St George's University of London

St George's Healthcare NHS Trust

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

St George's Hospital

August 2011

55th edition

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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.

- **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 team: Teck Khong (editor: tkhong@sgul.ac.uk), Vivien Perkins, Jo Harding, Lisa Britton

ADULT CARDIAC ARREST – extracted from RCUK guidelines*
***available to download free: http://www.resus.org.uk/pages/iResusDt.htm**
(Link: Paula McLean, Resuscitation Service Manager EXT 1648)

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 charges, 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):

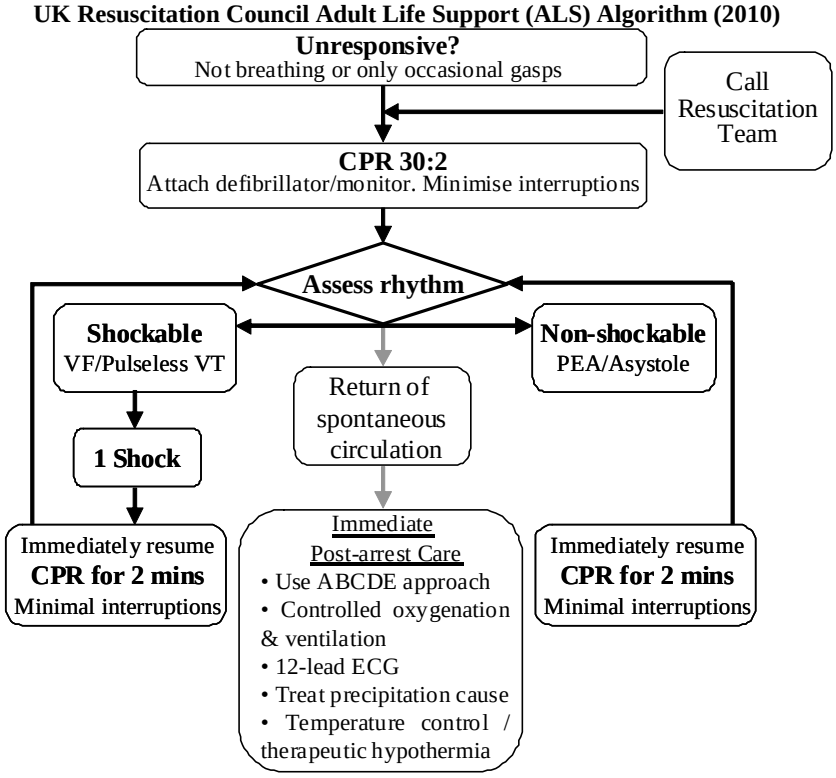
- Check a central pulse and end-tidal carbon dioxide [ET CO_2] trace if available
- If there is evidence of ROSC, start post-resuscitation care (induced hypothermia and primary percutaneous coronary intervention (PPCI) should be considered).
- If no signs of ROSC, continue CPR and switch to the non-shockable 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 CO_2]). 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)



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), intra-cranial haemorrhage, aortic dissection, acute coronary syndromes (unstable angina/acute myocardial infarction), acute left ventricular failure with pulmonary oedema, or pre-eclampsia/eclampsia. The initial aim of treatment is to lower blood pressure in a rapid (within 2-6 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 nitoprusside 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.*** Patients should be admitted to a medical bed and blood pressure reduced slowly; ideally 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 3341 or blp. 7961/6045/6602).

MANAGEMENT OF ACUTE CORONARY SYNDROMES (ACSs)

Link consultant: Dr Nicholas Bunce

All patients arriving at the hospital with chest pain suggestive of myocardial ischaemia (central or retrosternal pressure, tightness, heaviness, radiating to neck, shoulder orjaw, 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 (NST**E**-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, NST**E**-ACS or neither.

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

- ST elevation of ≥ 0. **2mm** in leadsV1-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.
- 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

Link consultant: Dr Nicholas Bunce

Refer the patient immediately to Cardiology for Primary Percutaneous Intervention (1° PCI); the target door-balloon time is within 90 mins. 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.

Clopidogrel As soon as possible give clopidogrel 600mg. This should be followed by clopidogrel 75mg 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 repiratory failure controlled oxygen should be administered if oxygen saturation **(SpO_2) is <94%. Target SpO_2 94-98%**. In patients with chronic obstructive pulmonary disease and who are at risk of hypercapnic respiratory failure the target **SpO_2 is 88–92%** 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 All patients with STEMI with a known history of diabetes mellitus or a blood glucose >11.0mmol/L should be managed with tight glycaemic control - but avoid hypoglycaemia. 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).

If blood glucose >11.0mmol/L, use a background infusion of 5% glucose at 50-100ml/hr. Additionally, using a solution of 50 units Actrapid in 50mls 0.9% sodium chloride (1unit/ml), titrate on a sliding scale as below:

BLOOD GLUCOSE	***ACTRAPID INFUSION RATE (1 unit/ml)***
BM >17	**5mls/hr**
BM 14 – 17	**4mls/hr**
BM 10 – 13.0	**3mls/hr**
BM 6 – 9.9	**2mls/hr**
BM <6	**1ml/hr**

After 24 hours convert to sc insulin or oral anti-diabetic medication as appropriate.

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 rampiril 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 rampiril 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.

MANAGEMENT OF NSTE-ACS
Link consultant: Dr Nicholas Bunce

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.
- Give clopidogrel 300mg 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 on page 8).

- 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 80 mg) in patients with confirmed NSTE-ACS. Reduce dose or use Pravastatin 40mg od in patients receiving interacting drugs (eg 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, page 8) 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)

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, 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).

MANAGEMENT OF ACUTE HEART FAILURE

Link consultant: Dr Lisa Anderson

Community heart failure nurse follow-up reduces the 3 month risk of readmission by 35%. Please contact heart failure nurse specialists (blp7376/ x.4404) when patients are admitted.

DIAGNOSIS

Heart failure is one of the commonest medical admissions (up to 5%) and one in seven people >85y 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.

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

If the NTproBNP is normal (below 300ng/L), search for an alternative diagnosis. If the NTproBNP is significantly elevated (see above) acute heart failure is likely and should be confirmed by echo if not already documented. If the NTproBNP concentration is intermediate (above 300ng/L but below acute heart failure levels), reconsider the diagnosis. If, after reassessment, ventricular failure is likely, request an echo.

Heart failure echo requests

1. NTproBNP level must be documented on the request form.
2. If significant LV impairment is known a repeat echo is not necessary unless a new lesion (such as new murmur) is being investigated.

MANAGEMENT - Initial treatment

Acute pulmonary oedema:

- O_2 to maintain SaO_2 (95-98%)
- IV GTN infusion (10-200micrograms/min) - titrate to highest tolerable dose (systolic BP 90-100mmHg)
- IV furosemide 40-100mg bolus followed by an infusion at 5-20mg/h if required
- IV morphine as a 2.5mg bolus can be given if patient is distressed/in pain, and repeated as necessary until symptoms are controlled
- CPAP (with intubation if respiratory failure develops and appropriate for the patient)

General measures

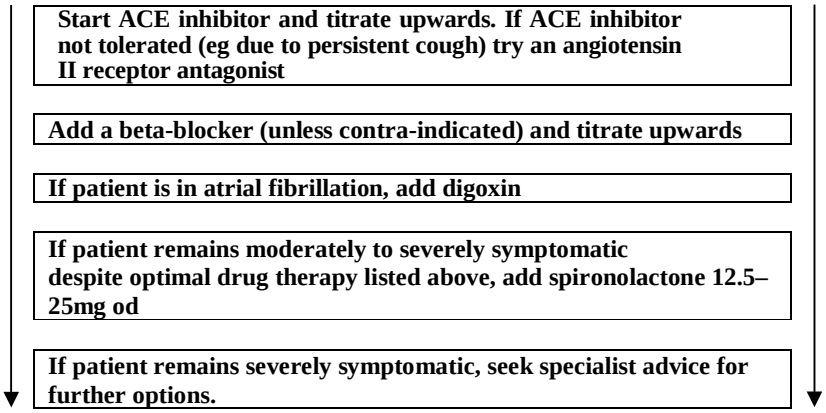
- *Monitor* ECG continuously; check oximetry and BP every 5 mins. If cardiogenic shock develops, contact cardiology SpR immediately.
- *Review* medication: stop Ca^{2+} channel blockers and NSAIDs where possible. In unstable patients with diabetes, switch to sliding scale insulin.
- *Request* chest X-ray; plasma U&E's, creatinine, FBC, TFTs, LFTs, NTproBNP, troponin, glucose and lipids; arterial blood gases if in respiratory distress; urinalysis; peak flow or spirometry.
- *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.

Follow-up treatment

Once the patient is stable, start 'long-term' therapy. For those with isolated right ventricular failure, the key objective is to maintain optimal diuretic therapy. For those with left ventricular impairment, follow the management of chronic heart failure algorithm below.

Management of chronic heart failure

Diuretics are used for the relief of congestive symptoms and fluid retention in patients and should be titrated (up and down) according to need, following the initiation of heart failure therapies:



DISCHARGE AND FOLLOW-UP

- There are now heart failure nurse teams in all local districts and any patient admitted with acute decompensation of heart failure should be referred for early community follow-up to avoid re-admission (currently 36% at 3 months).
- If ACE inhibitors or beta-blocker doses have been reduced or discontinued during the admission, the reasons should be documented in the discharge summary.
- Record the patient's weight on discharge and the presence of any residual oedema at this weight.
- Follow-up arrangements should be clearly documented.

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 600-1200micrograms IV in the first instance. Persistent symptomatic bradycardia requires pacing (temporary or permanent). If temporary pacing is required, transvenous pacing under X-ray control is optimal. For advice, contact the Cardiology registrar on call.

ATRIOVENTRICULAR BLOCK

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

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.

Indications for Temporary Pacing – Emergency/Acute
Acute MI with: ▪ **Asystole** ▪ **Symptomatic bradycardia not responsive to atropine** ▪ **Bilateral bundle branch block (alternating BBB or RBBB with alternating LAHB/LPHB** ▪ **New or indeterminate age bifascicular block with 1st degree AV block** ▪ **2nd or 3rd degree AV block after an acute anterior MI**
Bradycardia not associated with acute MI: ▪ **Asystole** ▪ **Any symptomatic bradycardia resistant to medication** ▪ **2nd or 3rd degree AV block with haemodynamic compromise or syncope at rest** ▪ **Asymptomatic AV block with severe bradychardia (<30bpm) +/- QRS>120ms+/- QTc prolongation >500ms**
VT secondary to bradycardia eg Torsades de Pointes
Suppression of drug-resistant VT or SVT
Drug overdose, eg. digoxin, beta blockers, verapamil

Indications for Temporary Pacing – Elective
▪ **Support for procedures that may promote bradychardia** ▪ **General anaesthesia with: 2nd or 3rd degree AV block; intermittent AV block; 1st degree AV block with bifascicular block; 1st degree AV block and LBBB** ▪ **Cardiac surgery when epicardial pacing has failed** ▪ **Rarely considered for coronary angioplasty**

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). 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; 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 (200-360J) under sedation is the best therapy (if help needed contact the cardiac registrar for advice). Whatever method is used, full facilities for resuscitation should be to hand. Further cardiological assessment is mandatory in all cases not associated with acute ischaemia or infarction. Remember to check electrolyte levels. The administration of magnesium, initial dose 8mmol (4mL of 50%) may help when the arrhythmia is refractory.

NB: DO NOT TREAT A POSSIBLE VT WITH VERAPAMIL

Some patients presenting with ventricular arrhythmias who have an ICD implant may have received shocks from the device. The presence of an ICD does not prevent the use of

emergency defibrillation or cardioversion in the event of a cardiac arrest or compromising VT that has not responded to ICD therapy. 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. 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**, page 3).
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**, page 3)
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 Muriel Shannon

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, consult 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 and sent to the Ultrasound (US) Department – the study can then be done on the next inpatient list.

- A&E and out-patients seen during weekdays from 9am-5pm are assessed by the DVT nurse in Richmond Ward using the Outpatient DVT Investigation guidelines. If an ultrasound scan is indicated a time is arranged by ringing ext 1473; scans are done between 10-12am or 2-4pm. The patient is sent to US Department with a completed radiology request form, which must include the pre-test probability score (PTP) score. Details of the scoring system are available from the DVT nurse (blp7380;x1332), in A&E **and on the Anticoagulant web page on the Intranet** **http://stginet/Units%20and%20Departments/Haematology/ANTICOAGULATION/ANTICOAGULATION.aspx**

- A&E and out-patients seen after **4**pm or on weekends should be managed using Outpatient DVT Investigation guidelines and D dimer sent. The patients must be referred to the DVT nurse on the next working day. Referral to ultrasound will then be made if indicated. If on clinical grounds (history and examination) and the D-dimer result, the diagnosis is probable, give dalteparin according to schedule below before discharge and until seen by the DVT nurse.

Treatment

1. 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 once daily dalteparin and warfarin, using the Warfarin Dosing Chart. Those presenting to A&E will be re-assessed by the DVT nurse (blp 7380; x1332) with the results of the ultrasound and ambulatory treatment commenced. Patients at enhanced risk of bleeding (those with liver disease, peptic ulcer, alcohol abuse, hypertension, heart failure, recent major trauma, or on drugs that enhance warfarin's effects), 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 abuser, is demented, has a 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.

In patients in whom anticoagulants can be started immediately, warfarin tablets should be taken in a dose of 5mg daily with the aim of achieving an INR >2.
This 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, depends on body weight according to the following schedule:

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

The dose of dalteparin should be continued until the INR has been >2.0 for two consecutive days. If dalteparin is given for more than 5 days, assess renal function and if it is impaired, reduce dose or use unfractionated heparin. For patients at home the injections are usually self-administered, but may be given by a district or practice nurse.

2. If the initial compression ultrasound is negative, management will depend on an assessment of the likelihood of this being a false negative, and so whether the patient should have a further scan after 5–7 days. A scheme for this assessment, together with advice on the additional diagnostic tests that might be needed in this situation, are available from the DVT nurse. Out-of-hours advice can be given by the on-call haematology registrar.

The duration of anticoagulation varies and is summarised in the table above. For ward patients arrange for a Yellow Anticoagulant booklet to be issued by the Anticoagulant Clinic, and a follow-up appointment to be made with the Clinic before the patient is

discharged. **Details of discharge procedure is available on the Anticoagulant web page *(see previous page*)**. Draw the patient's attention to the common interactions with warfarin outlined in the booklet.

PRESENTING FEATURES	TARGET INR (range)	RECOMMENDED DURATION
Calf vein thrombosis - surgical (post-op), no risk factors	2.5 (2-3)	6 weeks and review
Calf vein thrombosis – non-surgical (post-op), no risk factors	2.5 (2-3)	3 months
Proximal DVT	2.5 (2-3)	6 months
DVT plus continued risk factors (eg. immobility, hypercoagulability)	2.5 (2-3)	Long term or until risk removed
Recurrent DVT, off warfarin	2.5 (2-3)	Long term
Recurrent DVT, on warfarin	3.5 (3-4)	Long term

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).

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_2 is 10.7 kPa or more)

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

Contraceptive pill	Malignancy/Cancer
Pregnancy or <6/52 post partum	Air travel >4hrs in previous 4/52
Surgery in last 4/52	Bedbound
Previous VTE	Family Hx of VTE
Known thrombophilia	Obesity
Smoker	IVDU

Use the Wells Score (*table below*) to assess clinical pre-test probability of PE.

