“THE GREY BOOK”

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

February 2015

62nd edition
<table>
<thead>
<tr>
<th>Topic</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardio-vascular emergencies</td>
<td></td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>3</td>
</tr>
<tr>
<td>Hypertension</td>
<td>6</td>
</tr>
<tr>
<td>Acute coronary syndromes</td>
<td>8</td>
</tr>
<tr>
<td>Management of STEMI</td>
<td>8</td>
</tr>
<tr>
<td>Management of NSTEMI</td>
<td>10</td>
</tr>
<tr>
<td>Acute heart failure</td>
<td>12</td>
</tr>
<tr>
<td>Disorders of cardiac rhythm</td>
<td>14</td>
</tr>
<tr>
<td>Acute deep vein thrombosis (DVT)</td>
<td>17</td>
</tr>
<tr>
<td>Acute pulmonary embolism</td>
<td>19</td>
</tr>
<tr>
<td>Respiratory emergencies</td>
<td></td>
</tr>
<tr>
<td>Respiratory arrest</td>
<td>22</td>
</tr>
<tr>
<td>Oxygen therapy in acute illness</td>
<td>24</td>
</tr>
<tr>
<td>Asthma</td>
<td>25</td>
</tr>
<tr>
<td>Spontaneous pneumothorax</td>
<td>27</td>
</tr>
<tr>
<td>Gastro-intestinal emergencies</td>
<td></td>
</tr>
<tr>
<td>Acute gastro-intestinal bleeding</td>
<td>29</td>
</tr>
<tr>
<td>Bleeding oesophageal varices</td>
<td>32</td>
</tr>
<tr>
<td>Acute bloody diarrhoea</td>
<td>34</td>
</tr>
<tr>
<td>Diabetic &amp; endocrine emergencies</td>
<td></td>
</tr>
<tr>
<td>Diabetic ketoacidosis (DKA)</td>
<td>36</td>
</tr>
<tr>
<td>Hypoglycaemia</td>
<td>39</td>
</tr>
<tr>
<td>Neurology emergencies</td>
<td></td>
</tr>
<tr>
<td>Acute stroke</td>
<td>40</td>
</tr>
<tr>
<td>Status epileptic (convulsive SE)</td>
<td>44</td>
</tr>
<tr>
<td>Anaphylaxis</td>
<td>46</td>
</tr>
<tr>
<td>Acute pain</td>
<td>47</td>
</tr>
<tr>
<td>Suggestions for the use of antimicrobial drugs in adults</td>
<td>49</td>
</tr>
<tr>
<td>Febrile neutropenia</td>
<td>52</td>
</tr>
<tr>
<td>Infective endocarditis</td>
<td>52</td>
</tr>
<tr>
<td>Upper respiratory tract infection</td>
<td>53</td>
</tr>
<tr>
<td>Antibiotic Management Table</td>
<td>54</td>
</tr>
<tr>
<td>Diabetic foot infection</td>
<td>55</td>
</tr>
<tr>
<td>Infectious diarrhoea</td>
<td>56</td>
</tr>
<tr>
<td>Management of C.Difficile</td>
<td>56</td>
</tr>
<tr>
<td>Endocarditis prophylaxis</td>
<td>59</td>
</tr>
<tr>
<td>Prophylactic antibiotics in surgery and trauma</td>
<td>60</td>
</tr>
<tr>
<td>Surgical wound infection</td>
<td>60</td>
</tr>
<tr>
<td>Malaria in returning travellers</td>
<td>60</td>
</tr>
</tbody>
</table>

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GENERAL POINTS

“Guidelines for the Management of Common Medical Emergencies and for the Use of Antimicrobial Drugs” or the Grey Book, was first published and edited by Professor Joe Collier in August 1979. It is probably the oldest established set of such guidelines in the UK.

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 http://stginet/greybook/

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.

- Clinically relevant material new to this edition is printed in bold type and 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.

- When asked to accept emergency/urgent referrals from GPs or other Trusts, priority should go to patients from Wandsworth and Sutton & Merton PCTs, and to anyone who has a significant history of previous care at St. George’s Hospital. Most patients who present with a medical emergency, and certainly all those on their initial visit to A&E, will be treated free under the NHS. Always seek advice on the eligibility of all non-UK residents for NHS treatment, by contacting the Overseas Patients Department (ext.4693/3439).

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.

**UK Resuscitation Council Adult Life Support (ALS) Algorithm (2010)**

**Unresponsive?**
Not breathing or only occasional gasps

- Call Resuscitation Team

**CPR 30:2**
Attach defibrillator/monitor. Minimise interruptions

**Assess rhythm**

- **Shockable**
  - VF/Pulseless VT
  - 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.
SEVERE HYPERTENSION
Link consultant: Dr Tarek Antonios

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.

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, seizures, coma), intracranial 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 2-6 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 a high dependency or intensive care 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** is the parenteral drug of choice for most hypertensive emergencies. It is an arteriolar and a venous dilator and has an immediate onset and short duration of action, t 1/2 2-3 min. It is administered by intravenous infusion starting at 0.3microgram/kg/min, increasing by 0.5microgram/kg/min every 5 minutes, to a maximum of 8micrograms/kg/min. The use of nitroprusside 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 2micrograms/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.
  - **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 5micrograms/min to be increased by 10micrograms/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.
  - **Labetalol,** a combined α- and β-blocker, is a logical option for patients with ischaemic heart disease, aortic dissection or dysphagic stroke patients; it is also safe in pregnancy. It is given either by slow intravenous injection: 20mg over 1 minute initially, followed by 20-80mg every 10 minutes to a total dose of 200mg; or by infusion at a rate of 0.5 to 2mg/min. Labetalol can cause severe postural hypotension.  
  - **Hydralazine,** an arteriolar dilator, is used particularly in hypertensive emergencies in pregnancy but labetalol is preferable. A bolus dose of 5mg 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 200-300micrograms/min; this usually requires a maintenance dose of 50-150micrograms/min.  
  - **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 2-5mg 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), nephropathy (malignant nephrosclerosis) with or without encephalopathy and microangiopathic haemolytic anaemia. 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. atenolol 50mg) 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 or bleep 6045).
All patients arriving at the 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. Management depends on whether the patient has ST-segment Elevation Myocardial Infarction (STEMI) or Non ST-segment Elevation Acute Coronary Syndromes (NSTE-ACS).

**INITIAL DIAGNOSTIC MEASURES FOR ALL PATIENTS.** A cardiac monitor should be attached to detect cardiac arrhythmias. By brief history, examination and 12-lead ECG, establish whether the patient is suffering from STEMI, NSTE-ACS or neither.

- **The ECG changes diagnostic of STEMI are:**
  - ST elevation of ≥ 0.2mm in leads V1-V3 or ≥ 0.1mm in other leads.
  - Left bundle branch block that is new or presumably new, in the context of a convincing history.

- **The ECG changes diagnostic of NSTE-ACS are:**
  - Symmetrical deep T wave inversion ≥ 2 mm.
  - Deep T wave inversion V1-V4/LAD syndrome
  - Persistent ST depression ≥ 1 mm
  - Transient ST elevation
  - Deep T wave inversion V1-V4/LAD syndrome
  - Persistent ST depression ≥ 1 mm

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

If STEMI is suspected but not definite, discuss urgently with A&E senior or on-call Cardiology registrar (blp 6002), phone the Coronary Care Unit (x3168/3166) or the Cardiac Catheter Lab on ext.1370/1703/3274.

**MANAGEMENT OF STEMI**

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 300mg to be chewed. This should be followed by aspirin 75mg daily. If the patient is allergic to aspirin seek advice.

**Ticagrelor** On arrival in the Cardiac Catheter Laboratory or Coronary Care Unit the patient will be given Ticagrelor 180 mg as a loading dose (contraindicated in patients with active bleeding or a history of intracranial haemorrhage). This should be followed by Ticagrelor 90 mg twice daily.

**Heparin** As soon as possible give unfractionated heparin 5000 IU by slow IV injection.

**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 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** Give Fondaparinux 2.5 mg SC od (Arixtra) for 48-72 hours or until discharge (maximum 8 days). If creatinine clearance < 20 ml/min: prescribe IV unfractionated heparin for 24-48 hours then DVT prophylactic dose SC unfractionated heparin.
**Blood glucose management** Manage hyperglycaemia by keeping blood glucose levels below 11.0 mmol/litre while avoiding hypoglycaemia. In the first instance, consider a dose-adjusted insulin infusion with regular blood glucose monitoring. Stop all existing oral hypoglycaemic therapy before, and for 48 hrs after, coronary intervention. 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** All patients with STEMI should be given an ACE inhibitor except those with renal failure or a systolic blood pressure (BP) <90mmHg. A reasonable choice is ramipril started at a dose of 1.25mg bd. Dosage should be slowly titrated upwards to the maintenance dose of 5.0mg bd, 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
- second or third degree heart block
- cardiogenic shock
- heart failure requiring therapy
- a history of bronchospasm
- allergy/hypersensitivity to β-blockers

A reasonable choice is metoprolol which should be given as an initial oral dose of 12.5mg tds. If there is persistent tachycardia or hypertension, metoprolol can be given IV at a dose of 5mg. A reasonable oral maintenance dose of metoprolol is 25mg tds.

**Statins and lipid-lowering agents** All patients should have a lipid profile on admission, then started on Atorvastatin 40mg od titrated to 80mg before discharge. Reduce dose or use Pravastatin 40mg od 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 <40%, consider an aldosterone antagonist such as Eplerenone 25mg od (contraindicated if the creatinine clearance is <50mls/min or potassium >5.0mmol/L).

**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 >90mmHg and the patient hasn’t received a phosphodiesterase inhibitor (eg. sildenafil) within 24 hours.

**Gastroprotection** All patients requiring GI protection should be prescribed ranitidine 300mg bd for gastro-protection, unless they have an active or recently healed peptic ulcer (< 6 months) in which case use lansoprazole 30 mg od.
Non ST-segment elevation acute coronary syndromes (NSTE-ACS) include unstable angina (UA) and non ST-segment elevation myocardial infarction (NSTEMI). Patients with NSTE-ACS may complain of rapidly worsening, prolonged and increasingly frequent episodes of cardiac chest pain, of cardiac pain occurring at rest, or of pain of recent onset occurring with trivial provocation.

**DIAGNOSIS**

Patients presenting with ischaemic chest pain and diagnostic ECG (persistent ST depression ≥1mm, symmetrical deep T wave inversion ≥2mm, transient ST elevation, deep T wave inversion V1-V4/LAD syndrome) should be admitted and treated for NSTE-ACS.

*Contact cardiology registrar (blp 6002); ACS practitioner (blp 7138) or phone Coronary Care Unit (x3168/3166).*

- Give aspirin 300mg on admission (unless previously taking aspirin, or aspirin contraindicated), and 75mg daily thereafter. If the patient is intolerant of aspirin, seek advice.

- **HIGH RISK NSTE-ACS PATIENTS** (on-going pain, ST depression dynamic or >2mm, early troponin elevation > 500 ng/L, haemodynamic instability or ventricular arrhythmia).
  - Give Ticagrelor 180 mg as a loading dose (contraindicated in patients with active bleeding or a history of intracranial haemorrhage or on warfarin or NOAC).
  - This should be followed by Ticagrelor 90 mg twice daily.

- **NOT HIGH RISK NSTE-ACS PATIENTS** - Give clopidogrel 600mg followed by clopidogrel 75mg od.

- Give morphine 2.5-5.0mg by slow IV injection and repeat if pain persists. To reduce the likelihood of vomiting, give either metoclopramide (10mg IV over 2 mins) or cyclizine (50mg over 3 mins).

- Give controlled oxygen therapy if appropriate (*see STEMI guideline*).

- Give Fondaparinux 2.5 mg SC od (Arixtra) for 48-72 hrs or until discharge (max. 8 days). If creatinine clearance <20 ml/min: prescribe IV unfractionated heparin for 24-48 hrs then DVT prophylactic dose SC unfractionated heparin.

- Give Atorvastatin 40mg od (titrated up to 80mg) in patients with confirmed NSTE-ACS.

Reduce dose or use Pravastatin 40mg od in patients receiving interacting drugs (e.g. clarithromycin, cyclosporin, protease inhibitors, diltiazem, amiodarone, verapamil). Prescribe simvastatin 40mg od (reduce doses similarly with concomitant interacting drugs) where NSTE-ACS is not confirmed and primary prevention is required.

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 contra-indications and suggestions for choice and dose)
- **Patients with diabetes mellitus or a blood sugar of >11** 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 contra-indications).
Further Risk Assessment
- Cardiac biomarkers (Creatine Kinase and Troponin I – 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)
  Patients at intermediate or high risk on the GRACE score, or patients with unstable symptoms, should have cardiac catheterisation performed within 24 hrs

- 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).

Normal or equivocal ECG
For patients presenting with ischaemic pain and a normal or equivocal ECG, should have cardiac biomarkers performed on admission and at 3 and 6 hours.
- If the initial troponin I concentration is >50 ng/L and the 3-hour troponin >increases by over 30%, the patient should be treated for NSTE-ACS.
- If the initial troponin I concentration is ≤50ng/L and the 3-hour troponin > increases by over 30% and is >50 ng/L, the patient should be treated for NSTE-ACS.
- If the initial troponin I is >50 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 ≤50 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 or ETT). Patients with a low-intermediate GRACE Score and stable symptoms should be suitable for out-patient assessment (open access ETT, RACPC, or Cardiology OPD).
Acute decompensated heart failure is a life-threatening condition with 30-day mortality of 15% in those with NTproBNP>5000ng/L and 5% in those with NTproBNP<5000ng/L. Patients with heart failure should generally be discharged from hospital only when their clinical condition is stable and the management plan is optimised.

Community heart failure nurse follow-up reduces the 3-month risk of re-admission by 35%. Please contact heart failure nurse specialists (blp7376/x.4404) as soon as patients are admitted for specialist review and for long-term management plan.

**DIAGNOSIS**

Acute heart failure is the leading cause of hospital admission in people 65 yrs 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 NTproBNP 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>NT-proBNP likely if</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 echocardiograph if not already documented. All patients admitted with a new diagnosis of heart failure (with raised NT-proBNP) should have an inpatient echocardiogram prior to discharge (ideally within 48 hours of admission). If the NT-proBNP concentration is intermediate (above 300ng/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**

**Acute pulmonary oedema:**
- Call the cardiology SpR (bleep 6002) to arrange admission to CCU
- O₂ to maintain SaO₂ (95-98%)
- IV furosemide 40-100mg bolus followed by an infusion at 5-20mg/h if required
- Consider IV GTN infusion (10-200micrograms/min) for patients with concomitant myocardial ischaemia, severe hypertension or regurgitant aortic/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)

**General measures**
- Monitor pulse, check oximetry and BP 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 TTFs, LFTs, troponin, glucose and lipids; arterial blood gases gases if oxygen saturation is low or oxygen is required to maintain saturation.
- Review medication: stop Ca²⁺ 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 uptitrated.

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 (eg. rampiril) and titrate upwards. If not tolerated (eg due to persistent cough) try an angiotensin II receptor antagonist (eg. candesartan).
2. Start a beta-blocker, unless contra-indicated, (eg. bisprolol) and titrate upwards
3. Add a mineralocorticoid receptor antagonist (spironolactone or eplerenone 12.5—25mg od).

For those with isolated right ventricular failure, fluid balance and diuretic therapy is all that is required.

DISCHARGE AND FOLLOW-UP
• All acute heart failure admissions need community heart failure nurse follow-up after discharge to reduce risk of re-admission. This can be arranged via the inpatient heart failure nurses (extension 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 (eg 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: Management of chronic heart failure in adults in primary and secondary care
NICE Clinical Guideline 108, August 2010
\(^2\) 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.

<table>
<thead>
<tr>
<th>Indications for Temporary Pacing – Emergency/Acute</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute MI with:</strong></td>
</tr>
<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>
<tr>
<td><strong>Bradydcardia not associated with acute MI:</strong></td>
</tr>
<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 bradycardia (&lt;30bpm) +/- QRS &gt;120ms +/- QTc prolongation &gt;500ms</td>
</tr>
<tr>
<td><strong>VT secondary to bradycardia eg Torsades de Pointes</strong></td>
</tr>
<tr>
<td><strong>Suppression of drug-resistant VT or SVT</strong></td>
</tr>
<tr>
<td><strong>Drug overdose, eg. digoxin, beta blockers, verapamil</strong></td>
</tr>
</tbody>
</table>

<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. Temporary pacing can be achieved rapidly by a balloon flotation wire but is rarely needed; contact the cardiology registrar for advice and XRay guided temporary pacemaker.

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 digoxin or other AV nodal blocking drugs (diltiazem, beta-blockers). AF of recent onset (<24 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 cardiac 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. 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. If the patient is hypotensive, in cardiac failure or has ischaemia, cardioversion should be undertaken. If stable then initially 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 procainamide (20mg/min at a dose of 10-15mg/kg up to a total of 1g, stopping infusion if arrhythmia resolves, hypotension develops or QRS complex widens significantly); or 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 helped 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. Follow Cardiac Arrest advice or as described above, but attempt to defibrillate away from the device itself (usually left infraclavicular site). Haemodynamically stable VT or successfully treated patients can 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.

