.6%</td>
</tr>
<tr>
<td>Class II: 66-85 points</td>
<td>Low 1.7-3.5%</td>
</tr>
<tr>
<td>Class III: 86-105 points</td>
<td>Moderate 3.2-7.1%</td>
</tr>
<tr>
<td>Class IV: 106-125 points</td>
<td>High 4.0-11.4%</td>
</tr>
<tr>
<td>Class V: &gt; 125 points</td>
<td>Very high 10.0-24.5%</td>
</tr>
</tbody>
</table>

Patients who are class I-II can be considered for early discharge.

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 require hospital monitoring whilst initiating Rivaroxaban for a few days with clinical judgement re discharge.
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). Importantly the Wells score has not been validated in pregnancy and D-Dimer measurement is not reliable. Please follow RCOG guidelines and algorithm below.

References
2014 ESC Guidelines on diagnosis and management of acute PE
http://eurheartj.oxfordjournals.org/content/ehj/35/43/3033.full.pdf
2015 RCOG Thromboembolic disease in pregnancy and puerperium
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 on 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 dyspnœa 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 antidepressants) 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 $\text{pCO}_2 \geq 7$
  - a 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](http://stginet/Policies/Clin 5-PatientMment/Clin_5_25.pdf)
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

<table>
<thead>
<tr>
<th>COPD or other risk factor for hypercapnic respiratory failure*</th>
<th>Non-hypercapnic respiratory failure/hypoxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Start 24-28% oxygen via Venturi. Assess for signs of hypercapnia and measure arterial blood gases (ABGs) urgently. Write a prescription specifying the target SpO₂ range. Initial target SpO₂ 88-92%</td>
<td>If acutely unwell, or if SpO₂ &lt;85%, start reservoir mask at 15 L/min; otherwise use nasal cannulae (2-6L/min) or variable flow mask (5-10 L/min). Write a prescription specifying the target SpO₂ range. Initial target SpO₂ 94-98%</td>
</tr>
</tbody>
</table>

Titrate oxygen therapy up or down to achieve and maintain target SpO₂ using appropriate oxygen delivery device(s). Document oxygen saturations and oxygen % and delivery device(s)
- Observe SpO₂ for 5 min after any change in O₂ therapy to ensure target SpO₂ is achieved
- If oxygen requirement increases, measure ABGs within 30-60 min of dose change
- Move between limbs of algorithm if necessary according to clinical evaluation and ABGs

Treat exacerbation of COPD. Nebulised bronchodilators should be driven with air, not oxygen, with simultaneous oxygen administered via nasal cannulae. Make sure the patient achieves oxygen saturations 88-92% during nebulisation. Consider NIV or invasive ventilation if PaCO₂ >6 kPa and pH <7.35; discuss with respiratory &/or ICU team as appropriate (see Respiratory Arrest; NIV policy on Intranet)

Investigate and treat underlying cause of hypoxia. If nebulised bronchodilators are indicated, they should be driven by oxygen. Refer to ICU if hypoxia persists, hypercapnia develops, patient tiring or if ICU management is required for the underlying condition.

- Once patient is stable, SpO₂ should be monitored at least every 4 hours; nursing staff should adjust oxygen therapy up or down to maintain the target SpO₂
- Wean O₂ (to air if appropriate) if patient stable and SpO₂ is within or above target range
- A medical review should be sought if a patient’s SpO₂ is repeatedly below the target range; if their O₂ requirement is rising; or if indicated by their clinical condition or EWS score
- Consider humidification if ≥35% oxygen required for ≥2 hours

The driving gas for nebulised medication should be prescribed. Hypoxic patients who are NOT at risk of carbon dioxide retention should be prescribed oxygen at 6 litres/ min to drive the nebuliser. Patients at risk of hypercapnic respiratory failure (for example a small portion of patients with COPD, obesity hypoventilation syndrome, respiratory muscle weakness should be prescribed medical air at 6 litres/ min or via a compressor, with controlled oxygen being delivered via nasal cannulae to achieve oxygen saturations of 88-92

*Other risk factors for hypercapnic (type 2) respiratory failure include severe chest wall or spinal disease, neuromuscular disease, severe obesity, cystic fibrosis and bronchiectasis.
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 β₂-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 achievable 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 PCO₂ 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 SaO₂ 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 advise 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 anteriorally in the 2nd intercostal space.

See footnote 1 for equipment required.
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.


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

1. 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

2. PLEURAL PROCEDURE CHECKLIST
(PLEURAL ASPIRATION AND CHEST DRAIN INSERTION)

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

**SUSPECTED GI BLEED**

**HIGH RISK**
- Acute severe
  - Tachycardia >100
  - Hypotension: systolic <100
  - Haematemesis/melaena
  - Co-morbidity

**LOW RISK**
- <60 years
- Coffee ground vomitus
- CVS stable

**SECOND PHASE**

**Varices**
- See varices management gastro firm to take over NEXT working day

**Bleeding continues**
- High risk 'stable'
  - Tachycardia >100
  - Postural hypotension
  - Co-morbidity

**Bleeding stopped**
- Low risk 'stable'
  - Tachycardia >100
  - Postural hypotension
  - Co-morbidity

**OPTIONS**
- Consult endoscopy
- Surgery
- Radiological intervention

**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.
Each episode of acute variceal bleeding is associated with a 30% mortality at time of admission. 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
     - Packed red cells to maintain the haemoglobin at approximately 8-10g/l.
   - Correct clotting problems:
     - Maintain platelet count >50 x 10^9/L, with platelets
     - Give vitamin K (phytomenadione) 10mg IV slowly.
     - Give fresh frozen plasma (12mls/kg) if clotting is abnormal.
     - Introduce a CVP line to guide intravascular filling.
   
   This is especially valuable if the patient has renal, pulmonary or cardiac dysfunction. NB: ascites may result in an overestimate in the CVP reading. Aim for hourly urine output (as measured by urinary catheter) of 0.5ml/kg/hr.

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, and continue for 2-5 days after endoscopy.
   - Antibiotic prophylaxis
     Blood and an MSU should be sent for microscopy, culture etc. Antibiotic prophylaxis is essential and should be started from admission, eg. Co-amoxiclav 625mg tds PO or 1.2g IV tds (or Ciprofloxacin 500mg PO or 400mg IV bd, only if Penicillin allergic).
   - Endoscopy
     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 1g qds after banding.
     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 15-20ms 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.
- Balloon tamponade
  Insert Sengstaken tube (available on emergency endoscopy trolley/ITU). Check tube position once at 50cm. Inject air down gastric port and ausculate over stomach. Cautiously inflate gastric balloon with 300mls of 1:1 Niopam and water, and pull back until resistance is felt at the gastroesophageal junction. Attach the tube firmly to the patient’s cheek with tape. Do not use traction. Put gastric and oesophageal port on free drainage. Do CXR to check gastric balloon is below the diaphragm. Re-scope within 24hrs. Do not leave gastric balloon inflated for more than 24hrs.
- Transjugular intrahepatic portosystemic stent shunt (TIPPS)
  If bleeding is still uncontrolled, contact on-call endoscopist

4. SECONDARY PROPHYLAXIS OF VARICEAL HAEMORRHAGE

Hepatology team (Clark/Forton) should take over care on the next working day. Do early ultrasound of abdomen and hepatic and portal dopplers, and liver screen if aetiology unknown. Start propranolol 20mg bd, increasing to 40mg bd if tolerated, once haemodynamically stable. Enter patient into variceal ablation.
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:

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

*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 Sharvanu Saha

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.

### Causes of hyperglycaemic emergencies and their differentiation

<table>
<thead>
<tr>
<th></th>
<th>Blood glucose</th>
<th>Urinary Ketones</th>
<th>Dehydration</th>
<th>pH</th>
<th>Serum Osmolality (osmol/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe DKA</td>
<td>11 to 33 mmol/L</td>
<td>≥ +++</td>
<td>+++</td>
<td>&lt;7.35</td>
<td>Variable</td>
</tr>
<tr>
<td>Normoglyc ketoacidosis</td>
<td>&lt;11mmol/L</td>
<td>+++</td>
<td>+++</td>
<td>&lt;7.35</td>
<td>Normal</td>
</tr>
<tr>
<td>HHS</td>
<td>≥33mmol/L</td>
<td>Negative</td>
<td>+++</td>
<td>≥7.35</td>
<td>&gt;320</td>
</tr>
<tr>
<td>HHS/DKA mixed</td>
<td>≥33mmol/L</td>
<td>++ to +++</td>
<td>+++</td>
<td>&lt;7.35</td>
<td>&gt;320</td>
</tr>
<tr>
<td>Lactic acidosis</td>
<td>Variable</td>
<td>0 to 1+</td>
<td>0 to 1+</td>
<td>&lt;7.35</td>
<td></td>
</tr>
</tbody>
</table>

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 6 units/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

**Discuss with HDU/ ICU if:**
- BG > 33 mmol/L
- Hypokalaemia (K+ < 3.5mmol/l)
- GCS < 15
- Pulse > 100bpm or < 60bpm
- SBP < 90 mmHg
- Evidence of sepsis
- pH < 7.1
- HCO3 < 5.0
- Lactate > 2.0

**Essential monitoring**
- 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 ≥ 3mmol/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 euvolaemia 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
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 (e.g., 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 (e.g., sulphonylurea screen, estimate of insulin concentration).
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 BLP 7778 for further on-going management.

References
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 i.e. 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 subarachnoid 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 (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 generalised tonic/clonic (convulsive) seizures. Convulsive SE may present as either a run of discreet generalised tonic/clonic seizures without full recovery in between (ie without regaining consciousness), or continuous generalised 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 generalised 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.

0-5 minutes: Stabilisation 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 or 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 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 anti-epileptics if already on treatment. NB: Benzodiazepines must be written up on the ‘stat dose’ rather than the ‘pm’ 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’).
- 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.

20-40 minutes, 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. Chose ONE of the following options and give as single dose. Of the listed options, only Phenytoin is licensed for status epilepticus. However, current evidence and consensus expert opinion support clinical equipoise between Phenytoin, Valproate and Levetiracetam in terms of efficacy and safety for this indication at stated doses. Further guidance for each is in the table.

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

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose; Rate (Maximum)</th>
<th>May be preferable</th>
<th>Contraindicated</th>
</tr>
</thead>
</table>
| Phenytoin       | 20mg/kg; 50mg/min (N/A) | ● Already taking Phenytoin, suspected poor adherence  
● Alternatives contra-indicated or previously ineffective | ● Significant hypotension  
● Bradycardia  
● Heart block  
● Porphyria  
● Generalized epilepsy*  
● Overdose of recreational drugs or antidepressants |
| Valproate       | 40mg/kg; 10mg/kg/min (3000mg) | ● Already taking Valproate, suspected poor adherence**  
● Generalized epilepsy*  
● Comorbid migraine, mood disorder  
● Alternatives contra-indicated 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 contra-indicated or previously ineffective | May not be best choice in  
● acute or prior brain injury  
● known mood./behaviour disorder (may exacerbate) |

*known or suspected idiopathic (genetic) generalized 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, 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 2\textsuperscript{nd} 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.

All adult (> 16 years): team. All adult patients with convulsive status epilepticus should be reviewed by a member of the epilepsy team (Epilepsy registrar - bleep 8134 Mon-Fri), or on-call neurology (24h/day) within 24 hours of presentation for advice on on-going management.