Level of clinical risk	Total score
'High'	>6.0
'Moderate'	2.0 – 6.0
'Low'	<2.0

Risk factor	Score
clinical signs and symptoms of DVT (objectively measured leg swelling and pain with palpation in the deep-vein region)	3.0
heart rate >100 beats/min.	1.5
immobilisation (bed rest, except access to bathroom, for 3 or more days; or surgery in previous 4 weeks)	1.5
haemoptysis	1.0
Previously objectively diagnosed DVT or PE	1.0
malignancy (patients with cancer receiving treatment or treatment stopped within previous 6 months or receiving palliative care)	1.0
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	3.0

Investigations

All patients should have a CXR and ECG and measurements made of arterial blood gases (ABGs) and plasma D-dimer levels. Plasma D-dimer levels are useful in patients with low or moderate clinical risk**.** Depending on the results of these (see flow diagram), the patient may then need a V/Q scan. Those patients in whom a massive pulmonary embolism is suspected should also have measurements made of BNP and troponin.

The primary purpose of the CXR and ECG is to exclude other diagnoses. If the plasma D-dimer level (test) is below 0.3 mg/L, 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 any two of D-dimers, ABG and respiratory rate are normal, PE is very unlikely. In patients with a D-dimer level of <0.3mg/L and PaO_2 of ≥ 10.7 Kpa a lung scan is usually unnecessary.

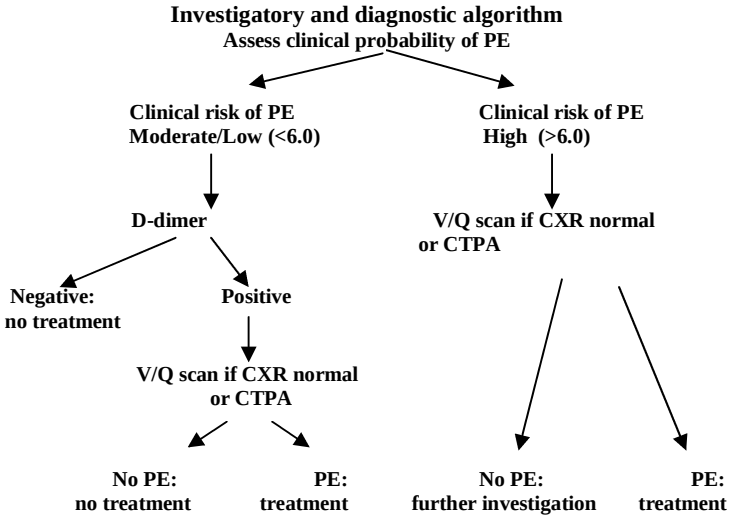
Any patient with a risk factor for pulmonary thromboembolic disease and unexplained tachypnoea or dyspnoea should be investigated for the possibility of pulmonary thromboembolic disease, especially when clinical signs in keeping with this diagnosis are present. Once clinical risk of PE has been assessed, investigations should follow the algorithm on page 19.

Management

Patients will require oxygen therapy if hypoxic, and analgesia if in pain (paracetamol is often sufficient). While awaiting confirmation of PE, the patient should be given a 'treatment dose' of LMW heparin subcutaneously***. 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 PE specialist nurse Rosanna Salinas x5543 or blp 8409 and assess pulmonary embolism severity score, as patient may be considered for outpatient therapy. Start warfarin; 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 (these will have a raised BNP and/or troponin) 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 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. If it is decided to give a thrombolytic, then, 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.

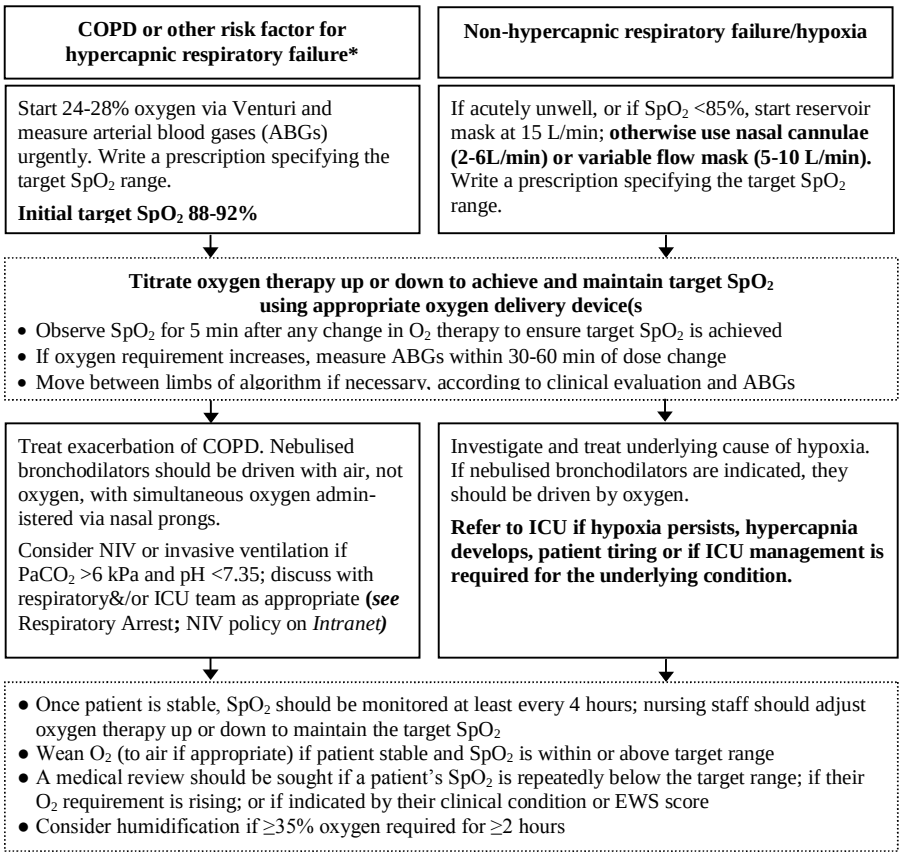


OXYGEN THERAPY IN ACUTE ILLNESS
(Link: Samantha Prigmore, Respiratory Nurse Consultant)

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 (SpO_2) 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***.

Algorithm for Oxygen Therapy in Acute Illness



*Other risk factors for hypercapnic (type 2) respiratory failure include severe chest wall or spinal disease, neuromuscular disease, severe obesity, cystic fibrosis and bronchiectasis.

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 (200micrograms over 15 sec followed by 100micrograms every 60 sec if required, up to 1mg total dose). Use with caution, if other psychotropic drugs (especially tricyclic anti-depressants) may have been ingested as their toxic effects may be potentiated; if the patient is known to be benzodiazepine dependent; or if the patient is epileptic and has been taking a benzodiazepine for a prolonged period. Flumazenil has a short duration of action, the patient should remain under close observation until all possible central benzodiazepine effects have subsided.

Tracheostomy problems

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

Ongoing management

Most patients who survive a respiratory arrest will require intermittent positive pressure ventilation. This should be carried out on the Intensive Therapy Unit under the strictest supervision. Even if the patient is deemed not to require intermittent positive pressure ventilation, any patient who has had a respiratory arrest should be closely watched for the next 24 hours and their management discussed with a member of the respiratory, or ITU, team.

Respiratory failure

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

The indications for non-invasive ventilation (NIV) are:
- acute hypercapnic respiratory failure in the acute, or acute-on-chronic, patient who does not yet require tracheal intubation and who has
  - a $pCO_2 \geq 7$
  - a pH <7.35
  - an increased respiratory rate despite optimisation with oxygen therapy
- acute hypercapnic respiratory failure with chest wall deformity, neuromuscular disorder or decompensated obstructive sleep apnoea
- cardiogenic pulmonary failure refractory to CPAP

- patients who might otherwise receive tracheal intubation, but in whom this is better avoided or not appropriate
- patients being weaned from mechanical ventilation

Patients requiring NIV should be discussed with the respiratory registrar or, if out-of-hours, with the respiratory consultant on call. Full NIV guidelines are on the Intranet *http://stginet/Policies/Clin 5-PatientMment/Clin_5_25.pdf*

ASTHMA

Link consultant: Dr Yee Ean Ong

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 ß$_2$-agonists (less effective and relief shorter lasting)
- decreased efficacy of rescue medication (such as corticosteroids)

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

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

The features of potentially fatal asthma include:

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

MANAGEMENT – Monitoring

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

MANAGEMENT – Treatment

Oxygen. Patients with acute severe asthma are hypoxaemic and this should be corrected urgently with a high concentration of oxygen (usually 40-60%) and a high flow mask keeping oxygen saturations $\geq$92%.

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 should be continued 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. The administration of bronchodilators IV is only indicated in patients who fail to respond or deteriorate, despite repeated treatment given by nebuliser, and in whom intubation is imminent.

***Corticosteroids*.** Patients should be given hydrocortisone 100 mg IV 6-hourly or prednisolone 30-60 mg od by mouth as soon as the initial assessment is made. No material benefit can be expected for several hours but it is essential not to delay administration. Whichever steroid is given initially, after 2 days all patients should be

taking 30 mg of prednisolone daily by mouth and this should be continued for a minimum of 5 days. 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 tenaciousness 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.

SPONTANEOUS PNEUMOTHORAX

Link consultant: Dr Adrian Draper

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

MANAGEMENT. For most patients there is no immediate threat. Once a pneumothorax is suspected, X-ray the chest to confirm the diagnosis, to assess the degree of any collapse (small – a rim of air <2cm around the lung; large >2cm), 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 cardiopulmonary resuscitation 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 (>2cm)

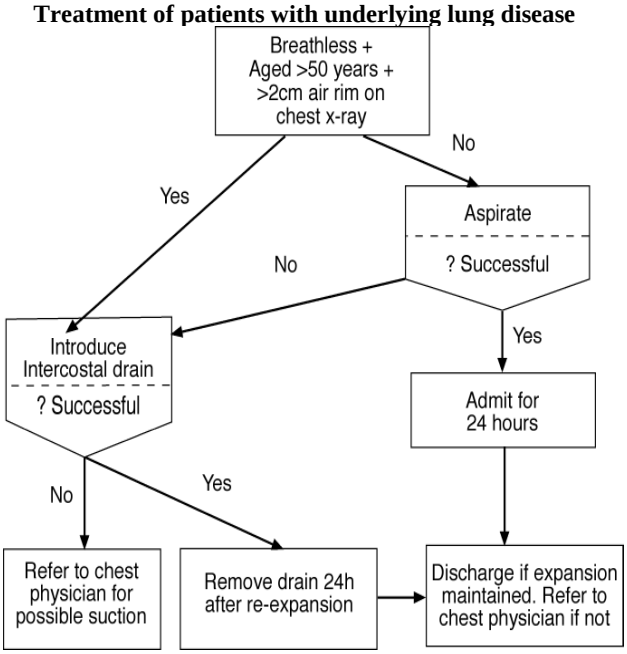
pneumothorax**,** aspirate the air through a wide bore needle introduced under local anaesthesia. If aspiration with a needle fails an intercostal drain may have to be introduced (seek advice).

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.

Treatment of patients with underlying lung disease



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 above*. 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

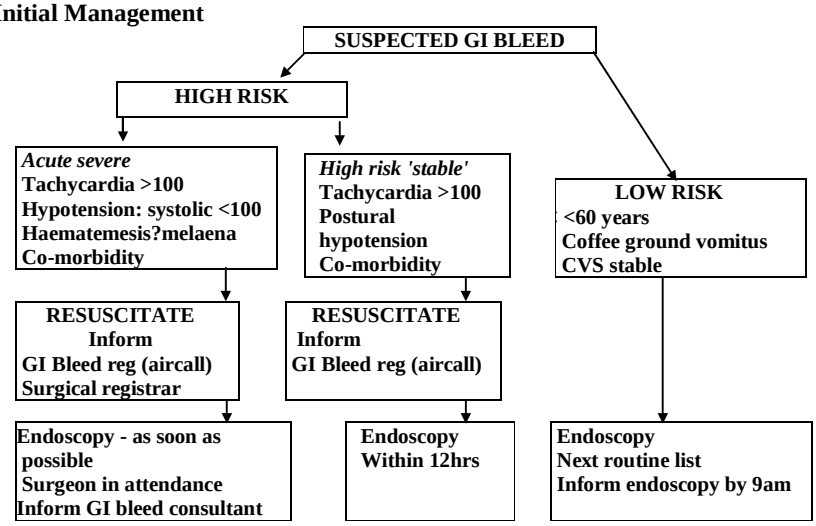
Immediate Assessment

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).

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

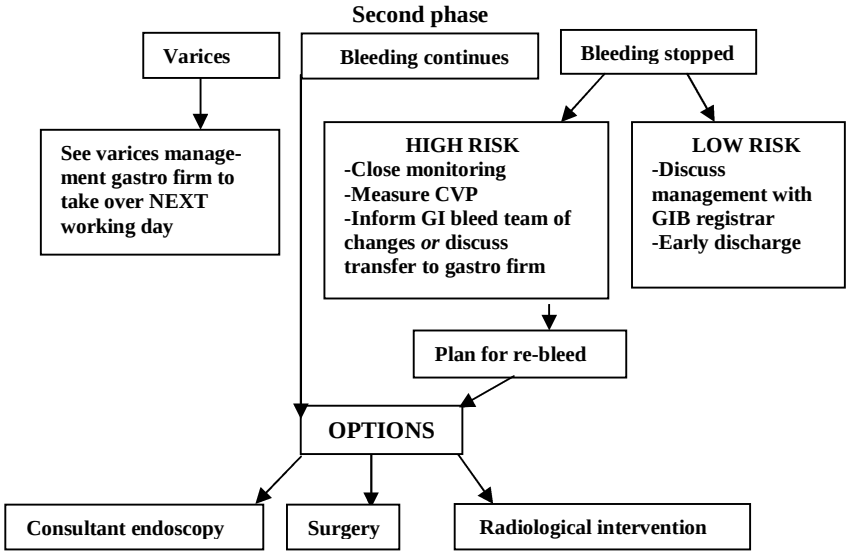
The next stage of management depends on the state of the patient, his or her 'risk assessment' and the findings on endoscopy, (see 'Second Phase' diagram below).

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, prothrombin time, U&Es, LFTs. Crossmatch 6 units of blood or inform haematology of 'CODE BLUE'.
- 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 SpO_2 above 90%.
- Correct blood volume cautiously and carefully, using plasma expanders to maintain haemodynamic stability, and packed red cells to maintain the haemoglobin at approximately 8-10g/dl.

  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

  Give vitamin K (phytomenadione) 10mg IV slowly.
  Give fresh frozen plasma (12mls/kg) if clotting abnormal.
  Give platelets (1-2 pools) if platelet count <50x109/L.
- 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.

3. FAILURE TO CONTROL ACTIVE BLEEDING

- ET Tube

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

  Insert Sengstaken tube (available on emergency endoscopy trolley/ITU). Check tube position once at 50cm. Inject air down gastric port and ausculate over stomach. Cautiously inflate gastric balloon with 300mls of 1:1 Niopam and water, and pull back until resistance is felt at the gastroesophageal junction. Attach the tube firmly to the patient's cheek with tape. Do not use traction. Put gastric and oesophageal port on free

drainage. Do CXR to check gastric balloon is below the diaphragm. Re-scope within 24hrs. Do not leave gastric balloon inflated for more than 24hrs.

