St George's University of London

St George's Healthcare NHS NHS Trust

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

St George's Hospital
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46th Edition

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GENERAL POINTS

- "Guidelines for the Management of Common Medical Emergencies and for the Use of Antimicrobial Drugs" have been produced at St George's Hospital since August 1979 and are probably the oldest established set in the UK. To keep them up-to-date they are reviewed every 6 months. Then, as now, articles are written by specialists, edited to ensure clarity, approved by colleagues and designed to "advise junior staff what to do when confronted with some of the more common medical emergencies".
- With all clinical guidelines, questions are asked about their validity, about the sources of the advice given and whether it is evidence based. In the "Grey Book" (a name coined by the students when the cover went grey in 1984), the words, sentiments, sequences and priorities are the product of repeated honing. Changes in hospital organisation (wards, contact numbers, pharmacy provision), staffing (specialist teams), facilities (endoscopy, scanning, infusion pumps) and the findings of clinical audit all have a profound effect on advice. But all of these have to be integrated with evidence-based material. In editing the Grey Book every attempt is made to ensure that statements are fully compatible with the advice given by the British National Formulary, the Drug and Therapeutics Bulletin, the various professional bodies (such as the British Thoracic Society), the Royal Colleges (particularly the Royal College of Physicians; RCP), National Service Frameworks and NICE. The references used to support the advice are on the Intranet version, which can be found at the St George's NHS Trust Intranet website. If you have any comments or questions please send them to the link consultant named at the beginning of the section concerned.
- **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.
- A patient admitted as a medical emergency who has been a medical in-patient **in the last 3 months** should, in general, be returned to the care of the consultant of the previous admission. Handback should be by 9.00am of the next working day, or straight away (but not later than 2.00pm) if admitted during the morning and the receiving team agree. If the patient is currently attending other medical outpatient departments the relevant consultant should be informed and the patient offered back. These handback rules do not apply if the patient is admitted with a drug overdose or alcohol intoxication.
- When medical problems arise the arrangements for seeking advice are as follows. During the working day, or when on in-take, always 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 intaking 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 or urgent referrals from General Practitioners, priority should be given to patients from the Wandsworth and Merton and Sutton areas, as well as to those from elsewhere who have 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 Accident & Emergency, will be treated free under the NHS. Some, such as visitors from outside the EU and from countries without a reciprocal arrangement, may need to pay. If you believe that a patient may not reside permanently in the UK, please contact the Overseas Patients Department to check eligibility for NHS treatment – Monday to Friday, ext. 0895, 3439; bleeps 7335, 7060; or aircall SG 303. Weekends and Bank Holidays (8am-10pm) via aircall SG 303.

Joe Collier (Editor; jcollier@sgul.ac.uk)

CARDIAC ARREST

(Link: Paula McLean, Resuscitation Service Manager)

Cardiac output must be restored within minutes if the patient is to have any chance of survival. As soon as cardio-respiratory arrest is **confirmed through a simultaneous 10-second breathing and pulse check**, and provided that there are no contra-indications to proceeding (see Appendix 2), call for the arrest team (St. George's Hospital dial 2222) and a defibrillator/resuscitation trolley. If the arrest was witnessed **and** monitored, and **in the absence of a defibrillator**, deliver a firm, sharp blow with the fist to the lower half of the patient's sternum. **If life signs are absent then immediately start external chest compressions regardless of the rhythm on the monitor**. **Compressions should be applied at a rate of 100/min with a ratio of 30 compressions to 2 ventilations**. **If the arrest was not witnessed go straight to the compression stage**.

Cardiac arrest is associated with four underlying disorders of heart rhythm: ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), asystole, and pulseless electrical activity (PEA). The treatment of VF and VT are identical, as is the treatment of asystole and PEA (see flow diagram on page 5).

Ventricular fibrillation is the most common immediate cause of sudden cardiac death and the most amenable to treatment. Patients can be successfully defibrillated for some time after cardiac arrest but the chances of success and also of a favourable long-term outcome are optimal only in the first ninety seconds, unless basic life support is instituted. In a patient with ventricular fibrillation or pulseless ventricular tachycardia it is of paramount importance that there is minimum delay in the administration of defibrillating shocks. Even basic cardio-pulmonary resuscitation (CPR) using cardiac massage and bag-**valve**-mask ventilation is of secondary importance. Basic CPR should start whilst the patient and defibrillator are being prepared but must cease as soon as the charge is ready to be administered.

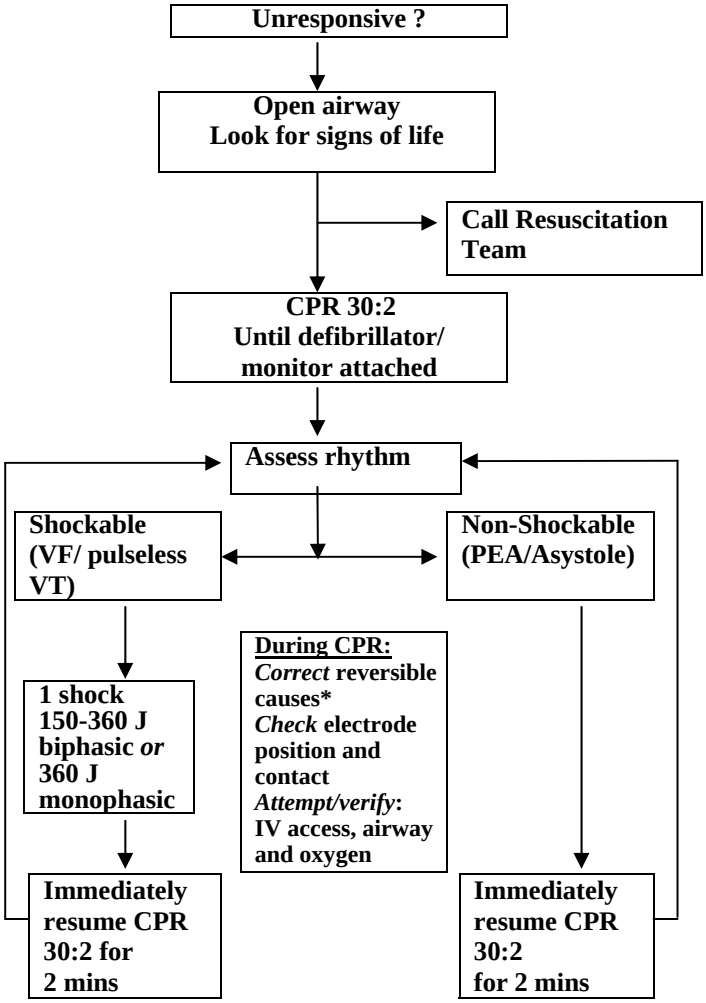
To defibrillate, **place self-adhesive pads** (**the preferred approach**) **or defibrillator gel pads on the patient's chest – one below the right clavicle, one in the V6 position in the midaxillary line**. **If using defibrillator paddles** these should be applied with firm pressure to ensure uniform contact. The person using the defibrillator is responsible for informing team members that the defibrillator is to be charged, giving the order to "stand back" and checking that everyone has done so before discharging the machine. Remember to check cardiac rhythm for VF or VT immediately before the defibrillator is discharged. After each shock (**150-360J biphasic**, **or 360J monophasic**) **and in the absence of life signs**, **immediately resume CPR regardless of rhythm**. Each shock should be followed by 2 mins of CPR ('single shock strategy').