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

- Give oxygen and establish IV access
- Give Pabrinex if suggestion of alcohol abuse or impaired nutrition
- Measure glucose; if hypoglycaemic, give 100 ml of 10% glucose rapidly. If still fitting or unconscious, repeat and then start 10% glucose at 100 ml/hr (Refer to management of Hypoglycaemia)

- **Focal/non-convulsive status**
  - Call neurology SpR on-call for advice on appropriate drug management

- **Convulsive status**
  - Lorazepam 4mg IV* (Paed 0.1mg/kg)
  - Lorazepam 4mg IV* (Paed 0.1mg/kg)

- If still fitting or unconscious, repeat and then start 10% glucose at 100 ml/hr (Refer to management of Hypoglycaemia)

- **Phenytoin 20 mg/kg at rate of 50 mg per minute. Cardiac monitor OR**
- **Valproate 40 mg/kg at a rate of 10 mg/kg/min (max 3000 mg) OR**
- **Levetiracetam 60 mg/kg at a rate of 6 mg/kg/min (max 4500 mg)**

- **ITU GENERAL ANAESTHESIA**
  - Seizures persisting by end of infusion, or 20 mins since infusion started (whichever soonest)

*If Lorazepam is unavailable, give 10 mg Diazepam iv (paed 300-400 micrograms/kg - max. 10 mg) or 10 mg 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)

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

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

- Entacapone, Opicapone
- Selegiline, Rasagiline
- Amantadine

Refer to PD team if long term omission

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

**Apomorphine**
Subcutaneous infusion
DO NOT change dose or stop pump until discussed with PD CNS/neurology registrar
APO-go (apomorphine) 24 hour helpline: 08448801327

**Duodopa®**
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:

**AVOID**
- Metoclopramide
- Prochlorperazine
- Promethazine
- Haloperidol

**USE**
- Domperidone
- Ondansetron
- Cyclizine
SHORT TERM ONLY (cardiac side effects)

USEFUL CONTACT 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.

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

• Intravenous fluid challenge 500-1000 mL immediately and more as needed.
• Give chlorphenamine 10 mg by IM or slow IV injection.
• Corticosteroids may help prevent or shorten protracted reactions. In asthma, early corticosteroid treatment is beneficial. Give hydrocortisone in a dose of 200mg by slow IV or IM injection.
• An inhaled β2 agonist (salbutamol) is a useful adjunct if bronchospasm is a major feature which has not responded rapidly to other treatment.

• **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 as 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 Lenny Ng

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


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 3g 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 (Max1.25g/day)

The lowest possible dose should be used for the shortest period of time to control symptoms. Patients should be on gastro-prophylaxis for extended courses of NSAIDS and 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 an antiemetic and regular laxatives.

- Weak Opioids
  Dihydrocodeine: 30mg PO 4-6 hourly (maximum 240mg/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)

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

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:

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Dose IV Morphine</th>
<th>IM/ SC Morphine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>In A&amp;E, ICUs and Theatres ONLY</td>
<td>Age (yrs)</td>
</tr>
<tr>
<td>&lt; 70</td>
<td>Pain severe: 2mg</td>
<td>20-39</td>
</tr>
<tr>
<td></td>
<td>Less severe: 1mg</td>
<td>40-59</td>
</tr>
<tr>
<td>&gt; 70</td>
<td>Pain severe: 1mg</td>
<td>60-69</td>
</tr>
<tr>
<td></td>
<td>Less severe: 0.5mg</td>
<td>70-85</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt;85</td>
</tr>
</tbody>
</table>
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 email acute.painservice@stgeorges.nhs.uk to seek further advice.

**OPIOID OVERDOSE**

**a. In the Acute Pain setting**

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

1. Stop the opioid.
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 ≥ 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 morphine.

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 standard ampoule containing naloxone 400microgram to 10ml with sodium chloride 0.9%
4. Administer 0.5ml (20microgram) IV every 2 minutes until the patient’s respiratory status is satisfactory.

Naloxone is shorter-acting than Morphine 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).
Email: acute.painservice@stgeorges.nhs.uk
SUGGESTIONS FOR THE USE OF ANTIMICROBIAL DRUGS IN ADULTS
Link Consultant: Dr Matthew Laundy

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/sgh/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.
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:

1. **Stop** antibiotics if there is no evidence of infection
2. **Switch** antibiotics from intravenous to oral when:
   - temperature has been < 38°C for 48 hours or more;
   - oral foods/liquids are tolerated;
   - there is no unexplained tachycardia (HR <90bpm 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: ARHAII 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 (beep 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.

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

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

<table>
<thead>
<tr>
<th>Red Flag Sepsis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Responds only to voice or pain/ unresponsive</td>
</tr>
<tr>
<td>Systolic B.P ≤ 90 mmHg (or drop &gt;40 from normal)</td>
</tr>
<tr>
<td>Heart rate &gt; 130 per minute</td>
</tr>
<tr>
<td>Respiratory rate ≥ 25 per minute</td>
</tr>
<tr>
<td>Needs oxygen to keep SpO2 ≥92%</td>
</tr>
<tr>
<td>Non-blanching rash, mottled/ ashen/ cyanotic</td>
</tr>
<tr>
<td>Not passed urine in last 18 hours</td>
</tr>
<tr>
<td>Urine output less than 0.5 ml/kg/hr</td>
</tr>
<tr>
<td>Lactate ≥2 mmol/l</td>
</tr>
<tr>
<td>Recent chemotherapy in past 6 weeks</td>
</tr>
</tbody>
</table>

- 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 appropriate
**Amber Flag Sepsis**

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 Intensive Care Unit
Patients with neutropenic sepsis should be treated according to Trust protocol (see new guideline below). This guideline should be applied to immunocompromised patients with evidence of an infection by an unknown organism.

These guidelines are applicable to unwell or febrile patients who:
- have received chemotherapy within the last 6 weeks; or,
- are otherwise at risk of neutropenia eg bone marrow failure due to primary haematological disorder (eg. Leukaemia)
- have immunosuppression

Neutropenic sepsis is a medical emergency. Patients can be critically unwell with minimal signs. If the patient is unwell, resuscitate immediately and give IV antibiotics – DO NOT WAIT FOR NEUTROPHIL COUNT.

If you are not experienced in the management of these patients you must contact a senior clinician or consultant medical microbiologist to discuss further.

- When patients are admitted, review previous microbiology tests for positive culture results
- Screen for fungal infection (HRCT in all high risk haematology patients) and consider anti-fungals as per the anti-fungal policy.

**1. First-Line Treatment**

Piperacillin-tazobactam (Tazocin) 4.5g IV 8 hourly
+ Amikacin 15mg/kg IV stat dose (based on dose determining weight if obese - see Trust amikacin guidelines). If renal impairment, consider dose reductions and/or frequency amendments in line with clinical history and presentation.

Review need for regular daily amikacin dosing in line with clinical picture and Trust amikacin guidelines

**Alternative if allergic to 1st line**

**a) Non-severe allergy (e.g. rash):**

Meropenem 1g IV 8 hourly
+ Amikacin 15mg/kg IV stat dose (based on dose determining weight if obese - see Trust amikacin guidelines)

**b) Severe allergy (e.g anaphylaxis):**

Ciprofloxacin 400mg IV 12 hourly
+ Tigecycline 100mg stat IV, then 50mg 12 hourly
+ Amikacin 15mg/kg IV stat dose (based on dose determining weight if obese - see Trust amikacin guidelines). If renal impairment, consider dose reductions and/or frequency amendments in line with clinical history and presentation.

- Review need for regular daily amikacin dosing in line with clinical picture and Trust amikacin guidelines
• Review at 48 hours or earlier if condition deteriorates. Consider culture results and/or discuss with microbiology. Investigate sources of infection and consider source control.

2. If still pyrexial after 48 hours
Meropenem 1g IV 8 hourly
+ Vancomycin as per Trust Vancomycin guidelines

If severe penicillin allergy (e.g. anaphylaxis) please discuss with microbiologist.

• If remains pyrexial after another 48 hours, then discuss with microbiologist.
• If not already done so, investigate sources of infection and control of the infection source.

3. Oral switch
• Consider an oral switch after 48 hours of IV therapy for patients who are apyrexial and have neutrophils > 1.0. If source of infection is known, treat as per Trust empirical guidelines.
• Always check sensitivities of any significant organisms cultured first and treat accordingly.

a) If source of infection is unknown:
Co-amoxiclav 625mg PO 8 hourly to complete a total antibiotic course of 5 days

b) Penicillin allergy (e.g. rash or anaphylaxis)
Ciprofloxacin 500mg PO 12 hourly to complete a total antibiotic course of 5 days

4. In addition
a) Patients with indwelling venous lines should receive adequate anti-staphylococcal cover:
if not already prescribed, add Vancomycin if known or suspected MRSA (as per Trust Vancomycin guidelines)

b) In patients with diarrhoea:
add Metronidazole 500mg IV 8 hourly or 400mg PO 8 hourly

c) In patients with perineal or gingival infection:
add Metronidazole 500mg IV 8 hourly

d) In patients with Evidence of herpetic lesions:
add Aciclovir 5mg/kg IV 8 hourly

Please also consult the Trust Intranet: [http://microguide.horizonsp.co.uk/viewer/sguh/adult](http://microguide.horizonsp.co.uk/viewer/sguh/adult): Speciality Guidelines> Sepsis and septic shock> Neutropenic sepsis

**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](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 MANAGEMENT OF COMMON INFECTIONS IN ADULTS

THIS SECTION IS CURRENTLY UNDER REVIEW

PLEASE REFER TO TRUST INTRANET GUIDELINES USING THIS LINK:

http://microguide.horizonsp.co.uk/viewer/sguh/adult
MANAGEMENT OF C. DIFFICILE

Guidelines link: [http://microguide.horizonsp.co.uk/viewer/sguh/adult](http://microguide.horizonsp.co.uk/viewer/sguh/adult)

Healthcare-associated infection> Clostridium difficile infection

---

**Clostridium difficile Infection**

*Clostridium difficile* infection (CDI) usually occurs following the use of antibiotics which disrupt the normal bacterial flora of the gut, allowing colonisation by *C. difficile*.

All antibiotics are implicated, particularly broad spectrum agents such as quinolones and cephalosporins. The crude mortality rate of CDI is around 20%.

**All patients with potentially infectious diarrhoea must be clinically reviewed and this documented in the patient notes and on the diarrhoea request form.**

---

**Severity Assessment**

**Mild to Moderate CDI**
- WCC < 15 x10⁹/L
- < 5 stools of type 5 to 7 on the Bristol Stool Chart.

**Severe CDI** is associated with the following
- WCC > 15 x10⁹/L
- an acute rising serum creatinine (i.e. > 50% increase above baseline)
- a temperature of > 38.5°C
- evidence of severe colitis (abdominal or radiological signs)
- The number of stools is a less reliable indicator of severity.

**Life-threatening CDI includes:**
- hypotension,
- partial or complete ileus,
- toxic megacolon or CT evidence of severe disease.

---

**Suspected CDI - 1st Line Treatment**

If CDI is suspected do not wait for toxin testing results to come back and start the patient empirically.

- **Vancomycin** 125 mg PO/NG *qds* for 10-14 days if patient has recently received.

If toxic megacolon/perforation suspected request urgent surgical opinion.
- If oral/NG treatment is impossible give IV **Metronidazole** 500mg *tds*.
- Change to oral as soon as possible.