**POLYMORPHIC VT**

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

**VENTRICULAR FIBRILLATION** *(see Cardiac Arrest)*.

**VENTRICULAR ECTOPIC BEATS.**

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

**ASYSTOLE** *(see Cardiac Arrest)*

Patients with acute MI who develop CARDIAC FAILURE or CARDIOGENIC SHOCK, should be referred to the on-call cardiology registrar as soon as possible.
ACUTE DEEP VEIN THROMBOSIS (DVT)
Link consultant: Dr James Uprichard

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. 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.
- Out-patients suspected of DVT are assessed by A&E or Urgent Care Centre according to the Outpatient DVT Investigation 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. Details of the scoring system are available on the Trust Intranet in A&E and on the Anticoagulant web page, http://stginet/Units%20and%20Departments/Haematology/ANTICOAGULATION/ANTICOAGULATION.aspx When DVT is confirmed by US Doppler, the DVT Nurse should be contacted on bleep 7380 for further plan of care.

Treatment
1. For positive compression ultrasound
If a compression ultrasound done within working hours confirms a DVT, and provided anticoagulants are not contraindicated (due to an enhanced risk of bleeding or suspected active bleeding), immediately start rivaroxaban (provided that creatinine clearance is ≥ 15 ml/min). The dose is 15mg twice daily for three weeks and then 20mg once daily for maintenance (if creatinine clearance ≤ 50 ml/min then use 15 mg once daily for maintenance).
Details on the use of rivaroxaban for VTE treatment are available on the Trust Intranet: file:///stg1nas01/formulary/DVT%20and%20PE%20Treatment%20Pathway%20%20updated%202014.pdf

For cancer-associated thrombosis or pregnancy-related thrombosis, therapeutic dose dalteparin should be given (rather than rivaroxaban).
If there is a contraindication to rivaroxaban therapy then once daily dalteparin (or unfractionated heparin) should be used, followed by warfarin, using the Warfarin Dosing Chart.

Those presenting to A&E will be re-assessed by the Thrombosis clinical nurse specialist (blp 7380; x2826) with the results of the ultrasound; ambulatory management will be commenced if appropriate. Patients at enhanced risk of bleeding (those with liver disease, peptic ulcer, alcohol abuse, 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.
For patients initiated on rivaroxaban

For patients initiated on rivaroxaban, a ‘Screening and notification of initiation of treatment’ form needs to be completed and sent to the patient’s GP; available via:

file:///\stg1nas01\formulary\Rivaroxaban%20for%20VTE%20-%20Notification%20of%20Initiation%20of%20Treatment.pdf

The patient 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’ (or ‘Rivaroxaban: A patient’s guide to pulmonary embolism treatment’).

These are available from pharmacy or the anticoagulation clinic, and are also kept as stock on ED and 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. The dose of dalteparin, which should be given subcutaneously daily, 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 has been >2.0 on two consecutive days, 24 hours apart. If dalteparin is given for more than five days, assess renal function and if creatinine clearance is ≤ 30 ml/min use unfractionated heparin. For patients needing ambulatory management of DVT, dalteparin injection can be self-administered. However, patients should be referred to the district nurse team if they are unable to self-administer the injections.

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. Details of discharge procedure are available on the anticoagulant web page (see above). Draw the patient’s attention to the common interactions with warfarin outlined in the booklet.

For most indications an INR of 2-3 is sufficient, but this may need to be tailored to the individual patient circumstances.

2. For negative compression ultrasound

If the initial compression ultrasound is negative, refer to the out-patients 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. Out-of-hours advice can be given by the on-call haematology registrar via switchboard.
ACUTE PULMONARY EMBOLISM
Link consultant: Dr Adrian Draper

Pulmonary embolism (PE) should be considered in anyone presenting with:
- breathlessness
- chest pain
- cough/haemoptysis
- hypotension (this occurs if embolism sufficient to compromise cardiac output, in this instance assume a massive pulmonary embolism (see last paragraph).

Various risk factors increase the likelihood of the patient having a PE:

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contraceptive pill</td>
<td></td>
</tr>
<tr>
<td>Malignancy/Cancer</td>
<td></td>
</tr>
<tr>
<td>Pregnancy or &lt;6/52 post partum</td>
<td></td>
</tr>
<tr>
<td>Air travel &gt;4hrs in previous 4/52</td>
<td></td>
</tr>
<tr>
<td>Surgery in last 4/52</td>
<td></td>
</tr>
<tr>
<td>Bedbound</td>
<td></td>
</tr>
<tr>
<td>Previous VTE</td>
<td></td>
</tr>
<tr>
<td>Family Hx of VTE</td>
<td></td>
</tr>
<tr>
<td>Known thrombophilia</td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td></td>
</tr>
<tr>
<td>Smoker</td>
<td></td>
</tr>
<tr>
<td>IVDU</td>
<td></td>
</tr>
</tbody>
</table>

The following clinical signs are associated with PE:
- tachycardia
- tachypnoea (PE is most unlikely if the respiratory rate is less than 20/min)
- pleural rub
- right ventricular heave or accentuated pulmonary component to second heart sound
- hypoxia (PE is most unlikely if the PaO\textsubscript{2} is 10.7 kPa or more)

Use the two level PE Wells Score table to estimate the clinical probability of PE:

<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>PE as likely or more likely than an alternative diagnosis (based on clinical information, chest X-ray, ECG and any blood tests required to diagnose PE)</td>
<td>3.0</td>
</tr>
</tbody>
</table>

The primary purpose of the CXR and ECG is to exclude other diagnoses. If the plasma D-dimer level (test) is below 300 ng/ml, it is most unlikely that the patient has had a PE.
A raised D-dimer level is not diagnostic in itself as it also occurs in patients after recent surgery, in the presence of malignant disease or infection, and in patients with a total bilirubin above 34 micromol/L.

When the Wells score is 4 points or less and D-Dimer is negative the patient is unlikely to have a PE and further imaging is not indicated. Any patient with a risk factor for pulmonary thromboembolic disease and unexplained tachypnoea or dyspnoea should be investigated for the possibility of pulmonary thromboembolic disease, especially when clinical signs in keeping with this diagnosis are present. Once clinical risk of PE has been assessed, investigations should follow the algorithm below.

Investigatory and diagnostic algorithm
Assess clinical probability of PE

Clinical risk of PE
(4 points or less)

D-dimer

Negative: no treatment

Positive

V/Q scan if CXR normal or CTPA

No PE: no treatment

PE: treatment

Clinical risk of PE
(more than 4 points)

V/Q scan if CXR normal or CTPA

No PE: further investigation

PE: treatment

Management
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 active cancer or is pregnant (use LMW heparin). While awaiting confirmation of PE, the patient should receive their first ‘loading dose’ of rivaroxaban at 15mg twice daily.

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 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), as patient may be considered for ambulatory therapy. 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 15-50ml/min). Unfractionated heparin should be used in renal failure CrCl< 14ml/min. Warfarin can be used if Rivaroxaban is contraindicated – see PE guidance on intranet. This should then be continued for at least six months. Advice on the duration of anticoagulant therapy can be obtained from the haematology department or respiratory physicians.

Massive pulmonary embolism
Patients with suspected massive PE will require immediate additional investigation, and should usually be managed in an ITU or HDU setting. An echocardiogram should be
performed. If this is not diagnostic then CT pulmonary angiography should be performed. A high venous filling pressure is required to maintain cardiac output; insert a central line (internal jugular approach) and maintain the CVP at 15-20 mmHg. When the diagnosis is confirmed the management options are: offer unfractionated heparin and consider thrombolytic therapy with either tissue plasminogen activator (rt-PA; alteplase) or, if this is not available, streptokinase, or surgical pulmonary embolectomy. The decision should be taken in conjunction with the on-call respiratory consultant or cardiologist, and, if appropriate, the cardiothoracic surgeon. Unfractionated IV heparin should be administered and a thrombolytic should only be given if patient is haemodynamically unstable, i.e they are hypotensive with a systolic BP <90mmHg (in a previously normotensive individual) or a drop in systolic BP of 40mmHg for 15 minutes.

Provided there are no contraindications, give alteplase as a 10mg IV injection over 1-2 minutes, followed by infusion of 90mg over 2 hours; maximum dose 1.5mg/kg in patients weighing less than 65kg. 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 up to 72 hours. If a cardiac arrest seems imminent, give a 50mg bolus dose of alteplase.

Thrombolysis should only be used in patients who are haemodynamically unstable at presentation or whom subsequently deteriorate. Stable patients should not be thrombolysed; the risk of bleeding complications do not justify this intervention.

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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 dyspnoea may be absent until the failure is well advanced.
- Secondary to cardiac arrest.
- Plugging of a tracheostomy.

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

Treatment of the cause
The underlying cause of the arrest should be treated as appropriate. Non-specific respiratory stimulants are of little value. However, when the arrest has been caused by an opiate, naloxone should be given. The initial dose is 0.4mg IV and if the patient fails to respond, the dose should be repeated every 2-3 mins until depression is reversed. If IV access is not available, naloxone can be given IM or subcutaneously. The drug is not effective in buprenorphine overdose but will occasionally work in patients with alcohol overdose. If arrest is secondary to benzodiazepine overdose try flumazenil IV (200 micrograms over 15 sec followed by 100 micrograms 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
  - $\text{pCO}_2 > 7$
  - $\text{pH} < 7.35$
  - an increased respiratory rate despite optimisation with oxygen therapy
- acute hypercapnic respiratory failure with chest wall deformity, neuromuscular disorder or decompensated obstructive sleep apnoea
- cardiogenic pulmonary failure refractory to CPAP
- patients who might otherwise receive tracheal intubation, but in whom this is better avoided or not appropriate
- patients being weaned from mechanical ventilation

Patients requiring NIV should be discussed with the respiratory registrar or, if out-of-hours, with the respiratory consultant on call. Full NIV guidelines are on the Intranet [http://stginet/Policies/Clin_5-PatientMment/Clin_5_25.pdf](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 Emergency Oxygen Guidelines (2008) [www.brit-thoracic.org.uk](http://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. <strong>Assess for signs of hypercapnia and</strong> 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)  
  - 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 prongs. Consider NIV or invasive ventilation if PaCO₂ >6 kPa and pH <7.35; discuss with respiratory/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 | | |

*Other risk factors for hypercapnic (type 2) respiratory failure include severe chest wall or spinal disease, neuromuscular disease, severe obesity, cystic fibrosis and bronchiectasis.
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 achieved by patient
- cyanosis/hypoxia
- silent chest on auscultation
- bradycardia/hypotension

**MANAGEMENT – Monitoring**

Measure arterial blood gases on admission and repeat as necessary to assess progress. A 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 a high concentration of oxygen *adjusted to keep SaO₂ 94-98%.*

*Bronchodilators.* A bronchodilator, such as salbutamol (2.5-5mg) should be started as soon as possible via an oxygen-driven nebuliser (drive at a flow rate of at least 6L/min). This dose can be repeated at 15-30 min intervals if no improvement is seen. Nebulised ipratropium bromide (500micrograms) helps in about 30% of patients with acute asthma and may be given every 6 hours. Parenteral 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 40-50mg 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.* Patients tend to become dehydrated because of decreased fluid intake and extra loss through hyperventilation. This may increase the tenaciously of the bronchial secretions. Give IV fluids in amounts to maintain 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 2g (8mmol) in 250mL of NaCl 0.9% over 20 minutes.

Aminophylline. This 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. Therapeutic monitoring is essential.

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 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.
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 cardiopulmonary 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 – a visible rim of >2 cm between the lung margin and the chest wall (at the level of the hilum) is large (small = a rim of air <2 cm around the lung and is easily measured with the PACS system), and to check for fluid levels. Treatment varies according to the symptoms, the degree of the collapse, and whether there is underlying lung disease or bleeding.

**Tension pneumothorax.** Patients with a tension pneumothorax will require immediate aspiration of the entrapped air followed by intercostal tube drainage. 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.

**History of trauma.** Admit any patient in whom the pneumothorax might be the result of trauma (eg road traffic accident, assault). Check for bleeding (see below).

**Healthy young adults.** Admit the patient to hospital if there is shortness of breath on slow walking, or if a fluid level is found. In those with shortness of breath or large (>2 cm) pneumothorax, aspirate the air through a wide bore needle introduced under local anaesthesia. If aspiration with a needle fails an intercostal drain may have to be introduced (seek advice). In those with a suspected bleed, monitor the heart rate and blood pressure and repeat the X-ray to check whether bleeding has stopped. If it hasn’t, seek advice. There is no need to admit an otherwise healthy young adult if:

- there is no shortness of breath at rest or when walking slowly,
- pain is mild or diminishing,
- collapse is small or moderate (less than 50%),
- fluid on the chest X-ray is only sufficient to blunt the costophrenic angle.

Before a patient leaves A&E explain the cause of the symptoms, arrange for outpatient review in 7-10 days and advise the patient to return promptly to hospital if symptoms worsen.

**Patients with underlying lung disease.** All patients with underlying lung disease should be admitted to hospital with a view to aspiration or drainage, depending on their age, the level of their dyspnoea and the results of their chest X-ray. **Management should follow the scheme in the flow diagram below.**

For greatest safety the chest drain should be inserted in the 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. Use a Seldinger 12 French Portex drain when possible. Seek advice from a respiratory specialist registrar or consultant if:

- the lung fails to expand
- the patient develops surgical emphysema
- pleurodesis is being considered
On discharge give the patient an appointment for the chest clinic in 7-10 days. The patient should be told to report back to hospital immediately if symptoms deteriorate, and advised not to travel by air for 6 weeks.
ACUTE UPPER GASTROINTESTINAL BLEEDING
Link consultant: Dr Chris Groves

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

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

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

Initial Management
- Patients at ‘low risk’ include those with no sign of haemodynamic compromise; Hb > 10g/dl; aged < 60 years, and previously fit. In low-risk patients allow oral fluids, observe for signs of continued or re-bleeding and arrange an OGD for the next routine list. Referral for endoscopy should be made on an endoscopy request form. It is important to complete all sections of the form to allow appropriate prioritisation of the patient. Inform the Endoscopy Unit of the need for endoscopy by 9am. Start patient on oral Omeprazole 40mg BD.
- Patients at ‘high risk’ include those with haematemesis or fresh melaena; systolic hypotension (<100mmHg); tachycardia (pulse >100 beats per min); postural drop in diastolic BP; Hb<10g/dL; severe concomitant disease (liver/cardiovascular/respiratory); age >60 years. In high-risk patients restore blood volume with blood/blood substitutes, admit to high dependency ward, monitor closely (pulse rate, blood pressure, CVP), inform GI bleed registrar and discuss/arrange emergency endoscopy. High risk patients or those with haematemesis who are vomiting, where endoscopy is planned but not imminent, can be given IV Omeprazole 40mg BD until ready for an OGD. The endoscopist should enter the OGD findings in the Endoscopy Unit computer. If the endoscopist sees a bleeding ulcer, the patient should be given omeprazole (80mg) as a stat injection IV, followed by an infusion at 8mg/h for 72 hours.

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

A patient with a gastric or duodenal ulcer who has had endoscopic treatment of a visible vessel should have high dose PPI. Omeprazole 8mg/ hr via a syringe driver for 72 hrs is recommended. Eradication therapy for H.Pylori should be given either now or at discharge.
A patient with a visible vessel or endoscopic evidence of recent or active bleeding is at high risk of rebleeding. Observe for continued bleeding or rebleeding as indicated by a fall in systolic BP, rise in pulse rate, fall in CVP or overt evidence of bleeding.
Surgery
Surgery should be considered if bleeding continues or recurs after hospital admission despite endoscopic therapy, since this is associated with a tenfold increase in mortality. A high transfusion requirement (>4 units if patient older than 60 years; >8 units if younger) should also alert the team to the possible need for surgery. A consultant surgeon should be involved in the decision on whether to operate.
**General Measures**
The patient may be allowed to drink water and start a light diet as soon as the initial endoscopy has been performed and surgery is not contemplated. Gastric ulcers require endoscopic follow up at 8 weeks to ensure healing. There is no need to rescope duodenal ulcers unless symptoms recur in which case an H.Pylori breath test is indicated.
BLEEDING OESOPHAGEAL VARICES
Link consultant: Dr Daniel Forton

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 auscultate 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.
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>Causes</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>&gt;33mmol/L</td>
<td>Negative</td>
<td>++++</td>
<td>≥7.35</td>
<td>&gt;320</td>
</tr>
<tr>
<td>HHS/DKA mixed</td>
<td>&gt;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 6units/hr via infusion pump
- If BG at presentation is <14mmol/litre then infuse 10% glucose at 100ml/hr in addition to 0.9% sodium chloride, and reduce IV insulin rate to 3 units/hr
- (Only give a stat dose of Actrapid (SC) insulin if a delay of ≥ 1 hour is anticipated in setting up an insulin infusion)

Continue long-acting subcutaneous (SC) insulin – glargine (Lantus®) or detemir (Levemir®) – if the patient was taking this before admission; withhold short and intermediate-acting insulins
### OTHER ASPECTS OF MANAGEMENT

- 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).