- Transjugular intrahepatic portosystemic stent shunt (TIPPS)

If bleeding is still uncontrolled, contact Liver Unit to discuss what to do next.

4. SECONDARY PROPHYLAXIS OF VARICEAL HAEMORRHAGE

Liver 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.

BLOODY DIARRHOEA (ACUTE ULCERATIVE COLITIS)

Link consultant: Dr Richard Pollok

Management of patients with severe bloody diarrhoea, (passing 6 or more bowel motions /day) 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:

Common	***Less common***	***Rare***
Ulcerative colitis	Crohn's disease	Haemolytic-uraemic syndrome
Pseudomembranous colitis	Ischaemic colitis	Yersiniosis
Bacterial dysentery (eg camylobacter, salmonella, shigella, etc.)	Amoebic dysentery	TB enteritis
	Colorectal cancer	Schistosomiasis
	Diverticular disease	HIV-related opportunistic Infection, eg. CMV, HSV, etc.

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.

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 $\geq$ 6cm)
- a labelled white cell scan may also be of value in assessing the extent and severity of the disease

Management - on admission:
- start hydrocortisone 100mg qds IV immediately
- start appropriate fluid replacement with normal saline and potassium supplement
- request early surgical review (ideally from a colorectal surgeon)
- perform (and view) 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 3 times a week

Further management should be instituted by a gastroenterologist and the team contacted promptly. Remember that patients should not usually be kept nil by mouth unless surgery is imminently scheduled.

DIABETIC KETOACIDOSIS (DKA)

Link consultant: Dr Natasha Patel

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/ITU.

Causes of hyperglycaemic emergencies and their differentiation

	Blood glucose	Urinary Ketones	Dehydration	pH	Serum Osmolality (osmol/kg)
Severe DKA	**>13mmol/L**	**++ to +++**	**+++**	**<7.35**	**Variable**
Normoglyc ketoacidosis	**<11mmol/L**	**+++**	**+++**	**≥7.35**	**Normal**
HHS	**>33mmol/L**	**Negative**	**++++**	**≥7.35**	**>320**
HHS/DKA mixed	**>33mmol/L**	**++ to +++**	**++++**	**<7.35**	**>320**
Lactic acidosis	**Variable**	**0 to 1+**	**0 to 1+**	**<7.35**	

STAGE 1: IMMEDIATE MANAGEMENT (On presentation – Hour 1)

STEP 1: Initial investigations
- Insert iv cannula and take bloods for U&E's, blood glucose (BG), venous gases with lactate, FBC, blood cultures
- Ensure urinalysis, ECG, CXR are done

STEP 2: Fluid replacement
- Give sodium chloride 0.9% at a rate of 1000ml/hr
- If $K^+ < 5.5$ mmol/L, give pre-prepared bag of 40mmol/L potassium and 0.9% sodium chloride

STEP 3: Start insulin
- Make up insulin infusion by adding 50units Actrapid® to 49.5mls of 0.9% sodium chloride
- Infuse iv insulin at a fixed rate of 6units/hr via infusion pump. (Only give a stat dose of Actrapid insulin if there is a delay in setting up an insulin infusion)

• Continue subcutaneous insulin glargine (Lantus®) or insulin detemir (Levemir®) if the patient is already receiving this.

Discuss with HDU/ ITU if:		**Essential monitoring**
• **BG > 33 mmol/L**	• **Evidence of sepsis**	• **Regular observations**
• **Hypokalaemia (K^+ <3.5mmol/l)**	• **pH <7.1**	• **Fluid Balance Chart**
• **GCS < 15**	• **HCO3 <5.0**	• **Monitor conscious level**
• **Pulse > 100bpm or < 60bpm**	• **Lactate >2.0**	• **Regular blood chemistry (esp K^+)**
• **SBP < 90 mmHg**		

Consider central line, continuous cardiac monitoring, nasogastric tube, urinary catheter.

STAGE 2: ON-GOING MANAGEMENT (Hours 2-4)

STEP 1: re-assess patient – (at 2hrs and then 4hrs)

• Monitor patient vital signs using EWS. Alert a senior member of staff should a patient trigger a response. Be especially aware of conscious level

• Repeat venous blood gases for accurate blood glucose and potassium and to monitor trends in acid base balance

• Catheterise if oliguric (urine output < 0.5 ml/kg/hr)

• Continue sodium chloride 0.9% 1000mls/hr for Hour 2, then 500mls/hr for Hours 3 & 4

• Caution in the elderly, pregnant, adolescent, heart or kidney failure, other serious co-morbidities

• Give pre-prepared infusion bag of 40mmol/L potassium and 0.9% sodium chloride unless anuric or potassium is >5.5mmol/L.

(If K^+ remains <3.5mmol/L despite replacement, continue with 40 mmol/L K^+ per litre of 0.9% NaCl and call HDU/ITU)

• Call HDU if there is a deterioration in conscious level

STEP 2: Further monitoring, continuation of insulin

• Hourly Capillary Blood Glucose (CBG) (if CBG records HI, send laboratory venous blood glucose for accurate results)

• Continue intravenous insulin at 6units/hr

IF CBG is not falling by 3mmol/hr, increase rate by 1unit/hr (check infusion pump is working and connected)

• Once CBG <14mmol/L, *add 10% Glucose infusion at 100mls/ hr* (this is alongside the sodium chloride 0.9% infusion) and *reduce insulin infusion to 3units/hr* or a rate to maintain CBG at 9-14mmol/L

• Venous blood gas for pH, HCO_3 and K^+ at the end of the 1^{st}, 2^{nd} and 4^{th} hours.

STAGE 3: SUBSEQUENT MANAGEMENT (HOURS 5 ONWARDS)

STEP 1:Re-assess patient, monitor vital signs

• Allow oral intake if bowel sounds are present

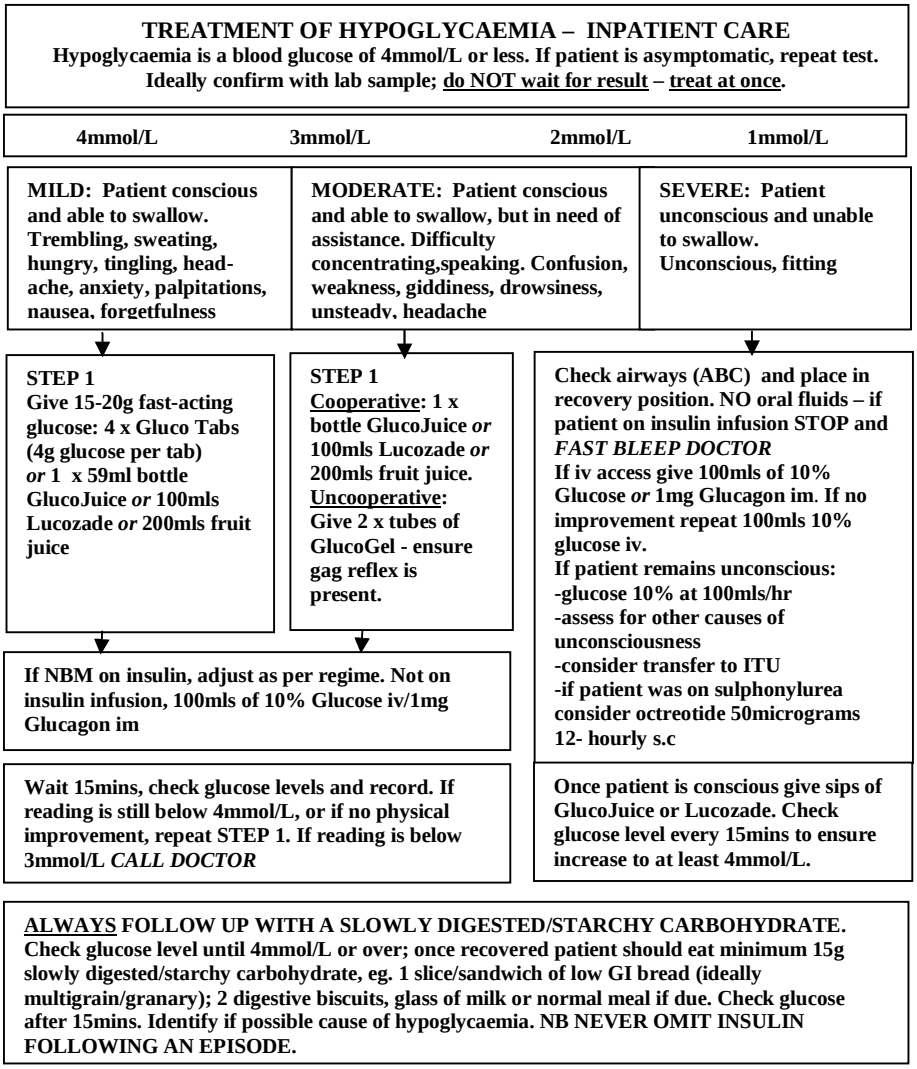
• Continue to monitor bicarbonate until within the reference range

• Continue 10% glucose infusion at 100mls/hr to maintain CBG in conjunction with sodium chloride 0.9% <250mls/hr (adjust according to patient's fluid status) until bicarbonate is within the reference range and patient is eating

• Continue K^+ supplementation to ensure K^+ remains within the reference range

- Convert to subcutaneous insulin when biochemically stable and eating
- Stop iv fluids and insulin 1 hour AFTER the first dose of subcutaneous 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).

ACUTE STROKE

Link consultant: Dr Barry Moynihan

Stroke is a clinical syndrome in which an acute focal cerebral deficit lasts for 24 hours or results in death that occurs secondary to cerebrovascular disease. In a transient ischaemic attack (TIA) symptoms and signs are similar but resolve within 24 hours (most commonly within 1 hour). Causes of stroke include cerebral infarction, primary intracerebral haemorrhage, subarachnoid haemorrhage and cerebral venous thrombosis. To direct management it is essential to know the underlying pathology (haemorrhage or infarction), the site (e.g. carotid or vertebrobasilar territory), underlying aetiology (eg carotid stenosis or cardiac embolism) and disability. All potential stroke patients in SW London are 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.

Admission

Good management of patients with stroke reduces mortality by 25% and the risk of recurrence by up to 75% and reduces complications and residual disability. To this end any patient (whatever age) who has developed features of stroke (whatever severity), or a TIA, with the exception of those in whom the episode is not the major current condition, should be admitted directly to the Hyper Acute Stroke Unit (HASU, William Drummond Ward, 3rd Floor AMW)**.** 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 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, during working hours (9am-5pm Monday-Friday) contact the Stroke Unit SpR (blp 7317) or SHO, or out-of-hours the Neurology on-call SpR (blp 7277). Inpatients should also 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, or directly from A&E. 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 and Examination

The history should be recorded in the stroke proforma (available from HASU), include time and mode of onset (sudden /gradual), progression since onset and vascular risk factors (including the presence/absence of hypertension, diabetes, hyperlipidaemia, smoking, alcohol, heart disease, claudication and family history of stroke or ischaemic heart disease). The neurological examination should assess the patient's conscious level (use the Glasgow coma scale), gait, cognitive function (orientation, language, memory, visuospatial skills, ATMS), 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 the presence/absence of carotid bruits and cardiac murmurs.

Investigations

All patients should have a CT or MRI scan. 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 patients being considered for thrombolysis (*see below*). 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. In others, it should be undertaken as soon as possible and always before admission to the HASU. 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 department of neurology). 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 full blood count, ESR, U&Es, glucose and cholesterol levels and an 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 should have a thrombophilia screen (protein C, protein S, antithrombin III, APC resistance, lupus anticoagulant), auto 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 in whom the pattern of infarction is consistent with embolism, i.e in multiple cerebral vascular territories). 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 patient under the age of 50, and should be performed in the AMW neuroradiology department after referral to the neurology team.

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) or Neurology SpR (blp 7277 or 07717 291256 mob) before arranging investigations. They will organise brain imaging, CT or MRI scanning, 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 fofr 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 and aspirin is stopped at this stage**. Full heparinisation should be reserved for patients with cerebral venous thrombosis, or where 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* p5).

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

- In patients at high risk of DVT and pulmonary embolism, *provided there is no haemorrhage on brain imaging*, give low-dose anticoagulation with low molecular weight heparin, according to St George's thromboprophylaxis protocol. Fill out the VTE form. Calf pumps can be used in patients with haemorrhage.
- Ensure swallowing is adequate before giving oral fluids and food. If swallowing is not safe, 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. Patients are currently randomised into the CADISS trial, to compare antiplatelet agents with anticoagulation.

- **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 scan, to look for evidence of clot within the sinuses, and magnetic resonance venography. Refer suspected patients to the neurology SpR. Most patients should be anticoagulated with heparin and then warfarin even if there is 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.

- **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. Repeat imaging between 1 and 2 months post event, 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 first.*

Prevention/reduction of risk of recurrence:

- Hypertension should be investigated and treated after the acute stage (*see above*).
- Patients with carotid stenosis demonstrated on Duplex should be referred urgently to the stroke SpR or the cerebrovascular disease clinic for consideration for carotid surgery or angioplasty.
- Consider anticoagulation in patients with atrial fibrillation (age alone should not be seen as a contraindication to anticoagulation).
- Treat other risk factors: eg. diabetes, smoking, cholesterol.
- Patients with ischaemic stroke who are not anticoagulated should be treated with appropriate anti-platelet therapy. **First-line treatment is clopidogrel (75mg per day). Aspirin and dipyridamole is an alternative. The choice of antiplatelet is made after stroke team review**.
- All patients admitted with stroke should be followed up in the stroke follow up clinic (fax discharge letter to x4591).

STATUS EPILEPTICUS

Link consultant: Dr Hannah Cock

Generalised status epilepticus is defined as either a run of discreet generalised tonic/clonic seizures without full recovery in between fits (ie without gaining consciousness), or continuous generalised tonic/clonic seizure activity lasting for 30 mins. As most seizures terminate spontaneously within 3 minutes, *the following measures should only be instituted for seizures lasting longer than 7-10 minutes*, 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.