Secure the airway as soon as possible, give 100% oxygen, and get vascular access. Chest compressions should continue uninterrupted once the airway is secure. Central venous access (e.g. internal jugular) is optimal for drug delivery, once the necessary expertise is available. IV access should be secured initially via an antecubital vein. Keep the line open with 0.9% saline which should also be used to "flush through" any drug.

After 2 minutes of CPR, if the patient is still in VF or has pulseless VT, repeat the single shock at 150-360J biphasic, or 360J monophasic; if intubation and/or IV

access has not been achieved, they should be attempted **during the next 2 minutes of CPR**. Give adrenaline 1mg every **4** minutes throughout the resuscitation attempt.

Adult Advanced Life Support Algorithm



***Reversible causes:**
Hypoxia, hypovolaemia, hypo/hyperkalaemia/metabolic, hypothermia, tension pneumothorax, tamponade (cardiac), toxins, thrombosis (coronary or pulmonary).

If VF/VT, **give the adrenaline immediately before the 3rd shock**, **and every alternate cycle thereafter**. **If PEA/asystole**, **give it as soon as IV access is achieved and then every alternate cycle thereafter**. If IV access has not been obtained, but an endotracheal tube is in place, the adrenaline may be given down the tube. Give **3mg diluted in 10-20ml sterile water**. Atropine (3mg) should be given for asystole or a bradycardic (heart rate <60/min) PEA. Amiodorone (**300mg**) should be given after the third shock for refractory VF/pulseless VT. Magnesium should be given as a **2g IV bolus** (4ml of 50% magnesium sulphate) for refractory VF if there is any suspicion of hypomagnesaemia (e.g. patients taking a potassium-losing diuretic).

Measure blood gases and pH using a sample from a central venous line. Bicarbonate (50mL of 8.4% solution) may be given if the central venous pH is less than 7.0 and base-excess less than –10, or if the arrest is associated with tricyclic poisoning or hyperkalaemia, or is prolonged (>25 minutes). During resuscitation look for, and if possible treat, reversible causes. These include hypoxia, hypovolaemia, hypo-glycaemia, hyper/hypokalaemia, hypothermia, tension pneumothorax, tamponade, thrombo-embolic/mechanical obstruction.

In persistent VF/VT, consider changing the paddle positions to a front-back axis. Trying a different defibrillator can also help. Attempts to resuscitate should not be abandoned while the patient is still in ventricular fibrillation or pulseless VT. The length of time attempting resuscitation is a matter for clinical judgment. However, resuscitation attempts should not usually last for more than one hour unless the patient is profoundly hypothermic.

SEVERE HYPERTENSION

(Link consultant: Professor Graham MacGregor)

Patients require admission and urgent treatment when blood pressure is known to have risen rapidly or is raised, such that the systolic pressure is equal to or above 220mmHg and/or diastolic equal to or above 120mmHg. Treatment depends on whether or not there is evidence of life-threatening end-organ damage

WHEN THERE IS ACUTE, LIFE-THREATENING ORGAN DAMAGE. The situation is a true emergency when there is acute and life-threatening organ damage as manifest by, for example, encephalopathy (headache, lethargy, seizures), intra-cranial haemorrhage, aortic dissection, unstable angina/acute myocardial infarction, acute left ventricular failure with pulmonary oedema or pre-eclampsia/eclampsia. The initial aim of treatment is to lower the diastolic pressure to about 110-115mmHg. This goal should be achieved within 2-6 hours. Too rapid a fall in pressure may precipitate cerebral or myocardial infarction or acute renal failure.

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 administered by intravenous infusion starting at

0.3μg/kg/min, increasing by 0.5μg/kg/min every 5 minutes, to a maximum of 8μg/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 2μg/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), a venodilator, is particularly useful in patients with either symptomatic coronary artery disease or acute left ventricular failure. The dose of GTN is 2-10mg/hr.

- Labetalol, a combined α- and β-blocker, is a logical option for patients with ischaemic heart disease or aortic dissection; 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. 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 200-300μg/min; this usually requires a maintenance dose of 50-150μg/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.

WHEN THERE IS NO LIFE-THREATENING ORGAN DAMAGE. If there are features of malignant hypertension, which may include retinal changes, mild LVF or renal impairment, but no evidence of life-threatening target organ damage, then the situation requires urgent medical attention rather than the 'emergency' approaches outlined above. Patients should be admitted to a medical bed and blood pressure reduced slowly; ideally the diastolic pressure should be lowered to about 110-115mmHG over 24-48 hours.

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. 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 (retard/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 or the long-acting (LA) preparations 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 reponse 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 either the Blood Pressure Unit or the Renal Unit.

- Diuretics should also 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 110-115mmHg, 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 months.

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 or other adrenal pathology, 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 5413).

ACUTE CORONARY SYNDROMES

(Link consultant: Dr Nicholas Bunce)

All patients arriving at the hospital with chest pain suggestive of myocardial ischaemia 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-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-ACS or neither.

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

- ST elevation of $\geq$ 0. 2mV in leadsV1-V3 or $\geq$ 0.1mV 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 NST-ACS are:**

- ST depression >0.05mV or symmetrical deep T wave inversion >0.2mV.

Repeat the 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 who can be contacted via bleep, switchboard or by phoning the catheter lab (ext 1370/1703/3274).

MANAGEMENT OF STEMI Refer the patient immediately to Cardiology for Primary Percutaneous Intervention (1° PCI); the target door-balloon time is within

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. Arrange for an echo-cardiagram 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 (contraindicated if the creatinine clearance is <50mls/min or potassium >5.0mmol/L).

Aspirin
As soon as possible give soluble aspirin 150-300mg 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.

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 hours as required). To reduce the likelihood of vomiting give it with either metoclopramide (10mg IV over 2 minutes) or cyclizine 50mg IV.

Oxygen
High flow oxygen should be given to all patients during the first 2 to 3 hours and thereafter to patients with overt pulmonary congestion and those with an arterial oxygen saturation of less than 90%.

Anticoagulation after 1° PCI
Give dalteparin (Fragmin) 120 IU/kg SC bd (maximum 10,000 IU bd) for 24-48 hours.

Blood glucose management
All patients with STEMI with a known history of diabetes mellitus or a blood glucose (BG) >11.0mmol should be managed with tight glycaemic control. Stop all existing oral hypoglycaemic therapy before, and for 48 hours after, coronary intervention. Refer newly diagnosed diabetic patients to the diabetes nurse specialist (bleep 6236).

If blood glucose (BG) >11.0mmol, 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 (1 unit/ml), titrate on a sliding scale as follows:

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 antidiabetic 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 <90mmHg. A reasonable choice is rampiril started at a dose of 2.5mg bd. Dosage should be slowly titrated upwards to the maintenance dose of 5.0mg bd, taking care to avoid a fall in blood pressure or reduction in renal function. If rampiril is not tolerated try candesartan (4mg od) or valsartan (80mg bd).

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 (ie. sildenafil) within 24 hours.

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

- a history of bronchospasm
- heart failure requiring therapy
- bradycardia of less than 50bpm
- second or third degree heart block
- cardiogenic shock
- allergy or hypersensitivity to beta-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
All patients should have a lipid profile performed on admission, and then started on a statin. For patients with an LDL <4.5mmol/L give simvastatin 40mg od; for patients with an LDL >4.5mmol/L give atorvastatin 40mg od.