<table>
<thead>
<tr>
<th>Discuss with HDU/ ICU if:</th>
<th>Essential monitoring</th>
</tr>
</thead>
<tbody>
<tr>
<td>• BG &gt; 33 mmol/L</td>
<td>• Hourly EWS &amp; BG</td>
</tr>
<tr>
<td>• Hypokalaemia (K⁺ &lt;3.5mmol/l)</td>
<td>• Hourly fluid balance</td>
</tr>
<tr>
<td>• GCS &lt; 15</td>
<td>• Electrolytes (esp K⁺) every 2 to 4 hours (venous blood gas analysis)</td>
</tr>
<tr>
<td>• Pulse &gt; 100bpm or &lt; 60bpm</td>
<td>• Evidence of sepsis</td>
</tr>
<tr>
<td>• SBP &lt; 90 mmHg</td>
<td>• pH &lt;7.1</td>
</tr>
<tr>
<td></td>
<td>• HCO3 &lt;5.0</td>
</tr>
<tr>
<td></td>
<td>• Lactate&gt;2.0</td>
</tr>
</tbody>
</table>

#### **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

---

37
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 euvoalaemia 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
Link consultant: Dr Natasha Patel

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

TREATMENT OF HYPOGLYCAEMIA – INPATIENT CARE
Hypoglycaemia is a blood glucose of 4mmol/L or less. If patient is asymptomatic, repeat test. Ideally confirm with lab sample; do NOT wait for result – treat at once.

<table>
<thead>
<tr>
<th>4mmol/L</th>
<th>3mmol/L</th>
<th>2mmol/L</th>
<th>1mmol/L</th>
</tr>
</thead>
<tbody>
<tr>
<td>MILD: Patient conscious and able to swallow. Trembling, sweating, hungry, tingling, headache, anxiety, palpitations, nausea, forgetfulness</td>
<td>MODERATE: Patient conscious and able to swallow, but in need of assistance. Difficulty concentrating, speaking. Confusion, weakness, giddiness, drowsiness, unsteady, headache</td>
<td>SEVERE: Patient unconscious and unable to swallow. Unconscious, fitting</td>
<td></td>
</tr>
</tbody>
</table>

**STEP 1**

Cooperative: Give 15-20g fast-acting glucose: 4 x Gluco Tabs (4g glucose per tab) or 1 x 59ml bottle GlucoJuice or 100mls Lucozade or 200mls fruit juice.

Uncooperative: Give 2 x tubes of GlucoGel - ensure gag reflex is present.

If NBM on insulin, adjust as per regime. Not on insulin infusion, 100mls of 10% Glucose iv/1mg Glucagon im.

Wait 15mins, check glucose levels and record. If reading is still below 4mmol/L, or if no physical improvement, repeat STEP 1. If reading is below 3mmol/L CALL DOCTOR.

**ALWAYS FOLLOW UP WITH A SLOWLY DIGESTED/STARCHY CARBOHYDRATE.** Check glucose level until 4mmol/L or over; once recovered patient should eat minimum 15g slowly digested/starchy carbohydrate, eg. 1 slice/sandwich of low GI bread (ideally multigrain/granary); 2 digestive biscuits, glass of milk or normal meal if due. Check glucose after 15mins. Identify cause of hypoglycaemia. NB NEVER OMIT INSULIN FOLLOWING AN EPISODE.

Check airways (ABC) and place in recovery position. NO oral fluids – if patient on insulin infusion STOP and FAST BLEEP DOCTOR. If iv access give 100mls of 10% Glucose or 1mg Glucagon im. If no improvement repeat 100mls 10% glucose iv.

If patient remains unconscious:
- glucose 10% at 100mls/hr
- assess for other causes of unconsciousness; consider transfer to ITU
- if patient was on sulphonylurea consider octreotide 50micrograms 12-hourly s.c

Once patient is conscious give sips of GlucoJuice or Lucozade. Check glucose level every 15mins to ensure increase to at least 4mmol/L.
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.

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, TIA’s may be admitted depending on the clinical view at the time. If the patient is an inpatient, we can often arrange the tests before they go home.

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.

**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). Inpatients should be referred immediately for stroke assessment. If the patient cannot be admitted directly to the HASU, care should be started in a general ward but every effort made to transfer the patient to the HASU as soon as possible. If the HASU is full, a patient (usually the one who has been there the longest) will be moved to make way for the new admission. The ‘moved’ patient will either be transferred to a stroke unit bed, or if unavailable, a general medical bed. If the transfer occurs out of hours, the stroke team will hand over to the receiving team on the next working day with clear details of diagnosis, secondary prevention and ongoing management plan, and a discharge letter.

**History & Examination**

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

**Investigations**

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

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

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

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

**Acute Medical Management - 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 (blp 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.

**Other acute treatment**

**Antiplatelet therapy** should be given to all patients with ischaemic stroke, and in whom imaging has excluded a haemorrhage. An initial Aspirin dose of 300 mg (given orally or rectally) is followed by a daily dose in the range 75-300 mg. However, Clopidogrel 75 mg daily is now started after review by the stroke team after loading with 300mg. Aspirin is stopped at this stage. Full heparinisation should be reserved for patients with cerebral venous thrombosis, or where the risk of a cardioembolic source is high. In patients with atrial fibrillation or other cardioembolic source, anticoagulation should be delayed for two weeks if the stroke is large. If the stroke is small it can be started sooner. If in doubt, seek advice from the stroke SpR.

Patients already on antihypertensive medication should continue their usual treatment unless their blood pressure (BP) is low. **Acutely elevated BP is common following 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).
Much of the mortality and morbidity following stroke is from secondary complications. To minimise these:

**Fill out 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. 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.

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

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.

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

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

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 antiplatelet 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 x4591)
STATUS EPILEPTICUS (Convulsive SE)
Link consultant: Dr Hannah Cock

Status epilepticus (SE) is defined as continuous seizure activity which has failed to self-terminate leading to a risk of neurological damage. The risks are highest with generalised tonic/clonic (convulsive) seizures. Convulsive SE may present as either a run of discrete 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 have longer seizures with self termination (this information may be obtained 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, but not to over-treat patients in whom seizures have terminated but are slow to recover.

GENERAL MANAGEMENT
1. Protect the patient from damage during the seizures - make the environment safe by using padded bed rails. Do not restrain the patient. Once the flurry of seizures has ceased, place the patient in a semi-prone position with the head down to prevent aspiration and to help maintain the airway. The patient should be kept in this position until full consciousness is restored. Note the time.
2. Initially concentrate on respiratory support. During an inter-ictal period insert an airway and then administer oxygen. Do not attempt to insert anything in the patient’s mouth during a seizure, even if the tongue is injured.
3. Set up an IV line as soon as possible to gain access to the circulation.
4. If there is any suggestion of alcohol abuse or impaired nutrition, give thiamine as high potency intravenous Pabrinex BEFORE GLUCOSE.
5. 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, p.29).
6. Draw venous blood for full blood count, clotting, glucose, urea, sodium, potassium, calcium, liver function and anticonvulsant drug levels (irrespective of known history at this stage). Save a sample of blood and urine for toxicology.
7. Measure body temperature, take an ECG, monitor respiration and BP.
8. Gain information – is there evidence of previous epilepsy, any anticonvulsant drugs, diary or wallet card or bracelet.

DRUGS
1. The drug of first choice is lorazepam given as an IV bolus injected at 2mg/min, ideally in a dose of 4mg for adults or 0.1mg per kg for children. See flow chart for alternatives if Lorazepam is unavailable or no iv access. Also check if benzodiazepines have been given prior to hospital (community/paramedics) to avoid overdose.
2. If seizures persist or recur, repeat lorazepam at 5-10 minutes. Benzodiazepines (including pre-hospital doses) should not be used more than twice in any 24 hour period.
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’). If fits persist or recur despite two doses within 24hrs, move to stage 3 and contact neurologist on call.
3. If fits persist after further 5 mins, call the anaesthetist and neurology SpR on-call. Immediately start an IV infusion of phenytoin a total dose of 20mg/kg given at a rate of 50mg per minute, with cardiac monitoring. If phenytoin is contra-indicated because of sinus bradycardia, a heart block or porphyria, or patient is already taking phenytoin, phenobarbitone may be given by IV infusion (dissolved 1:10 in water for injection, max
rate 100mg/min) in boluses of 200mg every 5 mins, to a maximum total dose of 10-15mg/kg, taking care to monitor respiratory function.

4. If, despite intravenous lorazepam & phenobarbitone or phenytoin, seizures persist or recur over 30-60 minutes, the patient should be transferred immediately to an ITU.

5. If status persists or recurs after 30-60 minutes the patient will need to be sedated and ventilated. This will require the active involvement of an anaesthetist, and infusions of midazolam, propofol or thiopentone. **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. EEG monitoring is necessary for refractory status. The anaesthetic will need to be continued for 12-24 hrs after the last clinical or electrographic seizure, and the dose then tapered.

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**Treatment of Status Epilepticus**

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

<table>
<thead>
<tr>
<th>Partial status</th>
<th>Generalised status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Call neurology SpR on-call for advice on appropriate drug management</td>
<td>7-10 mins</td>
</tr>
<tr>
<td></td>
<td>Lorazepam 4mg IV* (Paed 0.1mg/kg)</td>
</tr>
<tr>
<td></td>
<td>5mins</td>
</tr>
<tr>
<td></td>
<td>Lorazepam 4mg IV* (Paed 0.1mg/kg)</td>
</tr>
<tr>
<td></td>
<td>5mins</td>
</tr>
<tr>
<td></td>
<td>Phenytoin 18-20mg/kg at rate of 50mg per minute. Cardiac monitor OR</td>
</tr>
<tr>
<td></td>
<td>Phenobarbitone in boluses of 200mg (injected over 2mins) every 5mins to a max. total dose of 10-15mg/kg, if known heart block or already on phenytoin</td>
</tr>
<tr>
<td></td>
<td>ITU GENERAL ANAESTHESIA</td>
</tr>
<tr>
<td></td>
<td>seizures persisting 30-60mins after onset</td>
</tr>
</tbody>
</table>

* If Lorazepam is unavailable, give 10mg Diazepam iv (paed 300-400 micrograms/kg-max.10 mg) or **10mg Buccal Midazolam (paed 300 micrograms/kg)** – whichever is quickest/easiest for individual patient. If no iv access, give Buccal Midazolam.
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 anterolateral 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 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 Jeremy Cashman

*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 or very severe and treated accordingly. In general it is more realistic to *strive for comfort* rather than complete abolition of pain.

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**THE “ANALGESIC LADDER”**

**Mild pain**
- paracetamol or an NSAID

**Mild-to-moderate**
- combination analgesic + an NSAID

**Moderate**
- oral opioid or combination analgesic + an NSAID

**Moderate-to-severe**
- oral opioid + paracetamol + an NSAID

**Severe**
- parenteral opioid (IM, SC or IV) + paracetamol + an NSAID

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**TREATMENT DETAILS**

**Simple Analgesic**
- Paracetamol: 1g PO/NG/PR 4-6 hourly (maximum 4g/day).
  - *Note: For adult patients <50kg especially those who are malnourished, we advise dosing at 15mg/kg PO/NG/IV 4-6 hourly.*

**Non-Steroidal Anti Inflammatory Drugs (NSAIDs)**
- Ibuprofen: 200-400mg PO 4-6 hourly (maximum 2.4g /day).
- Naproxen: 250-500mg PO 6-8 hourly (maximum 1.25g/day)
- Diclofenac:
  - 75mg IV twice a day or
  - 75-100mg PR per day (maximum 150mg/day)

**Contraindications:** Bleeding diathesis, peptic ulceration, renal dysfunction, allergy to NSAIDs (care in asthma), severe heart disease (especially with diclofenac).
Combination Analgesic
Co-dydramol: 1-2 tablets PO 4-6 hourly (maximum 8 tablets/day).
(10mg dihydrocodeine + 500mg paracetamol/tablet)

Opioids – Oral
Dihydrocodeine: 30mg PO 4-6 hourly (maximum 240mg/day)
Codeine Phosphate: 30mg PO 4-6 hourly
Tramadol: 50-100mg PO 4-6 hourly

Opioids – Parenteral
Morphine is the preferred opioid. It may be given on the wards IM, SC or IV-Patient-Controlled Analgesia (PCA). In A&E, ICUs and Theatres, morphine is also administered as an IV bolus or infusion. If the patient is hypotensive or has signs of shock, treat these before starting as it may reduce blood pressure further.

Note: additional oral opioid analgesic with PCA should only be dihydrocodeine or codeine 30mg 6 hourly.

Injection: Severe acute pain often requires morphine to be given by injection to give adequate control. Use the dosage regimens given in the following tables:

<table>
<thead>
<tr>
<th>*IV morphine</th>
<th>IM morphine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>Dose</td>
</tr>
<tr>
<td></td>
<td>Pain severe</td>
</tr>
<tr>
<td>&lt; 70</td>
<td>2mg</td>
</tr>
<tr>
<td>&gt; 70</td>
<td>1mg</td>
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</table>

*A&E, ICU and Theatres Only

Assess the patient 60min after IM, and 5min after IV, injection.
Assuming there is no evidence of opiate overdose (see section below for diagnosis and treatment), then if:

- *pain relieved*, repeat same dose up to 2-4 hourly PRN after IM injection. Check for overdose post injection as below.
- *pain persists*, for IM administration repeat injection but at a dose no more than 50% of the original dose; for IV administration (only in A&E, ICUs and Theatres) repeat injection but at a dose no more than 50% of the original dose. Re-assess analgesic effect as above and/or check for overdose post-injection (see below).

Infusion: Infusions (morphine 1-6 mg/hour IV) should only be given where there is close supervision with adequate patient monitoring. O₂ should be administered continuously and O₂ saturation monitored. Monitor patient closely. A subcutaneous infusion may be used in patients without IV access.

PCA: Patient Controlled Analgesia allows titration of the opioid to the patient’s need with a higher degree of safety than a continuous infusion. Contact the Acute Pain Team for help with this regimen.

**OPIOID OVERDOSE**

If the opioid causes features of overdose such as drowsiness or respiratory depression (respiratory rate of less than 8 per minute) then:

1. stop the opioid,
2. administer oxygen by face mask,
3. give naloxone by IV injection 100mcg every 2-3 minutes until patient is rousable and respiratory drive returns. **Seek senior review if still unsuccessful after 3 to 4 doses.**
4. consider giving doxapram (1mg/kg) IV. This is a respiratory stimulant and does not reverse analgesia.

Both naloxone and doxapram are shorter-acting than morphine so observe the patient to ensure that the signs of overdose do not recur.

**Communications:** Acute Pain Team (bleep 6477); On-Call Anaesthetist (bleep 6111); Palliative Care Team (bleep 6508).
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:

• DO NOT START ANTIBIOTICS in the absence of clinical evidence of bacterial infection (does not apply to febrile neutropaenic patients)
  • If there is evidence/suspicion of bacterial infection, use the trust empiric antibiotic guidelines to initiate prompt effective antibiotic treatment within one hour of diagnosis (or as soon as possible) in patients with life threatening infections. (Avoid inappropriate use of broad-spectrum antibiotics)
  • 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
• 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:

Stop, Switch, Change, Continue and OPAT:

1. **Stop** antibiotics if there is no evidence of infection
2. **Switch** antibiotics from intravenous to oral when:
   - temperature has been < 38°C for 48 hours or more;
   - oral foods/fluids 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: ARHAI Antimicrobial Stewardship Guidance 18.11.11).*

- Remember the potential harm caused by antibiotics, in terms of side effects and selection of resistant organisms such as MRSA and *Clostridium difficile*. Cephalosporins and ciprofloxacin have particular risk of selecting *C.difficile* or MRSA.
- Treat the clinical condition and not the microbiology result – a positive culture may represent colonisation, normal flora or contamination, as well as infection.
- 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 ‘Dual-approval’ – 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

**Switch from IV to oral antibiotics**

For Trust policy for specific conditions see the Intranet website:
http://stginet/Units%20and%20Departments/Antimicrobial%20prescribing%20information/Antimicrobial%20prescribing%20for%20staff.aspx
(or follow quick link at bottom of Intranet homepage)

Seek further advice within working hours as appropriate from:

- Medical Microbiology Medical Specialities Registrar (including A&E): bleep 6480
- Surgical Specialities Registrar (including cardiology, cardiothoracic surgery, neurosciences, obstetrics and gynaecology): bleep 6959
- Intensive Care and Paediatric Specialities Registrar (covers GICU, CITU. NICU, PICU, NNU, Paediatrics, Paediatric Surgery): bleep 7118
- Clinical Infection Unit (adult infectious diseases Registrar: bleep 7568
- Ward Pharmacists, or Infection Control Nurses

For out-of-hours advice contact the respective department via switchboard.
Complex infections in inpatients such as TB, meningitis or infections in returned travellers should generally be managed by CIU. 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 nurse (blp 8170) or consultant (aircall SG278). 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>
ANTIMICROBIAL POLICY FOR PATIENTS WITH NEUTROPENIA
Link consultant: Dr Matthew Laundy

Patients with neutropenic sepsis should be treated according to Trust protocol (see new guideline below)

The guideline for neutropenic sepsis is applicable to unwell or febrile patients (T>38°C) 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 & give IV antibiotics – DO NOT WAIT FOR NEUTROPHIL COUNT.
**INFECTIVE ENDOCARDITIS**

Suspected endocarditis: take blood cultures (several sets, 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 Microbiology team, blp 6959/x1970, 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 & Infectious Diseases, with input from Medical Microbiology in all cases.