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 rapidly infuse a 50% solution of glucose to give 1-2mg per kg body weight.
6. Draw venous blood for full blood count, clotting, glucose, urea, sodium, potassium,

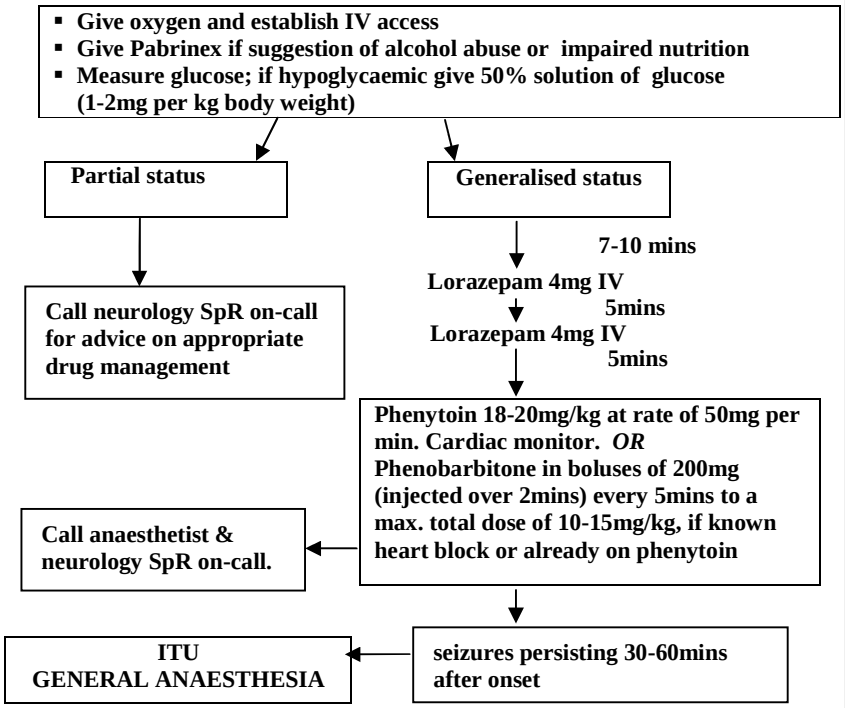
calcium, liver function and anticonvulsant drug levels (irrespective of known history at this stage). Save a sample of blood and urine for toxicology.
7. Measure body temperature, take an ECG, monitor respiration and BP.
8. *Gain information* – is there evidence of previous epilepsy, any anticonvulsant 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.
2. If seizures persist or recur, repeat lorazepam at 5-10 minutes. Lorazepam, however, 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.

Treatment of Status Epilepticus



1. 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 18-20mg/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, whilst monitoring respiratory function.

2. If, despite intravenous lorazepam & phenobarbitone or phenytoin, seizures persist or recur over 30-60 minutes, the patient should be transferred immediately to an ITU.
3. 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, with the possible use of midazolam, propofol or thiopentone. Phenobarbitone may also be considered. 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.

SUBSEQUENT MANAGEMENT

1. Reinstitute any recently-stopped anticonvulsant medication. Check that the patient is taking the medicines as prescribed and that there have been no interactions reducing drug efficacy.
2. If this is a new presentation, a cause must be sought. Intracranial bleeding, infection or drug toxicity are the major causes; consider investigations such as CT scanning, EEG monitoring and lumbar puncture as appropriate.
3. *All* patients should be discussed with the on-call neurology registrar or an epilepsy team member, and arrangements for follow-up made. No patient should be discharged without an adequate explanation of their presentation and agreeing a plan of management. Discussion, which ideally involves the patient's partner/parent, should include basic seizure safety information and driving regulations.

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, antisera, 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
- Give adrenaline (epinephrine), 0.5mL of a 1:1000 solution (ie 0.5mg) IM into lateral 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 in whom IV access can be obtained without delay. The dose should be given slowly, by a doctor with appropriate training, in a dilution of at least 1:10,000 and with ECG and BP monitoring.*
- Give chlorphenamine by IM or slow IV injection in a dose of 20mg.
- For patients with a severe or recurrent reaction, and in all patients with asthma, give hydrocortisone (sodium succinate) in a dose of 100-300mg (depending on body size) by slow IV or IM injection.
- For patients in shock who do not respond rapidly to drug treatment give 1-2 litres of 0.9% NaCl.
- An inhaled β_2 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 minimus of 8-24hrs. *Write the name of the agent that caused the reaction prominently in the patient's notes and drug chart.*

ACUTE PAIN

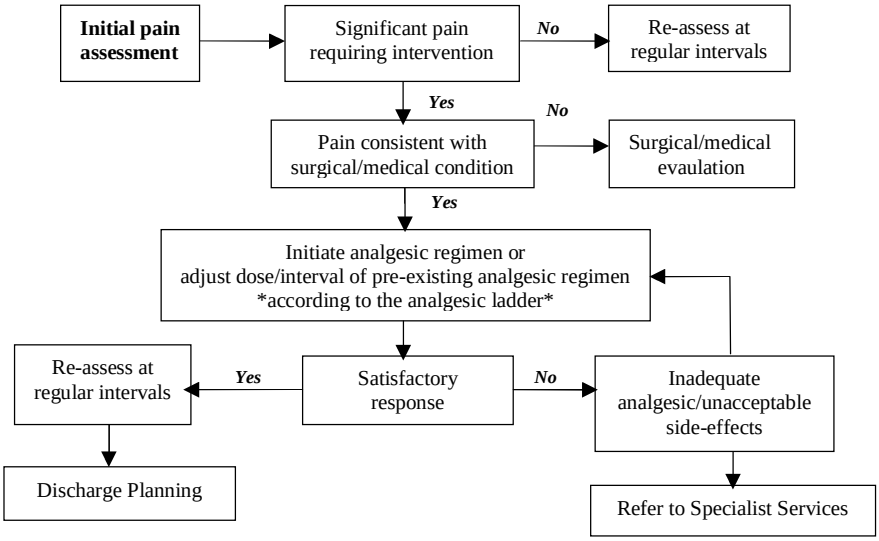
Link consultant: Dr Jeremy Cashman

Note that for some conditions, such as acute coronary syndromes** (page 7), **acute painful joints** (page 56), **and sickle cell crises** (page 63), **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 according to the following '***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**

In general it is more realistic to *strive for comfort* rather than complete abolition of pain.



TREATMENT DETAILS

Simple Analgesic

Paracetamol: 1g PO/NG/PR 4-6 hourly (maximum 4g/day).

Non-Steroidal Anti Inflammatory Drugs (NSAIDs)

Diclofenac: 50mg PO 8-hourly or 12.5mg, 25mg, 50mg, 100mg PR/day (max 150mg/day)
Ibuprofen: 200-400mg PO 4-6 hourly (maximum 2.4g /day).
Contraindications: Bleeding diathesis, peptic ulceration, renal dysfunction, allergy to NSAIDs (care in asthma), congestive cardiac failure.

Combination Analgesic

Co-dydramol (10mg dihydrocodeine + 500mg paracetamol/tablet): 1-2 tablets PO 4-6 hourly (maximum 8 tablets/day).

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.

- 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:

IV morphine		
Age (yrs)	Dose	
	Pain severe	Less severe
< 70	2mg	1mg
> 70	1mg	0.5mg

A&E, ICU and Theatres Only

IM morphine	
Age (yrs)	Dose
20-39	7.5-12.5mg
40-59	5-10mg
60-69	2.5-7.5mg
70-85	2.5-5mg
>85	2-3mg

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 immediately repeat injection but at a dose no more than 50% of the original dose; for IV administration (only in A&E, ICUs and Theatres) immediately repeat same or at a dose no more than 50% of the original dose. Check for analgesia or overdose post-injection as above.

- Infusion: Infusions (morphine 1-6 mg/hour IV) should only be given where there is close supervision with adequate patient monitoring. O_2 should be administered continuously and O_2 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 100micrograms every 2-3 minutes until patient is rousable and respiratory drive returns,
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 6477/6159); On-call anaesthetist (bleep 6111); Palliative Care team (bleep 6796/6508 or ext 3313).

SUGGESTIONS FOR THE USE OF ANTIMICROBIAL DRUGS IN ADULTS

Link consultant: Dr Aodhan Breathnach

- Try to take microbiology samples before starting antibiotics, but do not delay treatment in a severely ill patient.
- Use strict asepsis when taking blood cultures – contaminated samples lead to clinical confusion and inappropriate antibiotics. CSF, joint fluid and other clinically urgent samples can be processed out of hours – contact the on-call lab staff to discuss.
- Remember the potential harm caused by antibiotics, in terms of side effects and selection of resistant organisms such as MRSA and *Clostridium difficile*.
- 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. Base initial antibiotic choice on your judgement of the most likely pathogen(s), and guidance below. Use a narrow-, rather than broad-spectrum antibiotic whenever possible. Cephalosporins and ciprofloxacin have particular risk of selecting *C.difficile* or MRSA.
- 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.
- Write Stop/Review date and indication on the medicines chart for *all* antimicrobial prescriptions.
- Switch IV to oral when appropriate (*see below*).
- Penicillin allergy – check and document nature of reaction: mild = rash; moderate to severe = angioedema, swollen tongue, anaphylaxis.
- If pathogens are identified, modify treatment accordingly.
- If patient is colonised with MRSA consider if empiric treatment with an antibiotic with activity against MRSA is required.

<u>Switch from IV to oral antibiotics</u>

Seriously ill patients should be given parenteral drugs initially. Remember to switch to oral therapy once:

- 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.

For Trust policy for specific conditions see pages 42-43 and the Intranet website:
http://stginet/Units%20and%20Departments/Antimicrobial%20prescribing%20information/Antimicrobial%20prescribing%20info%20for%20staff.aspx
(or follow quick link at bottom of Intranet homepage)

Seek further advice from Medical Microbiology (blp 6480), the Clinical Infection Unit (adult infectious diseases: McEntee Ward, x3280), the Ward Pharmacists, Infection

Control Nurses, as appropriate. 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.

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

ANTIMICROBIAL POLICY FOR PATIENTS WITH NEUTROPENIA

See the Trust Intranet for the full care pathway: http://stginet/Units%20and%20Departments/Antimicrobial%20prescribing%20information/Neutropenic%20sepsis%202011.doc; or use the quick link to the antibiotic prescribing pages from the homepage.

The care pathway definition of neutropenia is: **a neutrophil count of <1.0.**

The diagnosis of neutropenic sepsis must be assumed for any patient who is unwell and febrile and: (i) has cancer and receiving chemotherapy, or has received chemotherapy within the last 28 days; or, (ii) is at risk of neutropenia, for example, secondary to bone marrow failure (e.g. due to primary haematological disorder) or immunosuppression.

Patients can be critically ill with minimal signs and without a pyrexia. Observe for pallor, mottled skin, tachycardia, anxiety, altered GCS, increased respiratory rate.

IF CLINICALLY SHOCKED AT ANY TIME, RESUSCITATE IMMEDIATELY & GIVE IV ANTIBIOTICS. DO NOT WAIT FOR NEUTROPHIL COUNT

Ensure samples are sent for culture (peripheral + line bloods, MSU, stool, sputum, throat swab) & urgent FBC, U & Es, LFT, Ca. IV antibiotics should be administered promptly.

Ensure samples are sent for culture (peripheral + line bloods, MSU, stool, sputum, throat swab) & urgent FBC,U&Es, LFT, Ca. IV antibiotics should be administered promptly.

ANTIBIOTIC REGIME for patients with infection by unknown organism

1st line: Piperacillin-Tazobactam 4.5 grams IV tds (if penicillin allergy use ceftazidime) **AND** Amikacin 15mg/kg IV once daily. If grossly obese use dose = 15x[IBW+0.4(TBW-IBW)] where IBW is ideal and TBW is total bodyweight for amikacin dose. Check amikacin level prior to giving the 2nd dose- aim for trough level <5microgram/ml. If renal impairment, consider ceftazidime or reduced amikacin dose (see amikacin dosing guidelines) or see antibiotic prescribing pages on the intranet. **If still pyrexial after 48 hrs 2nd line:** Meropenem 1g IV tds **AND** Vancomycin 1g IV bd. Consider increasing the vancomycin dose for larger patients with good renal function and reducing dose in renal impairment - see App.10 for detailed guidelines. Monitor vancomycin levels immediately before the 3rd dose.

Neutropenia continued on page 44.....

Empirical Antibiotic Management of Common Infections in Adults: Med Microbiology x

Infection	1st line Antibiotics
Community acquired pneumonia (CAP) Record CURB-65 score and evidence of Chest X-Ray consolidation. (If clear, treat as COPD/LRTI) • *Confusion (new onset)* °*Age ≥65yrs* • *Urea>7mmol/l* • *Respiratory rate≥30/min* • *BP<90mmHg (systolic) or ≤60 (diastolic)* **If severe** send blood & sputum cultures, & urine for pneumoccocal and legionella antigen detection **& sputum if no prior antibiotics**	**LOW SEVERITY (CURB-65 score 0-1)** Doxycycline PO 200mg STAT then 100-200mg OD **MODERATE-SEVERE (CURB-65 score 2-5)** Benzyl Penicillin IV 1.2g, 4-hrly + Clarithromycin IV/**PO** 500mg, 12-hrly **Patients with moderate CAP (CURB=2) may be suitable for a more rapid IV to oral switch**
Infective Exacerbation of COPD and LRTI No Chest X-Ray changes **Send sputum**	Doxycycline PO 200mg STAT then 100-200mg OD (*or* Amoxicillin IV 1g 8-hrly if severe or unable to take orally)
Hospital Acquired Pneumonia (HAP) Occurring >5 days in hospital Record Chest X-ray evidence of consolidation	Benzyl Penicillin IV **1.2g**, 4-hrly + once-daily Gentamicin IV (if severe or unable to take orally) *OR* Co-amoxiclav PO 625mg 8hrly
Aspiration Pneumonia Record Chest X-Ray evidence of consolidation 48-72hrs following aspiration	Treat as severe **CAP** or **HAP** +Metronidazole IV 500mg, 8-hrly (unless already on Co-amoxiclav)
Urinary Tract Infection Urine dipstick Always collect urine specimen before starting antibiotics **Only treat positive CSU if features of urinary sepsis** Change treatment according to microbiology results	**Uncomplicated UTI** Trimethoprim PO 200mg, 12-hrly **Complicated UTI** Co-amoxiclav IV 1.2g, 8-hrly + Gentamicin IV 5mg/kg STAT if shocked **Pyelonephriti** Co-amoxiclav IV 1.2g 8-hrly + Gentamicin IV **5mg/kg**
Intra-abdominal Sepsis (Hepatobiliary, peritonitis, diverticulitis, gastro-intestinal sepsis associated with surgery)	Amoxicillin IV 1g, 8-hrly + once-daily Gentamicin IV +Metronidazole IV 500mg, 8-hrly
Cellulitis Wound swab if skin is broken If cellulitis associated with diabetic foot ulcer, see specific guidelines for diabetic foot infection. **Contact microbiology if patient is shocked and for necrotic skin infections**	**NON SEVERE** Flucloxacillin PO 500mg, 6-hrly **SEVERE** Benzyl Penicillin IV 1.2-1.8g, 4-hrly +Flucloxacillin IV 1-2g, 6-hrly
Osteomyelitis/ Septic Arthritis **Refer patient to OPAT (SG278) if fit for discharge on IV antibiotics**	Flucloxacillin IV 1-2g, 6-hrly + Sodium Fusidate PO 500mg-1g, 8-hrly
Clostridium difficile	Metronidazole PO 400mg, 8-hrly
Suspected Sepsis – site unknown	Co-amoxiclav PO 625mg *or* IV 1.2g, 8-hrly +Gentamicin IV 5mg/kg STAT
Meningitis (Start antibiotics immediately)** Take blood cultures plus blood in EDTA for molecular studies and **a throat swab**. Seek advice on need for a CT scan, timing of LP and need for dexamethasone.	Cefotaxime IV 2g 6hrly + Aciclovir IV 10mg/kg 8hrly if viral encephalitis suspected + Amoxicillin IV 2g 4hrly if immunocompromised or >55 years to cover for listeria. ****Remember tuberculous meningitis - seek advice from CIU if suspected**

1970 (***emergency blp*** **6480**). **Pharmacy blp 7508**. **Out of hours reg. SG395, Pharm blp 6267**