Aldosterone receptor antagonists
Patients with clinical signs of heart failure and left ventricular function of <40% should be considered for an aldosterone receptor antagonist if the creatinine clearance is greater than 50mls/hr and the serum potassium is below 5.0mmol/L. A suitable agent is spironolactone 25mg od.

MANAGEMENT OF NST-ACS
Non ST-segment elevation acute coronary syndromes (NST-ACS) include unstable angina and non ST-segment elevation myocardial infarction (NSTEMI). Patients with NST-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. The risk of MI or death within 6 weeks of developing NST-ACS may be as high as 10%.

Troponin measurement (see Appendix 1)
All patients with normal or equivocal ECG should have their troponin levels measured.

- If the initial troponin T concentration is >0.05ng/mL, the patient is likely to be having a NST-ACS (specifically a NSTEMI) and should be admitted for further management and continuous ECG monitoring.
- If the initial troponin T is <0.05ng/mL, the patient needs to have a repeat troponin

performed at 12hrs. If the second level is ≥0.05ng/mL, admit for NST-ACS.

- If the initial troponin T is <0.05ng/mL, but the second sample is between 0.01 and 0.05ng/ml, the patient's history should be reviewed and if 'cardiac', discuss with cardiology. If 'non-cardiac', consider alternative diagnoses.
- If both troponin T samples are <0.01ng/mL, review the patient's history. If definitely cardiac, arrange for an exercise tolerance test (as an in-patient if possible). If positive refer to cardiology for possible coronary angiography.

Management of NST-ACS

- 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 it with either metoclopramide (10mg IV over 2 mins) or cyclizine (50mg over 3 mins).
- Give high flow oxygen to all patients during the first 2-3 hours and thereafter to patients with overt pulmonary congestion and those with an arterial oxygen saturation of <90%.
- Give low molecular weight heparin in the form of dalteparin (Fragmin) subcutaneously in a dose of 120 IU/kg every 12 hours (up to a maximum dose of 10,000 IU 12 hourly) for 24-72 hours.
- Intravenous GTN in a dose of 1-10mg/hr can be given for continuous chest pain or pulmonary oedema if the systolic BP is >90mmHg and the patient hasn't received a phosphodiesterase inhibitor (ie. sildenafil) within 24 hours.
- Beta-blockers are recommended for all patients except those with:
  - a history of bronchospasm
  - heart failure requiring therapy
  - bradycardia of >50bpm
  - 2nd or 3rd degree heart block
  - cardiogenic shock
  - allergy or hypersensitivity to beta blockers

  A reasonable choice is metoprolol given as 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.
- For all patients with NST-ACS do a lipid profile, then start a statin. For patients with LDL <4.5mmol/L give simvastatin 40mg od; for those with LDL >4.5mmol/L give atorvastatin 40mg od.
- Patients with continuing pain and dynamic ECG changes should be admitted to the coronary care unit, where a decision will be made whether to add a glycoprotein IIb/IIIa receptor antagonist.
- Patients with diabetes mellitus or a blood suger of >11 should be started on IV insulin (see STEMI section, page 9).

Patients with NST-ACS should be considered for coronary angiography and re-vascularisation unless contra-indicated because of significant morbidities. Refer patients with NST-ACS to cardiology SpR (bleep 6002).

MANAGEMENT OF ACUTE HEART FAILURE

Link consultant: Dr Lisa Anderson

DIAGNOSIS

The diagnosis of acute heart failure relies on the symptoms and signs, and ultimately on the findings of echocardiography. The characteristic symptoms of heart failure are posturally-dependent dyspnoea (worst on lying flat, eased by sitting up or standing), cough (sometimes productive of frothy pink, or blood-stained, sputum), and ankle swelling. The signs include raised central venous pressure, bilateral crepitations, a fourth heart sound and a displaced apex beat. In a patient with these features, and a known history of ventricular dysfunction (confirmed by an earlier echocardiograph), assume an acute exacerbation, and manage as below.

If the clinical features are new, and heart failure is suspected, measure the serum concentration of brain natruiretic peptide (N-terminal pro-BNP; NTproBNP). If the NTproBNP is normal (i.e below 35pmol/L), assume the patient does not have heart failure and search for an alternative diagnosis. If the NTproBNP is **>210pmol/L** (or **>110pmol/L** if younger than 75 years), heart failure is likely and should be confirmed by echo. If the NTproBNP concentration is between 35–212pmol/L, reconsider the diagnosis. If, after reassessment, ventricular failure is likely, request an echo. When the echo findings are compatible with ventricular impairment, assume a diagnosis of heart failure and treat as such.

MANAGEMENT

Initial treatment

1. *Acute left ventricular failure*:

- O_2 to maintain SaO_2 (95-98%)
- IV GTN infusion (10-200mcg/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 3mg bolus repeated as necessary till symptoms controlled
- CPAP/BIPAP (with intubation if respiratory failure develops and appropriate)

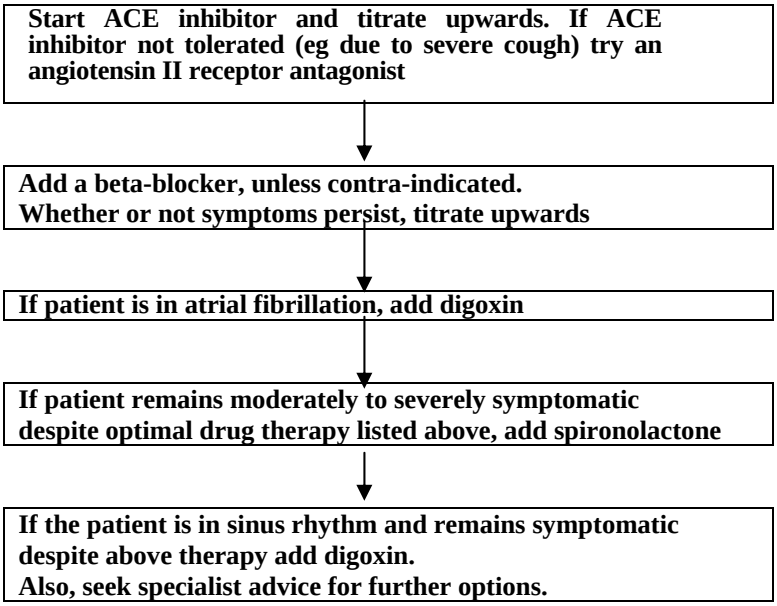
General measures:

- *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 patients with diabetes, switch to sliding scale insulin.
- *Request* chest X-ray; plasma U& E's, creatinine, FBC, TFTs, LFTs, glucose and lipids; urinalysis; peak flow or spirometry.

2. Acute right ventricular failure:

Treatment consists essentially of optimising diuretic therapy.

Follow-up treatment:
Once the initial phase is over, start 'long-term' therapy. For those with isolated right ventricular failure, the key target is to maintain optimal diuretic therapy. For those with left ventricular impairment, whether it is the sole problem or is in association with right ventricular failure, follow the algorithm below.



DISCHARGE AND FOLLOWUP
Patients should be discharged with a clear management plan for continuing care (including titration of ACE inhibition and β-blockade, fluid balance monitoring and review) which is communicated to the GP. Clear instructions should be given as to how the patient/carer can access advice particularly in the high-risk period immediately after discharge. To aid co-ordination of chronic heart failure care in the community, and to provide telephone help for the patient on discharge, contact the heart failure nurse specialist (bleep 7376) as soon as possible with details of the case, so that s/he can establish the necessary support arrangements.