---

**Clinicians should use this mnemonic (SIGHT) when managing CDI:**

- **S**- Suspect a case may be infective when there is no other obvious cause for diarrhoea
- **I**- Isolate the patient & consult the Infection control team while determining the cause of diarrhoea
- **G**- Gloves and apron must be used for all contacts with the patient and their environment
- **H**- Hand washing with soap and water should be carried out before and after each contact with patient and their environment
- **T**- Test the stool for *C. difficile* toxin by sending a specimen immediately to Microbiology.
**ACTIONS**
CDI should be managed as a diagnosis in its own right, a full examination and review must be conducted by the SpR including:
- Severity assessment
- Review the need to continue antibiotics for other indications (if any) in conjunction with medical microbiology
- Look for signs of acute abdomen, ileus or toxic megacolon and order abdominal X-ray and refer to surgeons urgently if required.
- Stop contributory agents (i.e. laxatives)
- Stop antimotility agents where possible (i.e. loperamide, opiates)
- Need for fluid resuscitation and electrolyte replacement,
- Nutritional status
- Presence of signs of increasing severity of disease - refer to ITU early as patients may deteriorate very rapidly

**Testing**
Up to three different types of tests are used for diagnosis of CDI
1. A test for the GDH antigen of *C. difficile*
2. A test for *C. difficile* toxins A and B
3. A PCR test for the detection of *C. difficile* DNA
All tests that are required will be performed and reported on the same day

**Laboratory confirmed *C. difficile***

**Switch treatment to:**
- Fidaxomicin 200mg PO 12 hourly for 10 days.
- If oral therapy is impossible give Metronidazole 500mg IV 8 hourly.
  Fidaxomicin may be crushed and given via feeding tubes (unlicensed method of administration)
- Metronidazole 500mg tds. Change to oral as soon as possible.

---

**EMPIRICAL TREATMENT OF DIABETIC FOOT INFECTION**
*Refer patients urgently to Diabetic Foot Team x1859; Vascular SpR blp6640*

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**INFECTION 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
**UPPER RESPIRATORY TRACT INFECTION**
Remember that most throat infections are caused by viruses, so do **NOT** require an antibiotic.

<table>
<thead>
<tr>
<th>Infection</th>
<th>Antibiotics</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Microbiologically confirmed streptococcal pharyngitis</strong></td>
<td>Penicillin V PO 500mg qds (or clarithromycin PO 500mg bd if allergic to Penicillin) Guideline link: <a href="http://microguide.horizonsp.co.uk/viewer/sguh/adult">http://microguide.horizonsp.co.uk/viewer/sguh/adult</a> Speciality Guidelines&gt;ENT Treatment Guidelines&gt; Head and Neck</td>
</tr>
</tbody>
</table>
| **Acute otitis media & bacterial infection proven or strongly suspected** | **Acute Otitis Media**  
When not responded to treatment in primary care  
**First Line**  
Co-amoxiclav PO 625mg TDS  
+/- **Ofloxacin** ear drops ṪṪ TDS  
**Second Line / Alternative**  
**Levofloxacin** PO 500mg OD  
+/- **Ofloxacin** ear drops ṪṪ TDS  
Guideline link: [http://microguide.horizonsp.co.uk/viewer/sguh/adult](http://microguide.horizonsp.co.uk/viewer/sguh/adult) Speciality Guidelines>ENT Treatment Guidelines> Otology |
| **Suspected acute epiglottitis** | **Epiglottis/ Supraglottis**  
Treatment duration = 10 days  
**First Line**  
Ceftriaxone IV 2g OD  
**Second Line / Alternative**  
**Levofloxacin** IV/PO 500mg OD (levofloxacin has excellent oral bioavailability - early switch is advised)  
Guideline link: [http://microguide.horizonsp.co.uk/viewer/sguh/adult](http://microguide.horizonsp.co.uk/viewer/sguh/adult) Speciality Guidelines>ENT Treatment Guidelines> Head and Neck |
| **Suspected diphtheria** | Call for Consultant help. |
| **Sinusitis** | **First Line / #Acute**  
Supportive treatment only - most resolve without antibiotics  
**Second Line / If severe**  
**Amoxicillin** PO 1g TDS for 7 days and consider nasal steroid  
**OR** **Doxycycline** PO 200mg stat then 100mg OD if penicillin allergy  
Guideline link: [http://microguide.horizonsp.co.uk/viewer/sguh/adult](http://microguide.horizonsp.co.uk/viewer/sguh/adult) Speciality Guidelines>ENT Treatment Guidelines> Rhinology |
PROPHYLAXIS TO PREVENT ENDOCARDITIS

St George’s Hospital NHS Trust has carefully reviewed the current NICE guidance and recent American Heart Association (AHA) and BSAC guidelines on antibiotic prophylaxis for endocarditis in dental and other surgery, and continues to recommend prophylaxis to patients with the highest risk of adverse outcome from infective endocarditis

<table>
<thead>
<tr>
<th>THE FOLLOWING HIGH-RISK PATIENT GROUPS SHOULD RECEIVE ANTIBIOTIC PROPHYLAXIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Previous Infective Endocarditis</td>
</tr>
<tr>
<td>• Prosthetic valve</td>
</tr>
<tr>
<td>• Acquired valvular heart disease with stenosis or regurgitation</td>
</tr>
<tr>
<td>• Unrepaired or incompletely repaired cyanotic congenital heart disease</td>
</tr>
<tr>
<td>• Congenital heart disease repaired with prosthetic material (for 6 months after procedure)</td>
</tr>
<tr>
<td>• Valve disease in recipients of a cardiac transplant</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>HIGH RISK PATIENTS REQUIRE PROPHYLAXIS FOR THE FOLLOWING PROCEDURES</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Dental procedures involving dento-gingival manipulation or endodontics</td>
</tr>
<tr>
<td>- - dental extractions</td>
</tr>
<tr>
<td>- - sub-gingival scaling</td>
</tr>
<tr>
<td>- - placement of restorations in relation to the gingival mucosa</td>
</tr>
<tr>
<td>• All surgery to the jaw and oral cavity*</td>
</tr>
<tr>
<td>• ENT – tonsillectomy and adenoidectomy*</td>
</tr>
<tr>
<td>• Invasive procedures of respiratory tract needing incision or biopsy of mucosa</td>
</tr>
<tr>
<td>• All gastrointestinal and genitourinary surgical procedures</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prophylactic regimens:</th>
<th>1st line prophylaxis</th>
<th>If allergic to Penicillin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral – single dose given 1hr before procedure</td>
<td>Amoxicillin 3g</td>
<td>Clindamycin 600mg</td>
</tr>
<tr>
<td>Unable to take oral therapy Single IV dose 0-30 mins before procedure</td>
<td>Amoxicillin 3g</td>
<td>Clindamycin 600mg</td>
</tr>
</tbody>
</table>

* If co-amoxiclav or vancomycin is indicated as standard surgical prophylaxis, additional amoxicillin or clindamycin is not necessary.

Please also consult the relevant surgical antibiotic prophylaxis policy on Trust Intranet: [http://microguide.horizonsp.co.uk/viewer/sguh/adult](http://microguide.horizonsp.co.uk/viewer/sguh/adult) : Speciality Guidelines> Endocarditis Guideline – Prophylaxis
PROPHYLACTIC USE OF ANTIBIOTICS IN SURGERY AND MINOR TRAUMA

<table>
<thead>
<tr>
<th>Infection</th>
<th>Antibiotics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drainage of abscess</td>
<td>Not routinely required (unless septic or surrounding cellulitis)</td>
</tr>
<tr>
<td>Bites (Human or Animal)</td>
<td>Co-amoxiclav (625 mg tds) for 3-5 days.</td>
</tr>
<tr>
<td></td>
<td>In patients allergic to Penicillin give doxycycline (100 mg bd) plus</td>
</tr>
<tr>
<td></td>
<td>metronidazole (400 mg tds) by mouth for 3-5 days.</td>
</tr>
<tr>
<td></td>
<td>Remember tetanus prophylaxis, and rabies if patient from endemic region</td>
</tr>
<tr>
<td></td>
<td>(seek advice).</td>
</tr>
<tr>
<td></td>
<td>For human bites give accelerated hepatitis B immunisation.</td>
</tr>
<tr>
<td>Lacerations</td>
<td>Nil</td>
</tr>
</tbody>
</table>

Please refer to latest Trust guidelines on the Microguide App or web page for empiric treatment. [http://microguide.horizonsp.co.uk/viewer/sguh/adult](http://microguide.horizonsp.co.uk/viewer/sguh/adult): Speciality Guidelines>ENT Treatment Guidelines> Rhinology

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](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.
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](http://www.spib.axl.co.uk) or the National Poisons Information Service on 0844 892 0111.

**PRIMARY ASSESSMENT**

- **Is airway protected?**
  If not, crash bleep 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 IT fails, use back up “poisons file” in Resuscitation Room in A & E.
  c) 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 Accident & Emergency 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 A&E 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

*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 45o
- 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 <10mg/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.
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.

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 <1 hour 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 disproportionally prolonged in overdose.
**Management**

NB. For management of opioid overdose in the setting of treatment of acute pain and in palliative care, please refer to the management of *Acute Pain*. Otherwise,

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 30 mins 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. See flowchart below.
   - Some patient groups may require reduced doses of naloxone: 1) 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 hypertension, cardiac arrhythmias, pulmonary oedema and cardiac arrest; 2) regular illicit opiate users are also at risk of withdrawal with naloxone. If such patients have a respiratory rate ≥8, are easily rousable and not cyanosed/normal pCO2 on ABG, consider omitting any further opiate doses and monitoring the patient closely. If required, use smaller doses of naloxone titrated to effect – dilute one ampoule of naloxone (400 micrograms) to 10ml in 0.9% sodium chloride (40 micrograms/ml) and give 0.5-1.0ml every 2-3mins titrated to effect.
   - In the acute pain setting naloxone boluses of 100 micrograms are advised (see guidelines link below)
   - 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:

<table>
<thead>
<tr>
<th>Preparation Type</th>
<th>Observation Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Standard Preparation</td>
<td>6hrs</td>
</tr>
<tr>
<td>Prolonged Release</td>
<td>12hr</td>
</tr>
<tr>
<td>Methadone</td>
<td>24hrs</td>
</tr>
</tbody>
</table>

Prior to discharge, ensure the patient has been symptom-free for at least 6 hours after the last dose of naloxone.

**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
2) 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: Dr 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 heptocellular carcinoma (HCC).

**Investigations**

**Blood Tests**
1. FBC
2. Clotting screen
3. Urea, electrolytes, creatinine
4. Liver function tests, γGT, 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 where they might help establish aetiology
8. Septic screen – blood cultures, urine cultures, sputum cultures and ascitic tap

**Radiology**
1. CXR
2. Early abdominal ultrasound to: 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).
1. Do diagnostic paracentesis (ask for urgent cell count to check for spontaneous bacterial peritonitis (SBP) defined as >250 neutrophils/mm$^3$ or >300 lymphocytes/mm$^3$. Send sample for culture/biochemistry/cytology)
2. If *moderate volume ascites* and if plasma Na$^+$ >130mmol/L and renal function is normal, give spironolactone 100mg plus furosemide 40mg daily. Measure weight daily, target weight loss at ~500g/day. The dose of both diuretics can be increased simultaneously every 3–4 days to achieve target weight loss; maintain a 100:40 ratio up to a maximum of 400mg spironolactone: 160mg furosemide. Do daily U&E; rapid changes can lead to encephalopathy. If hyponatraemic, restrict Na$^+$ to 88mmol (2000mg)/day and fluid to 1.5litres/day (arrange with dietician).
3. If there is *massive ascites* – seek advice about total paracentesis from hepatology team (Dr Clark/Dr Forton). Note that 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 – if the WBC is >250/mL (neutrophils) or >300/mL (lymphocytes), the patient is likely to have SBP. While awaiting culture results (send ascites inoculated in culture-medium bottles to increase diagnostic yield) start IV co-amoxiclav 1.2g bd or tds (ciprofloxacin 750mg bd PO only if Penicillin allergic).
**Jaundice**
1. Exclude haemolysis, do conjugated bilirubin and blood film
2. Exclude biliary obstruction

**Coagulopathy**
1. Give vitamin K (menadione sodium phosphate) 10mg PO daily for 3 days. If severe coagulopathy, Vit 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.