**Empirical Therapy (if organism known, discuss with microbiology)**

<table>
<thead>
<tr>
<th>Clinical criteria</th>
<th>Antibiotics</th>
<th>Duration</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;1 week duration/indolent presentation</td>
<td>Amoxicillin 2g 4hrly &amp; Gentamicin 1mg/kg bd</td>
<td>4 weeks (Stop Gent at 2 weeks)</td>
<td>Aim for Gent levels Trough &lt; 1mg/L &amp; Peak 3-5 mg/l</td>
</tr>
<tr>
<td>&lt; 1 week, Acute, severely ill, IVDU</td>
<td>Flucloxacillin 2g 4-6hrly* &amp; Gentamicin 1mg/kg 8 hrly Replace flucloxacillin with vancomycin if history of MRSA colonisation. *(give 4hrly if pt&gt;85kg)</td>
<td>4 weeks (Stop Gent at 1 week)</td>
<td>Aim for Gent levels Trough &lt; 2mg/L &amp; Peak 5–10 mg/L</td>
</tr>
<tr>
<td>Prosthetic Valve, pacemaker, other implanted foreign material</td>
<td>Vancomycin as per Trust guidelines &amp; Rifampicin 300 -600mg 12hrly PO &amp; Gentamicin 1mg/kg 8hrly</td>
<td>6 weeks (Stop Gent at 2 weeks)</td>
<td>Aim for Gent levels Trough &lt;1mg/L &amp; Peak 5–10mg/L Aim for Vanc levels of 15-20mg/L (start treatment as per Trust guidelines but adjust doses according to levels)</td>
</tr>
</tbody>
</table>

Penicillin Allergy
As for Prosthetic valve

Suspected MRSA
As for Prosthetic valve

**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>Microbiologically confirmed streptococcal pharyngitis</td>
<td>Penicillin V PO 500mg qds (or clarithromycin PO 500mg bd if allergic to penicillin)</td>
</tr>
<tr>
<td>Acute otitis media &amp; bacterial infection proven or strongly suspected</td>
<td>Amoxicillin PO 500mg tds for 3 days</td>
</tr>
<tr>
<td>Suspected acute epiglottitis</td>
<td>1) Co-amoxiclav 1.2g IV tds OR Ceftriaxone 2g daily IV 2) Ertapenam 1g IV once daily if penicillin-allergic (contact microbiology if history of anaphylaxis with penicillin)</td>
</tr>
<tr>
<td>Suspected diphtheria</td>
<td>Call for Consultant help.</td>
</tr>
<tr>
<td>Sinusitis</td>
<td>Antibiotics should only be prescribed if symptoms are severe or persistent with a purulent discharge for 7 days or more. Give 3-7 days treatment with oral amoxicillin 500mg tds. If allergic to penicillin give oral Clarithromycin 500mg BD.</td>
</tr>
<tr>
<td>Infection</td>
<td>1st line Antibiotics</td>
</tr>
<tr>
<td>-----------</td>
<td>----------------------</td>
</tr>
<tr>
<td>Community acquired pneumonia (CAP)</td>
<td>LOW SEVERITY (CURB-65 score 0-1) Doxycycline PO 200mg STAT then 100mg OD</td>
</tr>
<tr>
<td>Infective Exacerbation of COPD and LRTI</td>
<td>MODERATE-SEVERE (CURB-65 score 2-5) Benzyl Penicillin IV 1.2g, 4-hrly + Doxycycline PO 200mg STAT then 100-200mg OD (use Clarithromycin IV 500mg, 12-hrly if unable to take oral)</td>
</tr>
<tr>
<td>Hospital Acquired Pneumonia (HAP)</td>
<td>Doxycycline PO 200mg STAT then 100mg OD (or Amoxicillin IV 1g 8-hrly if severe or unable to take orally)</td>
</tr>
<tr>
<td>Aspiration Pneumonia</td>
<td>Doxycycline PO 200mg STAT then 100mg OD OR (if severe or unable to take oral) Benzyl Penicillin IV 1.2g, 4-hrly + Gentamicin IV OD as per dosing guidelines</td>
</tr>
<tr>
<td>Urinary Tract Infection</td>
<td>Uncomplicated UTI Trimethoprim PO 200mg, 12-hrly</td>
</tr>
<tr>
<td>Catheter-associated UTI</td>
<td>Complicated UTI (structural abnormality or post-urological surgery) &amp; Pyelonephritis Co-amoxiclav IV 1.2g, 8-hrly + Gentamicin IV 5mg/kg STAT if shocked</td>
</tr>
<tr>
<td>Intra-abdominal Sepsis</td>
<td>Amoxicillin IV 1g, 8-hrly + Gentamicin IV OD as per dosing guidelines + Metronidazole IV 500mg, 8-hrly</td>
</tr>
<tr>
<td>Clostridium difficile</td>
<td>NON SEVERE Flucloxacillin PO 500mg, 6-hrly</td>
</tr>
<tr>
<td>Ceftriaxone IV 4g OD + Aciclovir IV 10mg/kg 8hrly if viral encephalitis suspected</td>
<td>Seek microbiology advice</td>
</tr>
<tr>
<td>Meningitis (Start antibiotics immediately)</td>
<td>Co-amoxiclav PO 625mg or IV 1.2g, 8-hrly + Gentamicin IV 5mg/kg STAT</td>
</tr>
<tr>
<td>Clinical symptoms of infection (sweats, chills, malaise, rigors etc) plus 2 or more of the following: Temp &gt;38° or &lt;36°, HR &gt;90bmp; RR &gt;20/min WCC ≤4 or &gt;12. Order Chest X-ray, send blood and urine cultures</td>
<td>Doxycycline PO 200mg STAT then 100mg OD + Gentamicin IV STAT</td>
</tr>
<tr>
<td>Meningitis</td>
<td>Ceftriaxone IV 4g OD + Aciclovir IV 10mg/kg 8hrly if viral encephalitis suspected</td>
</tr>
</tbody>
</table>

*Note: CURB-65 score includes: Confusion (new onset), Age ≥65 yrs, Urea ≥7mmol/L, Respiratory rate ≥30/min, BP <90/60mmHg (systolic) or ≤60 (diastolic)*
**EMPIRICAL TREATMENT OF DIABETIC FOOT INFECTION**

Refer patients urgently to Diabetic Foot Team x1859; Vascular SpR blp6640

<table>
<thead>
<tr>
<th>Clinical syndrome (+general comments)</th>
<th>Empirc Antibiotic Choice — if pathogen is known adjust treatment</th>
<th>Category of infection</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cellulitis</strong></td>
<td></td>
<td>‘Routine’</td>
<td></td>
</tr>
<tr>
<td>Mild erythema, warmth oedema. Moderate lymphatic streaking, large areas involved</td>
<td>Fluclaxacillin PO 1g QDS</td>
<td>Doxycycline PO 200mg stat then 100-200mg OD</td>
<td>1 week</td>
</tr>
<tr>
<td>Severe systemic features</td>
<td>Benzylpeniciln IV 1.2 g 4hrly + Fluclaxacillin IV 1g 6hrly</td>
<td>Vancomycin IV + Benzylpeniciln IV 1.2g 4hrly + Fluclaxacillin IV 1g 6hrly</td>
<td>1 week</td>
</tr>
<tr>
<td><strong>Ulcer + Cellulitis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>The presence of an ulcer without cellulitis does not warrant antibiotics</td>
<td>Co-amoxiclav PO 625mg tds</td>
<td>Doxycycline PO 200mg stat then 100-200mg OD + Trimethoprim PO 200mg BD</td>
<td>1-2 weeks then review (preferably in the diabetic foot MDT)</td>
</tr>
<tr>
<td>Severe</td>
<td>Co-amoxiclav IV 1.2g tds</td>
<td>Vancomycin IV + Co-amoxiclav IV 1.2g tds</td>
<td>2 weeks then review, as above</td>
</tr>
<tr>
<td><strong>Ulcer + definite osteomyelitis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suspect if deep tissues involved or failure to improve, consider imaging and seek specialist advice</td>
<td>Co-amoxiclav IV 1.2g tds</td>
<td>Vancomycin IV + Co-amoxiclav</td>
<td>Minimum of 2 weeks may need longer if slow to improve</td>
</tr>
<tr>
<td>Initial IV phase</td>
<td>Co-amoxiclav IV 1.2g tds</td>
<td>Vancomycin IV + Co-amoxiclav</td>
<td>Minimum of 2 weeks may need longer if slow to improve</td>
</tr>
<tr>
<td>Continuation on PO phase</td>
<td>Co-amoxiclav IV PO 625mg tds</td>
<td>Doxycycline PO 200mg OD + Co-amoxiclav PO 625mg tds</td>
<td>When ready to switch to oral, continue treatment for a minimum 6 weeks in total (including IVs)</td>
</tr>
</tbody>
</table>

- Review MRSA status at 48hrs and relevant microbiology (eg.deep swabs, tissue and bone samples) – discuss with microbiology for further antibiotic advice if resistant organisms have been isolated or if the patient deteriorates clinically. For **Vancomycin** dosing, refer to the dosing guidelines in Appendix 10.
INFECTIOUS DIARRHOEA

The following advice relates directly to trust policy and guidelines:

Patients with suspected infectious diarrhoea should be managed according to the
ST GEORGE’S NHS TRUST ADULT INPATIENT DIARRHOEA PROTOCOL
http://stginet/Units%20and%20Departments/Infection%20Control/diarrhoea%20protocol%202012%20v3.doc

MANAGEMENT OF C. DIFFICILE

*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%.

Empirical antimicrobial therapy should be guided by the St George’s protocol. When used, antibiotics should always have a stop/review date and a reasonable attempt should be made to make a microbiological diagnosis prior to commencing antibiotics. Antimicrobial therapy should be guided by microbiology results.

**Suspected CDI**

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 while determining the cause of diarrhoea and consult the Infection Control Team only if an infectious cause is suspected
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, assess severity and treat according to Management Chart and ensure the patient is reviewed daily regarding:

- Need for fluid resuscitation and electrolyte replacement,
- Nutritional status,
- Signs of acute abdomen, ileus or toxic megacolon, (if present refer to surgeons urgently).
- Signs of increasing severity of disease and if present refer to ITU early as patients may deteriorate very rapidly

**Testing**

A stool sample should be sent to microbiology when CDI is suspected. The laboratory will then use up to three different types of tests for diagnosis of CDI from the stool sample:

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.
CDI TESTING ALGORITHM

Suspected CDI
- Profuse watery diarrhoea which represents a change in bowel habit
- Recent antimicrobial therapy

Send stool for toxin test to Microbiology
Initiate treatment if CDI is suspected on clinical grounds

GDH Antigen Test

+ve

No evidence of CDI
Review antibiotic treatment. If clinical suspicion is high, continue treatment and re-send stool for C.diff tox in once

-ve

Toxin Test

+ve

Result on EPR:
C.difficile antigen (GDH) DETECTED
C.difficile cytotoxin in (A/B) DETECTED
These results are compatible with CDI – Treatment AND infection control precautions are required

-ve

PCR Test

+ve

Result on EPR:
C.difficile antigen (GDH) DETECTED
C.difficile cytotoxin in (A/B) DETECTED
C.difficile DNA DETECTED
No evidence of CDI:
Treatment not usually required BUT
If the results are compatible with C.diff carriage:
Infection control precautions are required until diarrhoea resolves

-ve

MANAGEMENT OF C.DIFFICILE

See chart below

NOTE: The oral route is the best option in the treatment of Clostridium difficile as it results in adequate drug concentrations.
- Metronidazole IV 500mg TDS can be used in vomiting patients and those who are unable to tolerate oral or NG route. Change to oral metronidazole as soon as the patient is able to tolerate oral or NG route.
- IV vancomycin has no role in the treatment of Clostridium difficile diarrhoea.
Suspected CDI
• Profuse watery diarrhoea (which represents a change in bowel habit)
• Recent antimicrobial therapy

Actions
S- Suspect a case may be infective when there is no other obvious cause for diarrhoea
I- Isolate the patient until asymptomatic for 48h & consult the Infection control team (x5724) 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.

A full examination and review should 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 acute abdomen, toxic megacolon (consider abdominal X-ray and surgical review if abdominal pathology found)
✓ Stop contributory agents i.e. laxatives
✓ Stop antimotility agents where possible (loperamide, opiates)
✓ Commence appropriate supportive therapy i.e. fluid resuscitation
The patient should be review daily by medical team until symptoms resolve

1st-line Treatment Mild – Moderate CDI
• Oral/NG metronidazole 400mg tds for 10-14 days
• If oral/NG therapy is impossible give IV metronidazole 500mg tds
• Consider oral/NG vancomycin* if patient has recently received metronidazole

1st-line Treatment Severe CDI
• If toxic megacolon suspected request urgent surgical opinion
• Treat with Oral/NG vancomycin* 125mg qds for 10-14 days
• If oral/NG therapy is impossible give IV metronidazole 500mg tds (IV vancomycin has no role in the treatment of CDI)

Treating Failure
Mean time to improvement is 2-3 days. If no improvement in symptoms after 5 days contact microbiology

NO

C.DIFF confirmed by microbiology?
YES

Severity Assessment
Mild – Moderate CDI
• WCC < 15 x 10⁹/L
• <5 stools of type 5-7 on the Bristol Stool Chart per day.
Severe CDI is associated with any one of the following:
• a WCC ≥15 x 10⁹/L,
• an acutely 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 may be a less reliable indicator of severity.

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

*Vancomycin NG administration
Vancomycin capsules cannot be opened up and administered via a feeding tube.
Vancomycin injection can be used orally/via feeding tubes. Reconstitute a 500mg vial with 10ml of Water for Injections. Dilute dose 125 mg (2.5ml) in 20ml of water. Reconstituted solution stored in fridge can be used up to 24h after reconstitution. Discard remaining solution 24h after reconstitution.

Fidaxomicin NG administration
Fidaxomicin tablets can be crushed & mixed with water immediately prior to administration via NG tube

If CDI confirmed by microbiology:
Switch treatment to:
• Oral fidaxomicin 200mg bd for 10 days
• If oral/NG therapy is impossible give IV metronidazole 500mg tds

Consider alternative diagnosis.
If clinical suspicion high send repeat stool and continue current therapy

Recurrence disease
contact microbiology
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.

THE FOLLOWING HIGH-RISK PATIENT GROUPS SHOULD RECEIVE ANTIBIOTIC PROPHYLAXIS

- Previous Infective Endocarditis
- Prosthetic valve
- Acquired valvular heart disease with stenosis or regurgitation
- Unrepaired or incompletely repaired cyanotic congenital heart disease
- Congenital heart disease repaired with prosthetic material (for 6 months after procedure)
- Valve disease in recipients of a cardiac transplant

HIGH RISK PATIENTS REQUIRE PROPHYLAXIS FOR THE FOLLOWING PROCEDURES

- Dental procedures involving dento-gingival manipulation or endodontics
  - Dental extractions
  - Sub-gingival scaling
  - Placement of restorations in relation to the gingival mucosa
- All surgery to the jaw and oral cavity*
- ENT – tonsillectomy and adenoidectomy*
- Invasive procedures of respiratory tract needing incision or biopsy of mucosa
- All gastrointestinal and genitourinary surgical procedures

Prophylactic regimens:

<table>
<thead>
<tr>
<th></th>
<th>1st line prophylaxis</th>
<th>If allergic to penicillin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral – single dose</td>
<td>Amoxicillin 3g</td>
<td>Clindamycin 600mg</td>
</tr>
<tr>
<td>1hr before procedure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unable to take oral</td>
<td>Amoxicillin 3g</td>
<td>Clindamycin 600mg</td>
</tr>
<tr>
<td>therapy Single IV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>dose 0-30 mins before procedure</td>
<td></td>
<td></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://stginet/Units%20and%20Departments/Antimicrobial%20prescribing%20information/Endocarditis%20prophylaxis.pdf](http://stginet/Units%20and%20Departments/Antimicrobial%20prescribing%20information/Endocarditis%20prophylaxis.pdf)
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. In patients allergic to penicillin give doxycycline (100 mg bd) plus metronidazole (400 mg tds) by mouth for 3-5 days. Remember tetanus prophylaxis, and rabies if patient from an endemic region (seek advice). For human bites give accelerated hepatitis B immunisation.</td>
</tr>
<tr>
<td>Lacerations</td>
<td>Nil</td>
</tr>
</tbody>
</table>

See the Trust Intranet for guidelines on surgical prophylaxis. Use the quick links at the bottom of the Trust homepage and click on antibiotic prescribing.