DISORDERS OF CARDIAC RHYTHM

(Link consultant: Dr Edward Rowland)

1. SINUS BRADYCARDIA.

This requires no treatment unless it is causing symptoms. If treatment is deemed necessary, give atropine 600-1200mcg 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 firm on call.

2. 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 pacing if the block is impairing cardiac function.
Complete (third degree) AV block requires careful evaluation. Patients with symptomatic block usually require immediate pacing even if symptoms have resolved upon arrival. This is preferably achieved by prompt implantation of permanent pacing (contact cardiology firm on call). Complete AV block associated with inferior myocardial ischaemia is usually transient but will require 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 temporarily 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.

3. SUPRAVENTRICULAR TACHYCARDIAS.

The commonest types are:
a) atrial fibrillation, atrial flutter and atrial tachycardia
b) junctional 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 ($\leq$24 hours) is best terminated by IV flecainide (1-2mg/kg over 10 min**;** maximum dose 150mg).
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 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.

4. VENTRICULAR TACHYCARDIA.

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). 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.

5. VENTRICULAR FIBRILLATION (see Cardiac Arrest; page 4).

6. 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.

7. ASYSTOLE (see Cardiac Arrest; page 4)

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

ACUTE DEEP VEIN THROMBOSIS
(Link consultant: Dr Muriel Shannon)

Deep vein thrombosis (DVT) is common, particularly in hospital. About 20% of thromboses extend proximally and 2% embolise. Treatment aims to reduce the risk of embolism and to 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. Arrangements will vary between hospitals.
- Inpatients should have an ultrasound request form completed and sent to the Ultrasound (US) Department in Lanesborough Wing – the study can then be done on the next inpatient list.
- A&E and out-patients seen during weekdays from 9am-5pm should be assessed using the protocol available in A&E. Those in whom a scan is indicated should be sent to the US Department with a completed radiology request form, when the scan will be done during the list. It helps to contact the department and tell

them of your request. When completing request forms from A&E it is essential to include the risk probability assessment (RPA) score. Details of the scoring system are available in A&E (and will soon be available on the intranet)

- A&E and out-patients seen after 5pm, or on weekends from 9am–5pm, should be given a completed radiology request form and asked to report to the ultrasound reception desk in Lanesborough Wing on the next working day between 10-11am.

When compression duplex ultrasound is not available, initial treatment should be based on a clinical diagnosis plus an assessment of the patient's risks and a D-dimer test.

Treatment

1. If a compression ultrasound done within hours confirms a DVT, and provided anticoagulants are not contraindicated (because of an enhanced risk of bleeding), immediately start once daily dalteparin and warfarin. Those presenting to A&E can then be discharged to continue the course at home. The thrombosis nurse (bleep 7380) will assess the patient after the results of the ultrasound become available and start treatment (in her absence this will be done by A&E). Patients at enhanced risk of bleeding (those with liver disease, peptic ulcer, alcohol abuse, hypertension, heart failure, or on drugs that enhance warfarin's effects), need to be assessed before treatment is started and those in A&E may require admission to hospital. Admission will also be required if the patient is an IV drug abuser, is demented, has a pulmonary embolus, if the DVT is bilateral or extending to the IVC, or if the patient is pregnant.

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 greater than 2. This should be checked in the Anticoagulation Clinic within 3 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	7,500iu
46-56kg	10,000iu
57-68kg	12,500iu
69-82kg	15,000iu
83-110kg	18,000iu
over 110kg	seek advice

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 alter dose. For patients at home the injections can be given either by the patient or a district or practice nurse.

2. If the initial compression ultrasound cannot be performed because presentation is out-of-hours, but on clinical grounds (examination, history etc) and the D-dimer result the diagnosis is probable, give dalteparin according to schedule above. Arrange for the patient to have ultrasonography on the next working day. Definitive treatment will then depend on the findings and if DVT confirmed should follow advice outlined in "1." above.

3. 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 Anticoagulant Clinic (at St George's, ext. 5443, and out-of-hours from on-call haematology registrar).

The duration of anticoagulation varies and is summarised in the table below.
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. Draw the patient's attention to the common interactions with warfarin outlined in the booklet. For A&E patients the Yellow Anticoagulant book will be given at the Anticoagulant Clinic at their first visit which should be within 3 days of treatment being started.

PRESENTING FEATURES	TARGET INR (range)	RECOMMENDED DURATION
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
Calf vein thrombosis - surgical (post-op), no risk factors	2.5 (2-3)	6 weeks and review
Calf vein thrombosis - Non-surgical, no risk factors	2.5 (2-3)	3 months

ACUTE PULMONARY EMBOLISM

(Link consultant: Dr Charlotte Rayner)

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 note on page 19).

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. The following scoring system allows clinical risk to be graded as 'low', 'moderate' or 'high'.

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
taking oral contraception or hormone replacement therapy	1.0

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

Investigations

All patients should have a CXR and ECG and measurements made of arterial blood gases (ABGs) and plasma D-dimer levels. 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.

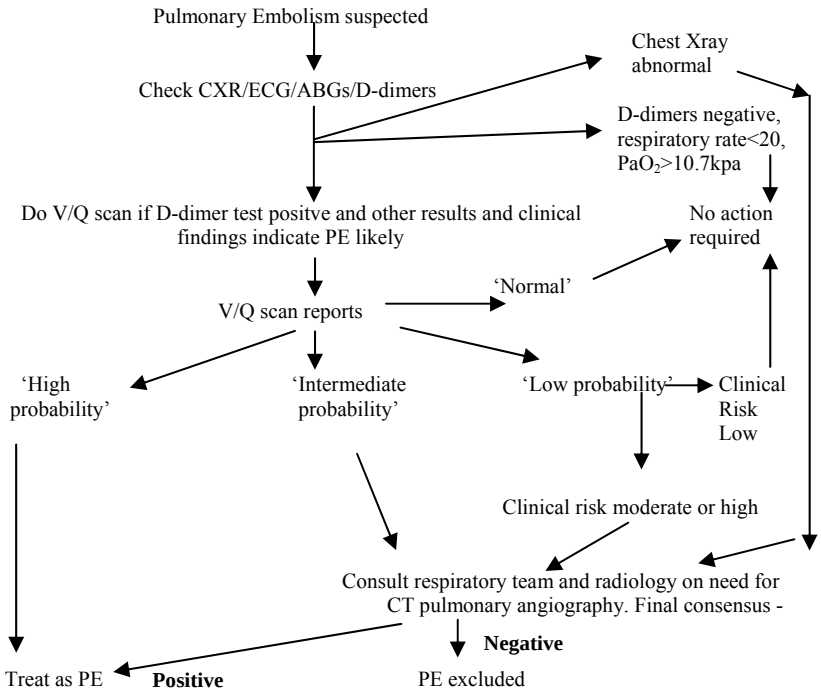
When any two of D-dimers, ABG and respiratory rate are normal, PE is very unlikely. In patients with a D-dimer level of <0.3mg/L and PaO_2 of ≥ 10.7 Kpa a lung scan is usually unnecessary.

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.

Management

Patient 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 PE confirmed 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.