**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 antibiotics (broad spectrum): co-amoxiclav (or ciprofloxacin only if Penicillin allergic)
6. If grade 3 or 4 encephalopathy, consider intubating to protect the airway
7. Remember other causes of reduced Glasgow Coma Scale, eg. sepsis, Wernicke’s (give Pabrinex), intercranial bleed (consider CT head)

**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
2. Stop NSAIDs; they are contraindicated in liver failure
3. Catheterise bladder
4. Check urine sodium
5. Insert central venous line (internal jugular) and use it as one indicator of volume control; remember that in massive ascites the CVP will read higher than the true clinical position. Give human albumin solution (HAS) if CVP suggests hypo-volaemia
6. If fluid replacement does not result in an adequate urine output (>0.5mL/kg/hr) consider giving bolus of furosemide (50-100mg)
7. If adequately fluid resuscitated and still oliguric, start terlipressin 1mg qds: reduce dose in patients with ischaemic heart disease or peripheral vascular disease
8. Give infusion of N-acetylcysteine (150mg/kg over 24 hrs) if patient having CT, to prevent contrast nephropathy
9. Patients in whom decompensated chronic liver disease is secondary to alcohol and renal impairment should be given pentoxyphylline 400mg tds orally

**Portal hypertension** (defined by the presence of varices on endoscopy)
1. Give propranolol 20mg bd. Aim to reduce resting pulse rate by 20% or aim for pulse rate of 60bpm. If a β-blocker is contraindicated give isosorbide mononitrate 20mg bd
2. Give antibiotic prophylaxis (co-amoxiclav) to patients who have cirrhosis plus bleeding varices

**Acidosis**
The commonest cause is a metabolic acidosis due to fluid depletion. This should be treated by fluid resuscitation as for renal failure.
**Fluid replacement**
In liver failure there is total body sodium excess, therefore avoid saline 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 (Na⁺ <125mmol/l) seek specialist advice.

**Nutrition**
Patients are often malnourished. Feeding should be enterally, if necessary with a nasogastric tube provided the airway can be protected. With dietician’s advice give:
1. High protein diet (unless known to worsen encephalopathy)
2. High calorie diet
3. No added salt diet
4. Thiamine replacement (Pabrinex 1&2 IV over 10 mins for 3 doses, then thiamine 100mg po bd for 2 weeks)

**Analgesia**
Pain is not usually a feature of liver failure. If analgesia needed:
1. Paracetamol is safe in the conventional doses (NB NSAIDS are contra-indicated)
2. Opioids may be used, but may precipitate encephalopathy (less likely with dihydrocodeine than codeine phosphate). Remember that opioids may accumulate even when given at traditional doses

**Referral to Hepatology team**
All patients with decompensated liver disease should be referred to the hepatology team. 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 and are likely to need total paracentesis
5. Recent-onset encephalopathy (<12 weeks of onset of jaundice)
6. Incipient renal failure
7. Alcoholic hepatitis
ACUTE PAINFUL SWOLLEN JOINT(S)  
Link Consultant: Dr 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.
- Indomethacin 50mg/8 hourly; (6 hourly for acute gout)

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.
EARLY RECOGNITION AND DIAGNOSIS
Consider GCA in patients fulfilling the criteria below where other more common causes of headache have been considered and ruled out.

Mandatory criteria:
1) Age over 50
2) CRP>10
3) New and unilateral temporal region headache

Plus at least one of:
• Scalp tenderness
• Jaw and/or tongue claudication
• New onset visual deficit - Amaurosis fugax, diplopia, blurring
• Temporal artery abnormalities (tender on palpation or absent/difficult to palpate temporal artery pulse)
• Preceding PMR symptoms

INITIAL INVESTIGATIONS
• Serology: FBC, U&E, ESR, CRP, LFT, Bone profile + Vitamin D
• Urine dip:
  - + Blood → red cell casts
  - + Protein → Urine protein: CR
  - + leucocytes/nitrites → MCS
• CXR : ?aortic aneurysm
• USS Temporal arteries (urgent)
  - Request on EPR and call Vascular coordinator on ext. 0092 OR 4312 to arrange on the same day as assessment if in hours (Monday to Friday 9am-5pm)
  - If out of hours, to arrange admission/review as soon as possible during the week and contact coordinators on the day

INITIAL MANAGEMENT
• Rheumatology review in working hours (Bleep 7787)
  - Rheumatology team will review and advise within 24 hours of receiving the referral call, on whether Temporal artery Biopsy, further treatment and Rheumatology follow up is indicated
  - Biopsy can be organised by emailing vascular coordinator Andrea Browne (Andrea.Browne@stgeorges.nhs.uk)
• If out of hours, patient to be brought back into ambulatory care or ED in hours at earliest opportunity and Rheumatology team to be contacted
• Urgent Ophthalmology review if any visual compromise
  - Moorfields emergency line in hours: extension 6115
  - Via switchboard to Ophthalmology on-call Out of hours
Initial treatment
• GCA without visual compromise:
  ➢ Prednisolone 40- daily until resolution of symptom and improvement in inflammatory markers
• GCA with evolving visual loss and/or amaurosis fugax
  ➢ Consider IV Methylprednisolone 500mg -1g daily for 3 days before switching to oral steroid following senior clinician or Rheumatology review
• GCA with established visual loss:
  ➢ Prednisolone 60mg or 1mg/kg daily

Please ensure patients are ALL on the following additional treatments if steroid treatment commenced:
• Aspirin 75mg OD
• Bone protection if not already prescribed
  ➢ Alendronic acid 70mg po weekly plus
  ➢ Adcal D3 T po BD
• Gastrointestinal protection
Management of suspected Giant Cell Arteritis (GCA)

Mandatory criteria
1) Age over 50; 2) CRP>10; 3) New and abrupt onset headache

Plus one or more of:
- Jaw and/or tongue claudication
- New onset visual deficit
- Temporal artery abnormalities
- Preceding PMR symptoms

Immediate investigations
- Serology: FBC, U&E, ESR, CRP, LFT, Bone profile + Vitamin D
- Urine dip:
  + Blood → red cell casts
  + Protein → Urine protein: CR
  + Leucocytes/nitrites → MCS
- CXR
- USS Temporal arteries (urgent)

IF high clinical suspicion for GCA

Immediate start of steroid
- **GCA without visual compromise:**
  Prednisolone 40-daily until resolution of symptom and improvement in inflammatory markers
- **GCA with evolving visual loss and/or amaurosis fugax:**
  Consider IV Methylprednisolone 500mg -1g daily for 3 days before switching to oral steroid following senior clinician or Rheumatology review
- **GCA with established visual loss:**
  Prednisolone 60mg or 1mg/kg daily PLUS
  - Aspirin 75mg od
  - Bone protection: Alendronic acid + Adcal D3
  - Gastrointestinal protection

Urgent referral
- Rheumatology review in in hours (Bleep 7787)
  - Rheumatology team will review and advise within 24 hours of receiving the referral call, on whether Temporal artery Biopsy, further treatment and Rheumatology follow up is indicated
  - Biopsy can be organised by emailing vascular coordinator Andrea Browne (Andrea.Browne@stgeorges.nhs.uk) and must be performed within 10 days of starting steroid treatment
  - If out of hours, patient to be brought back into ambulatory care or ED in hours at earliest opportunity and Rheumatology team to be contacted
  - Urgent Ophthalmology review if any visual compromise
  - Moorfields emergency line in hours :extension 6115
  - Via switchboard to Ophthalmology on-call Out of hours
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:

1. Stage 1: Creatinine rise 1.5-2 fold from baseline or ≥26.4µmol/L and/or urine ≤0.5ml/kg/hr > 6h
2. Stage 2: Creatinine rise 2-3 fold from baseline and/or urine ≤0.5ml/kg/hr > 12 h
3. Stage 3: Creatinine rise >3 fold from baseline or ≥354µmol/L and/or urine ≤0.3ml/kg/hr > 24 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 (26µmol/L) or >1.5 times from baseline (>x3- 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

**SEPIS:** 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$^+ > 6$ mmol/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://www.londonaki.net), [http://publications.nice.org.uk](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
The most serious clinical manifestations associated with raised serum potassium are cardiac arrhythmias, which include asystole, ventricular fibrillation and muscle weakness and paralysis.

Indication for treatment. Attempts should be made to lower potassium when serum K\(^+\) > 6 mmol/L. There are a number of causes of pseudohyperkalaemia including haemolysis of the blood sample or delay in transit to the laboratory. If hyperkalaemia is an unexpected finding, a repeat sample should be sent but should not delay treatment.
Causes. Potassium rises when there is:

a) Reduced renal excretion
   eg. renal failure; when taking potassium-sparing diuretics, ACE inhibitors/angiotensin-II receptor blockers or NSAIDS, and, in Addison’s disease); or when,

b) Potassium leaves cells
   eg. acidosis, diabetic hyperglycaemia and cell damage (ie. trauma, burns, haemolysis). Remember that where there has been movement of K⁺ between body compartments, the total body K⁺ may be normal (or even low). Measure arterial pH, pCO₂ and pO₂ if in doubt.

Treatment.
1. If the ECG is abnormal, or K >6.5MMOL give 10 mL of 10% calcium gluconate slowly iv (at a maximum rate of 2 mL/min), repeating the dose if necessary until the ECG normalises up to a maximum dose of 40 mL. Ideally, cardiac monitoring should be instituted.

2. To move potassium into the cells give Dextrose/Insulin infusion, 50 mL of 50% Dextrose with 10 units of soluble human Insulin, over 15 mins. Check blood glucose every hour. If hyperkalaemia persists after a treatment, the infusion can be repeated and patients should be discussed with medical or renal registrar.

In addition to potassium, arterial pH and plasma bicarbonate should be measured in all patients.

i) If there is mild to moderate acidosis (pH 7.1-7.3) and the patient is not fluid overloaded, give 500 mL 1.4% NaHCO₃ over 2-3 hours and recheck bicarbonate.

ii) If there is severe acidosis (arterial pH <7.1), give 500 mL 1.4% NaHCO₃ over one hour (in patients with volume overload or cardiac arrest give 50-100 mL of 8.4% NaHCO₃ slowly IV into a central vein).

- Sodium bicarbonate should not be given in the presence of type 2 respiratory failure or hypocalcaemia (corrected calcium < 2.0 mmol/L).
- Consider adjunct use of 10-20 mg nebulised salbutamol. This must not be used instead of the above interventions as 40% of patients do not respond and caution is required in patients with tachycardia or ischaemic heart disease.
- The use of Calcium Resonium is no longer recommended as there is little evidence for efficacy and it often causes constipation leading to increased re-absorption of potassium.
- Once the acute episode is managed check serum K⁺ concentration at least twice daily, then once daily when K⁺ is < 6.0 mmol/L.
- Stop all potassium-retaining/containing drugs where possible and arrange dietary review of potassium in diet where appropriate.