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

MALARIA OR FEVER IN RETURNING TRAVELLERS
Link consultant: Professor Derek Macallan
Malaria should be considered in any ill or febrile patient who has travelled in a malaria-endemic area.
For country information, see http://nathnac.org/pro/index.htm. Note that the incubation can be very prolonged.

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

All febrile adult travellers returning from the tropics should be referred to Infectious Diseases: CIU registrar, bleep 7568 during daytime, or via switch after hours.
If malaria is diagnosed in an adult, enquire if there are children in the family who are currently unwell.

Children suspected of having malaria should be evaluated in paediatric A&E the same day. Inform the Paediatric Registrar on duty, bleep 7474.
DRUG OVERDOSAGE/ACUTE POISONING
Link consultant: Dr Arv Sadana

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 (username: H1082, password: 4BUFM8), 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 O\textsubscript{2} 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
- Continue charcoal until patient’s clinical and laboratory parameters, including plasma drug concentrations, are improving
- Give a laxative to prevent constipation

**B. Whole bowel irrigation**

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

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

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

**SPECIFIC MEASURES FOR COMMON DRUG OVERDOSES PARACETAMOL**

Paracetamol overdose, even in small amounts, can cause fatal liver damage. To prevent this:
- Paracetamol should be suspected as a component of all overdoses.
- Plasma concentrations should be measured and compared against a paracetamol treatment graph (see below)

Patients with plasma paracetamol concentrations above the treatment line are at risk of liver damage and require antidote treatment.
- N-acetylcysteine, which acts as an antidote and prevents paracetamol-induced liver damage, should be used as described below.
- The treatment of patients who have taken a paracetamol overdose depends on the timing of presentation after overdose, as well as the way in which the overdose was taken.

**Within 4 hours of ingestion**
- <1 hour, give activated charcoal
- 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:
  - 150mg/Kg (maximum 16.5g) in 200mL 5% glucose as IV infusion over 60 minutes
  - 50mg/Kg (maximum 5.5g) in 500mL 5% glucose as IV infusion over next 4 hours
  - 100mg/Kg (maximum 11g) in 1L 5% glucose as IV infusion over next 16 hours

**Within 4–8 hours of ingestion**
- Measure plasma concentrations at presentation
- Compare concentrations with treatment graph to determine whether N-acetylcysteine

**Within 8-15 hours of ingestion**
- Take blood for paracetamol concentrations
- Start N-acetylcysteine infusion immediately
- Stop treatment if level is below the treatment line on the treatment graph.

**Within 15-24 hours of ingestion**
- Take blood for paracetamol concentrations
- Start N-acetylcysteine infusion immediately
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.

![Graph showing plasma paracetamol concentration over time](image)

**Presenting after 24 hours**
Take blood for 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. If the patient is symptomatic, or any blood tests are abnormal, discuss management with NPIS.

**Situations where N-acetylcysteine should be given without guidance of the treatment graph**
- 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
**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**

*Gastric decontamination*
- If <1hour since overdose and there are no contraindications, perform gastric lavage and give 50g activated charcoal, if >1 hour just give activated charcoal.
- 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
Give 25-50g charcoal every 4 hours until plasma salicylate level reaches its peak and starts to fall.

*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.
Give 1 litre of 1.26% or 1.4 % sodium bicarbonate (isotonic) with 40mmol K⁺ IV over 4 hours. 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.
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.
Patients with chronic impaired liver function can remain stable (compensated) for many months but can also decompensate rapidly. The commonest causes of acute (rapid) decompensation are hypovolaemia (sometimes secondary to a GI bleed), alcohol, sepsis, drugs and renal impairment. Rapid ‘decompensation’ may also occur with the development of hepatocellular carcinoma (HCC).

**Investigations**

**Blood Tests**
1. FBC
2. Clotting screen
3. Urea, electrolytes, creatinine
4. Liver function tests, \( \gamma \)GT, albumin
5. \( \alpha \)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. Ascetic 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 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 (menadiol 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 pentoxifylline 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 30-60mg/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**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
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<tbody>
<tr>
<td>Ibuprofen</td>
<td>400mg 6-8 hourly.</td>
</tr>
<tr>
<td>Indomethacin</td>
<td>50mg/8 hourly; (6 hourly for acute gout)</td>
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</table>

*Alternatively, for gout, give*

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colchicine</td>
<td>500micrograms/2 hourly (maximum 8 daily)</td>
</tr>
</tbody>
</table>

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.
RECOMMENDATION FOR MANAGEMENT OF SUSPECTED GIANT CELL ARTERITIS (GCA)

Link Consultant: Dr Nidhi Sofat

Early recognition and diagnosis
- Consider GCA in patients presenting with at least 3 symptoms listed in figure below.
- Particular attention should be paid to features like jaw claudication, visual symptoms and temporal artery abnormalities which predict neuro-ophthalmic complications.

Investigation and temporal artery biopsy (TAB)
- FBC, U+E, ESR, CRP, LFT, bone profile, glucose, urinalysis, CXR (for aortic aneurysm)
- Rheumatology SpR will liaise with Vascular surgeon (bleep: 6640) to organize TAB in either Ambulatory Care area in Richmond ward or Dermatology Theatre in clinic B (xx day).

Initial steroid therapy
- **Uncomplicated GCA:** Prednisolone 40-60mg (not less than 0.75mg/kg) daily until resolution of symptom and lab abnormalities.
- **Complicated GCA (Evolving visual loss/ amaurosis fugax):** IV Methylprednisolone 500mg -1g daily for 3 days before switching to oral steroid.
- **Complicated GCA (Established visual loss):** Prednisolone 60mg or 1mg/kg daily

Follow up and monitoring of treatment
- Follow up in Rheumatology clinic in 2-3 weeks following initial referral.
- **TAB positive:** add bone protection (calcium/ vitamin D supplement & bisphosphonate) and gastrointestinal protection (proton pump inhibitor).
- **TAB negative, clinical suspicious high:** treat as TAB positive GCA.

Steroid reduction regime
- 40-60mg prednisolone for 4 weeks (or until resolution of symptoms and lab abnormalities).
  - Then reduce the dose by 10mg every 2 weeks to 20mg
  - Then taper by 2.5mg every 2-4 weeks to 10mg
  - Then taper by 1mg every 1-2 months

Treatment of relapse
- If symptoms relapse, treat with previous higher steroid dosage.
- **Large vessel GCA-** investigate with PET. Consider treatment using systemic vasculitis protocol
  - Consider steroid sparing agent (Methotrexate, Leflunamide) in the case of recurrent relapse or failure to wean steroid dose.

References
Management of suspected Giant Cell Arteritis (GCA)

Suspected GCA
- Age > 50
- Abrupt new & persistent headache
- Jaw/ tongue claudication
- Scalp sensitivity/ tenderness
- Temporal artery (TA) abnormalities
- Visual symptoms *
- Symptom of Polymyalgia Rheumatica
- ESR > 50, CRP > 4mg/L

Immediate start of steroid
- **Uncomplicated** (without visual symptoms/ jaw claudication): Prednisolone 40mg to 60mg daily
- **Complicated** (Visual symptoms/ jaw claudication): Pulsed IV Methylprednisolone 500mg to 1g daily for 3 days; then Prednisolone 1mg/kg or 60mg daily
- Aspirin 75mg OD in both groups

Temporal Artery (TA) biopsy
(Ideally within 10 days of commencing steroid)

- Biopsy positive
  - Prednisolone 40-60mg for 4 weeks
  - Dose reduction regime (see table 1)
  - Bone protection
  - Gastrointestinal protection
  - Monitor disease activity, relapses, large vessel GCA
  - Consider steroid sparing agent

- Biopsy negative
  - Clinical suspicion HIGH
  - Treat as biopsy positive GCA
  - Clinical suspicion LOW
  - Consider alternative diagnosis
  - Taper steroid within 2 weeks

*Visual symptoms: Amaurosis fugax, blurring, diplopia

Urgent referral
- **All cases**: Contact Rheumatology SpR by bleep 7787 AND email referral to: rheumatologyregistrars@stgeorges.nhs.uk who will organise TA biopsy and follow up appointment.
- **If any visual symptoms**: Phone Moorfield eye clinic on ext 6115 or 6119 (Mon-Fri, 9a.m-5p.m); or Ophthalmology on-call via switchboard 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, myoglobinemia or haemo-globinaemia). Other less common causes of AKI are obstruction, acute interstitial nephritis (due to drugs or infection), and glomerulonephritis occurring as a primary event or complicating multi-system disease.

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

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

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.

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 blp 6415 or ext 1080; if uncontrolled hyperkalaemia (>6.0), acidosis (pH<7.2), fluid overload +/- anuria, significant haematuria proteinuria, low Hb, or AKI Stage 3
4. Treat hyperkalaemia (K+ greater than 6mmol/L) - see management of **Hyperkalaemia**.
5. Order urgent ultrasound scan (if no other obvious cause is found) and relieve obstruction if present (using catheter and urology advice). Order ANA, ANCA, anti-GBM antibodies, complements, serum electrophoresis, urine Bence Jones Protein – if haematuria and proteinuria present; LDH, bilirubin, retics, CK if necessary.
6. Diuretics can help to reduce fluid overload.

If dehydrated and urine sodium is <10mmol/L or fractional excretion of sodium <1% think of prerenal failure and administer adequate fluids. Renal biopsy to be considered if there are atypical clinical features or features suggesting multisystem disease.

**Indications for dialysis:**
- 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 renal disease.

For patients due to receive radiocontrast, the following should be implemented to reduce the risk of AKI due to contrast nephropathy:

**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
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 below 3 mmol/L, or below 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, beta stimulants, 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⁺/h (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.

N.B. 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⁺ exceeds 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 reduced renal excretion (as in renal failure, when taking potassium-sparing diuretics, ACE inhibitors/angiotensin-II receptor blockers or NSAIDS, and in Addison’s disease), or when potassium leaves cells as in acidosis, diabetic hyperglycaemia and cell damage (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, pCO$_2$ and pO$_2$ if in doubt.

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

2. To move potassium into the cells give dextrose/insulin infusion, 50mL 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), give 500 mL 1.4% NaHCO$_3$ over 2-3 hours and recheck bicarbonate.
   ii) If there is severe acidosis (arterial pH<7.1), give 500 mL 1.4% NaHCO$_3$ over one hour (in patients with volume overload or cardiac arrest give 50-100mL of 8.4% NaHCO$_3$ 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 10-20mg 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 parasthesia, then leading to muscle cramps, carpo-pedal spasm, laryngeal stridor and convulsions. These effects are determined by the concentration of ionised calcium and are influenced by plasma pH (available calcium concentration falls the more alkaline the plasma).

Indications for treatment. Attempts to raise the available calcium should be made if the plasma ‘adjusted’ calcium is below 1.8 mmol/L or the patient has unequivocal signs of hypocalcaemia with a low calcium, ie tetany, positive Chvostek or Trousseau’s sign, or seizures. Calcium levels should always be corrected against albumin.To calculate ‘adjusted’ calcium: adjusted calcium (mmol/L) = unadjusted calcium (mmol/L) + 0.02 x (40 – serum albumin (g/L)).
**Causes.** While alkalosis increases the likelihood of symptoms and signs, and occasionally (eg prolonged hyperventilation) is the sole cause of the clinical picture, other causes include primary hypoparathyroidism, renal failure, vitamin D deficiency and malabsorption. A low plasma Mg\(^{2+}\) can also cause hypocalcaemia without any change in total body calcium. Measure magnesium if in doubt – hypomagnesaemic hypocalcaemia should be treated with intravenous magnesium alone. Seek specialist advice.

**Treatment.** Supplements can be given either by mouth or intravenously.
- **Oral route.** Give 12.5g of CaCO\(_3\) (5g of elemental Ca) over 24h not with food. One Calcichew tablet contains 0.5g of elemental Ca. Alfacalcidol should be given in a dose of 1-5micrograms daily.
- **Intravenous infusion.** Give 10mL of 10% calcium gluconate (2.2mmol 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 produce no symptoms or cause symptoms such as thirst, polyuria, nausea, vomiting, constipation and abdominal pain. There may be confusion or coma.

**Indications for treatment.** Attempt to lower the serum calcium in anyone with an ‘adjusted’ serum calcium of greater than 3 mmol/L unless the value is stable and the patient completely asymptomatic. (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 compartments. Common causes are primary hyperparathyroidism, thiazide diuretics and malignant disease. Rarer causes include sarcoidosis, thyrotoxicosis, vitamin D intoxication, calcium-containing drugs and cortisol deficiency.

**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/h. Consider giving furosemide (40-80mg orally or iv), to increase urine flow and calcuiuresis. If diuretic is given it is essential that the patient is not rendered hypovolaemic. If the serum calcium is still raised after 24 hours give iv pamidronate over 2-3 hours in a dose of 15-90mg (15-30mg if serum calcium up to 3.0mmol/L; 30-60mg if 3-3.5mmol/L; 60-90mg if 3.5-4.0mmol/L and 90mg if above 4mmol/L) dissolved in 500mL 0.9% NaCl. If the patient has renal impairment the rate should not exceed 20mg/h. 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. Where hypercalcaemia is due to hyperparathyroidism associated with renal disease, surgical parathyroidectomy or medical parathyroidectomy, using Cinacalcet may be considered and advice from the renal team sought.

**HYPONATRAEMIA**

Hyponatraemia (Na\(^+\)<135mmol/L) results from H\(_2\)O retention, Na\(^+\) loss or a combination of the two. Although the definition of hyponatraemia is Na\(^+\)<135mmol/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 and an increase in intracellular fluid ie cerebral oedema. The concentration of plasma sodium does not give any indication of volume status, i.e. 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
• 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)

Hyponatraemia is usually associated with hypo-osmolality (plasma osmolality <275 mosmol/kg). The combination of hyponatraemia and normal or elevated plasma osmolality indicates the presence of an additional, osmotically active, substance (e.g. glucose, mannitol.)

**Clinical assessment**
1. Confirm plasma sodium below 135mmol/L
2. Measure urinary sodium concentration
3. Measure plasma osmolality and assess volume status;
   a) if osmolality greater than 275mosmol/kg, assume the problem is hyperglycaemia 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 serum [Na]) + (serum [glucose]/18) + (blood urea nitrogen*/2.8)
*blood urea nitrogen as mg/dl (1mg/dL = 0.36mmol/L)

**Therapy**
Principles
- Treatment of underlying disease where possible
- Initial therapy to raise the serum sodium
- 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:

**Hypovolaemic hyponatraemia:** give iv 0.9% NaC1 stop diuretics give antiemetics 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 (150mmol/L) to be given over 24h from this formula.
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 300mg - 600mg 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.

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 rather than to Na\(^+\) gain.
The causes include:

- H\(_2\)O loss without adequate H\(_2\)O intake
- diuretics/laxatives
- upper GI loss from vomiting or drainage
- osmotic diuresis (e.g. hyperglycaemia)
- diabetes insipidus
- Na\(^+\) gain (ingestion of sea water, infusion of large volumes of intravenous
  NaHCO\(_3\) 8.4%)

Management

1. stop H\(_2\)O loss. Depending on the cause this may involve giving an anti-emetic,
   stopping diuretics or treating diarrhoea
2. calculate the H\(_2\)O deficit, where
   \[
   H_2O\ deficit(L) = \text{body weight in kg} \times 0.6 \times \frac{\text{actual Na}^+(mmol/L) - 140}{140}
   \]
3. replace fluid with 5% dextrose plus 0.18% sodium chloride (contains Na\(^+\) 30mmol/L), alternating with 0.9% sodium chloride (contains Na\(^+\) 150mmol/L). In the
   first 24 hours replace one third of the calculated water deficit and maintain usual fluid
   replacement.
4. check serum Na\(^+\) daily; it should not fall by >8mmol/L in 24 hours.
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. Intaking 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 oxycodeone 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 levofloxacin 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 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
1. the analgesia regimen is followed
2. the patient has fluid input maintained
3. the patient has antibiotic regimen maintained
4. the patient is observed regularly to ensure all vital signs are maintained and pain levels assessed.

*If a patient is discharged* from, or leaves A&E, then:
- contact the haemoglobinopathy specialist nurse (SGH blp 7520; or via Balham Health Centre on 0208 700 0615 if community-based) and give details of the admission and assessment.
- give the patient sufficient analgesia to ensure effective pain management until the patient may see their GP or a specialist nurse counsellor.

**LIFE-THREATENING CRISIS**

Patients can present with a variety of other acute manifestations which may be rapidly fatal if not recognised and treated quickly.

**Infection**

Patients prone to sickling have reduced splenic function and are at risk of overwhelming septicaemia (pneumococcus, meningococcus, rarely haemophilus) even if taking penicillin prophylaxis. Peak risk is in childhood. The patient may present with fever, shock, seizures, coma, meningitis (often with delayed CSF pleocytosis) or even profuse diarrhoea. Early IV antibiotics to cover pneumococcus and staphylococcus (Co-amoxiclav and clarithromycin, or if penicillin allergic then levofloxacin alone) and volume support are vital. If osteomyelitis suspected, discuss with Microbiology.