Investigatory and diagnostic algorithm



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.

RESPIRATORY ARREST

(Link consultant: Dr Charlotte Rayner)

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.

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 ventilation is the best option if intubation skills not available.

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 (200mcg over 15 sec followed by 100mcg every 60 sec if required, up to l mg 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.

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.

Most patients 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.

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
- patients being weaned from mechanical ventilation

Patients with type one respiratory failure who are tiring should be moved urgently to the high dependency unit as they may need invasive ventilation.

ASTHMA

(Link consultant: Dr Charlotte Rayner)

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.

Treatment.

Oxygen. Most patients will have a low arterial oxygen tension so give a high concentration of oxygen at a flow rate of 6 L/min; a Ventimask should not be used.

Bronchodilators. A bronchodilator, such as salbutamol (2.5mg) should be started as soon as possible via an oxygen-driven nebuliser (drive at a flow rate of at least 7L/min). This dose should be continued if no improvement is seen. Nebulised ipratropium bromide (250mcg) 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 200 mg IV or prednisolone 30-60 mg by mouth as soon as the initial assessment is made. No material benefit can be expected for several hours and it is essential not to delay administration. If hydrocortisone is given the same dose should be repeated every 6 hours for 12 hours then 100 mg 6 hourly. Whichever steroid is given initially, after 2 days all patients should be taking 20-30 mg of prednisolone daily by mouth and this should be continued for 14-21 days. Thereafter the dose should be reduced.

Hydration. Patients tend to become under-hydrated 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.

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. The patients normally should be transferred from nebulised to aerosol therapy 24 hours after admission and started on inhaled steroids. 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.

Magnesium. In patients with severe asthma who respond poorly to nebulised bronchodilators, and only after 'approval' by a respiratory consultant, consider giving intravenous magnesium at a dose of 2g (8mmol) in 250mL of NaCl 0.9% over 20 minutes. This approach is endorsed by the BNF but not by DTB.

Discharge. Patients should be discharged on inhaled and/or oral steroids (as appropriate to their previous history and current severity), and followed up after 2-3 weeks. Home 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 or consultant.

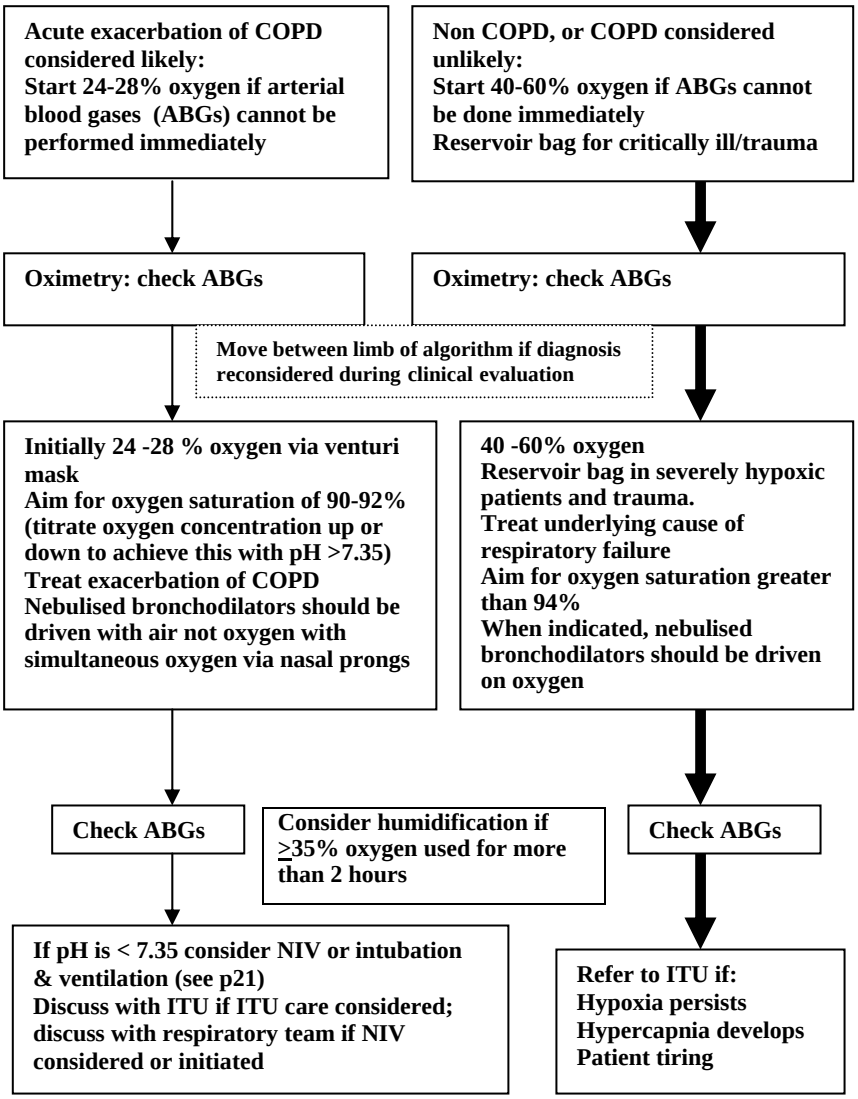
OXYGEN THERAPY IN THE ACUTELY BREATHLESS ADULT

(Link consultant: Dr Charlotte Rayner)

In patients who are acutely breathless, the aim is to give sufficient oxygen to support

their needs. The algorithm below is designed to help optimise such treatment. Too little oxygen can lead to cardiac arrythmias and excessive oxygen can be dangerous in some patients with respiratory failure.

Algorithm for Oxygen Therapy in the Acutely Breathless Adult



SPONTANEOUS PNEUMOTHORAX

(Link consultant: Dr Charlotte Rayner)

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 around the lung; moderate – collapse halfway to the heart border; complete – airless lung separated from the diaphragm), 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, if the X-ray shows greater than 50% collapse, or if a fluid level is found.. In those with shortness of breath or complete collapse aspirate the air through a wide bore needle introduced under local anaesthesia. If aspiration with a needle fails an intercostal drain may have to be introduced (seek advice).

In those with a suspected bleed, monitor the heart rate and blood pressure and repeat the X-ray to check whether bleeding has stopped. If it hasn't, seek advice. There is no need to admit an otherwise healthy young adult if:

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

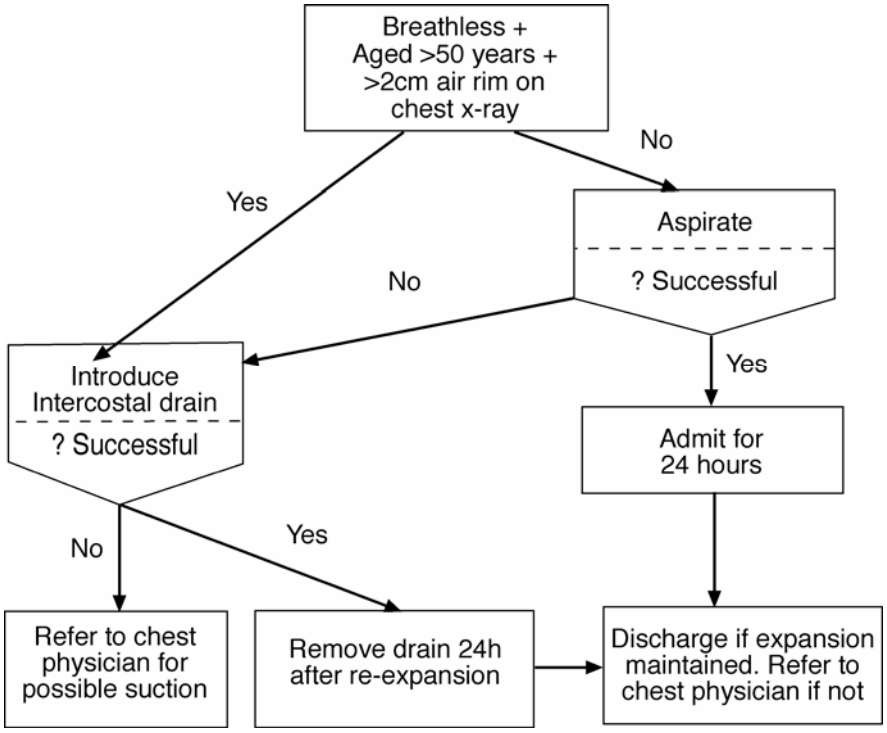
Before a patient leaves A&E explain the cause of the symptoms, arrange for outpatient review in 7-10 days, and advise the patient to return promptly to hospital if symptoms worsen.

Patients with underlying lung disease. All patients with underlying lung disease should be admitted to hospital with a view to aspiration or drainage, depending on their age, the level of their dyspnoea and the results of their chest X-ray. Management should follow the scheme in the flow diagram on page 26. 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, who should be told to report back to hospital immediately if symptoms deteriorate, should be advised not to travel by air for 3 months.

Treatment of patients with underlying lung disease



ACUTE UPPER GASTROINTESTINAL BLEEDING

(Link consultant: Dr Chris Groves)

Immediate Assessment

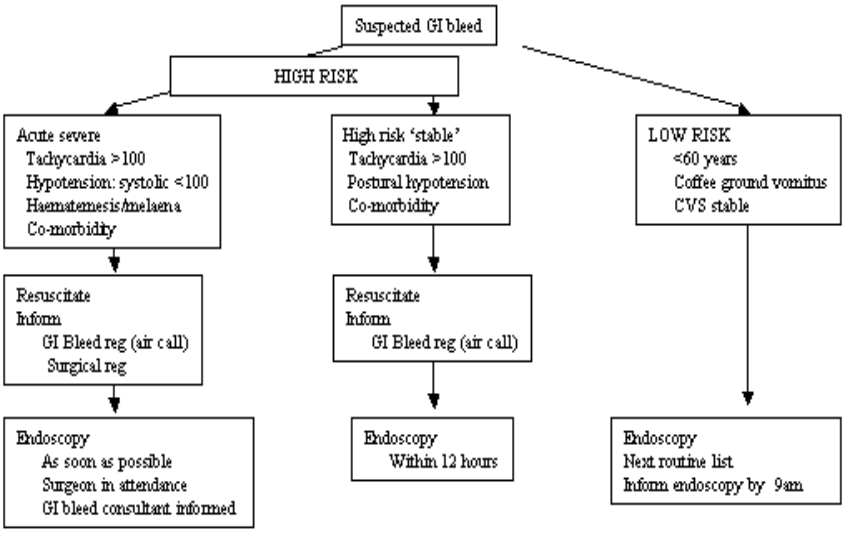
Once the diagnosis of a bleed has been made, take blood for haemoglobin, blood grouping/cross match, and coagulation studies. Enquire about drug usage (especially NSAIDs) and alcohol, retching (Mallory Weiss tear) and previous dysphagia. Examine for signs of chronic liver disease and portal hypertension (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 so the urgency for endoscopy and possible surgical intervention (see 'Initial Management' flow diagram).

- 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 rebleeding 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 9.00am.

Initial management



- 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; age > 60 years; severe concomitant disease (liver/cardiovascular/respiratory).
  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.

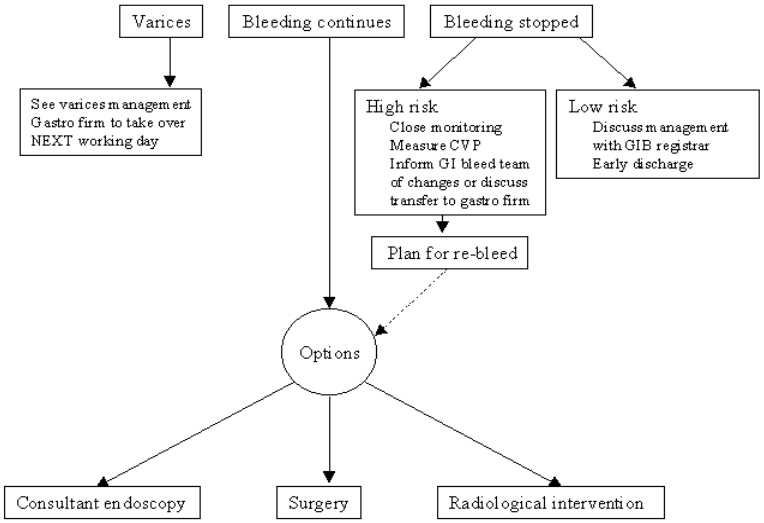
The endoscopist should enter the OGD findings in the Endoscopy Unit computer. If the endoscopist sees a bleeding ulcer, the patient should be given omeprazole (80mg) as a stat injection IV, followed by an infusion at 8mg/h for 72 hours.

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.

Second phase



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. ciprofloxacin 500mg po or 400mg IV bd.
- Endoscopy
  For general advice and to arrange endoscopy, contact endoscopy unit/GI SpR (bleep 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 programme.

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 2500-5000 units 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.

SEVERE DIABETIC KETOACIDOSIS/HYPEROSMOLAR COMA

(Link consultant: Dr Arshia Panahloo)

In many respects the treatment of severe ketoacidosis and hyperosmolar coma are similar; the few instances where they differ are indicated in the text.

IMMEDIATE admission to ITU must take priority over all except life-saving interventions. Contact a member of the diabetic "team" (Prof. Nussey SG127, Dr

Panahloo SG156, Dr Earle SG552. Dr Seal SG153) through the switchboard; it is better to seek advice early than late.

REMEMBER – diabetic ketoacidosis may develop very rapidly in insulin-dependent diabetics who omit their injections or have intercurrent illnesses, or slowly in patients with previously undiagnosed diabetes, but all patients need continuous observation during the first hour, then half hourly for 2 hours and, after that, hourly until out of danger.

URGENT INVESTIGATIONS

Measure blood glucose using a ward glucose meter. This will be accurate up to about 30mmol/L but if values are above 25mmol/L it is important to obtain a value from the laboratory as soon as possible.

1) Measure arterial blood gases and pH.
2) Measure ketones in urine; semiquantitative estimations using serial dilutions can be used for monitoring progress.
3) Measure blood urea, electrolytes (especially K^+) and creatinine, together with a Full Blood Count.
4) Do CXR, ECG, blood & urine cultures and take swabs from other sites as indicated.