Alternative if allergic to 1st line	**Oral switch**	**Duration**
Clarithromycin PO 500mg, 12-hrly *OR* Amoxicillin PO 500mg – 1g 8-hrly		5 – 7 days
Seek microbiology advice	**Amoxycillin 500mg –1g 8-hrly (+/- clarithromycin 500mg 12-hrly) OR Doxycycline PO 200mg STAT then 100-200mg OD (if penicillin-allergic)**	7-10 days in total IV + PO *(review needed for macrolide on oral switch)*
Amoxycillin PO 500mg – 1g 8-hrly *OR* Clarithromycin PO 500mg 12-hrly	Amoxicillin 500mg – 1g, 8-hrly	5 – 7 days
SEVERE / unable to take orals: Seek microbiology advice. NON SEVERE: Doxycycline PO 200mg STAT then 100-200mg OD	Co-amoxiclav 625mg, 8-hrly **OR contact microbiology if penicillin allergic**	5-7 days in total IV + PO
Seek microbiology advice	Co-amoxiclav 625mg, 8-hrly	5-7 days in total IV + PO
Nitrofurantoin PO 50-100mg, 6-hrly		3 – 7 days
Ciprofloxacin PO 500mg 12hrly + Gentamicin IV 5mg/kg STAT if shocked	**Co-amoxiclav 625mg, 8-hrly (Check results of urine MC&S)**	7 – 10 days
Ciprofloxacin PO 500mg, 12-hrly **+ Gentamicin IV 5mg/kg**	Co-amoxiclav PO 625mg 8hrly	**10-14** days in total IV + PO
Seek microbiology advice	Co-amoxiclav PO 625mg, 8-hrly	Review IV after 48 hrs
Clarithromycin PO 500mg, 12-hrly		7 days
Clindamycin IV/PO 600mg 6hrly Clindamycin has excellent oral bioavailability so early switch is recommended	**Flucloxacillin 500mg, 6-hrly *OR* Clindamycin 450-600mg 6-hrly**	Review IV after 5-7 days depending on response
Vancomycin IV 1g, 12-hrly + Sodium Fusidate PO 500mg, 8-hrly	Flucloxacillin 500mg, 6-hrly + Sodium Fusidate 500mg, 8-hrly *Seek microbiology advice if Penicillin allergic*	Usually 2 weeks IV then 4 weeks PO (*seek microbiology advice*)
Vancomycin PO 125mg 6-hrly Recurrence – seek microbiology advice	***switch antibiotic if no improvement after 5 days initial therapy***	10 –14 days but review after 5dys
Seek microbiology advice		Review after 24hrs
		Dependent on culture results *(seek advice from microbiology/CIU)*

NEUTROPENIA - additional notes

- For new patients, review previous microbiology tests for positive culture results
- Patients with indwelling venous lines should receive adequate anti-staphylococcal cover
- Give vancomycin if known or suspected MRSA
- In patients with diarrhoea prescribe metronidazole 500mg IV tds or 400mg po tds
- In patients with herpetic lesions prescribe aciclovir 5mg/kg IV tds
- In general patients should continue on IV antibiotics until neutrophils >1.0, switch to ciprofloxacin by oral route once patient is apyrexial for 4 days

INFECTIVE ENDOCARDITIS

Suspected endocarditis: take blood cultures (several sets, plus a serum sample), request an 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** Endocarditis is best managed by Cardiology or jointly by Cardiology & Infectious Diseases, with input from Medical Microbiology.

Empirical Therapy (if organism known, discuss with microbiology)

Clinical criteria	Antibiotics	Duration	Comment
>1 week duration/indolent presentation	Benzylpenicillin 1.2g 4hrly & Gentamicin 80 mg bd	4 weeks (Stop Gent at 2 weeks)	Aim for Gent levels Trough < 1mg/L & Peak 3-5 mg/l
< 1 week, Acute, severely ill, IVDU	Flucloxacillin 2g 4-6hrly (give 4hrly if pt>85kg) & Gentamicin 1mg/kg 8 hrly	4 weeks (Stop Gent at 1 week)	Aim for Gent levels Trough <2mg/L & Peak 5–10 mg/L
Prosthetic Valve, pacemaker, other implanted foreign material	Vancomycin 1g 12hrly & Rifampicin 300 -600mg 12hrly PO & Gentamicin 1mg/kg 8hrly	6 weeks (Stop Gent at 2 weeks)	Aim for Gent levels Trough <1mg/L & Peak 5– 10mg/L
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.

Infection	Antibiotics
Microbiologically confirmed streptococcal pharyngitis	Penicillin V PO 500mg qds (or clarithromycin PO 500mg bd if allergic to penicillin)
Acute otitis media & bacterial infection proven or strongly suspected	Amoxicillin PO 500mg tds for 3 days
Suspected acute epiglottitis	**Co-amoxiclav 1.2g IV tds *OR*** Cefotaxime IV 1g tds
Suspected diphtheria	Call for Consultant help.
Sinusitis	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.

EMPIRICAL TREATMENT OF DIABETIC FOOT INFECTION

Refer patients urgently to Diabetic Foot Team x1859; Vascular SpR Blp 6640

Clinical syndrome (+general comments)	**Empiric Antibiotic Choice – if pathogen is known adjust treatment**				
	Severity of infection	Category of infection			Duration
		'Routine'	MRSA+ (EPR '*X* ')	Penicillin allergy	
Cellulitis Clinical diagnosis. Exclude critical limb ischaemia, Charcot or DVT (all of which can coexist with infection)	Mild erythema, warmth oedema. Moderate lymphang-itis	Flucloxacillin PO 1g QDS	Doxycycline PO 200mg stat then 100-200mg OD	**Doxycycline PO 200mg stat then 100-200mg OD**	1 week total treatment. Review patient at 48 hrs for oral switch (if pt on IVs) and *MRSA* status with updated microbiology results. Adjust antibiotics accordingly.
	Severe systemic features	Benzylpenicillin IV 1.2 g 4hrly + Flucloxacillin IV 1g 6hrly	Vancomycin IV* + Benzylpenicillin IV 1.2g 4hrly + Flucloxacillin IV 1g 6hrly	Vancomycin IV* + Doxycycline PO 200mg OD	
Ulcer + Cellulitis	Mild/ Moderate	Co-amoxiclav PO 625mg tds	Doxycycline PO 200mg stat then 100-200mg OD + Trimethoprim PO 200mg BD	**Doxycycline PO 200mg stat then 100-200mg OD** + Trimethoprim PO 200mg BD	1 week total treatment. Review patient at 48 hrs for oral switch (if pt on IVs) and *MRSA* status with updated microbiology results. Adjust antibiotics accordingly.
	Severe	Co-amoxiclav IV 1.2g tds	Vancomycin IV*) + Co-amoxiclav IV 1.2g tds	Ertapenem IV 1g OD	
Ulcer probing to bone	No specific antibiotic, unless either cellulitis, or evidence of osteomyelitis (radiological or high index of clinical suspicion e.g. extruding bone fragments). Investigate for osteomyelitis radiologically or by bone biopsy				
Ulcer + definite osteomyelitis Confirm radiologically or by bone biopsy.	Co-amoxiclav IV 1.2g tds for 2 weeks ⇒ Co-amoxiclav PO 625mg tds	Vancomycin IV * + Co-amoxiclav IV 1.2g tds for 2/52 ⇒ Doxycycline PO 200mg OD + co-amoxiclav PO 625mg tds	**Ertapenem IV 1g OD for 2 /52** ⇒ Doxycycline PO 200mg OD + ciprofloxacin PO 500-750mg BD	2 weeks IV followed by 4 weeks PO Review after 6 weeks total treatment (clinical/ biochemical/ radiological) – may need to extend if not fully resolved. Review again 2 weeks after Rx ends.	
Vascular implant and superficial infection: Complex & rare – needs case-by-case discussion between vascular surgeons and microbiologists					

see Vancomycin dosing guidelines in Appendix 10

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. *(This guidance is contrary to NICE but compatible with that of the AHA **and BSAC** guidance).*

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 procedures		
Prophylactic regimens:	**1st line prophylaxis**	**If allergic to penicillin**
Oral – single dose given 1hr before procedure	Amoxicillin 3g	Clindamycin 600mg
Unable to take oral therapy Single IV dose 0-30 mins before procedure	Amoxicillin 3g	Clindamycin 600mg

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/Antimicrobial%20prescribing%20info%20for%20staff

PROPHYLACTIC USE OF ANTIBIOTICS IN SURGERY – MINOR TRAUMA

Infection	**Antibiotics**
Drainage of abscess	Not routinely required (unless septic or surrounding cellulitis)
Bites (Human or Animal)	Co-amoxiclav (625 mg tds) for 3-5 days. In patients allergic to penicillin give doxycyclin**e** (100 mg bd) plus metronidazole (500mg 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.
Lacerations	Nil

GASTRO-ENTERITIS

No antibiotic as a routine. If in doubt, or patient is severely ill, consult specialist, eg. paed-iatrician, microbiologist, Clinical Infection Unit, depending on situation. Patients with a clinical diagnosis of infectious gastroenteritis/diarrhoea should be isolated – see Trust Infection Control guidance.

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. Wounds related to the lower bowel or pelvis should be treated initially with co-amoxiclav, gentamicin and metronidazole. With other wounds, where anaerobic infection is not suspected, flucloxacillin should be given in the first instance (vancomycin if MRSA suspected). For contaminated soft tissue injuries use flucloxacillin and penicillin together with appropriate tetanus prophylaxis.

MALARIA OR FEVER IN RETURNING TRAVELLERS

Any ill or febrile traveller returning from an endemic area should be considered as at risk of having malaria. Other conditions, such as typhoid, should also be considered. Take a malaria blood film, blood cultures and a serum sample, and seek specialist advice from an adult or paediatric infectious disease physician.

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 (user name: H598; password: GEORGESW17), or the Guy's and St Thomas' Poisons Unit on 0870 243 2241.

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 $\pm$ arterial blood gases. If ventilation inadequate, consider giving naloxone (up to 2mg) to reverse opiates, and providing ventilatory support. Give O_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 a 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^{o}
- 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 (reproduced on page 51 with permission of Alun Hutchings). Patients with plasma paracetamol concentrations above the normal treatment line (use high-risk treatment line if the patient has liver disease or is malnourished, anorexic, HIV+ve, takes chronic alcohol in excess, i.e. >14 units/wk for women or 21 units/wk for men, takes liver enzyme inducers e.g. phenytoin, phenobarbital, carbamazepine, rifampicin, primidone, St John's Wort), 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 appropriate treatment line on treatment graph, give N-acetylcysteine intravenously using the following regimen:
  -150mg/Kg in 200mL 5% glucose as IV infusion over 15 minutes
  -50mg/Kg in 500mL 5% glucose as IV infusion over next 4 hours
  -100mg/Kg 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 should be given

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.

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

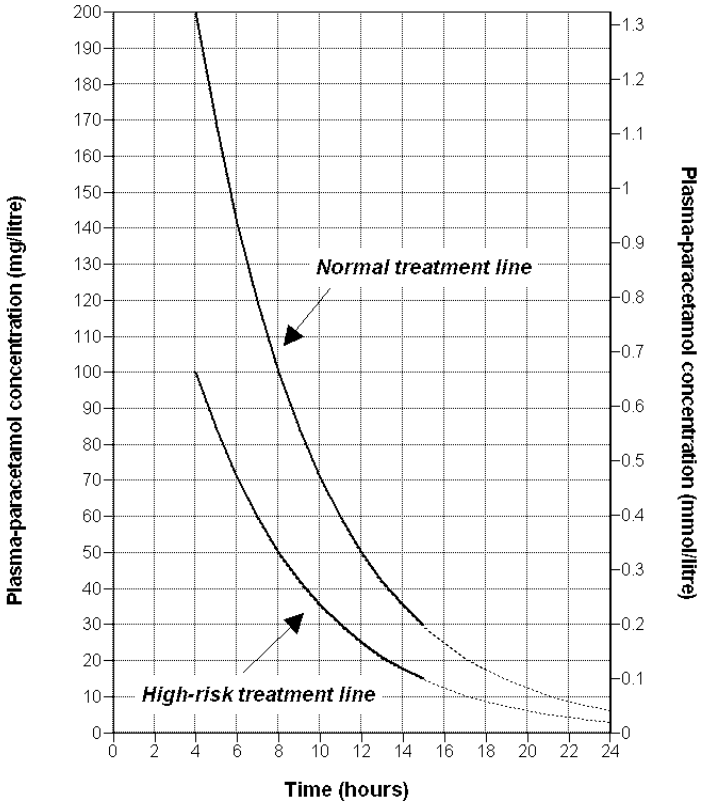
- Where timing of overdose is unknown
- Where overdose was staggered (tablets taken at 2 or more times)
- All patients presenting with evidence of severe toxicity or fulminant hepatic failure regardless of the time post overdose

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 $\leq$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 $NaHCO_3$: 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.

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 O_2, avoid IV sodium bicarbonate. Monitor ECG.
- Anticipate cerebral oedema; if necessary give mannitol 1g/kg (as 20% solution over 20 minutes).
- Discuss hyperbaric oxygen treatment with NPIS (tel. 0870 600 6266) if:
  Unconscious at any time since exposure
  Carboxyhaemoglobin > 20%
  Any neuro/psychiatric symptoms (particularly check for cerebellar signs.)
  CVS complications (including ischaemic ECG)
  Pregnancy

WHAT TO DO IF THE PATIENT REFUSES TREATMENT

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

1) Questions when the patient refuses treatment:

a. Does the patient have the capacity to consent?

-assess patient's capacity to consent and mental illness state
-document assessment in the notes
-ensure these processes are witnessed by a third party e.g. senior nurse
-consider independent second medical opinion and/or psychiatric opinion

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

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

Capacity may be affected by:

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

b. Does the patient have a psychiatric illness?

If in doubt obtain *early* psychiatric opinion

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

2) The treatment options

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

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

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

b. When the patient has psychiatric illness

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

c) When the patient is unconscious or medically unwell

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

MANAGEMENT OF DECOMPENSATED CHRONIC LIVER DISEASE

Link consultant: Dr Daniel Forton

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

Investigations

Blood Tests

1. FBC
2. clotting screen
3. urea, electrolytes, creatinine
4. liver function tests, γGT, albumin
5. α feto-protein (HCC marker)
6. arterial blood gases if patient has encephalopathy, renal impairment or sepsis
7. viral screen/autoantibodies/transferrin saturation/copper studies as appropriate where they might help establish aetiology
8. septic screen – blood cultures, urine cultures, sputum cultures and ascitic tap

Radiology

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

Management

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

1. Do diagnostic paracentesis (ask for urgent cell count to check for spontaneous bacterial peritonitis (SBP) defined as >250 neutrophils/mm^3 or >300 lymphocytes/mm^3. Send sample for culture/biochemistry/cytology)
2. If *moderate volume ascites* and if plasma Na^+ >130mmol/L and renal function is normal, give spironolactone 100mg plus furosemide 40mg daily. Measure weight daily, target weight loss at ~500g/day. The dose of both diuretics can be inc-reased 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 pentoxyphylline 400mg tds orally

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

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

Acidosis

The commonest cause is a metabolic acidosis due to fluid depletion. This should be treated by fluid resuscitation as for renal failure.

Fluid replacement
In liver failure there is total body sodium excess, therefore avoid saline or sodium-containing colloids if possible, unless the patient requires urgent fluid resuscitation, as this will worsen ascites or oedema. If the patient is hyponatraemic (Na^+ <125mmol/l) seek specialist advice.