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 – hypomagnesaeemic 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 (2.2mmol 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 hypercalceamia 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 < 3mmol/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, Phaeochromocytomas, 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 euvoalaemic, 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 Zolendronic 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 zolendronic acid.**
Maximum frequency of maintenance IV Zoledronic dose is 3-4 weekly.

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

<table>
<thead>
<tr>
<th>eGFR (mL/min/1.73m²)</th>
<th>&gt;60</th>
<th>50-59</th>
<th>40-49</th>
<th>30-39</th>
<th>&lt;30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose of IV Zolendronic acid</td>
<td>4mg</td>
<td>3.5mg</td>
<td>3.3mg</td>
<td>3.0mg</td>
<td>Do not use*</td>
</tr>
</tbody>
</table>

* 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

<table>
<thead>
<tr>
<th>Serum calcium</th>
<th>Up to 3 mmol/L</th>
<th>3-3.4 mmol/L</th>
<th>3.5-3.9 mmol/L</th>
<th>≥ 4 mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose of IV Pamidronate over 2-3 hrs</td>
<td>15-30 mg</td>
<td>30-60 mg</td>
<td>60-90 mg</td>
<td>Mg</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>Serum calcium (mmol/L)</th>
<th>&lt; 4 mmol/L</th>
<th>≥ 4 mmol/L</th>
<th>&lt; 4 mmol/L</th>
<th>≥ 4 mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose of IV Pamidronate</td>
<td>40 mg</td>
<td>60 mg</td>
<td>Do not give</td>
<td></td>
</tr>
<tr>
<td>Rate of infusion</td>
<td>&lt; 20 mg/hr</td>
<td></td>
<td>n/a</td>
<td>n/a</td>
</tr>
</tbody>
</table>
HYPONATRAEMIA

Hyponatraemia (Na⁺ < 135 mmol/L) results from H₂O retention, Na⁺ loss or a combination of the two. Although the definition of hyponatraemia is Na⁺ < 135 mmol/L, it is only clinically significant if the sodium concentration is < 125 mmol/L, or has fallen rapidly (> 20 mmol/L in 24 hours). Hyponatraemia can lead to shift of H₂O 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:

a) renal loss of Na⁺ (caused by, for example, diuretics, tubular disorder)
b) gain of H₂O 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 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
1. Confirm plasma sodium below 135 mmol/L
2. Measure urinary sodium concentration
3. Measure plasma osmolality and assess volume status;
   a) If osmolality greater than 275 mmol/kg, assume the problem is hyper-glycaemia or renal failure and treat as such
   b) 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 x s. [Na] + glucose + 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 SIADH

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:

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

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 > 145mmol/L, but is usually only clinically significant if the concentration is > 155mmol/L, or there has been a rapid rise (>20mmol/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 H\(_2\)O loss (deficiency) rather than to Na\(^+\) gain (excess). It is important to determine whether acute or chronic loss when assessing cause(s).

**Causes include:**
- H\(_2\)O loss without adequate H\(_2\)O intake
- Diuretics/Laxative abuse
- Osmotic diuresis (e.g. hyperglycaemia: beware pseudohypernatremia)
- Na\(^+\) gain (ingestion of sea water, infusion of large volumes of intravenous NaHCO\(_3\) 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 H\(_2\)O 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

\[ H₂O \text{ deficit}(L) = \text{body weight in kg} \times 0.6 \times \frac{(\text{actual Na}^+(\text{mmol/L}) - 140)}{140} \]

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 hypernatremia, 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. Hypernatremia 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.
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.

**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 SpaO₂s 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 Leovofloxacin alone if Penicillin allergic. (People with sickle cell disease are effectively asplenic and therefore susceptible to infection with encapsulated organisms such as Streptococcus pneumonia 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 $O_2$ tensions – a falling $PaO_2$ 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 sequestrating), 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)

Oral Medication:

Step 2 >65yrs

Lorazepam 0.5-1.0mg

Continue non-drug approaches. If little or no effect after 30 mins

Step 3 >65yrs

2nd Oral Medication

Further Lorazepam 0.5-1.0mg

Continue non-drug approaches. If little or no effect after 30 mins

Step 4 >65yrs

Consider alternative oral medication

Eg Quetiapine 12.5-50mg

(Discuss with a senior doctor before administration of any antipsychotic if a patient is known to have Parkinson’s Disease or Lewy Body dementia)

Continue non-drug approaches. If little or no effect after 30mins

Step 5 >65yrs

If no response, seek advice from a consultant physician, medical registrar or the psychiatry registrar on-call (via 02035135000).

Intra-muscular injection in severe cases ONLY

Lorazepam 0.5mg – 2mg IM or Haloperidol 0.5mg – 1mg IM

(Only use haloperidol if dementia with Lewy Bodies has been ruled out and no history of 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 contraindications to benzodiazepines and patients with cardiac disease:

**Step 2**

**18-65 yrs**

**Oral Medication:**
- Lorazepam 1-2mg or
- Buccal Midazolam 10mg

**Step 3**

**18-65 yrs**

**2ND**

**Oral Medication**
- Further Lorazepam 1-2mg or
- Buccal Midazolam 10mg

**Step 4**

**18-65 yrs**

**Intramuscular medication**
- Lorazepam 1-2mg i/m*
  or
- Midazolam 7.5mg i/m*
  (*if total daily dose not exceeded in Steps 2 & 3)

**Step 5**

**18-65 yrs**

For very disturbed patients, an enhanced sedative effect can be achieved by giving both benzodiazepine i/m and haloperidol i/m (if not contra-indicated & maximum doses not already reached)

**or**, Promethazine 25-50mg oral or 50mg I/M is an option if step 4 fails.

Seek advice from a more experienced doctor. Consider contacting the Psychiatry Registrar On-call (via 020 3513 5000) or Pharmacy.

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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**

**Oral Medication:**
- Promethazine 25-50mg oral or
- Haloperidol 5mg oral or
- Olanzapine 5-10mg oral

**Oral Medication:**
- If Haloperidol or Olanzapine given in step 2, give: Promethazine 25-50mg oral or
- If Promethazine given in step 2 above, give either Haloperidol 5mg oral, or
- Or Olanzapine 5-10mg oral

Wait 45-60 minutes to assess the response to oral medication. If the first dose of oral medication fails to produce an adequate effect, move to step 3.

**Oral or Intramuscular Medication:**
- Haloperidol 5mg oral or i/m
  (Where possible, obtain ECG before i/m administration)

Wait 45-60 minutes to assess the response to oral medication. If the first dose of oral medication fails to produce an adequate effect, move to step 3.
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.
<table>
<thead>
<tr>
<th>DRUG</th>
<th>(a) For patients aged &gt;65 years old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maximum oral dose in 24hrs</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>2mg</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>50mg</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>10mg</td>
</tr>
<tr>
<td>Midazolam</td>
<td>n/a</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>n/a</td>
</tr>
<tr>
<td>Promethazin</td>
<td>n/a</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DRUG</th>
<th>(b) For patients aged 18 - 65 years old</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maximum oral dose in 24hrs</td>
</tr>
<tr>
<td>Lorazepam</td>
<td>4mg</td>
</tr>
<tr>
<td>Haloperidol</td>
<td>20mg</td>
</tr>
<tr>
<td>Midazolam</td>
<td>20mg</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>20mg</td>
</tr>
<tr>
<td>Promethazin</td>
<td>100mg</td>
</tr>
</tbody>
</table>

** 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.
Delirium

Delirium 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.

The Delirium Assessment will be ordered automatically on iClip when an adult patient is admitted to a ward and must be filled out for all patients within 24 hours, find it by going to ‘Tasks’ and then ‘Medical’ tab. If the patient has any of the risk factors listed above the assessment will ask you to look for behavioural indicators of delirium, fill out a 4AT screen and decide if you can make a diagnosis of delirium, then ensure all precipitating factors are addressed. If they have no risk factors then the assessment is complete at that stage.

**Behavioural Indicators of 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**
Alertness
- Normal-0, mild sleepiness-0, clearly abnormal-4
AMT4 (DOB, age, place, current year)
- No mistakes-0, 1 mistake-1, 2 or more or untestable-4
Attention (months of the year backwards)
- Greater than7-0, <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.** Take a full collateral history of the confusion and behavioural changes, especially the symptoms listed in the behavioural indicators above. Use the 4AT as a screening tool or use CAM if you are confident in its use. Think about whether you can diagnose delirium based on the DSM-V criteria (disturbance in consciousness, change in cognition that develops over a short period of time and fluctuates).

Identify and treat the cause of the delirium. Ensure as full a physical examination as possible. Consider: urinary retention, constipation, pain, metabolic disturbances (sodium, calcium, renal, liver, thyroid, glucose), infection, cardiac or respiratory problems, 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 (including
head injury, epilepsy, meningoencephalitis etc), Wernicke’s encephalopathy. Remember delirium is often multifactorial.

Provide delirium information leaflet to relatives or patient. Document diagnosis of delirium in patient’s notes – if iClip Delirium Assessment is completed this will happen automatically.

Optimise the patient’s environment:
• Encourage presence of family or friends, ensure translator if needed
• Aim for continuity of care, minimising ‘new faces’, avoid ward and bed transfers, especially after 8pm
• Nurse in well-lit bay close to nursing station, use frequent reorientation to place and reason for admission
• Encourage early mobilisation, assess pressure ulcer risk
• Consider one to one nursing if the patient is very distressed
• Encourage adequate hydration and good nutrition, avoid constipation
• Minimise polypharmacy and review medication every 24 hours
• Use glasses and ensure hearing aids working and ears clean

If the patient is very agitated, refer to the section above on ‘Managing acutely agitated patients’, always starting with non-pharmacological intervention in the first instance.

If the patient has had more than two episodes of agitation requiring sedation during their inpatient stay they should be referred to the Liaison Psychiatry team, Dementia and Delirium team or Senior Health for advice on further management (Liaison Psychiatry are available out of hours, as is the Geriatrician on call via telephone)

If delirium symptoms are not resolving, seek help from Psychiatry liaison team or Senior Health.

Consult the intranet for patient information leaflet on delirium and further resources: http://stginet/Units%20and%20Departments/Delirium%20and%20Dementia/Delirium%20and%20Dementia.aspx
MANAGEMENT OF ALCOHOL WITHDRAWAL
Link Consultant: Dr Sarah Hughes

Advice on managing alcohol withdrawal is available from SGH Drug & Alcohol Liaison Team on Ext. 0595 or Bleep 6915, Monday to Saturday 8am to 5pm. 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.

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 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

<table>
<thead>
<tr>
<th>Questions</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Your score</th>
</tr>
</thead>
<tbody>
<tr>
<td>How often do you have a drink containing alcohol?</td>
<td>Never</td>
<td>Monthly or less</td>
<td>2-4 times per month</td>
<td>2-3 times per week</td>
<td>4+ times per week</td>
<td></td>
</tr>
<tr>
<td>How many units of alcohol do you drink on typical day when you are drinking?</td>
<td>1-2</td>
<td>3-4</td>
<td>5-6</td>
<td>7-9</td>
<td>10+</td>
<td></td>
</tr>
<tr>
<td>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?</td>
<td>Never</td>
<td>Less than monthly</td>
<td>Monthly</td>
<td>Weekly</td>
<td>Daily or almost daily</td>
<td></td>
</tr>
</tbody>
</table>

ASSESSMENT OF WITHDRAWAL
The following symptoms and signs suggest the possibility of acute alcohol withdrawal:

- Anxiety/agitation/irritability
- Nausea/vomiting/anorexia
- Tremor of hands, tongue or eyelids
- Insomnia
- Confusion
- 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
- Blood/Urine for other recreational drugs, where indicated
- 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. Printable versions of this are found on the Drug and Alcohol team page on the intranet. 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.