MANAGEMENT IN ITU

1. Introduce IV line – central if possible. Start fluids (see below).
2. If patient comatose, aspirate stomach via a naso-gastric tube; use a wide bore tube as contents may be viscous.
3. If no urine is passed by 3 hours catheterise bladder and monitor urine output hourly.
4. Weigh patient if humanly possible.
5. Monitor BP & ECG.
6. Look for infection by urinalysis, chest X-ray, blood cultures. Do not rely on temperature and leucocytosis. Have a low threshold for antibiotic treatment.
7. Give oxygen if arterial pO_2 less than 80mmHg (11kPa).
8. Give subcutaneous heparin (5000units bd) if patient is comatose or very hyperosmolar (>350mosmol/L) unless there are clear contraindications.

FLUID REPLACEMENT. Patients with a rapid onset of diabetic ketoacidosis are not necessarily severely dehydrated although dehydration (often gross) is particularly likely when the patient has gone gradually out of control. The urine output is a good guide as to the state of hydration – measurement of CVP is less
helpful. If urine output is good, be careful about giving large amounts of fluid. Remember, a dry mouth can have other causes – eg hyperventilation. Usually give 2 litres 0.9% NaCl over the first 4 hours, 2 litres over the next 8 hours, then 1 litre 8 hourly. Consider giving colloid if systolic blood pressure less than 100mmHg after 2 hours, or if there is other clinical evidence of circulatory collapse (peripheral shutdown, oliguria). Consider giving blood if available. Patients in hyperosmolar coma may remain in a state of circulatory collapse despite clinically adequate fluid replacement. If this occurs give 500mL colloid, monitoring the CVP. Use hypotonic solutions only if plasma Na^+ greater than 155mmol/L and then only as 1 litre over 8 hours, and only after discussion with a consultant from the diabetes service. Monitor CVP if the patient has heart disease, and always replace fluid cautiously in older patients.

Potassium. If plasma K^+ is less than 3.5mmol/L immediately start KCl infusion at 20mmol/hr. Do NOT give insulin until K^+ is greater than 3.5mmol/L. Accompany all insulin infusions with K^+ at a rate of at least 20mmol/hr. Monitor plasma K^+ hourly initially, later 2-hourly. Discontinue K^+ infusion if plasma K^+ greater than 6.0mmol/L. ECG monitoring may be a helpful guide to acute changes.
Acidosis. Ketoacidosis will usually correct itself once normal circulation and metabolism are restored. There is rarely, if ever, an indication for giving sodium bicarbonate; there may be a place after cardiac arrest but if worried in other circumstances seek specialist advice. If it is required, give 100mLs $NaHCO_3$ (8.4%) with 20mmol KCl over 30 minutes and repeat blood gases 30 minutes later.
Insulin. Use soluble insulin by intravenous infusion, preferably with a syringe pump, so that the infusion rate can be varied independently of the rate of fluid administration. Start at 6 units/hr and double this if there is no response in 2 hours. Check stick or lab blood glucose hourly and aim to reduce the blood glucose by about 5mmol/hr.

LATER CARE
When blood glucose falls to about 13mmol/L, start to give IV dextrose; start with **1L 5%** dextrose over 4-6 hours with 20mmol KCl and continue the insulin infusion at about 4 units/hr, adjusting this with regular blood glucose estimations. If this regimen is continued beyond 24 hours make sure that each day's infusion includes 1L 0.9% NaCl (150mmol Na & Cl). When the patient is able to eat, stop insulin by IV infusion and replace it with bd subcutaneous injections using human biphasic isophane 30/70. Under no circumstances should subcutaneous insulin be given using a sliding scale. In selecting an initial s/c dose schedule a useful guide is to calculate the total IV dose over the past 24 hours and give 60% of this in the morning and 40% in the evening. Advice can be obtained from the diabetic team (contact via Diabetic Unit on ext 1429 during working hours, or page consultant).

HYPOGLYCAEMIA

(Link consultant: Dr Arshia Panahloo)

Hypoglycaemia is unusual except in patients with diabetes who commonly suffer the excessive effects of their own hypoglycaemic drugs. Symptoms include sweating, tremor, palpitations, inco-ordination, convulsions and coma, and in any diabetic patient with impaired consciousness hypoglycaemia should be assumed to be the cause until it has been excluded by measurement of blood glucose (send sample to laboratory), most simply done on a finger prick test. Occasionally hypoglycaemia is induced by these drugs used in suicide bids by patients who are not diabetic. Other drugs, such as alcohol and aspirin, may also cause hypoglycaemia, and the problem may also arise as part of an underlying disease such as insulinoma, carcinoid or sepsis (particularly in children and neonates). If you suspect that the hypoglycaemia is iatrogenic, send blood/urine for screening, e.g. sulphonylurea screen, estimate of insulin concentration.

Whatever the cause, IMMEDIATELY give glucose, if possible by mouth. Diabetic patients on insulin often carry a supply of glucose for self-administration. If the patient is semiconscious, give the glucose gel formulation (Hypostop), which is well absorbed and unlikely to be inhaled. If oral administration is not possible, give 20-30mL of a 50% glucose solution IV, taking care that the needle is in a vein as

extravascular infusion can cause tissue necrosis. If the administration of glucose IV proves difficult give 1mg glucagon IM (or by any other parenteral route). Patients who have taken insulin should fully recover consciousness in 5 min. If they do not, insert a central venous line and infuse 10% glucose to keep the blood glucose around 10mmol/L. Any patient who does not recover fully in 30min should be transferred to ITU and a specialist consulted, or an SR on the endocrinology team informed.

If the hypoglycaemia is caused by a sulphonylurea such as chlorpropamide or glibenclamide, the fall in blood sugar may be profound and prolonged and further complicated by the administration of glucose which stimulates insulin release. In these circumstances give octreotide (50mcg 12 hourly) subcutaneously to block insulin release and allow blood glucose to return to normal. Such patients should only be given IV glucose if blood glucose is below 5mmol/L.

ACUTE STROKE

(Link consultant: Professor Hugh Markus)

Stroke is a clinical syndrome in which an acute focal cerebral deficit that occurs secondary to cerebrovascular disease lasts for more than 24 hours or results in death. In a patient with a transient ischaemic attack (TIA) the symptoms and signs are similar but resolve within 24 hours (most commonly within one hour). The 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), the underlying aetiology (e.g. carotid stenosis or cardiac embolism) and the disability.

Admission

Good management of patients with stroke reduces overall mortality by one quarter and the risk of recurrence by up to three quarters. It also reduces complications and residual disability. To this end any patient (no matter what age) who has developed features of stroke (no matter what 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 Acute Stroke Unit (**William Drummond Ward**, **3rd Floor AMW**).

The admitting medical Registrar should assess the patient and then, during working hours (9am-5pm Monday-Friday) contact the Stroke Unit Registrar (bleep 7317) or SHO, or out-of-hours the Neurology on-call Registrar (bleep 7210) or SHO to arrange for transfer. If the patient cannot be admitted directly to the Acute Stroke Unit, care will need to be started in a general ward, but every effort should be made to transfer the patient to the Acute Stroke Unit as soon as possible, and directly from A&E. If the Acute Stroke Unit is initially full, a patient (usually the one who has been on the Unit longest) will be moved to make way for the new admission. The 'moved' patient will either be transferred back to the Acute Medical Team on call when the patient was admitted, **or to a geriatric medicine bed**.