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

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

Referral to Hepatology team
All patients with decompensated liver disease should be referred to the hepatology team. They should also be referred if they have:
1. Organ failure in addition to liver disease
2. Hepatocellula+r 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: Professor John Axford

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

Ibuprofen	400mg 6-8 hourly.
Indomethacin	50mg/8 hourly; (6 hourly for acute gout)
	Alternatively, for gout, give
Colchicine	500micrograms/2 hourly (maximum 8 daily), especially useful where an NSAID is not tolerated or does not work.

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

Antibiotics

In adults the antibiotics of choice are flucloxacillin plus fusidic acid or clindamycin (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.

ACUTE KIDNEY INJURY (AKI)

Link consultant: Dr Iain MacPhee

Acute kidney injury (AKI), which is characterised by a sudden rise in blood urea and creatinine secondary to an underlying fall in glomerular filtration rate (GFR), is relatively common in patients in hospital. The most frequent cause, and one from which recovery is eminently possible, is acute tubular necrosis (ATN). This is usually the result of hypovolaemia (surgery, haemorrhage, burns), sepsis or nephrotoxic insult (*eg* drugs, iv contrast media, myoglobinaemia or haemo-globinaemia). Other less common causes of AKI are obstruction, acute interstitial nephritis, as seen with drug hypersensitivity, and rapidly progressive glomerulonephritis occurring as a primary event or complicating multi-system disease.

AKI is sometimes associated with a normal urine output or even polyuria. More often there is oliguria (urine output less than 400 mL/day) and occasionally anuria. If there is complete anuria exclude obstruction by ultrasound examination or, if there could be bladder outlet obstruction, by passing a bladder catheter (note the urine volume passed).

Management

1. Treat hyperkalaemia (K^+ greater than 6.0mmol/L).
   a) 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.
   b) To move potassium into the cells give dextrose/insulin infusion, 50mL of 50% dextrose with 10 units of soluble human insulin, over 30 mins. If hyperkalaemia persists after a few hours, the infusion can be repeated. Check blood glucose every hour.
   c) Measure arterial pH and plasma bicarbonate. To help correct 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).
   d) Consider adjunct use of 10-20mg nebulised salbutamol. This must not be used instead of the above interventions as 40% patients do not respond.
   e**)** Start oral polystyrene sulphonate resin (Calcium Resonium), in a dose of 15g four times daily, to remove potassium from the body. The resin can also be given as a retention enema (30g once daily). Give lactulose to prevent constipation.
   f) Check serum K^+ concentration at least twice daily.
   g) Stop all potassium-retaining drugs.
2. Assess status of patient's circulating blood volume. Simple clinical assessment may be misleading and the best guide is given by measurement of CVP. However, in patients who are clearly volume depleted it is probably safest (and technically easier) to go some way to achieving repletion before attempting central venous access.
3. Correct hypovolaemia using 0.9% saline to achieve CVP (mid-axillary line as zero) of 8-10cm H_2O.
4. Insert a bladder catheter. If there is oliguria/anuria it need not remain *in situ*.
5. If the systolic BP is < 100mmHg despite optimal intravascular volume, discuss the position with the ICU-SpR with a view to inotropic support.
6. If a diuresis does not occur despite achieving optimal intravascular volume, give fluid hourly on the basis of replacing measured losses plus estimated insensible losses (approximately 30mL/h) appropriate to clinical state. The primary goal is to achieve optimal (blood) volume; urine flow is of secondary importance. The use of a diuretic or dopamine to increase urine flow in these circumstances is of no benefit to the glomerular filtration rate. However, diuretics can help to reduce fluid overload.
7. Stop nephrotoxic drugs.
8. Give all patients an H_2-blocker or proton pump inhibitor to prevent gastrointestinal haemorrhage.

Urinary and other sepsis should be sought, so it is important to do urinalysis, microscopy and culture. Renal ultrasound must be performed at the earliest possible opportunity to exclude obstructive nephropathy and to assess renal size. Loss of parenchymal mass suggests chronic renal disease. Renal biopsy should be considered if there are atypical clinical features or features to suggest a multisystem disease.

Indications for dialysis or haemofiltration:

- Life-threatening or intractable pulmonary oedema.
- Uncontrollably rising K^+.
- Severe (pH < 7.2) or worsening acidosis.
- Uraemia (eg. uraemic pericarditis).

Specialist advice. Early referral to the consultant renal physician/ registrar (contact at St George's through Buckland ward extension 0062, 1080 or 4119, or via switch-board), should be considered in any patient with –

- Oliguria or anuria
- Creatinine > 250μmol/L
- K^+ > 6.0mmol/L

Remember 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.

Prevention of radiocontrast nephropathy

Patients receiving contrast, in particular those at high risk of AKI, probably benefit from being given N-acetylcysteine 1.2 g twice daily orally on the day before, and on the day of, administration of the contrast agent. They should also be given iv fluids (the preferred fluid is sodium bicarbonate 1.4%, but if not available sodium chloride 0.9% may be used) at 3mL/kg for 1 hr before the procedure, and 1mL/kg/hr for 6hrs after.

ELECTROLYTE DISTURBANCES

Link consultant: Dr Iain MacPhee

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 occuring immediately after haemodialysis may be transient and correct itself. Hypokalaemia in those with end-stage renal failure is complex and supplements should not be given without first discussing the case 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– 600mmol). 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 creatinine (expect more rapid rate of rise of K^+ in patients with renal failure).

HYPERKALAEMIA
The only clinical problems associated with raised serum potassium are cardiac arrythmias, which include asystole and ventricular fibrillation.
Indication for treatment. Attempts should be made to lower potassium when serum K^+exceeds 6.0 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 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. See the section on *Acute Kidney Injury* on page 58.

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. 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. 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 euvolaemic aiming to increase urine volume to 200 mL/h. Consider giving furosemide (40-80mg orally or iv), to increase urine flow and calciuresis. 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.

HYPONATRAEMIA

Hyponatraemia (Na^+<135mmol/L) results from H_2O 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 H_2O 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, euvolaemic 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 H_2O 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)
- euvolaemic (*causes*: diuretics, hypothyroidism, primary polydipsia, cortisol deficiency, SIADH or irrigation with glycine or sorbitol during TURP)
- hypervolaemic (*causes*: congestive cardiac failure, renal failure, conditions associated with hypoalbuminaemia)

Therapy

Treatment is aimed at raising serum Na^+ by no more than 8mmol/L in 24 hours.
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^+ requirement (mmol) = 0.6 x body weight in kg x (desired Na^+ - 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 demeclocycline 300mg tds if no response to fluid restriction

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 SpR 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_2O loss rather than to Na^+ gain. The causes include:

- H_2O loss without adequate H_2O intake
- diuretics
- 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_2O loss. Depending on the cause this may involve giving an anti-emetic, stopping diuretics or treating diarrhoea
2. calculate the H_2O deficit, where

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

3. replace fluid with 5% dextrose plus 0.18% saline (contains Na^+ 30mmol/L), alternating with 0.9% saline (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.

SICKLE CELL CRISES

Link consultant: Dr Fenella Willis

At least 500 patients with sickle cell diseases (HbSS, HbSC, HbSBthal) live in the St George's catchment area. 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). The pain can lead to mute despair or aggressive panic. If pain is bad enough to bring the patient to hospital, the patient usually warrants admission.

In the Accident and Emergency Department

Assessment

- Patients with sickle cell disease should be triaged as urgent.
- Pain needs to be controlled to enable thorough history-taking and clinical examination.
- Rapid initial examination should focus on detecting medical complications requiring specific therapy – infection, dehydration, acute chest syndrome (temperature, oxygen saturations on and off oxygen, tachypnoea, chest signs), severe anaemia, cholecystitis, splenic enlargement, abdominal crisis, neurological events and priapism.
- Target-time for presentation-to-medical assessment by an A&E doctor and analgaesia is within 30 mins.
- If the patient has pain, ask whether it is similar to that of previous crises. If not, look for other (non-sickle) causes.

Initial Management

- Patients will usually have tried simple analgesics and opiates prior to presentation.
- Initial management should be aimed at achieving rapid pain control with parenteral opiates.
- If pain severe administer IM morphine (10mg for adults unless indicated otherwise on the patient's personal protocol). For children get paediatric advice. The dose should be repeated every 2 hrs.
- In opiate naïve patients give 0.1mg/kg IM or SC at 20 min intervals until pain controlled.
- Monitor at 20-30 min intervals for pain, respiratory rate and sedation until patient is stable with adequate pain control; then monitor 2 hourly.
- If breakthrough dose required, give 50% of maintenance dose.
- Administer adjuvant non-opioid analgesic: ibuprofen, diclofenac.
- Also prescribe laxatives, anti-pruritics, antiemetics.
- In all patients given regular parenteral opiates, monitor respiration clinically and oxygen saturation with pulse oxymetry. If respiratory rate less than 10, omit

maintenance analgesia. If severe respiratory depression and sedation, give 100micrograms naloxone every 2 mins as necessary.

- Do not delay starting analgesia while awaiting the results of investigations, transfer to the ward, etc.
- Pethidine is metabolised to norpethidine, a cerebral irritant which can cause grand mal seizures in susceptible patients. It is also short acting with poor bioavailability. **Pethidine should *not* be given – contact haematology team for suitable alternatives.**
- If a new patient, or a patient without a personal protocol, requests pethidine and refuses any alternative, they should be referred to the Haemotology team.
- Nitrous oxide (Entonox) should not be given after leaving the ambulance; in patients with sickle cell disease it can cause an acute, irreversible neuropathy.

After analgesia, perform a full medical assessment. This should include:

- clinical assessment focusing on the chest, abdomen and CNS,
- measurement of body temperature, BP, pulse and respiratory rate,
- pulse oxymetry measuring O_2 saturation,
- blood samples for FBC,U&Es, blood culture and group, and save,
- a chest x-ray if the pain is in the chest. Do not x-ray painful bones as it is rarely useful,
- checking for clinical signs of any of the life-threatening crises (see below).

Further action

- *Oxygen*. Ensure airway and ventilation. Start 24% O_2 at 4L/min via a facemask. If pulse oxymetry shows saturation of <92% increase concentration of inhaled O_2.
- *Fluids*. Give 5% dextrose/w to correct volume depletion followed by 1.0-1.5 x maintenance fluid requirements (Level III). If no venous access give equivalent orally. Cannulation of veins in legs, ankles and feet should be avoided because of the risk of venous thrombosis and leg ulceration. For children get paediatric advice.
- *Antibiotics*. People with sickle cell disease are effectively asplenic and therefore susceptible to infection with encapsulated organisms such as *Streptococcus pneumonia* and *Haemophilus influenzae B.* Fever is usual in crisis and infection often present. Start a broad spectrum antibiotic (e.g amoxicillin 500mg IV qds or cefotaxime 1g IV tds). If chest signs are present, add a macrolide.
- *Blood Transfusion* is NOT indicated in uncomplicated pain crisis.

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. **Cohorted sickle beds are available on Dalby Ward and Ruth Myles Ward.** After admission to the ward, and if personally controlled analgesia is not available, continue 2 hourly sc morphine. Give at the dosage indicated on the patient's personal protocol if available (usually 10–15mg/hr), with additional 5–10mg boluses for breakthrough pain. If personally controlled analgesia is available, give morphine up to 10mg/hr as continuous infusion bolstered, as necessary, by 2-10mg/hr as an sc PCA bolus with a lockout of 20 mins.

The patient should wait no more than 4 hours in A&E. If, for unavoidable reasons, this delay is extended then the patient should:

1. be given a 2 hourly programme of analgesia
2. have fluid input maintained
3. have antibiotic regimen maintained
4. be observed regularly to ensure all vital signs are maintained.

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 (broad-spectrum beta-lactams such as ampicillin or cefotaxime) and volume support are vital. If osteomyelitis suspected, discuss with Microbiology.

Splenic 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.

Chest crisis

Severe shunting & hypoxia caused by intra-pulmonary sickling and mimicking pulmonary embolus/pneumonia, may start in one lobe and then spread to others. It sometimes begins as a pain crisis affecting ribs or shoulders. Treat with fluids and oxygen; observe arterial O_2 tensions – a falling PaO_2 will require exchange transfusion and needs expert advice. Encourage patients with chest pain to attempt one maximal inhalation every 5-10 mins ('incentive spirometry') to aerate basal lung segments; this reduces the risk of progressive sickle chest syndrome.

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.

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.

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 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.

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 PSYCHIATRIC EMERGENCIES
Link consultants: Prof. Mohamed Abou-Saleh, Dr Marcus Hughes

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.

Management. In addition to physical reassessment, assess for cognitive impairment, using MMSE or AMT. Identify and treat the cause of the delirium. Consider: infection, medication, electrolyte disturbance, alcohol or drug withdrawal, hypoxia, CNS disease, Wernicke's encephalopathy, hypoglycaemia, epilepsy, head injury and poisoning.

Optimise the patient's environment:

- Encourage presence of family or friends
- Aim for continuity of care, minimising 'new faces'
- Nurse in well-lit bay close to nursing station
- Use frequent reorientation to place and reason for admission
- Consider one to one nursing if the patient is very distressed
- Encourage adequate fluid intake and monitor food intake
- Minimise polypharmacy and review medication every 24 hours
- Avoid routine sedatives, including sleeping tablets.

Medication for delirium should not be a first line treatment, but is sometimes necessary if the patient is very agitated or distressed. Use one drug at the minimum effective dose – usually haloperidol is the drug of choice.

For elderly patients start haloperidol at 0.5mg, and repeat if necessary after an interval of at least 2 hours.

For adults aged 18-65, start at 2mg. The preference is always for oral administration, but the same dose can be given IM if necessary. If the agitation or distress persists, haloperidol can be given two or three times daily. Maximum recommended doses are 1.5mg BD in the elderly or 5mg BD in adults aged 18-65.

Contact liaison psychiatry on bleep 6501 for review if regular antipsychotic medication is needed. Repeat the cognitive assessment (AMT) every few days at least.

For the management of delirum tremens and Wernicke's encephalopathy, see *Alcohol withdrawal* below.

In complex presentations (eg. diagnostic uncertainty, atypical symptoms, psychiatric co-morbidity, marked behavioural disturbance), as well as patients who require medication and have contra-indications to haloperidol (such as possible Lewy Body dementia), contact Liaison Psychiatry (bleep 6501 in working hours, bleep 6969 for out of hours emergencies). Such patients usually require joint management with the psychiatric team, and regular psychiatric review if the delirium persists.

Sedation of aggressive or violent patients

Sedative medication should only be used when attempts to verbally de-escalate the situation have failed, and enforced medication is only used as a last resort, where there is risk to the patient, staff, or others. Obtain advice from the psychiatry team (office hours bleep 6501, out of hours bleep 6969).

Agitated manic, delirious or psychotic patients can usually be sedated with lorazepam 1-2mg PO or IM (up to a total dose of 4mg/day). If the patient is violent or aggressive, an enhanced sedative effect is achieved by giving an antipsychotic in addition to lorazepam, eg. olanzapine 5-10mg PO, or haloperidol 5mg PO or IM. If an antipsychotic is given, check ECG (particularly QTc interval) when possible after administration. For elderly patients and those with cardiac disease, use lorazepam alone, and for elderly patients use lorazepam 0.5-1mg PO or IM. For all patients careful monitoring is needed after sedation – pulse, BP and respiratory rate at 15 minute intervals initially.