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. Total daily dose of chlordiazepoxide should not exceed 240mg. Patients should monitored at least 2 hourly in the first 24 hours, and until symptoms stable.
Table 2. Recommended chlordiazepoxide fixed dose regimens

<table>
<thead>
<tr>
<th>Severity</th>
<th>MODERATE</th>
<th>SEVERE</th>
<th>VERY SEVERE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol units/day</td>
<td>15 – 25</td>
<td>30 – 49</td>
<td>50 – 60</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Chlordiazepoxide prescribing (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1 (mg)</td>
</tr>
<tr>
<td>20 QDS</td>
</tr>
<tr>
<td>30 QDS</td>
</tr>
<tr>
<td>40 QDS</td>
</tr>
<tr>
<td>Day 2</td>
</tr>
<tr>
<td>15 QDS</td>
</tr>
<tr>
<td>25 QDS</td>
</tr>
<tr>
<td>35 QDS</td>
</tr>
<tr>
<td>Day 3</td>
</tr>
<tr>
<td>10 QDS</td>
</tr>
<tr>
<td>20 QDS</td>
</tr>
<tr>
<td>30 QDS</td>
</tr>
<tr>
<td>Day 4</td>
</tr>
<tr>
<td>5 QDS</td>
</tr>
<tr>
<td>15 QDS</td>
</tr>
<tr>
<td>25 QDS</td>
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<td>Day 5</td>
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<tr>
<td>5 BD</td>
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<td>10 QDS</td>
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<td>5 BD</td>
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<td>Day 10</td>
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<td>Prn</td>
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<td>10-20</td>
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<td>20-30</td>
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<tr>
<td>30</td>
</tr>
<tr>
<td>Max daily dose</td>
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<td>240</td>
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</tbody>
</table>

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 ≈ 10mg Diazepam ≈ 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 ≈ 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.
• 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 plus Vitamin B Co strong 2 tabs daily

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 plus Vitamin B Co strong 2 tabs daily
  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: Professor Paul Collinson

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: Isabelle Hamilton-Bower, 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)
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
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:


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 (HbA1c < 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 be 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 is 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.
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, with Malnutrition Universal Screening Test (MUST) scoring or iNUT (renal patients) complete.

Full guidance on nutritional support is detailed in the Trust policy available on the intranet via the following link:
http://stginet/Procedural%20Documents/Patient%20related/Patient_Management/Clin_5_41_nutrition%20support.pdf

In addition, Trust guidance and policy on use of naso-gastric tubes can be found via the following link:

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 cost, 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 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. For Critical Care unit referrals are to be made directly to the Nutrition Team Dietitian (bleep 6171).

The cut-off time for the submission of Parenteral Nutrition orders for feeding to initiate the same working day is 09:30am therefore referrals should be made in a timely manner. 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 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 (link above) for guidance on replacement of electrolytes and provision of thiamine (IV Pabrinex®). 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 daily (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. 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 (Ext. 3569)
Dr Jamal Hayat, Consultant Gastroenterologist (Ext. 3569, Dr Kamal Patel (Ext. 3429 or 1206)); Tracey Marshall & Emma Pindard, Nutrition nurse specialists (Bleep 8050/8498); the Gastroenterology SpR (Bleep 6590 or 7593), Sheung Him Lee, Lead Pharmacist Adult PN (Bleep 8580/7554)/ PN Pharmacist (Bleep 7554); Alison Green, Principal Dietitian Nutrition Team, Intestinal Failure and GI Services (Bleep 6171), and Evanna Leavy, Dietician Nutrition Team (Blp 6171).

**Out of hours**
For questions or concerns regarding existing patients contact the on-call pharmacist via Bleep 6267. The Dietitian’s weekend service is available 08:30 until 16.30 on Saturdays and Sundays and may be contacted via Bleep 8169.
APPENDIX 4b

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 – 1630

The 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:

Day 1-2: ½ breakfast, yoghurt, ¼ lunch, yoghurt, ¼ supper = 550-600kcal
Day 3-4: 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 intranet 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.
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 death\(^1\)(Figure 1\(^2\)).

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 electrolytes\(^1\). However, it is common for potassium, magnesium and phosphate to be within normal serum levels prior to feeding due to homeostatic mechanisms\(^1\). The consequence of these shifts can result in many different presentations from fluid retention, cardiac arrhythmias, respiratory insufficiency and possibly death\(^1\).

Clinical signs of starvation include\(^1\):
- 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 Syndrome\(^2\)
Identifying at-risk groups

- 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 steatorrhoea
- Diabetic hyperosmolar states resulting from poorly controlled or undiagnosed diabetes
- Morbid obesity with large weight loss weight loss (e.g. after gastric banding)

The following has been based on NICE CG36 guidelines.

Step 1:

<table>
<thead>
<tr>
<th>Identifying Refeeding Risk</th>
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<tbody>
<tr>
<td>At Risk</td>
<td>• Very little intake for greater than 5 days</td>
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<tr>
<td>High Risk</td>
<td><strong>The Patient has one or more of the following:</strong></td>
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<td>• BMI less than 16kg/m²</td>
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<td>• Unintentional weight loss greater than 15% within the previous 3-6 months</td>
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<tr>
<td></td>
<td>• Very little nutritional intake for greater than 10 days</td>
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<tr>
<td></td>
<td>• Low levels of potassium, phosphate or magnesium prior to feeding</td>
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<tr>
<td></td>
<td><strong>Or the patient has two or more of the following:</strong></td>
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<td></td>
<td>• BMI less than 18.5kg/m²</td>
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<tr>
<td></td>
<td>• Unintentional weight loss greater than 10% within the previous 3-6 months</td>
</tr>
<tr>
<td></td>
<td>• Those with very little intake for greater than 5 days</td>
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<tr>
<td></td>
<td>• A history of alcohol abuse or drugs including insulin, chemotherapy, antacids or diuretics (interpret with caution)</td>
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<tr>
<td>Extremely High Risk</td>
<td><strong>The patient has either of the following:</strong></td>
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<td></td>
<td>• BMI less than 14kg/m²</td>
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<tr>
<td></td>
<td>• Negligible intake for greater than 15 days</td>
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Step 2:

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<thead>
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<th>Vitamin and Mineral Supplementation</th>
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<tr>
<td><strong>Oral</strong></td>
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<tr>
<td>Thiamine</td>
<td>100 mg BD-TDS for 10 days ***</td>
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<tr>
<td>Vitamin B Co Strong</td>
<td>1 or 2 tablets TDS for 10 days</td>
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<tr>
<td>Multivitamin: Valupak Multivitamin</td>
<td>OD for 10 days</td>
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<tr>
<td><strong>NBM</strong></td>
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<tr>
<td>Pabrinex</td>
<td>Give one pair of ampoules OD for 10 days* ** ***</td>
</tr>
<tr>
<td>Multivitamin: Abidec Liquid</td>
<td>1.2 mL OD**</td>
</tr>
<tr>
<td><strong>Renal</strong></td>
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<tr>
<td>Dialysis pts</td>
<td>Renavit OD and Thiamine</td>
</tr>
</tbody>
</table>

*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:

| At risk   | • 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 | • 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 | • 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:

<table>
<thead>
<tr>
<th>Serum Levels 3.5-4.7mmol/L</th>
<th>Mild Higher than 3mmol/L</th>
<th>Moderate Asymptomatic 2.5-3 mmol/L</th>
<th>Severe Less than 2.5mmol/L or symptomatic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient able to tolerate oral or enteral therapy</td>
<td>Potassium effervescent tablets (Sando-K® 12mmol/tablet). Two tablets twice daily (48mmol).</td>
<td>Potassium effervescent tablets (Sando-K® 12mmol/tablet). Two tablets three times daily (72mmol).</td>
<td>Not appropriate. See i.v. therapy</td>
</tr>
<tr>
<td>NB: Use i.v. therapy if patient has diarrhoea or high output stoma.</td>
<td>Infuse potassium into a large vein at up to 20 mmol K+/hr (not more than 200 mmol/day). If plasma K+ &lt; 2 mmol/L with arrhythmia, 40 mmol K+ may be given over 1hr. Bags for IV potassium infusion are available through Pharmacy.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient on intravenous therapy only</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renal:</td>
<td>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.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

[References for this section]
## Replacing electrolytes: Phosphate

<table>
<thead>
<tr>
<th>Serum Levels 0.75-1.5mmol/L</th>
<th>Moderate (Asymptomatic) 0.4-0.6 mmol/L</th>
<th>Moderate Symptomatic 0.4-0.6 mmol/L</th>
<th>Severe Less than 0.4mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient able to tolerate oral or via enteral tube feeding <strong>NB: Use i.v. therapy if patient has diarrhoea or high output stoma.</strong></td>
<td>Phosphate Sandoz® Effervescent tablets (16.1mmol/tablet) Two tablets twice daily (64mmol)</td>
<td>Oral therapy not appropriate. Use i.v. therapy – See below</td>
<td></td>
</tr>
<tr>
<td>Patient on intravenous therapy only</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>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)</td>
<td>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.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### 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 predialysis bloods only to identify need for replacement. In renal impairment check dose of IV prescription with renal pharmacist.

## Replacing electrolytes: Magnesium

<table>
<thead>
<tr>
<th>Serum Levels 0.75-1.03mmol/L</th>
<th>Mild Greater than 0.5mmol/L</th>
<th>Severe Less than 0.5mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient on intravenous therapy only</td>
<td>10mmol Magnesium sulphate in 50ml sodium chloride 0.9% over 4 hours</td>
<td>20mmol Magnesium sulphate in 50mls sodium chloride 0.9% over 4 hours</td>
</tr>
<tr>
<td>Patient able to tolerate oral or via enteral tube feeding <strong>NB: Use i.v. therapy if patient has diarrhoea or high output stoma.</strong></td>
<td>Magnesium glycerophosphate 4mmol tablets/solution Two tablets three times a day (24mmol)</td>
<td>Not appropriate – See i.v above</td>
</tr>
</tbody>
</table>
Replacing electrolytes: Corrected Calcium

<table>
<thead>
<tr>
<th>Serum Levels 2.15-2.5mmol/L</th>
<th>Monitor corrected calcium when magnesium levels are low. It is important to look at corrected calcium when making any assessment of calcium supplementation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient able to tolerate oral or via enteral tube feeding</td>
<td>Sandocal “400” tablets containing 10mmol of calcium per tablet</td>
</tr>
</tbody>
</table>

Calcium must not be added to enteral/parenteral feeds due to stability issues. Please contact ward Dietitian or pharmacist for advice.

Monitoring and Discharge

- 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:
  - 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 | • 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 | • 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 | • Ensure correct medication and dose is prescribed for the individual patient  
|            | • Ensure refeeding medications are discontinued on discharge if appropriate |
| Nurses | • 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
10 – only px refeeding vitamins for 10/7
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\(^+\): <2.5 mmol/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\(^\circledR\) 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 1 hr. 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\(^\circledR\) 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, Thamine, Vit B co strong or replacement medication is not added to the TTO’s for cost efficiency

Refeeding Guide for Doctors: Bloods and Prescribing

APPENDIX 4d. HPN troubleshooting algorithms: see APPENDICES on Intranet
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 \(\text{HCO}_3^-\).