**Splenic or Liver Sequestration**

During infection children may suffer a rapid fall in haemoglobin and growth of the spleen – changes often noted by the mother. Death can result from hypovolaemia and
anaemia. Early transfusion is vital. In adults, liver sequestration is more common and can present similarly with profound anaemia and hepatomegaly. Transfusion is often required in these patients as well.

**Chest crisis** Severe shunting & hypoxia caused by intra-pulmonary sickling and mimicking pulmonary embolus/pneumonia, may start in one lobe and then spread to others. It sometimes begins as a pain crisis affecting ribs or shoulders. Treat with fluids and oxygen; observe arterial O₂ tensions – a falling PaO₂ will require exchange transfusion and needs expert advice. Encourage patients with chest pain to attempt one maximal inhalation every 5-10 mins (‘incentive spirometry’) to aerate basal lung segments; this reduces the risk of progressive sickle chest syndrome. Non invasive respiratory support may well be required, as well as urgent exchange transfusion. Discuss with haematology urgently.

**Girdle syndrome**
If sickling occurs in the splanchnic bed, abdominal pain with rigidity, loss of bowel sounds and increasing icterus may develop. IV fluids are vital. A surgeon should be consulted to exclude other abdominal events, but surgery should be withheld unless unavoidable, and then only after exchange transfusion and discussion with haematologists.

**Cerebral sickling**
Patients can present with strokes, fits, coma, bizarre behaviour or psychosis, and sickling should be excluded in any susceptible patient with such signs. IV fluids are vital and early exchange transfusion a possibility. Patients are at risk of both haemorrhagic and ischaemic strokes.

**Priapism**
Priapism typically affects only the corpora cavernosa. Major or prolonged attacks post puberty can result in permanent loss of erectile function. Urgent referral to Urology is essential as early decompression can be achieved by aspiration +/- intracavernosal phenylephrine.

**Blood transfusion**
In a patient with Sickle Cell Disease blood transfusion can be dangerous. Never give a simple transfusion for anaemia (except in those 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<5g/dl or if there has been a 2g/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 litres/24 hours) and ensure nephrotoxic drugs are withheld.
MANAGING ACUTELY AGITATED ADULT PATIENTS

New-onset acute psychiatric symptoms in an inpatient
Delirium should be considered as the most likely cause of new-onset confusion, or paranoia, or hallucinations in a hospital inpatient. Patients at particular risk are the elderly, acutely unwell, and those with CNS disease or alcohol dependence. It is important to identify underlying causes of agitation (See Step 1) before using drug treatment. Any factors or underlying causes contributing to delirium should be addressed.

- An agitated patient must have a senior medical review, preferably by a Consultant or Medical Registrar within one hour.
- Using medication to manage agitation can be harmful and not necessarily beneficial to the patient. It is always important to consider non-drug approaches first.
- If drug treatment is required, therapeutic choice and dosage will depend on a patient’s co-morbidities such as cardiovascular disease, the presence of dementia, liver and renal impairment. Minimise polypharmacy, avoid routine sedatives (including sleeping tablets) and review medication every 24 hours
- Benzodiazepines should be used first line unless contra-indicated. Risks with benzodiazepines include loss of consciousness, respiratory depression or arrest.
- Haloperidol can cause irreversible extrapyramidal symptoms and Parkinsonism in patients with dementia with Lewy Bodies and so, is contra-indicated in these patients. Haloperidol can also prolong cardiac QT interval and therefore, where possible, patients should have an ECG prior to commencing Haloperidol and be monitored.
- All antipsychotics are associated with increased risk of cardiovascular collapse, seizures, akathisia, dystonia, dyskinesia, neuroleptic malignant syndrome, excessive sedation; whilst Antihistamines can cause excessive sedation.
- Clinicians prescribing medication to sedate disturbed or violent patients should be familiar with the NICE guidelines on short-term management of disturbed/violent behaviour in in-patient psychiatric settings and emergency departments [http://guidance.nice.org.uk/CG25](http://guidance.nice.org.uk/CG25)


The following protocol is part of St George’s Trust policy and, with the exception of patients in Critical Care, should be used to guide management of acutely agitated adult patients.

Step 1 should be followed in all patients. Subsequent Steps 2-5 will depend on the patient’s age (ie above or below 65 yrs old). Patients receiving medication should be monitored according to guidance below.
Protocol for Managing Acutely Agitated Patients

STEP 1 - Non-drug approaches to de-escalate the situation
(for ALL Patients prior to Drug Treatment)

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 (17.00-20.00hrs daytime and 08.00-20.00hrs 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 the Trust Safeguarding Adults policy

If the above measures remain inadequate, then proceed to Steps 2-5 (a) for patients aged > 65 years old or (b) if aged 18-65 years. Should medication be required, ensure the patient is monitored according to the protocol below.
(a) Steps 2-5 (>65 yrs): Drug Management for Acutely Agitated Patients aged > 65 years old

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.

<table>
<thead>
<tr>
<th>Step 2 (&gt;65 yrs)</th>
<th>Oral Medication: Lorazepam 0.5-1mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Continue non-drug approaches. If little or no effect after 30 minutes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Step 3 (&gt;65 yrs)</th>
<th>2ND Oral Medication Further Lorazepam 0.5-1.0mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Continue non-drug approaches. If little or no effect after 30 minutes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Step 4 (&gt;65 yrs)</th>
<th>Consider alternative oral medication Eg Quetiapine 12.5-50mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(Discuss with a senior doctor before administration of any antipsychotic if a patient is known to have Parkinson’s Disease or Lewy Body dementia)</td>
</tr>
<tr>
<td></td>
<td>Continue non-drug approaches. If little or no effect after 30 minutes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Step 5 (&gt;65 yrs)</th>
<th>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</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lorazepam 0.5mg – 2mg IM or Haloperidol 0.5mg – 1mg IM</td>
</tr>
</tbody>
</table>
(b) Steps 2-5 (18-65yrs): Drug Management for Acutely Agitated Patients aged 18-65 years old

The following pharmacological interventions may be appropriate for patients aged 18-65 years who have not responded to Step 1 above. These 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 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**
(18-65yrs)

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

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.

**Step 3**
(18-65yrs)

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

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 4.

**Step 4**
(18-65yrs)

**Intramuscular medication**
Lorazepam 1-2mg i/m*
or
Midazolam 7.5mg i/m*

(* if total daily dose not exceeded in Steps 2 & 3)

If the patient remains very agitated after 1st dose of oral medication and if no contraindications, an enhanced sedative effect may be obtained by giving the following as 2nd oral drug:
Haloperidol 0.5-2mg
or
Olanzapine 5-10mg

If benzodiazepines have been given in steps 2/3 and further medication is needed, or if injectable medication is required and benzodiazepines are contra-indicated:

**Step 4**
(18-65yrs)

**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**
(10-65yrs)

**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 020 351 35000) or Pharmacy.

For patients in whom benzodiazepines are contra-indicated:
(Impaired respiratory function, paradoxical aggression can be caused by benzodiazepines.)

NB. HALOPERIDOL IS NOT SUITABLE FOR PATIENTS WITH CARDIAC DISEASE
Monitoring of patients following the use of medicines for controlling agitation (see dosing table below)

After medication has been given, the following must be monitored and 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
- Flumazenil (a benzodiazepine antagonist) 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) in 24 hours (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. Remember that patient may be unable 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
- 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>
<th></th>
<th></th>
<th>Maximum dose (oral and i/m) in one 24hr period (including prn and regular doses)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maximum oral dose in 24hrs</td>
<td>Maximum injectable dose in 24 hrs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lorazepam</td>
<td>2mg</td>
<td>2mg i/m</td>
<td>2mg</td>
<td></td>
</tr>
<tr>
<td>Quetiapine</td>
<td>50mg</td>
<td>n/a</td>
<td>50mg</td>
<td></td>
</tr>
<tr>
<td>Haloperidol</td>
<td>2mg</td>
<td>2mg i/m</td>
<td>2mg</td>
<td></td>
</tr>
<tr>
<td>Midazolam</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td></td>
</tr>
<tr>
<td>Olanzepine</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td></td>
</tr>
<tr>
<td>Promethazine</td>
<td>n/a</td>
<td>n/a</td>
<td>n/a</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DRUG</th>
<th>(b) For patients aged 18 - 65 years old</th>
<th></th>
<th></th>
<th>Maximum dose (oral and i/m) in one 24hr period (including prn and regular doses)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Maximum oral dose in 24hrs</td>
<td>Maximum injectable dose in 24 hrs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lorazepam</td>
<td>4mg</td>
<td>4mg i/m</td>
<td>4mg</td>
<td></td>
</tr>
<tr>
<td>Quetiapine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Haloperidol</td>
<td>30mg</td>
<td>10mg i/m*</td>
<td>20mg oral + 5mg i/m or 10mg oral + 10mg i/m</td>
<td></td>
</tr>
<tr>
<td>Midazolam</td>
<td>20mg</td>
<td>15mg i/m**</td>
<td>7.5 i/m + 10mg buccal</td>
<td></td>
</tr>
<tr>
<td>Olanzepine</td>
<td>20mg</td>
<td>n/a</td>
<td>20mg</td>
<td></td>
</tr>
<tr>
<td>Promethazine</td>
<td>100mg</td>
<td>100mg i/m</td>
<td>100mg</td>
<td></td>
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</tbody>
</table>

* A maximum of 10mg haloperidol I/M is recommended over a six hour period owing to a lack of beneficial effect above this dose and a greater risk of cardiovascular side effects.

** 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. Doses can be borrowed ‘out of hours’ from any adult Critical Care Unit but must be documented on the ‘Drugs Lent Out’ form.

** Alcohol dependence – Wernicke-Korsakoff’s syndrome**
All alcohol dependent patients admitted to hospital should be prescribed vitamin prophylaxis (see table below), and will need monitoring of fluid and electrolyte balance. In addition, for each patient you should individually consider the need for alcohol withdrawal management (see below). Many patients with Wernicke’s encephalopathy
do not present with the full triad of signs. Therefore give urgent Pabrinex treatment if there is an onset of confusion in an alcohol dependent patient. Pabrinex is diluted in 50-100ml normal saline, and given by IV infusion over 30 mins. If IV infusion not possible, initial doses may have to be given IM. Anaphylaxis is a rare but recognised complication.

<table>
<thead>
<tr>
<th>Patient’s condition</th>
<th>Dose required</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indication of Wernicke’s encephalopathy (Any one of three main symptoms: confusion, ataxia, ophthalmoplegia, or other reason for suspicion.)</td>
<td>Treatment: Two pairs High Potency Pabrinex amps three times per day for 3 days, followed by one pair daily for 3-5 days depending on response. then one pair daily for 3-5 days depending on response.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Dose required</th>
</tr>
</thead>
<tbody>
<tr>
<td>At-risk (Heavy drinking, significant weight loss, poor diet, signs of malnutrition)</td>
</tr>
</tbody>
</table>

| Oral vitamins should be used following parenteral vitamin treatment: Thiamine 100 mg orally four times per day, plus vitamin B compound strong 2 tablets daily. |

**Alcohol dependence – General Management**

Recent NICE guidelines on alcohol dependence and harmful alcohol use are advocated: [http://guidance.nice.org.uk/CG115/Guidance/pdf/English](http://guidance.nice.org.uk/CG115/Guidance/pdf/English)

For assisted withdrawal (detox), the patient is required to stop alcohol intake abruptly, and the ensuing withdrawal symptoms treated with medication, usually benzodiazepines. Once the withdrawal symptoms are controlled, the medication can be gradually reduced and stopped at a rate that prevents withdrawal symptoms re-emerging but without creating over-sedation. Key elements of the process are to provide a large enough initial dose to prevent severe withdrawal symptoms including seizures, DTs, severe anxiety or autonomic instability, but to withdraw the medication at a rate which prevents re-emergence of symptoms or serious complications (eg DTs or seizures).

NICE Guidelines also define: a) the symptom-triggered and b) the fixed-dose regimen methods for detox.

**a) Symptom-triggered regimen (ST)**

This approach involves tailoring the drug regimen according to the severity of withdrawal and complications displayed by the patient (eg Chlordiazepoxide 20-30mg hourly as needed, based on symptoms such as heart rate >90/min, diastolic BP >90mmHg or feature of withdrawal). The patient is monitored on a regular basis and medication administered according to the patient’s level of withdrawal symptoms. Withdrawal symptoms are usually assessed by clinical assessment including observation and interview.

**b) Fixed-dose regimen (FD)**

A fixed-dose (FD) regimen involves starting treatment with a standard dose determined by the recent severity of alcohol dependence and/or typical level of daily alcohol consumption, followed by reducing the dose to zero usually over 7 to 10 days according to a standard protocol.

Alcohol dependent patients showing signs of withdrawal or at high risk of developing withdrawals should be prescribed benzodiazepines (usually chlordiazepoxide).
Assessment of alcohol history should follow the St Georges Alcohol Screening Test (the 3 question AUDIT-C).

NB: for frequency and usual daily amount, note that there is an increased risk of dependence and withdrawal symptoms with around 15 units/day.

### Example of a fixed-dose (FD) regimen for chlordiazepoxide

<table>
<thead>
<tr>
<th>Stabilisation 1-3 days</th>
<th>Intermediate Reduction</th>
<th>Reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>DAY 1</td>
<td>DAY 2</td>
<td>DAY 3</td>
</tr>
<tr>
<td>8.00am</td>
<td>20-30 mg</td>
<td>20 mg</td>
</tr>
<tr>
<td>Noon</td>
<td>20-30 mg</td>
<td>20 mg</td>
</tr>
<tr>
<td>6.00pm</td>
<td>20-30 mg</td>
<td>20 mg</td>
</tr>
<tr>
<td>10.00pm</td>
<td>20-30 mg</td>
<td>20 mg</td>
</tr>
</tbody>
</table>

A single dose should not exceed 20 mg in the elderly or physically frail.

Withdrawals should be monitored 2 hourly or more frequently during the first 24 hours, until symptoms are stabilised. Regular doses may need to be increased if PRN doses initially required do not alleviate withdrawals. Therefore do not write up next day doses until symptoms have stabilised - in severe cases of withdrawal this may be after 72 hours.

PRN doses for breakout withdrawal symptoms should be in keeping with the regular dose (ie if regular dose is chlordiazepoxide 30 mg QDS, PRN should be 20-30 mg). Consideration should be given to increasing doses at night time and in the morning, as there is often 8 hours between drug dispensing times (eg 40 mg at 8am; 30 mg at noon; 30 mg at 6pm; and, 40 mg at 10pm). More frequent doses may be preferable to increasing above 40 mg due to the possibility of sedation/agitation between doses.

A common error in management of alcohol withdrawal is too rapid a reduction of dose, which can result in emergence or re-emergence of severe alcohol withdrawal symptoms. Another error is a starting dose of chlordiazepoxide that is too low. This can be avoided by taking account of typical daily alcohol consumption in determining the starting dose. In severe alcohol dependence the doses of chlordiazepoxide required may exceed BNF prescribing range. Increasing dosage of chlordiazepoxide is more clinically effective at controlling withdrawal symptoms than adding another type of medication (eg haloperidol). Medication should be started before withdrawal symptoms begin to emerge. Delay in initiating chlordiazepoxide treatment can result in withdrawal symptoms either becoming difficult to control or in the emergence of complications (eg DTs or seizures). Therefore, in people with severe alcohol dependence, it is not necessary to wait until blood or breath alcohol concentration falls to zero. Some people who are severely alcohol dependent can experience withdrawal with a blood alcohol concentration of 100 mg per 100 ml or more.

Note that due to the gradual rate of reduction, with higher starting doses, the duration of treatment is longer than with lower starting doses. A common error in management of alcohol withdrawal is too rapid reduction of chlordiazepoxide, which can result in emergence or re-emergence of severe alcohol withdrawal symptoms. Another common error is too low a starting dose of chlordiazepoxide. This can be avoided by taking account of typical daily alcohol consumption in determining the starting dose. In addition, the response to FD withdrawal regimes should be monitored and the dose of medication adjusted upwards or downwards accordingly in the early stages of
withdrawal. In severe alcohol dependence the doses of chlordiazepoxide required may exceed the British National Formulary (BNF) prescribing range.

It is more clinically effective to increase the dose of chlordiazepoxide to adequately control alcohol withdrawal symptoms than to add another type of medication (for example, haloperidol). The first dose of medication should be given before withdrawal symptoms begin to emerge. Delay in initiating chlordiazepoxide treatment can result in withdrawal symptoms either becoming difficult to control or the emergence of complications such as DTs or seizures. Therefore, in people with severe alcohol dependence, the first dose should be given before the breath alcohol concentration falls to zero, as withdrawal will emerge during the falling phase of breath alcohol concentration. The more severe the alcohol dependence, the earlier withdrawal symptoms emerge after last alcohol intake. Some people who are severely alcohol dependent can experience withdrawal with a blood alcohol concentration of 100 mg per 100 ml or more.