Oculogyric crisis and acute dystonia
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, but remember that the patient may be unable to swallow.
Response to IV administration will be seen within 5 minutes and IM in about 20 minutes. Eg. procyclidine 5-10mg by IM or IV injection; procyclidine 5-10mg orally, tablet or liquid (BNF maximum 30mg daily).

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*). High Potency Pabrinex is diluted in 50-100ml normal saline, and given by IV infusion over 30 mins.
Many patients with Wernicke's encephalopathy do not present with the full triad of signs, so onset of confusion in an alcohol dependent patient is a clear indication for urgent Pabrinex treatment. If IV infusion not possible, initial doses may have to be given IM. Anaphylaxis is a rare but recognised complication.
Oral vitamins should be used following parenteral vitamin treatment if required.

Patient's condition	Dose required
Incipient Wernicke's encephalopathy (Any one of three main symptoms: confusion, ataxia, ophthalmoplegia)	**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.**
At-risk (significant weight loss, poor diet, signs of malnutrition)	**Prophylaxis** **One pair High Potency Pabrinex ampoules OD for 3-5 days.**
Lower risk	**Thiamine 100 mg orally four times per day, plus vitamin B compound strong 2 tablets daily during detox.**

Alcohol withdrawal management

Example regimen	CHLORDIAZEPOXIDE starting dose		
Stabilisation phase	**20-25 mg**	**30 mg**	**40 mg***
Day 1 (starting dose)	**20 QDS**	**30 QDS**	**40 QDS***
Day 2	**15 QDS**	**25 QDS**	**35 QDS**
Day 3	**Continue below**	**20 QDS**	**30 QDS**
Day 4		**15 QDS**	**25 QDS**
Day 5		**Continue below**	**20 QDS**
			Continue below

*****Regular chlordiazepoxide in excess of 30 mg QDS should only be prescribed in cases where severe withdrawal symptoms are expected, ie patient reporting more than 50 units daily (eg two full bottles of spirits daily), follow the protocol above starting at 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.

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: i.e frequency, usual daily amount, and note that around 15 units there is an increased risk of dependence and therefore withdrawal symptoms. Initial dosage should be individually titrated against severity of withdrawal symptoms and signs (symptom-triggered). This should be monitored 2 hourly or more frequently during the first 24 hours and until symptoms stabilised. Regular dose may need to be increased if initial dose does not alleviate withdrawals, therefore do not write up subsequent reduced doses until symptoms have stabilised. In the most 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 30mg QDS, PRN should be 20-30mg).
Consideration should be given to raised doses at night time, as there is often 8 hours between drug dispensing times. (eg 40-30-30-40 instead of 35 QDS). More frequent doses may be preferable to increasing above 40mg due possibility of sedation/agitation between doses).

Reduction phase	**(or start dose 10-15mg)**		
Day 1	**10 QDS**		
Day 2	**10 TDS**		
Day 3	**5 TDS**		
Day 4	**5 BD**		
Day 5	**5 nocte**		

Liver impairment. Caution is required in prescribing benzodiazepines in hepatic impairment. Seek advice (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, following the regimes above, titrated to symptoms. If DTs have developed during a detox, additional doses of chlordiazepoxide are needed, for example 20mg PO stat, followed by close monitoring of the patient, and re-evaluation after 2 hours. If oral administration is not possible, an alternative is lorazepam 1-2mg IM. In patients who remain severely disturbed see "severe behavioural disturbance" above.
All patients with DTs should also be treated with Pabrinex (*see above*) as soon as practicable, in view of the difficulties in excluding Wernicke's encephalopathy.
Once the patient is stabilised, the regular chlordiazepoxide dose should be re-written to reflect the dose needed to control the DTs. For example, if a patient had reached 15mg QDS chlordiazepoxide in a detox before developing DTs, and then required two additional doses of 20mg over 24 hours, his daily dose requirement was 15mgx4 +

20mgx2 = 100mg. Therefore he should be prescribed 25mg QDS, and no further reductions made to the chlordiazepoxide dose until the symptoms begin to resolve.
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.
The Alcohol 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.

APPENDIX 1

CARDIAC MARKERS IN PATIENTS WITH SUSPECTED ACUTE CORONARY SYNDROMES

Link consultant: Dr Paul Collinson

In a patient in whom an acute coronary syndrome is suspected, measurements of cardiac markers should be used to confirm or exclude myocardial infarction. The tests currently available provide measurement of creatine kinase (CK) and cardiac troponin 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.

APPENDIX 2

DO NOT ATTEMPT (DNA) CARDIOPULMONARY RESUSCITATION(CPR)

Link: Paula McLean, Resuscitation Service Manager

All patients will be automatically assumed to be appropriate for CPR in the event of cardiac arrest unless a completed DNA CPR form is visible in the patient's notes.
Senior medical and nursing colleagues should support anyone initiating CPR where DNA CPR documentation has not been carried out. A DNA CPR decision only applies to CPR and *not* to other aspects of care (eg analgesia, antibiotics, suction, treatment of choking or anaphylaxis etc. – which are sometimes loosely referred to as resuscitation.
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 November 2007).
2. End of Life Guidance for Doctors – General Medical Coucil (July 2010)
http://www.gmcuk.org/End_of_life.pdf_32486688.pdf
3. Clinical Ethics Committee (email: cec@sghms.ac.uk or helpline ext 4971) or Legal Services (ext 2901).
*****http://stginet/Procedural%29documents/Patient%20related/Patient_Management/Clin_1_1.pdf

APPENDIX 3

PERIOPERATIVE MANAGEMENT OF DIABETES MELLITUS

Link consultant: Dr Grainne Nicholson

Patients with diabetes should ensure that they maintain good glycaemic control (**HbA1c < 8.5%**) in the months and weeks prior to surgery, as this improves the healing process post-operatively. Patients with diabetes in the peri-operative period and nil by mouth, should be managed according to the following protocol. (*For the management of diabetic ketoacidosis, see page 29).*

Pre-operative assessment

- Patients with type-2 diabetes managed with diet alone, need no intervention prior to surgery.
- Metformin should be stopped 48 hrs pre-operatively as it can cause lactic acidosis, especially when renal function is impaired.

Before surgery

- To ensure that blood glucose is controlled within normal limits before surgery (target range: 5-10mmol/L), a 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 diabetic team, the anaesthetic team or both.
- An intravenous cannula should be inserted the evening before surgery.
- Patients should receive their usual doses of oral anti-diabetic and/or insulin (except for long-acting) on the day prior to surgery, including evening doses. For long-acting insulins, normal evening doses should be halved and given with their evening meal.
- From 6am on the morning of surgery, maintain glycaemic control by starting a glucose/potassium/insulin (GKI) regimen, using the following infusions:
  (i) **500ml 10% dextrose with 0.15% KCl** (premixed bag at **85** ml/ hr. (If the patient has renal impairment, omit the potassium); *and*
  (ii) Soluble insulin (Human Actrapid) 50 units in 50ml 0.9% sodium chloride at the rate shown in the table below.
  These infusions should run simultaneously and through the same cannula.
- Bedside blood glucose measurements must be taken hourly from 6am and used to guide and adjust the rate of insulin infusion according to the table below.

Blood Glucose (mmol/l)	Rate of insulin infusion (units/hr)
<3.9 - 4	**0.5**
4 – 6.9	**1.0**
7 – 9.9	**2.0**
10 – 14.9	**3.0**
15 – 19.9	**4.0**
20	**5 and call doctor**

Patients with type-1 diabetes should not be left without any intravenous insulin running. If blood sugar is <4mmol/l, ***give Glucose 10% at 100mls/hr; if no increase after 30mins give glucose 20% 100mls as a bolus and repeat blood glucose after 15mins****. Continue insulin infusion at 0.5 units per hour.*

- If glucose is temporarily stopped, eg. on route to theatre, insulin must also be stopped temporarily.
- Never administer sodium chloride 0.9% as the sole intravenous fluid to a patient receiving insulin. If a patient is fasting for several days and there is concern regarding hyponatraemia, consult the anaesthetist.
- Diabetic patients should be first on the theatre list where possible, to minimise duration of starvation.

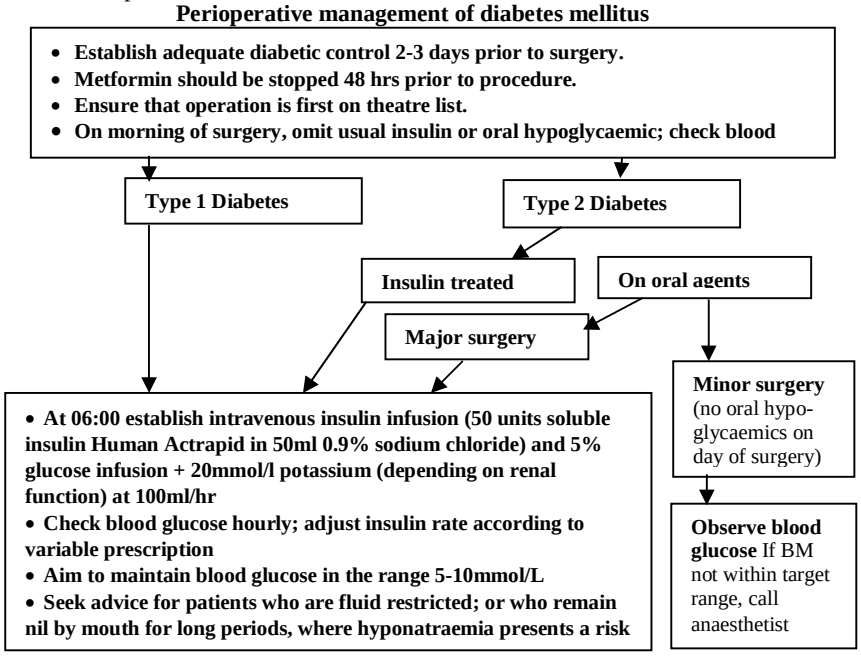
Treatment of hypoglycaemia for patients on a perioperative diabetic regimen

- If blood glucose is <4mmol/l, **give Glucose 10% at 100mls/hr; if no increase after 30mins give glucose 20% 100mls as a bolus and repeat blood glucose after 15mins**. Inform the doctor at once.
- Do not administer oral hypoglycaemics on the day of surgery. A patient on the afternoon list must still be stabilised on intravenous glucose, insulin and potassium.

Post-operative management

- Blood glucose measurements should be taken hourly until the reading is stable and within normal range. Readings can then be taken 2-hourly. Serum potassium should be measured on alternate glucose samples.
- The first meal following surgery should be eaten while the intravenous insulin infusion and glucose **10%** infusion continues, to check that the meal is tolerated. If tolerated, patients on subcutaneous insulin or oral hypoglycaemics can return to their usual regimen. **STOP IV insulin infusion 1hr AFTER the first dose of subcutaneous insulin**
- For patients taking metformin who receive contrast media, metformin may be restarted 48 hours after the procedure.

Perioperative management of diabetes mellitus



ENTERAL/PARENTERAL FEEDING APPENDIX 4

Link consultant: Dr Penny Neild

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 MUST score. 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, infectious complications and maintenance of gut function. There is no clinical advantage in embarking on IV feeding if the patient is expected to resume oral-enteral feeding within 5 days unless there is a history of malnutrition. The Nutrition Support Team operates at St.George's in order to provide advice on the management of difficult problems and to review patients for whom PN is being considered. Once a patient starts PN the ward dietitian and nutrition nurse specialist continues to review the patient and the NST will review the patient on a weekly ward round. It is important to be aware of the risk of re-feeding syndrome in patients initiated on nutrition support, particularly those with low BMI, significant weight loss or inadequate oral intake for >10 days. Please contact ward or NST pharmacist for advice on monitoring and replacement of electrolytes and other micronutrients **or refer to guidance in the Nutrition Support Policy on the Trust intranet**. PN should not be seen as an emergency intervention and will not be instigated outside weekday working hours or at weekends except on a pre-planned basis (Friday referrals should be made before midday). Any team member may be contacted via: Dr Penny Neild, Consultant Gastroenterologist (x3429); Tracey Marshall, nutrition nurse specialist (blp 8050); the Gastroenterology SpR (blp 6590); Emma Ryan/Russell Soanes, PN Pharmacist (blp 7554); Alison Green, Senior Dietitian (blp 6171).

APPENDIX 5

ASSESSING METABOLIC ACIDOSIS – THE ANION GAP

Link consultant: Dr Iain MacPhee

Metabolic acidosis, which may be fatal, will sometimes present acutely in the A&E department. The patient will be hyperventilating and, unusually for a 'breathless' patient, will be comfortable lying flat. The condition is characterised biochemically by a fall in arterial pH to less than 7.37 in association with a raised plasma concentration of H^+(> 43nmol/L) and a low plasma HCO_3^-.

Mechanisms:

- net gain of acid (increase in endogenous production or exogenous administration) *eg.* diabetic ketoacidosis, aspirin poisoning;
- net loss of alkali *eg.* loss from intestine (diarrhoea) or renal tract (renal tubular acidosis);
- failure of renal acid excretion in patients with normal production of acids, *eg*. chronic renal failure, renal tubular acidosis.

Calculations

In health the total for the positively or negatively charged electrolytes is around 150mmol/L. When the 4 major plasma electrolytes (sodium, potassium, chloride and bicarbonate) are considered the sum of $[Na]^+ + [K]^+$ is greater than $[C1]^- + [HCO_3]^-$ 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 HCO_3^- there will be an equivalent rise in $[C1]^-$ 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 HCO_3^-, as in diarrhoea, proximal renal tubular acidosis

· Decreased renal acid excretion *eg*, distal renal tubular acidosis

Increased anion gap

Condition	*Unmeasured Anions*
Lactic acidosis	Lactate, phosphate, urate
Ketoacidosis	
Diabetic	Ketone bodies (acetone, acetoacetate, ß–hydroxybutyrate)
Starvation	Acetoacetate, ß–hydroxybutyrate
Inborn enzyme defects	
Intoxication	
Methanol	Formate
Ethylene glycol	Glycolate, oxalate
Alcohol	ß–hydroxybutyrate, lactate, acetoacetate
Salicylates	Ketones, lactate, salicylate
Paraldehyde	Acetate
Uraemia	Sulphate, phosphate

It is important to realise that the ability to respond to the worsening acidosis by hyperventilation and elimination of CO_2 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 thera-peutic goal is to raise the arterial pH to about 7.20, a level at which arrhythmias are less likely and cardiac contractility is restored. Do not attempt to fully correct the pH as continuing hyperventilation will make the patient alkalotic and may precipitate tetany.

- In patients with renal failure who are acidotic and volume deplete, give $NaHCO_3$ 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 page 29*.
- 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 $NaHCO_3$ 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 $\geq$5.4.

APPENDIX 6

CONSIDERATIONS BEFORE ATTEMPTING LUMBAR PUNCTURE

Link consultant: Dr Hannah Cock

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 page 42*),

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 or MRI scan has excluded any clinically-suspected space-occupying pathology.
   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. In this syndrome it is often worth delaying the LP rather than doing it at the onset of symptoms, as this will improve the chances of a positive 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.