**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);

**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,

\[
\text{‘Anion Gap’} = \text{the sum of } \{ [\text{Na}]^+ + [\text{K}]^+ \} - \{ [\text{Cl}]^- + [\text{HCO}_3^-] \}
\]

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 \(\text{HCO}_3^-\) there will be an equivalent rise in \([\text{Cl}]^-\) 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 \(\text{HCO}_3^-\), as in diarrhoea, proximal renal tubular acidosis
- Decreased renal acid excretion *eg.* distal renal tubular acidosis

_B) With increased anion gap_

<table>
<thead>
<tr>
<th>Condition</th>
<th>Unmeasured Anions</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>a) Lactic Acidosis</em></td>
<td>Lactate, phosphate, urate</td>
</tr>
<tr>
<td><em>b) Ketoacidosis</em></td>
<td>Ketone bodies (acetone, acetoacetate, (\beta)-hydroxybutyrate)</td>
</tr>
<tr>
<td><em>c) Intoxication</em></td>
<td>(\beta)-hydroxybutyrate, lactate, acetoacetate, Glycolate, oxalate, Formate, Acetate, Ketone, lactate, salicylate, Sulphate, phosphate</td>
</tr>
</tbody>
</table>
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 acidic 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 (e.g. 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.
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 (see Antibiotic Management Table), 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 antimitotics 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 commencing any anticoagulant, evaluation of the relative risk of bleeding vs thromboembolism is required. If there is clinical suspicion of active major bleeding, anticoagulation should be withheld and urgent confirmatory tests performed.

HEPARIN
Monitoring heparin therapy.

i) Low molecular weight heparin
Patients receiving low molecular weight heparin (e.g. dalteparin) therapy do not routinely require monitoring. However, monitoring using the anti-Xa heparin assay should be carried out in:
- patients with a creatinine clearance below 30 ml/min
- patients with extremes of body weight
- women who are pregnant and taking therapeutic doses

Initially, weekly measurement is advised. The anti Xa assay (measured 3-4 hrs after injection) should be 0.2 – 0.4 units/mL for prophylaxis, or 0.5-1.0 units/mL for treatment of acute venous thromboembolism when given once daily.

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 used as its half life is 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 6 hours after infusion started (and after any dose change) and adjust as follows:

<table>
<thead>
<tr>
<th>APTT ratio</th>
<th>ACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;6</td>
<td>stop for 1 hr; reduce by 1mL/hr (500units/hr)</td>
</tr>
<tr>
<td>5.3-5.9</td>
<td>reduce by 0.6mL/hr (300units/hr)</td>
</tr>
<tr>
<td>4.7-5.2</td>
<td>reduce by 0.4mL/hr (200units/hr)</td>
</tr>
<tr>
<td>4.1-4.6</td>
<td>reduce by 0.2mL/hr (100units/hr)</td>
</tr>
<tr>
<td>3.6-4.1</td>
<td>reduce by 0.1mL/hr (50units/hr)</td>
</tr>
<tr>
<td>1.5-3.5</td>
<td>NO CHANGE</td>
</tr>
<tr>
<td>1.2-1.4</td>
<td>increase by 0.4mL/hr (200units/hr)</td>
</tr>
<tr>
<td>&lt;1.2</td>
<td>increase by 0.8mL/hr (400units/hr)</td>
</tr>
</tbody>
</table>

Repeat APTT ratio daily while on IV unfractionated heparin. Check platelet count at start of therapy and after 5 days. Where therapeutic doses of subcutaneous unfractionated heparin (e.g. 250 units/kg bd) is used, monitoring is again essential. An APTT ratio should be taken 6 hrs after injection, this can be reduced to daily once the APTT ratio is in range (target 1.5-3.5); discuss these patients with haematology (bleep 6068 or via switchboard out of hours).
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”, e.g. 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, no more than 50mg to 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 Department on-call 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.

Heparin Induced Thrombocytopenia (HIT). Up to 1 in 10 patients on IV or SC heparin may have a fall in platelet count which is usually transient and resolves spontaneously in 24-48 hours without thrombotic complication. Rarely the interaction between heparin and an antibody in the plasma causes platelet activation and thrombocytopenia. This can lead to an explosive thrombotic state called HIT. It is crucial to recognise this syndrome and immediately stop heparin by ALL routes and in ALL doses (including IV fluids and cannulae, eg. flushes). The alternative anti-thrombotic agents, argatroban (for all adult patients) or danaparoid (for pregnant patients) should be used in this situation. Seek urgent advice from the Haematology Department on-call registrar (bleep 6068 or via switchboard out of hours).

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 18-24 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 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 (phytomenadione) 1-3mg IV Repeat INR in 8 hours. Vitamin K administration is, however, not 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 5.0mg by slow IV injection. Prothrombin Complex Concentrate (PCC), held in the Haemophilia centre, should be given. Contact the on-call haematology registrar (bleep 6068 or via switchboard out of hours).
Recommended doses of PCC:
- INR ≤4.0: 20 units/kg;
- INR >4.0: 30 units/kg

The single dose should not exceed 3000 units. FFP (15 mL/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 unnecessary warfarin resistance.

OTHER ANTICOAGULANTS

Fondaparinux
Protamine has no effect on the anticoagulant effect of fondaparinux. Recombinant factor VIIa (*NovoSeven*) can be used for critical bleeding. Contact the on-call haematology registrar (Bleep 6068 or via switchboard out of hours).

Rivaroxaban, edoxaban, dabigatran and apixaban (Direct Oral Anticoagulants - DOACs)
The DOAC bleeding pathway should be followed:
http://stginet/Units%20and%20Departments/Haematology/ANTICOAGULATION/Management%20of%20Haemorrhage%20on%20NOACs.pdf

Dabigatran is the only DOAC to currently have a licensed specific reversal agent, Idarucizumab (Praxbind). This is kept in 2 locations at St Georges Hospital: ED resus and haematology labs. Please consult the full protocol:
http://stginet/Units%20and%20Departments/Haematology/ANTICOAGULATION/Idarucizumab.pdf

There are no reversal agents available for apixaban, edoxaban and rivaroxaban. If there is bleeding associated with these:
- 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 1 g 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 >80 g/L), platelets (aim for PLT > 60 x 10^9/L or if CNS bleed aim PLT > 100 x 10^9/L). Consider tranexamic acid 1 g po/iv. Consider PCC 25-50 units/Kg). **Urgent Haematology advice should be sought** (bleep 6068 or via switchboard out of hours).

**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 Blood Bank 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 Bank page and in the Blood Transfusion policy:
HIV POST-EXPOSURE PROPHYLAXIS (PEP)

Link Consultant: Dr Bojana Dragovic

APPENDIX 8

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 www.bashhguidelines.org/current-guidelines/hiv/post-exposure-prophylaxis-following-sexual-exposure/

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).

PEPSE is recommended where there is a significant risk of HIV transmission (risk>1/1000), see Table 3 in the national guidelines linked above.

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 A&E Department is the first point of contact for PEPSE, where following assessment, if PEPSE is required, an emergency 5-day pack containing Truvada and Raltegravir will be issued. To arrange follow up and further 23 day continuation pack contact the Sexual Health South West London Clinic (Monday to Friday 8am - 8pm) Telephone: 0333 300 2100

2. Occupational post-exposure prophylaxis

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 an undetectable viral load (<200 copies/ml) for more than 6 months, occupational PEP is not recommended. PEP should be offered to those who are anxious about the risk.

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 A&E and ask for immediate attention (Do not wait for triage)
- Complete an adverse incident form
- If the injury is reported to A&E 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/ A&E 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

Antiretrovirals prescribed for both PEPSE and occupational PEP

- **TRUVADA®** (tenofovir 300mg plus emtricitabine 200mg), one tablet once a day;
- **RALTEGRAVIR** 400mg, one tablet twice a day.

If the source patient is known, other combinations may be more appropriate – seek advice. PEP is still indicated in pregnancy but please seek advice from the Clinical Infection Unit. Emergency 5-day packs containing Truvada and Raltegravir are kept by Staff Health, Pinckney Ward, McEntee Ward, A&E, Courtyard Pharmacy and Laneborough Pharmacy.

WHEN TO TEST FOR HIV INFECTION
HIV testing should be considered for all general medicine admissions (15 to 59 years of age) in areas of high seroprevalence (>2 per 1,000 residents). The seroprevalence for London in 2017 was 5.7 per 1,000. A&E at St George’s Hospital are now offering opt out HIV testing to all patients having a blood test aged 15-59yrs.
Late HIV diagnosis, however, remains a serious problem – in 2014 40% of newly diagnosed adults had CD4 <350 cells/mm3. Testing should be offered in particular to people with indicator conditions. The link below is a useful resource for decision-making around testing:
https://www.bhiva.org/file/RHNUJgIseDaML/GlinesHIVTest08.pdf
**HIGHER RISK GROUPS**

While all general medicine admissions should be tested, particular at risk groups are:

1. Men who have sex with men (MSM)
2. Haemophiliacs
3. IV drug users
4. Heterosexual men and women who have partners from areas of high prevalence (e.g. sub Saharan Africa, SE Asia and parts of the Caribbean)
5. Sexual partners of the above (and children of 3 & 4)

Any patient thought to be at risk presenting with any of the above indicator conditions should be referred to the on-call resident CIU SHO/SpR.

**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.
APPENDIX 9

FIRST STEPS IN THE EVENT OF A MAJOR INCIDENT
Link Consultants: Dr Ainsley Heyworth & Dr Phil Moss

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

**Gentamicin**¹ 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).

**Vancomycin**¹ 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](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.

**Carbamazepine**² 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.

**Digoxin**² 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).

**Lithium**² 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.

**Phenytoin**² 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:
¹Microbiology, ext 5685/6 or Bleep 480 or via switchboard out of hours.
²Chemical Pathology, Bleep 6032 or pager SG 138 or via switchboard out of hours.
**ANTIFUNGAL MONITORING**

- Send sample in Yellow Cap Tube - SST to Rm 0.222, Ground Floor, Jenner Wing, SGUL using the form in appendix III.
- Assays are performed twice weekly on Tuesday and Friday, with results available the next working day. Use the blood form in appendix.
- If levels are outside the therapeutic range contact pharmacy (Bleep 7508) for advice on dose adjustment.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indication for monitoring</th>
<th>Time of measurement after start of therapy (days)</th>
<th>Target blood concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Efficacy</td>
</tr>
<tr>
<td>Flucytosine</td>
<td>Routine during first week of therapy, renal insufficiency, lacking response to therapy, co-administration of interacting medicines</td>
<td>3-5</td>
<td>Trough 20-40mg/L</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Peak 50-100 mg/L</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>An estimate of total antifungal activity using HPLC requires separate assays for both itraconazole and hydroxyitraconazole (an active metabolite of comparable potency to the parent compound).</td>
<td>5-7</td>
<td>For prophylaxis; Trough ≥0.5mg/L</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>For treatment; Trough ≥0.5–1.0mg/L</td>
</tr>
<tr>
<td>Voriconazole</td>
<td>Routine during first week of starting therapy, lacking response; GI dysfunction; co-medication; IV-to-oral switch; severe hepatic impairment; unexplained neurological symptoms/signs</td>
<td>4-7</td>
<td>Trough &gt;1-2mg/L</td>
</tr>
<tr>
<td>Posaconazole</td>
<td>Routine during first week of starting therapy, lacking response; GI dysfunction, co-administration of interacting medication</td>
<td>4-7</td>
<td>For prophylaxis; Trough &gt;0.7mg/L at steady state (or 0.35mg/L 48h after initiation of therapy)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>For treatment; Trough &gt;1.0 mg/L</td>
</tr>
</tbody>
</table>
AMIKACIN DOSING GUIDELINES (once daily dosing)

Exclusions:
- Paediatric patients
- Patients with ascities and cirrhosis
- Burns >15% BSA
- Pregnancy
- Patients allergic to amikacin or other aminoglycosides

Do not give more than 72 hours of amikacin without microbiology approval – prolonged courses are rarely necessary and increase the risk of toxicity (max licensed cumulative dose is 15g)

Instructions:
1) Calculate the patient’s creatinine clearance (CrCl) using the Cockroft-Gault* equation.