Regular chlordiazepoxide in excess of 30 mg QDS should only be prescribed when severe withdrawal symptoms are suspected, ie patient reporting more than 50 units per day (eg two full bottles of spirits daily). For such patients, follow the above protocol starting at chlordiazepoxide 40mg QDS, prescribe PRN and reassess the patient frequently during the first 24 hours (pulse, BP and signs of withdrawal or excessive sedation).

Daily total dose in excess of 200mg Chlordiazepoxide is only indicated with the most severe withdrawals – this is rarely necessary in women and never in the elderly, or where there is liver impairment.

Liver impairment. Caution is required in prescribing benzodiazepines in hepatic impairment. Seek advice regarding shorter acting drugs or lower doses.

Over sedation. If the patient is over sedated, dose will need to be reduced, this can sometimes be achieved by omitting one dose, but PRN should be available in case withdrawals occur on waking.

Severe behavioural disturbance or DTs. If benzodiazepines do not sedate, then add stat dose of haloperidol 5 mg PO or IM or olanzapine 5-10mg (as per aggression above).

Delirium Tremens (DTs). Alcohol-induced delirium with psychotic symptoms usually develops about 3–4 days after cessation of drinking, and can present with vivid visual and tactile hallucinations and sudden onset of paranoid delusions.

Delirium tremens is a medical emergency, requiring hospital admission. Patients with DTs who are already admitted should not be allowed to take their own discharge until assessed by a senior ward doctor and/or a psychiatrist. Pharmacological management is usually with chlordiazepoxide, with higher doses if indicated by symptoms. If oral administration is not possible, an alternative is lorazepam 1-2mg IM. Patients with DTs present particular risks to their own safety and the safety of those around them. There should be a low threshold for arranging one-to-one nursing by a registered mental health nurse. In patients who remain severely disturbed see “severe behavioural disturbance” in psychiatric emergency guidance. All patients with DTs should be given Pabrinex (see above) as soon as practicable, in view of the difficulties in excluding Wernicke’s encephalopathy. Once the patient is stabilised, regular chlordiazepoxide regimen should reflect the dose needed to control the DTs.

The Alcohol & Drug Liaison Team (ext 0595, blp 6915) will offer advice on managing withdrawal symptoms, as well as more comprehensive assessment and linking patients into community alcohol treatment. Further information is available under Alcohol Liaison on the St Georges Hospital Intranet.
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 I (cTnI). 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 cTnI also rises in other conditions where there is cardiac damage, such as myocarditis.

Measurement of cTnI 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 cTnI should be requested on admission, 3 and 6 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 12 hrs from admission. In accordance with the new universal definition of MI, an increase in troponin by more than 30% to above 50 ng/l with appropriate clinical features is required for a definitive diagnosis of MI. An increase from <50ng/l to more than 500 ng/l is highly suggestive of AMI. Re-infarction may be detected by repeat measurement.
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 (e.g., 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.

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).
3. Clinical Ethics Committee: email cec@sghms.ac.uk, helpline x 4971, 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:
1.1) Patients with diabetes should ensure that they maintain acceptable glycaemic control (HbA1C < 69mmol/mol) in the months and weeks prior to surgery, as this improves the healing process post-operatively.
1.2) 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.
1.3) 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.
1.4) 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 (before surgery)
2.1) Patients with diabetes should be, wherever possible, scheduled first on the operating list, in order to avoid unnecessarily long starvation periods.
2.2) 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 (DSN Blp 6236 or SpR Blp 7778), the anaesthetic team or both.
2.3) 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.
2.4) If VRIII is needed, then an intravenous cannula should be inserted and VRIII should be initiated either by 8 AM for morning operations or 12pm for afternoon procedures.
2.5) If glucose is temporarily stopped, e.g. en route to theatre, insulin must also be stopped temporarily.
2.6) 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.

2.7) 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:


2.8) No patient should be remain on VRII for more than 48 hours without discussion with diabetes team.

3) Post –operative management

3.1) 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.

3.2) Patients for whom VRII is been initiated should have their first meal following surgery while VRII continues, to check that meal is tolerated.

3.3) Usual insulin regimen should be re-started according to the VRII protocol and pre-operative guidance on prescribing anti-diabetic medications.

3.4) 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.

3.5) Usual anti-diabetic medications apart from insulin should be restarted as soon as patient is eating and drinking.
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 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

**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-ental 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. Complex patients can be discussed with any member of the Nutrition Support Team (NST), see contact details below. All referrals should be made before midday, weekend referrals must be made prior to midday on a Friday. All referrals should be made in a timely manner to allow an assessment to take place before the noon deadline to order PN. PN will not be instigated outside weekday working hours or at weekends except on a pre-planned basis.

**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.

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. Advice can be sought from the PN pharmacist (bleep 7554) or the on-call pharmacist (bleep 6267) for any questions or concerns, including cold chain storage.

**Contact details**

Any team member may be contacted during standard working hours via:

Dr Penny Neild, Consultant Gastroenterologist (x3429) Dr Sophie Barker, Consultant Gastroenterologist (x3750) Dr Jamal Hayat, Consultant Gastroenterologist (x3569); Tracey Marshall, Emma Pindard & Kirsty McCartney, nutrition nurse specialists (blp 8050/8498); the Gastroenterology SpR (blep 6590); Susan Spollen Home PN Pharmacist (blep 6052)/ Ben Blackburn PN Pharmacist (blep 7554); Alison Green, Specialist GI Surgery and Nutrition Team Dietitian (blep 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.
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:

I. 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.

II. 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.

III. 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.

IV. 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:
   a. Order daily U+E’s including potassium, phosphate and magnesium.
   b. Correct any electrolytes as required based on the below criteria.
   c. Monitor blood glucose levels every 4 hours until stable.
   d. 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 refeeding (see below)

Guide to electrolyte supplementation

<table>
<thead>
<tr>
<th>Potassium</th>
<th>Serum Levels</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.5-4.7mmol/L</td>
<td>Higher than 3mmol/L</td>
<td>Asymptomatic 2.5-3 mmol/L</td>
<td>Less than 2.5mmol/L or asymptomatic</td>
<td></td>
</tr>
</tbody>
</table>

Patient on intravenous therapy only
See Trust potassium supplementation guidelines (see Hypokalaemia)

Patient able to tolerate oral or enteral therapy
**NB: Use i.v. therapy if patient has diarrhoea or high output stoma**

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<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>
<td></td>
</tr>
</tbody>
</table>
### 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 on intravenous therapy only</td>
<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 specialist advice prior to supplementation. Not appropriate for patients with high plasma sodium. Not suitable for fluid restricted patients. Do not recheck phosphate for at least 6 hours post infusion (to allow for distribution). Halve dose in renal impairment or for patients less than 40kg.</td>
<td></td>
</tr>
<tr>
<td>Patient able to tolerate oral or enteral therapy <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 as above.</td>
<td></td>
</tr>
</tbody>
</table>

### 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 100mls sodium chloride 0.9% over at least 3 hours</td>
<td>20mmol Magnesium sulphate in 250mls sodium chloride 0.9% over at least 6 hours</td>
</tr>
<tr>
<td>Patient able to tolerate oral or enteral therapy <strong>NB: Use i.v. therapy if patient has diarrhoea or high output stoma.</strong></td>
<td>Magnesium glycerophosphate 4mmol tablets/solution (unlicensed product, needs form to be completed by doctor) Two tablets three times a day (24mmol)</td>
<td>Not appropriate. See i.v. therapy</td>
</tr>
</tbody>
</table>

### Calcium

<table>
<thead>
<tr>
<th>Serum Levels 2.15-2.5mmol/L</th>
<th>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 enteral therapy</td>
<td>Sandocal “400” tablets containing 10mmol of calcium per tablet</td>
</tr>
</tbody>
</table>

**NB:** Calcium must not be added to enteral/parenteral feeds due to stability issues. Please contact ward dietitian for advice.
**Pabrinex®/Thiamine**

Replace Thiamine at least 30 minutes prior to starting feeding (Stanga 2007)

<table>
<thead>
<tr>
<th>Patient on intravenous therapy only</th>
<th>Moderate risk of refeeding (See criteria in Adult Nutrition Support Policy, Appendix C)</th>
<th>High risk of refeeding (See criteria in Adult Nutrition Support Policy, Appendix C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High potency vitamins B &amp; C (Pabrinex®) IV injection</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Give ONE pair of ampoules ONCE a day for two days.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Administer in 100mls of sodium chloride 0.9% over at least 30 mins.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Please refer to Trust Smart Pump preparation guide if required</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Patients with severe high risk of refeeding syndrome can be given 2 pairs up to 3 times a day.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Please refer to NST for advice on these patients</td>
<td></td>
</tr>
<tr>
<td>Patient able to tolerate oral or enteral therapy</td>
<td>Thiamine 100mg, three times daily for 10 days with a multivitamin once daily until on full rate feeding.</td>
<td>High strength vitamins B &amp; C (Pabrinex®) Injection, as above Followed by oral thiamine 100mg three times a day and a multivitamin once daily for a total of 10 days.</td>
</tr>
</tbody>
</table>

**Multivitamin Supplementation**

Patients at high risk of re-feeding syndrome or those with prolonged poor nutritional intake may require additional supplementation as sub clinical deficiency is likely. This is on top of thiamine replacement already described above.

<table>
<thead>
<tr>
<th>Oral</th>
<th>Enteral</th>
</tr>
</thead>
<tbody>
<tr>
<td>One Sanatogen A to Z® tablet once daily</td>
<td>Abidec® liquid 1.2mL once daily</td>
</tr>
<tr>
<td>If severely high risk and on small amounts of enteral or parenteral nutrition discuss requirement for additional parenteral vitamin and mineral supplementation with dietitian or pharmacist</td>
<td></td>
</tr>
</tbody>
</table>

**Nutritional Treatment**

The regimen below outlines an out-of-hours nutrition plan.

On-call Dietitians are available on blp 8169, Saturdays and Sundays, 08.30 – 1630

*NOTE: Within working hours refer the patient to the Dietitian on bleep 7703*

*If out-of-hours make the referral the following working day.*

**Oral Re-feeding targets:**

A daily meal plan is as follows which provides an increased calorie intake every 2 days:

- **Day1-2:** ½ breakfast, yoghurt, ¼ lunch, yoghurt, ¼ supper = 550-600kcal
- **Day3-4:** ½ breakfast, yoghurt, ½ lunch, yoghurt, ½ dinner = 750-800kcal
- **Day 5:** ½ breakfast and glass of milk, yoghurt, ½ lunch, rice pudding, ½ supper, yoghurt = 1000kcal

Portion size to be checked by 1-1 nurse and not the patient

All food intakes should be recorded in the Food Record Charts by the patient’s named nurse

Each meal must contain a source of carbohydrate

Use phosphate rich foods e.g. milk, yoghurt

Allow the patient 3 specific food dislikes (individual foods not food groups e.g.: can dislike lamb but not all meat, or can dislike potatoes but not all starchy foods)
If patient is not eating meals the following sip feeds are appropriate:
Fortisip Compact 300 calories, 37 g carbohydrate
Fortisip Yoghurt Style 300 calories, 37 g carbohydrate
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: 500mls Nutrison 1.0 as 25mls per hour x 20 hours. Rest 4 hours and repeat on day 2.
Day 3: 800mls 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. Repeat day 2.
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.
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 $\text{H}^+$ (>43nmol/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 150mmol/L. When the 4 major plasma electrolytes (sodium, potassium, chloride and bicarbonate) are considered the sum of $[\text{Na}]^+ + [\text{K}]^+$ is greater than $[\text{Cl}]^- + [\text{HCO}_3^-]$ by 8-17mmol/L. This difference is described as the ‘anion gap’, with the difference 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, i.e 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**

### Normal anion gap:
- Loss of $\text{HCO}_3^-$, as in diarrhoea, proximal renal tubular acidosis
- Decreased renal acid excretion, *eg.* distal renal tubular acidosis

### Increased anion gap:

<table>
<thead>
<tr>
<th>Condition</th>
<th>Unmeasured Anions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lactic acidosis</td>
<td>Lactate, phosphate, urate</td>
</tr>
<tr>
<td>Ketoacidosis</td>
<td>Ketone bodies (acetone, acetoacetate, β–hydroxybutyrate)</td>
</tr>
<tr>
<td>Diabetic</td>
<td>Acetoacetate, β–hydroxybutyrate</td>
</tr>
<tr>
<td>Starvation</td>
<td>Anoence</td>
</tr>
<tr>
<td>Inborn enzyme defects</td>
<td></td>
</tr>
<tr>
<td>Intoxication</td>
<td></td>
</tr>
<tr>
<td>Methanol</td>
<td>Formate</td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td>Glycolate, oxalate</td>
</tr>
<tr>
<td>Ethanol</td>
<td>β–hydroxybutyrate, lactate, acetoacetate</td>
</tr>
<tr>
<td>Salicylates</td>
<td>Ketones, lactate, salicylate</td>
</tr>
<tr>
<td>Paraldehyde</td>
<td>Acetate</td>
</tr>
<tr>
<td>Uraemia</td>
<td>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
- In patients with lactic acidosis it is important to establish the reason for lactate accumulation (eg. cardiovascular compromise, ischaemic bowel) and to initiate resuscitation accordingly.
- Patients with normal anion gap metabolic acidosis secondary to profound diarrhoea or renal tubular acidosis should be treated with NaHCO₃ 1.4%.

When treating (reducing) the anion gap remember:
- Co-existing respiratory disease may lead to an inappropriately severe acidaemia and attention must be directed to the respiratory tract. The patient may even need ventilation.
- In a patient with a metabolic acidosis associated with a normal anion gap, measurement of urine pH should help distinguish between renal and non-renal causes. If the cause is renal the urine pH will be ≥5.4.
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 hsematoma
- 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 contraindicators. Correct and recheck 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).
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 are performed.

HEPARIN
Monitoring heparin therapy.

i) Low molecular weight heparin
Patients receiving low molecular weight heparin (eg dalteparin) therapy do not routinely require monitoring. However, monitoring using the anti Xa heparin assay should be carried out in:
- patients with a creatinine clearance below 30 ml/min
- patients weighing >100kg
- women who are pregnant on therapeutic doses

Initially, weekly measurement is advised. The anti Xa assay (measured 4 hrs after injection) should be 0.3 – 0.5 units/mL for prophylaxis, or 0.5-1.0 units/mL for treatment of acute venous thromboembolism.

Where therapeutic anticoagulation is required in the presence of a high bleeding risk, recent major surgery or severe renal impairment, 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 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 injection IV.
2. In patients at risk of bleeding consider omitting bolus dose.
3. Draw 25,000units of unfractionated heparin made up to 50mL with 0.9% sodium chloride (resulting concentration of 500units/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>

5. Repeat APTT ratio daily while on unfractionated heparin. Check platelet count at start of therapy and after 5 days. Where therapeutic dose subcutaneous unfractionated heparin (eg. 24 hour dose of 18 units/kg/hour given in two divided doses as, e.g 12,500units sc bd for a 58 kg patient) is used, it should be monitored 6 hrs after injection.
and then daily with the APPT ratio; 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, 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. 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 a heparin interaction with 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 ALL doses including that in IV fluids and cannulae, e.g flushes. Alternative anti-thrombotic agents such as argatroban or danaparoid 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 at least 2 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 review.
If INR >8.0 with no bleeding, give 0.5–1.0 mg Vitamin K orally and repeat INR in 24-48 hours. Omit warfarin until INR <4.0. The cause of the elevated INR should be investigated.

**IM injections should be avoided in patients taking anticoagulants**

**Minor haemorrhage, e.g. haematuria, epistaxis.** Reduce dose or, if INR >4.5, withhold warfarin for one or more days. If INR>8.0 give Vitamin K (phytomenadione) 0.5 to 2.0 mg orally (the IV preparation of Konakion can be given by mouth). Repeat INR in 24-48 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.

**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: 30units/kg

The single dose should not exceed 3000 units. FFP (15mL/kg) should only be infused if PCC is unavailable. Do not re-start warfarin until bleeding is controlled.

Repeat INR after 6 hours and after 24 hours; discuss with Haematology if bleeding persists. Further administration of Vitamin K may be necessary after 24 hours.

OTHER ANTICOAGULANTS

Fondaparinux

Protamine has no effect on the anticoagulant effect of fondaparinux.
Recombinant factor VIIa (NovoSeven) has been used with some success.
Contact the on-call haematology registrar (bleep 6068 or via switchboard out of hours).

Rivaroxaban, dabigatran and apixaban

The NOAC bleeding pathway should be followed:
file:///\stg1nas01\formulary\NOAC%20Bleeding%20Pathway.pdf

There are no reversal agents available for the novel oral anticoagulants. If there is bleeding associated with a NOAC:
• Stop NOAC and inform the haematologist (bleep 6068 or via switchboard out of hours)
• Document the time of last dose of NOAC
• Check FBC, coagulation screen, renal function and arrange group and save

Mild bleeding: Minor bleeding may only require withholding one or two doses of the drug. Tranexamic acid 1g po/iv should be considered.