Contraindications to lumbar puncture

1. A known intra-cranial mass lesion, for example tumour, haematoma, abscess or cerebral oedema. Remember that the swollen brain seen in patients with encephalitis or infarction may act as mass lesion.
2. Papilloedema (if benign intracranial hypertension is suspected contact neurologist).
3. Coma or rapidly increasing depression of consciousness (raised intracranial pressure is likely).
4. Focal neurological signs.
5. Prolonged or frequent epileptic seizures.
6. Any possibility of intra-spinal mass lesion.
7. Infection in lumbar region.
8. Anticoagulation, coagulation defect or low platelet count (see Appendix 7).

Potential hazards of lumbar puncture

1. Deterioration of brain stem function which may lead to death due to coning in the presence of raised intracranial pressure**.**
2. Deterioration of spinal cord function due to an obstructive intraspinal mass lesion.
3. Post LP CSF leakage through the puncture site. This may exacerbate 1, 2, or lead to 'low pressure' headache. The risk of leakage can be reduced by using a 22g blunt-tipped needle.
4. Iatrogenic infection.
5. Epidural haematoma.
6. Local damage to intraspinal structures.

APPENDIX 7

PROTECTING ANTICOAGULATED PATIENTS FROM BLEEDING

Link consultant: Dr Muriel Shannon

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 while urgent confirmatory tests are performed.

HEPARIN

Monitoring heparin therapy.

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 after 18-24 hours, in:

- patients with a serum creatinine above 150µmol/L
- patients weighing >100kg
- women who are pregnant and receiving therapeutic doses

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

Where therapeutic anticoagulation is required in the presence of a high bleeding risk, recent major surgery or severe renal impairment, *unfractionated heparin* can be used as it can be rapidly reversed with protamine sulphate. Given by IV infusion, it has a short half life of 1½ hours.

In patients receiving continuous IV infusion of *unfractionated heparin*, monitoring is essential. A scheme for instigating and monitoring use of unfractionated heparin is as follows:

1. Measure APTTR at start of therapy. Give a 5000 units loading dose as a bolus injection IV.
2. **In patients at risk of bleeding consider omitting bolus dose**.
3. Start IV infusion, at **2mL/hr into a 50mL syringe**; draw 25,000units of unfractionated heparin made up to 50mL with 0.9% sodium chloride (resulting concentration of 500units/mL).
4. The target therapeutic range for APTTR is 1.5-3.5. Check APTTR 6 hours after infusion started (and after any dose change) and adjust as follows:

APTTR	ACTION
>6	stop for 1 hr; reduce by 1mL/hr (500units/hr)
5.3-5.9	reduce by 0.6mL/hr (300units/hr)
4.7-5.2	reduce by 0.4mL/hr (200units/hr)
4.1-4.6	reduce by 0.2mL/hr (100units/hr)
3.6-4.1	reduce by 0.1mL/hr (50units/hr)
1.5-3.5	NO CHANGE
1.2-1.4	increase by 0.4mL/hr (200units/hr)
<1.2	increase by 0.8mL/hr (400units/hr)

5. Repeat APTTR daily while on unfractionated heparin. Check platelet count **at start of therapy and after 5 days**. Where therapeutic subcutaneous *unfractionated heparin* (15,000units sc bd) is used, it should be monitored 6 hrs after injection and then daily with the anti Xa assay or APPTR; discuss with haematology.

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", eg 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. Do not give any drugs by IM injection.

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 is less effective against LMWH but can still provide some reversal of anticoagulation given at the same dose. It may need to be repeated if bleeding persists as it has a shorter half-life

than LMWH. Administration of plasma products will **not** reverse heparin anticoagulation. The risk of thrombosis during the period of heparin withdrawal and control of bleeding does not outweigh the risk of continued bleeding – if bleeding persists seek Haematology advice.

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 clumping and loss of platelets. Which can lead to explosive thrombotic disease due to platelet emboli. This 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. Alternative anti-thrombotic agents such as danaparoid or **lepirudin** should be substituted. Seek **urgent** advice from the Haematology Department on-call registrar.

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 on 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 >4.5 with no bleeding. Reduce dose or withdraw warfarin for 1–2 days and review. Stop for 1-2 days if INR >5.0. In addition if INR >8.0 give 0.5–1.0 mg Vitamin K orally and repeat INR in 24-48 hours. Omit warfarin until INR <4.0.

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. Get advice from a cardiologist or haematologist.

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 Haematology Team: SpR blp 6068, out of hours SpR on call blp SG366 or via switchboard.

Recommended doses of PCC: INR <4.0: 20units/kg;
INR >4.0: 30units/kg

The single dose should not exceed 5000units. Only if concentrate is unavailable should FFP should be infused (15mL/kg). 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.

NEW ANTICOAGULANT AGENTS. **These include**:

Fondaparinux (being used for initial treatement of acute coronary syndromes).

Rivaroxaban (in use for prevention of VTE after major lower limb orthopaedic surgery).

Dabigatran is in limited used for stroke prevention in atrial fibrillation.

Bleeding in patients on these new anticoagulant agents is not reversible by current conventional means. Urgent Haematology advice should be sought.

Comment [MS1]: A list of such drugs in use in the UK and the test used to detect them is being prepared along with recommendations for treatment of major bleeding and reversal for emergency surgery.

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 Kaletra, (200/50mg) two tablets 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, Kaletra, domperidone and loperamide 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 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 A&E consultant (or deputy) or by the Nurse in Charge of the A&E department.

- 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 (ED consultant), Nursing Coordinator (Clinical Site Manager), 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).

- **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 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 Engineering and 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 Emergency Department 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 4454. 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. 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. 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:

PRIORITY	DESCRIPTION	COLOUR	AREA
1	*immediate*	red	Resuscitation Room
2	*urgent*	yellow	Majors
3	*delayed* (ambulatory)	green	Fracture Clinic
4	*expectant*	blue	

Children will be triaged as above, but directed to the Resuscitation Room, paediatric area of A&E and (if necessary) Fracture Clinic.
The Medical Coordinator will nominate a senior clinician to take responsibility for each of the clinical areas in the A&E department:
- Resuscitation Room – generally SpR in anaesthetics on call for St James Wing
- Majors – generally consultant surgeon or physician (or in his/her absence an SpR)
- Minor casualties area located in the Fracture Clinic – generally on-call SpR Plastic Surgery

A Triage Coordinator (A&E registrar) will allocate teams of doctors and nurses to patients in the treatment areas of the A&E department.
A Theatre Coordinator (consultant anaesthetist on-call for St James Wing) will coordinate work in theatres and allocate anaesthetic staff as required.
- **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 Ward (ext 3299). Vernon Ward (ext 3197) will provide a second admission ward if required. For children, Jungle Ward will be used (ext 2034/2035).

APPENDIX 10

GUIDE TO THERAPEUTIC DRUG LEVEL MONITORING

Gentamicin[1] *Once-daily regimen* - sampling time: trough 18-24 hours post dose; therapeutic range: trough <1 micrograms /mL.
Conventional regimen - sampling time between 3rd and 4th dose: trough immedi-ately prior to next dose, peaks 60min post IV dose. Therapeutic range: trough <2 micrograms/mL, peak 5-10 micrograms/mL for streptococcal or enterococcal endocarditis trough <1 microgram /mL, peak 3-5 micrograms/mL; time to steady state: 12-40 hrs (longer in renal failure).
Vancomycin[1] In patients receiving vancomycin by intermittent infusion - sampling time varies according to renal function (for further information *see*: http://stginet/Units%20and%20Departments/Antimicrobial%20prescribing%20information/Vancomycin%20guidelines%202009.pdf)
Trough levels should be taken immediately prior to next dose; therapeutic range: trough 10-15micrograms/mL, although levels of 15-20micrograms/mL may be required for deep-seated infections such as endocarditis and osteomyelitis – contact Microbiology for advice; time to steady state: 30-40 hrs (longer in renal failure). In patients receiving vancomycin by continuous infusion, sample during the infusion; therapeutic range 15-25 micrograms/mL.

Amikacin *Once-daily regimen* - sampling time: trough 18-24 hours post dose; therapeutic range: trough <5 micrograms /mL.
Carbamazepine[2] Sampling time: immediately prior to next dose; therapeutic range: single therapy **4**-12mg/L, multiple therapy (i.e. one or more drugs used in addition to carbamazepine) 4-8mg/L; time to steady state: 2-4 weeks after start of treatment, or 4-5 days after dose change.
Digoxin[2] Sampling time: at least 6 hours post dose or immediately pre-dose; therapeutic range: 0.9-**2.0** micrograms/L; time to steady state: 7 days (longer in renal failure).
Lithium[2] Sampling time 12 hours post dose; therapeutic range: as treatment - 0.4 –**1.0** mmol/L, as prophylaxis - 0.5-0.8mmol/L; time to steady state: 3-7 days.
Phenytoin[2] Sampling time: immediately prior to next dose; therapeutic range: **5**-20mg/L (interpretation difficult in renal failure, low albumin, raised bilirubin); time to steady state: 7 days or longer.
Theophylline Sampling time: liquid preps - peak 2 hours post dose, SR tablets – peak 4 hours post dose, trough immediately prior to next dose; therapeutic range: 10-20mg/L; time to steady state: 2 days.

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

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

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

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)

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
- 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.
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

CrCl	>60 ml/min	>60 ml/min	40 - 60 ml/min	10 - 40 ml/min	*<10 ml/ min*
Patient age	<65 yrs	≥ 65 yrs			*All patients*
Dose	5 mg/kg	5 mg/kg	4 mg/kg	3 mg/kg	*2 mg/kg*
Take levels	18-24 hrs post 1st dose	18-24hrs post 1st dose	18-24 hrs post 1st dose	18-24 hrs post 1st dose	*48 hrs post 1st dose*
Timing of 2nd dose	*24 hrs post 1st dose*	*Await levels < 1 mg/L before re-dosing*			

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

Level	*Trough level interpretation*
< 1.0 mg/L	*Continue current dosing regimen*
1-2 mg/L	*Recheck levels 12 hrs later – withhold dose pending results*
> 2mg/L	*Recheck levels 24 hrs later – withhold dose pending results*

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 to15mg/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 1: Loading dose

Weight (actual body weight)	< 60kg	60 - 90kg	*> 90kg*
Loading dose	1g	1.5g	*2g*
Fluid (NaCl 0.9% or glucose 5%)	250ml	500ml	*500ml*
Infusion period	*2 hours*	*3 hours*	*4 hours*

2) Calculate the initial maintenance dose based on the patient's creatinine clearance using the Cockroft-Gault* equation (page 80)

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

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

Table 2. Initial Maintenance Dose

Creatin-ine clear -ance* (ml/min)	Mainten-ance Dose	Start time after LD & future dosing interval	Volume of fluid (NaCl 0.9% or glucose 5%)	Infusion Period	*Time of first trough level*
>110	1.5g	12 hours	500mL	3 hours	*Before 4th dose*
90-110	1.25g	12 hours	250mL	2 ½ hours	*Before 4th dose*
75-89	1g	12 hours	250mL	2 hours	*Before 4th dose*
55-74	750mg	12 hours	250mL	1 ½ hours	*Before 4th dose*
40-54	500mg	12 hours	250mL	1 hour	*Before 4th dose*
30-39	750mg	24 hours	250mL	1 ½ hours	*Before 3rd dose*
20-29	500mg	24 hours	250mL	1 hour	*Before 3rd dose*
<20 (No dialysis)	*500mg*	*48 hours*	*250mL*	*1 hour*	*Before 2nd dose*

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 below.

Table 3. Trough level interpretation & maintenance dose adjustment when levels 10-15mg/L are required

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

- 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.

BLEEP AND PHONE NUMBERS

Medicine F1	**Bleep**	**Surgery F1**	**Bleep**
Drs Clark & Forton	6424/7856	Mr Reddy, Adamo, Wan	6364/6300/6367
Drs Bourke, Kiely & DeSilva	6576/6335	Mr Melville	7161
Drs Groves, Pollok, Poulis	6577	Mr Hagger	6581
Drs Patel & Simmgen	6225/6322/6252	Professor Kumar	6362
Drs Panahloo & Seal	6559/6578	Mr Mokbel	6361
Drs Baker, Chua & Shoults	6326	Mr Sharma & Mr Banerjee	6582/7352
Drs Ong & Draper	6563/6580	Mr Powell (plastics)	6279
Dr Antonios & Dr Khong	6135/6324	Miss Daly (orthopaedics	6556
Geriatrics		Mr Bircher (orthopaedics)	6583
Dr Hastie & Dr Coles	6539	**Vascular Surgery**	
Dr Myint & Dr Cloud	6221	Mr Loftus, Mr Loosemore, Mr	6536/6555
Drs Cottee & Martin-Marero	6554	McFarland, Prof. Thompson	
Ophthalmology		**Intensive Care** Dr Newman	7010
Mr Thompson	6252	**Paediatrics** Professor Walters	7011

BLEEP AND PHONE NUMBERS cont.

Specialty SpR Referrals	**Bleep**		**Bleep**		**Bleep**
Cardiology	6002	Neurology	7277	Haematology	6068
Respiratory	6614	Neurosurgery	7242	Infectious Disease	7568
Gastroenterology	7464	Stroke	7317	Surgical SpR on-call	7655
Care of Elderly	7662	Blood Pressure	6602	Surgical F2 on-call	7530
Diabetes/Endocrine	7778			Surgical F1 on-call	7630

Nurse Bleeps	**Bleep**		**Bleep**		**Bleep**
Heart Failure	7376	GI Bleed/Nutrition	7698	Diabetes	6236
Respiratory	SG302	Parkinson's	A8825	Infection Control	6797
DVT	7380	Anticoagulation	6200	HIV Counsellor	SG331
Palliative Care	6796	Sickle Cell	H3419	Psychiatry Liaison	6501
Renal Anaemia	7127			Neuro Ward Referrals	
Bed Manager 6447/6568/ 1667		Acute Rehab Team	7564 SG475	Hospital Intervention Team	6622
Venous Access (Ext 7199)	6099				

Other (Extensions)	**Ext**		**Ext**
Neuro Ward Referrals	2470	Multiple Sclerosis	4162

DEPARTMENTS	**Ext**
Audiological Medicine	
Receptionist	1880
Cardiothoracic	
Echo	3650
24 ECG	1386
ECG	1385/1387
St James **bleep**	6641
Lanesborough **bleep**	6403
Knightsbridge **bleep**	6436
Casualty X-ray	1296
Mobile X-ray blp	6345/ 6284
Chest Medicine	
Receptionist	3318/1273
Lung function tech	1667
Chest Clinic Fax	3769
Clinical Biochemistry	
Enquiries/results	5862/3/4
Urgent requests	5871
Diabetes/Endocrinology	
appointments	1429
Dermatology	1997
EEG/EMG	4632
Genito-urinary Medicine	3353/4
Haematology	
Blood bank	5471/5477
Enquiries	5468/5470
Haemostasis	5479
Histopathology	
Enquiries	5264
PMs	5240
Frozen section bookings	5257/ 5264
Cytology	5266/ 5268
ITU	1307
Nurses station	3294
Maxillofacial Unit	1244
Medical Microbiology	
Consultant/registrar	1970
Enquiries/results	5693
Norman Tanner Unit	1564
Nursing	1491
Patient Affairs	3411/ 3410
Palliative Care Team	3311
bleep	6796/ 6796
Medicines Information	1759
Psychiatry Emergency Clinic	5288
Switchboard	
SGH	1000
SGUL	5000
Springfield	#6600