Where dosing regimen is dependent on creatinine clearance (CrCl), this can be calculated using the Cockroft-Gault* equation:

*Creatinine clearance (ml/min) = \( \frac{(140 - \text{age}) \times \text{weight}^\# \text{(kg)}}{\text{Serum Creatinine} \text{ (umol/L)} \times 1.04 \text{ for females OR 1.23 for males}} \)

Obese patients (>30% over ideal body weight) should use adjusted weight for the creatinine clearance estimation
Adjusted wt = Ideal Body Wt + 0.4 x (Actual Wt – ideal body wt)
Ideal body wt (kg) = (2.3 x height in inches above 5 ft) + 45 (for females) OR + 50 (for males)

2) Select initial dose based on patients weight and renal function as shown in Table 1 below. Adjusted body weight should be used for obese patients (see formula above) but cap adjusted weight at 100kg.

- Patients with severe sepsis and those on ICU (where volume of distribution is increased) a single 15mg/kg dose may be used. Levels must fall below 5mg/L before the patient receives a second dose. Doses of up to 25mg/kg may be used in difficult to treat infections

<table>
<thead>
<tr>
<th>Table 1. Dosing regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CrCl (based on Cockroft- Gault equation)</strong></td>
</tr>
<tr>
<td><strong>Patient age</strong></td>
</tr>
<tr>
<td><strong>Dose</strong></td>
</tr>
<tr>
<td><strong>Take levels</strong></td>
</tr>
<tr>
<td><strong>Timing of 2nd dose</strong></td>
</tr>
</tbody>
</table>
3) Administer in 100ml sodium chloride 0.9% over 30 minutes
4) Take levels at the time indicated in table 1 (document sampling time on the blood form)
5) Give 2nd dose without waiting for level results in patients <65 yrs with good renal function. Await levels <5mg/L before re-dosing in elderly patients and those with renal impairment
6) Adjust maintenance dose according to level result (see Table 2 below)

### Table 2. Trough level interpretation

<table>
<thead>
<tr>
<th>Level</th>
<th>Trough level interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 5.0 mg/L</td>
<td>Continue current dosing regimen</td>
</tr>
<tr>
<td>5-10 mg/L</td>
<td>Recheck levels 12 hrs later – withhold dose pending results*</td>
</tr>
<tr>
<td>&gt; 10 mg/L</td>
<td>Recheck levels 24 hrs later – withhold dose pending results*</td>
</tr>
</tbody>
</table>

* Consider reducing dose or increasing dosing interval if levels are above the therapeutic range

7) Check serum creatinine & urea daily for patients on IV amikacin
8) Repeat levels every 3 days for haemodynamically stable patients with stable renal function whose last level was in range. More frequent monitoring may be necessary for other patients.

**VANCOMYCIN DOSING GUIDELINES (intermittent)**
These guidelines are designed to achieve trough levels of 10 to 15mg/L. For severe infections such as endocarditis, osteomyelitis, MRSA pneumonia, or bacteraemias, higher doses may be required – contact microbiology for advice.

**Exclusions:**
- If vancomycin therapy is required for Clostridium difficile infections, the oral route should be used [http://www.lhp.leedsth.nhs.uk/common/guidelines/detail.aspx?id=1254](http://www.lhp.leedsth.nhs.uk/common/guidelines/detail.aspx?id=1254)
- ITU patients - continuous IV vancomycin infusions are used (see local guidelines)
- Children under the age of 16 years (see local guidelines)
- Patients allergic to vancomycin or other glycopeptides
- Dialysis patients

**Instructions:**
1) Give an initial loading dose based on the patient’s actual body weight, as in Table 1

### Table 1: Loading dose

<table>
<thead>
<tr>
<th>Weight (actual body weight)</th>
<th>&lt; 60kg</th>
<th>60 - 90kg</th>
<th>&gt; 90kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loading dose</td>
<td>1g</td>
<td>1.5g</td>
<td>2g</td>
</tr>
<tr>
<td>Fluid (NaCl 0.9% or glucose 5%)</td>
<td>250ml</td>
<td>500ml</td>
<td>500ml</td>
</tr>
<tr>
<td><strong>Infusion period</strong></td>
<td>Max rate</td>
<td>Max rate</td>
<td>Max rate</td>
</tr>
<tr>
<td>Max rate</td>
<td>10mg/min</td>
<td>10mg/min</td>
<td>10mg/min</td>
</tr>
</tbody>
</table>

2) Calculate the initial maintenance dose based on the patient’s creatinine clearance using the Cockroft-Gault* equation

\[
*\text{Creatinine clearance (ml/min)} = \frac{(140 - \text{age}) \times \text{weight (kg)}}{\text{Serum Creatinine (µmol/L)}} \times 1.04 \text{ for females OR 1.23 for males}
\]
3) Give the 1st maintenance dose specified in Table 2 after the dosing interval specified in Table 2 below.

<table>
<thead>
<tr>
<th>Creatinine clearance* (ml/min)</th>
<th>Maintenance Dose</th>
<th>Start time after LD &amp; future dosing interval</th>
<th>Volume of fluid (NaCl 0.9% or glucose 5%)</th>
<th>Infusion Period</th>
<th>Time of first trough level</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;110</td>
<td>1.5g</td>
<td>12 hours</td>
<td>500mL</td>
<td>Max 10mg/min</td>
<td>Before 4&lt;sup&gt;th&lt;/sup&gt; dose</td>
</tr>
<tr>
<td>90-110</td>
<td>1.25g</td>
<td>12 hours</td>
<td>25mL</td>
<td>Max 10mg/min</td>
<td>Before 4&lt;sup&gt;th&lt;/sup&gt; dose</td>
</tr>
<tr>
<td>75-89</td>
<td>1g</td>
<td>12 hours</td>
<td>250mL</td>
<td>Max 10mg/min</td>
<td>Before 4&lt;sup&gt;th&lt;/sup&gt; dose</td>
</tr>
<tr>
<td>55-74</td>
<td>750mg</td>
<td>12 hours</td>
<td>250mL</td>
<td>Max 10mg/min</td>
<td>Before 4&lt;sup&gt;th&lt;/sup&gt; dose</td>
</tr>
<tr>
<td>40-54</td>
<td>500mg</td>
<td>12 hours</td>
<td>250mL</td>
<td>Max 10mg/min</td>
<td>Before 4&lt;sup&gt;th&lt;/sup&gt; dose</td>
</tr>
<tr>
<td>30-39</td>
<td>750mg</td>
<td>24 hours</td>
<td>250mL</td>
<td>Max 10mg/min</td>
<td>Before 3&lt;sup&gt;rd&lt;/sup&gt; dose</td>
</tr>
<tr>
<td>20-29</td>
<td>500mg</td>
<td>24 hours</td>
<td>250mL</td>
<td>Max 10mg/min</td>
<td>Before 3&lt;sup&gt;rd&lt;/sup&gt; dose</td>
</tr>
<tr>
<td>&lt;20 (No dialysis)</td>
<td>500mg</td>
<td>48 hours</td>
<td>250mL</td>
<td>Max 10mg/min</td>
<td>Before 2&lt;sup&gt;nd&lt;/sup&gt; dose</td>
</tr>
</tbody>
</table>

For fluid restricted patients please contact pharmacy for advice.

4) Monitor pre-dose (trough) level at time specified in Table 2. Levels must be taken 0-60 minutes pre-dose with the sampling time documented on the blood form.
- Do NOT wait for the result of the level before giving the next dose.

5) Adjust maintenance dose according to current dosing regime and guidance in Table 3

<table>
<thead>
<tr>
<th>Pre-dose (trough) level</th>
<th>Maintenance dose adjustment (table 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 5mg/L</td>
<td>Contact pharmacy</td>
</tr>
<tr>
<td>5 to 6.9mg/L</td>
<td>Move up two dosing levels in table 2</td>
</tr>
<tr>
<td>7 to 9.9mg/L</td>
<td>Move up one dosing level in table 2</td>
</tr>
<tr>
<td>10 to 15.9 mg/L</td>
<td>Continue at current dose.</td>
</tr>
<tr>
<td>16 to 19.9 mg/L</td>
<td>Move down one dosing level in table 2 without omitting any doses</td>
</tr>
<tr>
<td>20 to 25mg/L</td>
<td>Omit next dose &amp; decrease by 2 dosing levels in table 2</td>
</tr>
<tr>
<td>More than 25mg/L</td>
<td>Contact pharmacy</td>
</tr>
</tbody>
</table>

- Trough levels should be maintained above 10mg/L to ensure effective therapy and help avoid resistance. Repeat trough levels every 3 days in haemodynamically stable patients with stable renal function whose last level was in range. More frequent monitoring may be necessary for other patients.
- Daily serum creatinine & urea is recommended for patients on IV vancomycin
- Monitor FBC regularly as neutropenia or thrombocytopenia can occur after prolonged therapy.
- Contact microbiology or pharmacy if required for advice on dose adjustments, and before using doses above 1.5g twice daily.
Table 4. Trough level interpretation & maintenance dose adjustment when levels 15-20mg/L are required

<table>
<thead>
<tr>
<th>Pre-dose (trough) level</th>
<th>Maintenance dose adjustment (table 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 9mg/L</td>
<td>Ensure level is timed appropriately and no doses were omitted prior to sampling. If true trough level at steady state is &lt;9 double the daily dose. Do not give single doses of more than 2g. In instances where &gt;4g daily are required increase the frequency of administration (i.e. if levels are &lt;9 on 1.5g BD increase dose to 2g 8 hourly)</td>
</tr>
<tr>
<td>9 to 14 mg/L</td>
<td>Multiply the dose by 1.5 (i.e. if levels are 11 on 1g BD increase the dose to 1.5g BD)</td>
</tr>
<tr>
<td>15 to 20 mg/L</td>
<td>Continue at current dose.</td>
</tr>
<tr>
<td>21 to 28 mg/L</td>
<td>Multiple the current dose by 0.75 (i.e. if levels are 22 on 1g BD change dose to 750mg BD)</td>
</tr>
<tr>
<td>More than 28 mg/L</td>
<td>Omit doses until levels fall below 20mg/L then restart at half the current dose</td>
</tr>
</tbody>
</table>

- Trough levels should be maintained above 15mg/L in deep-seated infections to ensure effective therapy and help avoid resistance.
- Daily serum creatinine & urea is recommended for patients on IV vancomycin
- Repeat trough levels every 3 days in haemodynamically stable patients with stable renal function whose last level was in range. More frequent monitoring may be necessary for other patients.
- Monitor FBC regularly as neutropenia or thrombocytopenia can occur after prolonged therapy.

**APPENDIX 11**

**URGENT REMOVAL OF TUNNELLED OR HICKMAN/PORTACATH LINES**

Link: Jackie Nicolson, Independent Nurse Practitioner

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: http://stginet/Units%20and%20Departments/Central%20Venous%20Access/Homepage.asp