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 10⁹/l or CNS bleed aim PLT > 100 x 10⁹/l). Consider tranexamic acid 1g po/iv. Consider PCC 25-50 units/Kg). Dabigatran is potentially dialysable and this should be considered. 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 6311 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:
APPENDIX 8

HIV POST-EXPOSURE PROPHYLAXIS
Link consultant: Dr Phillip Hay

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 needlestick 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 needlestick 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.

Initial management
If the site of exposure is a wound or non-intact skin, liberally wash (but not scrub) with soap and water. Gently encourage any free bleeding. If exposed area is mucous-membrane, copiously irrigate with water (if conjunctiva, and contact lenses are worn, irrigate before and after they are removed).

Treatment following exposure to a known or high-risk source:
- should preferably be started within an hour of exposure, although it may still be effective if started up to 48 hours after exposure;
- involves taking a 4-week course of a combination of three drugs: Truvada (tenofovir 300mg plus emtricitabine 200mg), one tablet once a day; and Raltegravir 400mg, one tablet twice a day. If the source patient is known, other combinations may be more appropriate – seek advice;
- is complicated if the person exposed is pregnant – seek advice.

Emergency 5-day packs containing Truvada and Raltegravir for symptom relief, are kept by Staff Health, Pinckney Ward, McEntee Ward, A&E, Courtyard Clinic and Courtyard Pharmacy.

Post exposure prophylaxis after sexual exposure (PEPSE) can be offered in the same way if the partner is known to be HIV positive.

There is no epidemiological evidence relating to the use of post-exposure prophylaxis against HIV following rape (male or female) or failed barrier contraceptive methods. Individual cases should be clinically assessed. Contact A&E who will seek advice from the Courtyard Clinic or Clinical Infection Unit.

In the event of exposure of staff,
- during working hours, seek advice immediately from Staff/Student Occupational Health (8.30am-5pm, Monday to Friday; ext.1661- unit 1; ext.2663 – unit 2)
- if out-of-hours, attend Accident & Emergency. Inform triage nurse that you must be seen immediately. Staff at AMH should contact the duty doctor on Kent Ward.

WORKING WITH AIDS PATIENTS
Patients infected with HIV present either with symptoms of an HIV-associated disease, or with a coincidental, unrelated problem but requiring precautions because of their HIV
status. In this country there is a low incidence of HIV infection except in the following at-risk groups:

1. gay and bisexual men
2. haemophiliacs
3. IV drug users
4. heterosexual men and women who have partners from areas of high prevalence eg sub Saharan Africa, SE Asia and parts of the Caribbean
5. sexual partners of the above (and children of 3 & 4)

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 various 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.

Any patient in an at risk group presenting with any of the above should be referred to the on-call resident CIU SHO/SR.

Doctors and nurses caring for HIV-infected patients are at risk of contracting the disease if a patient’s blood or body fluids penetrates their skin or mucous membranes. Simple precautions such as wearing gloves when taking blood or dealing with a wound etc are usually all that are necessary.

The use of masks and goggles is needed when blood may be sprayed or aerosolised from an injured infected patient. The virus is very fragile outside the body and is inactivated by simple detergent solutions or ordinary sterilising measures, such as hypochlorite.

Advice on emergency prophylactic treatment following exposure to contaminated blood or body fluids is given earlier in this Appendix.

Outpatients who are seeking advice on HIV can be referred to the Courtyard Clinic, Department of Genito-urinary Medicine to speak to a Health Adviser (ext 3342 or 3353), and if they wish, to proceed to HIV testing. This is a free and confidential service which is available each weekday.

Inpatients who want or need an HIV blood test require pre-test counselling. This can either be arranged with a Health Adviser or through the CIU team.

Newly diagnosed HIV-infected patients should be referred to an HIV clinic. These are held each weekday in the Courtyard Clinic – ring ext 3140 to make an appointment.
APPENDIX 9

FIRST STEPS IN THE EVENT OF A MAJOR INCIDENT

Link consultant: Dr Phil Moss

St George’s Healthcare has a statutory duty to be prepared to deal with a major incident, broadly defined as any incident that results in the hospital having to handle numbers and/or types of patients in an emergency over and above those that can be managed by the A&E staff and normal intaking arrangements.

The Major Incident Plan may be activated by the London Ambulance Service (LAS) or the South East Coast Ambulance Service (SECAM), or alternatively a major incident may be declared by the Director On-call following consultation with A&E consultant (or deputy) or by the Nurse in Charge of the A&E department. As a major trauma centre St George’s is more likely to be involved in a major incident as the Ambulance Services strategy, in the event of a major incident, will be to fully utilise all major trauma centres first, before placing major trauma patients into trauma units, irrespective of the location of the incident.

- The initial response by the hospital to a major incident will be coordinated from the Hospital Control Centre, by a team led by a Medical Coordinator (Senior Medical Lead consultant), Nursing Coordinator (Clinical Site Manager or nominated senior nurse), and Incident Coordinator (General Manager on-call). The Hospital Control Centre (HCC) is located in the A&E Seminar Room, accessible only via the stairs in the back corridor of the A&E department, ground floor St James Wing (room 0.6.65). A control room will also be set-up in the Community; the location is dependent on the nature of the incident but options currently include Queen Mary’s Hospital, Roehampton or co-located with the London Borough of Wandsworth.

- ST GEORGE’S HOSPITAL ACCESS. During an incident, whatever its nature, all staff entering and leaving the hospital should do so via the Lanesborough Wing main entrance with their Trust ID card (other entrances may be locked). All patients involved in the incident should enter via the ambulance entrance to Emergency Department (ED). If there is a suspected chemical, biological or radiation hazard, the hospital and Emergency Department will be made ‘secure’. In these circumstances patients coming from the incident will be corralled outside the Emergency Department; a decontamination area with ‘clean’ and ‘dirty’ areas established; and decontamination facilities set up by ED staff with appropriate personal protective equipment. No patient who has been exposed to a chemical or biological hazard will be allowed into the Emergency Department until after they have been decontaminated. If patients potentially contaminated with radioactive material require admission to the Emergency Department or theatres for life-saving treatment, special decontamination precautions will be taken. All patients who leave ED must do so via the doors at the back of the department (opposite the St James’ Wing lifts), so that they pass an exit control point. A Discharge Area for patients discharged from ED will be located in the Neurosciences Outpatient area, Ground Floor Atkinson Morley Wing, ext 0634. A Relatives’ Information Centre (for friends and relatives) will be located in the Outpatient area, Ground Floor Atkinson Morley Wing, ext 4137.

- ASSEMBLY POINTS FOR CLINICAL STAFF. During normal working hours, on-call medical staff should stop what they are doing with as much urgency as is practical and report to A&E reception, where they will be supplied with a copy of their Action Card, appropriate identification and if necessary a radio. They will be asked to wait in
the waiting area until they are required to help treat patients. Staff not on-call should continue with their normal work unless contacted and asked to do otherwise.

Outside normal working hours, all on-call medical staff should report to A&E reception, where they will be supplied with a copy of their Action Card, appropriate identification and if necessary a radio. They will be asked to wait in the waiting area until they are required to help treat patients. Doctors not on-call who wish to come in should go in the first instance to the Waiting Area, A&E department, Ground Floor St James Wing.

- The assembly area for registered volunteers and medical students will be the John Parker Lecture Theatre, Atkinson Morley Wing (ext 1542).

- TRIAGE ARRANGEMENTS. A triage point will be established inside the ambulance entrance to A & E. Within the A & E department adult patients will be triaged as follows:

<table>
<thead>
<tr>
<th>PRIORITY</th>
<th>DESCRIPTION</th>
<th>COLOUR</th>
<th>AREA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Immediate</td>
<td>Red</td>
<td>Resuscitation Room</td>
</tr>
<tr>
<td>2</td>
<td>Urgent</td>
<td>Yellow</td>
<td>Majors</td>
</tr>
<tr>
<td>3</td>
<td>delayed (ambulatory)</td>
<td>Green</td>
<td>Urgent Care Centre</td>
</tr>
<tr>
<td>4</td>
<td>Expectant</td>
<td>Blue</td>
<td></td>
</tr>
</tbody>
</table>

Children will be triaged as above. In the event of a major incident involving significant numbers of children the dedicated paediatric area in A&E is likely to be used – this decision will be made dynamically by the A&E Consultant. The A&E Consultant will nominate senior A&E clinician to take responsibility for each of the clinical areas in the Accident & Emergency Department:

- Resuscitation Room
- Majors
- Minor casualties’ area located in the Urgent Care Centre

Paediatrics area - In the event of a major incident involving significant numbers of children the dedicated paediatric area in A&E is likely to be used. The A&E Consultant will also

- allocate teams of doctors and nurses to patients in the treatment areas of the Accident & Emergency Department.
- allocate on-call doctors who report to Accident & Emergency Department Reception to assist with clearing patients from Accident & Emergency.
- Work with the Theatre Co-ordinator (consultant anaesthetist on call for St James Wing) to co-ordinate the work in theatres.

- ADMISSION WARDS. The designated admission ward for all adults from a major incident – except those who go direct to theatres or ITU – will be Richmond Acute Medical Unit (ext 3299). Vernon Ward (ext 3197) will provide a second admission ward if required. For children, Jungle Ward will be used (ext 2034/2035).

- COMMUNITY SERVICES, INCLUDING QUEEN MARY’S HOSPITAL.

The focuses of the Community Services division will be to:

- ensure that community based services including Minor Injuries Unit at Queen Mary’s Hospital Roehampton continue to run as normally as possible to prevent further pressure on the Accident & Emergency Department at St. George’s Hospital
- maximise the availability of beds at Queen Mary’s Hospital by appropriately transferring patients to the community and creating additional bed capacity within Brysson Whyte Rehab Unit gym.
- Any request for Rest Centre nursing from the local authority can be met.
GUIDE TO THERAPEUTIC DRUG LEVEL MONITORING

**Gentamicin**  
*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**  
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 10-15 micrograms/mL, although levels of 15-20 micrograms/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**  
Sampling time: immediately prior to next dose; therapeutic range: single therapy 4-12 mg/L, multiple therapy (i.e. one or more drugs used in addition to carbamazepine) 4-8 mg/L; time to steady state: 2-4 weeks after start of treatment, or 4-5 days after dose change.

**Digoxin**  
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**  
Sampling time 12 hours post dose; therapeutic range: as treatment - 0.4 –1.0 mmol/L, as prophylaxis - 0.5-0.8 mmol/L; time to steady state: 3-7 days.

**Phenytoin**  
Sampling time: immediately prior to next dose; therapeutic range: 5-20 mg/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: 10-20 mg/L; time to steady state: 2 days.

1, 2 *Advice on these products can be obtained from:*

1 *Microbiology, ext 5685/6 or bleep 480 or via switchboard out of hours.*

2 *Chemical Pathology, bleep 6032 or pager SG 138 or via switchboard out of hours.*
GENTAMICIN DOSING GUIDELINES (once daily dosing)

Exclusions:
- Endocarditis (see endocarditis treatment guidelines)
- Paediatric patients (see local guidelines)
- Patients with ascites >10% body weight
- Burns >15% BSA
- Pregnancy: contact – microbiology to discuss the use of gentamicin; pharmacy for dosing advice
- Dialysis patients - give 1mg/kg (max 100mg) and await levels <2.0mg/L before re-dosing
- Patients allergic to gentamicin or other aminoglycosides
- Patients with severe sepsis or those on ITU – 5mg/kg may be used regardless of renal function – seek senior advice.

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:

\[
\text{Creatinine clearance} = \frac{(140 - \text{age}) \times \text{weight}^\# (\text{kg})}{\text{Serum Creatinine} (\mu\text{mol/L})} \times 1.04 \text{ for females OR } 1.23 \text{ for males}
\]

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) or cap weight at 100kg.

Table 1. Dosing regimen

<table>
<thead>
<tr>
<th>CrCl</th>
<th>&gt;60 ml/min</th>
<th>&gt;60 ml/min</th>
<th>40 - 60 ml/min</th>
<th>10 - 40 ml/min</th>
<th>&lt;10 ml/ min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient age</td>
<td>&lt;65 yrs</td>
<td>≥ 65 yrs</td>
<td>All patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dose</td>
<td>5 mg/kg</td>
<td>5 mg/kg</td>
<td>4 mg/kg</td>
<td>3 mg/kg</td>
<td>2 mg/kg</td>
</tr>
<tr>
<td>Take levels</td>
<td>18-24 hrs</td>
<td>18-24 hrs</td>
<td>18-24 hrs</td>
<td>18-24 hrs</td>
<td>48 hrs post 1st dose</td>
</tr>
<tr>
<td>post 1st dose</td>
<td>post 1st dose</td>
<td>post 1st dose</td>
<td>post 1st dose</td>
<td>post 1st dose</td>
<td>dose</td>
</tr>
<tr>
<td>Timing of 2nd dose</td>
<td>24 hrs post 1st dose</td>
<td>Await levels &lt; 1 mg/L before re-dosing</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3) Administer in 50ml sodium chloride 0.9% over 30 minutes
4) Take levels at the time indicated in Table 1 (remember to document the sampling time on the blood form)
5) Give 2nd dose without waiting for levels in patients <65 yrs with good renal function. Await levels <1mg/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; 1.0 mg/L</td>
<td>Continue current dosing regimen</td>
</tr>
<tr>
<td>1-2 mg/L</td>
<td>Recheck levels 12 hrs later – withhold dose pending results</td>
</tr>
<tr>
<td>&gt; 2 mg/L</td>
<td>Recheck levels 24 hrs later – withhold dose pending results</td>
</tr>
</tbody>
</table>

7) Do not give more than 72 hours of gentamicin without microbiology approval – prolonged courses are rarely necessary and increase the risk of toxicity. Daily serum creatinine & urea is recommended for patients on IV gentamicin. 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. Contact microbiology or pharmacy for advice.

**VANCOMYCIN DOSING GUIDELINES (intermittent)**

These guidelines are designed to achieve trough levels of 10 to 15 mg/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)
- 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>
<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>Infusion period</td>
<td>Max rate 10mg/min</td>
<td>Max rate 10mg/min</td>
<td>Max rate 10mg/min</td>
</tr>
</tbody>
</table>

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

\[
\text{Creatinine clearance (ml/min)} = \frac{(140 - \text{age}) \times \text{weight (kg)}}{\text{Serum Creatinine (µmol/L)}} \times \begin{cases} 1.04 & \text{for females} \\ 1.23 & \text{for males} \end{cases}
\]

3) Give the 1st maintenance dose specified in Table 2 after the dosing interval specified in Table 2 below.
Table 2. Initial Maintenance Dose

<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 4th dose</td>
</tr>
<tr>
<td>90-110</td>
<td>1.25g</td>
<td>12 hours</td>
<td>250mL</td>
<td>Max 10mg/min</td>
<td>Before 4th 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 4th 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 4th 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 4th 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 3rd 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 3rd 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 2nd dose</td>
</tr>
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</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 3. Trough level interpretation & maintenance dose adjustment when levels 10-15mg/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 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.
**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>
<th>Efficacy</th>
<th>Safety</th>
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<tbody>
<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 Peak 50-100 mg/L</td>
<td>Peak &lt;100mg/L</td>
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<tr>
<td>Itraconazole</td>
<td>Routine during first week of therapy, lacking response, gastrointestinal dysfunction, co-administration of interacting medication. Weekly during prophylaxis, potential drug toxicity</td>
<td>5-7</td>
<td>For prophylaxis; Trough ≥0.5mg/L For treatment; Trough ≥0.5–1.0mg/L</td>
<td>N/A</td>
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<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>
<td>Trough &lt;5.5mg/L</td>
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<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) For treatment; Trough &gt;1.0 mg/L</td>
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<td>Medicine F1</td>
<td>Bleep</td>
<td>Surgery F1</td>
<td>Bleep</td>
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<td>Dr Clark &amp; Dr Forton</td>
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<td>6364/6300/</td>
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<tr>
<td>Dr Bourke, Dr Kiely &amp; Dr</td>
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<td>Mr Wan</td>
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<td>Mr Melville</td>
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<td>Drs Groves, Pollok, Poulis</td>
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<td>Mr Hagger</td>
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<tr>
<td>Dr Patel &amp; Dr Simmgen</td>
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<td>Mr Mokbel</td>
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<td>Prof Baker, Dr Chua &amp; Dr</td>
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<td>Mr Sharma &amp; Mr Banerjee</td>
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<td>Dr Ong &amp; Dr Draper</td>
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<td>Dr Antonios &amp; Dr Khong</td>
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<td>Miss Daly (orthopaedics)</td>
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<td>Dr Cottee &amp; Dr Martin-Marero</td>
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## BLEEP AND PHONE NUMBERS cont.

### Specialty SpR Referrals

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### Nurse Bleeps

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### Venous Access

(Ext 7199)

### Other (Extensions)

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### DEPARTMENTS

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FEBRUARY 2015

PLEASE DISCARD WHEN 63rd EDITION IS PUBLISHED IN
AUGUST 2015