History and Examination

The history, which should be recorded in the stroke proforma (available from the acute stroke unit), should include time and mode of onset (sudden or gradual), progression since onset, vascular risk factors including the presence or absence of hypertension, diabetes, hyperlipidaemia, smoking, alcohol, heart disease, claudication and a 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), visual fields, speech, swallowing, limb weakness, cerebellar signs, reflexes, plantar responses and the presence or absence of incontinence, and check for neck stiffness and Kernig's sign if subarachnoid haemorrhage is suspected. The general examination must include vital signs (especially BP), cardiac or respiratory signs, peripheral pulses and the assessment of the presence or 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 urgently in patients with coma, deteriorating consciousness, brain stem or cerebellar signs or progression, or if subarachnoid haemorrhage is suspected. In others, it should be undertaken as soon as possible and always within 24 hours. A scan is needed to confirm diagnosis, distinguish infarction from haemorrhage and exclude non-vascular causes in order to determine treatment. The scan, if normal, confirms the safety of lumbar puncture where the history and findings on examination suggest subarachnoid haemorrhage. 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). It is essential to look for xanthochromia in the CSF if subarachnoid haemorrhage is suspected and the CT scan has not shown subarachnoid blood. Red cells alone in the CSF can occur with a traumatic lumbar puncture and can confuse diagnosis if the supernatant fluid is not examined.

All patients should have blood sent for a full blood count, ESR, urea, creatinine, sodium, potassium, glucose and cholesterol. They should also have 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 antibody screen and anticardiolipin antibody. Those you suspect of having a significant cardiac abnormality either from the history, or from your examination, or from an ECG, or who are under the age of 65, should have an echocardiogram. In those under the age of 50 or with recurrent unexplained stroke the echocardiogram should be transoesophageal.

Cerebral angiography, which should be performed in the AMW neuroradiology department after referral to the neurology team, may also be required in subarachnoid haemorrhage, intracranial haemorrhage, carotid stenosis, brain stem or cerebellar strokes or in any patient under the age of 50.

Acute Medical Management

Contact the stroke registrar (bleep 7317) or if unavailable the stroke SHO (bleep **7785**), to discuss all patients who present within 4½ hours of the onset of symptoms: the time before which thrombolytic therapy should be started if indicated. They will organise brain imaging, CT or MRI scanning, and will start tPA if appropriate. Aspirin **and dipyridamole** should be given to all patients with ischaemic stroke, and so 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. **The dose of dipyridamole MR is 200mg BD**.
Full heparinisation should be reserved for patients with carotid dissection, cerebral venous thrombosis, or where there is a high risk of a cardioembolic source. In patients with atrial fibrillation or another 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 registrar.
Patients already on antihypertensive medication should continue their usual treatment unless their blood pressure is low. Acutely elevated blood pressure is common following stroke and should not be treated aggressively. In patients with a systolic blood pressure of greater than 220mmHg, or a diastolic pressure greater than 110 mmHg, blood pressure should be reduced gradually (see page 6 sq.)
Much of the mortality and morbidity following stroke is from secondary complications. To minimize these:

- Fit thigh-length TED stockings as soon as possible to hemiplegic or otherwise immobile patients.
- In patients at high-risk of DVT and pulmonary embolism, consider giving low-dose anticoagulation with low molecular weight heparin.
- 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 > 10 mmol/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, a history of neck trauma, Horner's syndrome on the side of dissection. If this diagnosis is suspected, the imaging of choice is an MRI scan with cross-sectional views through the carotid artery in the neck (ask radiologist specifically for these) as well as carotid MRA. Patients with dissection should be referred to the neurology registrar for advice. Treatment includes anti-coagulation with heparin and then warfarin.
- **Cerebral Venous 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. Patients in whom the diagnosis is suspected should be referred to the neurology registrar. 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**
  Subarachnoid haemorrhage 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 at AMH.
- **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 one and two months post event to exclude an underlying lesion. In young patients cerebral angiography should be considered. Discuss with neurology team.

Prevention of Recurrence
To reduce the risk of recurrence:
- Hypertension should be investigated and treated after the acute stage (see above).
- Any patient with carotid stenosis demonstrated on Duplex should be referred urgently to the stroke registrar or the cerebrovascular disease clinic for consideration for carotid surgery or angioplasty.
- Consider anti-coagulation 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 aspirin (**75mg** per day), **plus dipyridamole MR** (**200mg BD**). Those with proven intolerance, or allergy to aspirin (a very small minority), should be given clopidogrel 75mg a day.

STATUS EPILEPTICUS
(Link consultant: Dr Tim Von Oertzen)

Status epilepticus is defined as either a run of discreet seizures without full recovery in between fits, or continuous seizures lasting for 30 minutes. As most seizures terminate spontaneously within three minutes, the following measures should only be instituted for seizures lasting longer than seven to ten minutes, unless the patient is known to have longer seizures with self termination (this information may be obtained from relatives or friends, or from the patient's epilepsy card or diary).
The mortality and morbidity of generalised tonic/clinic status is high, and it is important to control the 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. Estimate blood glucose rapidly using a blood glucose test. If the patient is hypoglycaemic rapidly infuse a 50% solution of glucose to give 1-2mg per kg body weight.
5. Draw venous blood for measurement of glucose, urea, sodium, potassium, calcium, liver function and anticonvulsant drug levels. Also send sample for full blood count and clotting studies.
6. Measure body temperature, take an ECG, monitor respiration and BP.
7. Gain information – is there evidence of previous epilepsy, any anti-convulsant 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 10 minutes. Lorazepam, however, should not be used more than twice in any 24 hour period.
3. If fits still persist – call the anaesthetist and immediately start an infusion of either phenobarbitone by intravenous infusion (dissolved in water) in a total dose of 10-15mg/kg, given at a rate of 100mg per minute, or phenytoin by intravenous infusion in a total dose of 15-18mg/kg given at a rate of 50mg per minute.
4. If, despite intravenous lorazepam plus phenobarbitone or phenytoin, the seizures continue or recur, then the patient should be transferred immediately to an Intensive Therapy Unit and discussed with a neurologist (do not forget to watch out for respiratory depression).
5. If status persists or recurs after 30-60 minutes the patient will need to be sedated and ventilated. This will require the active involvement of an anaesthetist.

SUBSEQUENT MANAGEMENT

1. Check that the patient is taking the medicines as prescribed and that there have been no interactions reducing drug efficacy. Reinstitute any recently-stopped anticonvulsant medication; 'reload' with the usual drugs if levels are low.
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 a member of the epilepsy team, and arrangements for follow-up made (most newly-diagnosed patients will need to take anticonvulsant(s) for at least 3 months). No patient should be discharged without being given an adequate explanation of his or her presentation and agreeing a plan of management. Discussion, which probably best involves the patient's partner/parent, should include basic seizure safety information and driving regulations.

ACUTE PAIN

(Link consultant: Dr Jeremy Cashman)

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, mild-to-moderate, moderate-to-severe or severe and treated accordingly. The use of combinations of analgesic drugs and techniques usually improves the quality of pain relief and may enable the use of lower doses of individual drugs thus minimising the risk of unwanted effects. Local anaesthetic techniques may help, and can decrease opioid requirements. In general it is more realistic to strive for comfort rather than complete abolition of pain. For advice on the management of acute pain contact the Acute Pain Team (bleep 6477/6159) or the on-call anaesthetist (bleep 6111). For the management of pain associated with end-stage disease, contact the palliative care team (bleep 6796/6508 or ext 3313). Note that for some conditions, such as acute coronary syndromes (p.8), arthritis (p.59), and sickle cell crises (p.66), analgaesic approaches differ.

THE "ANALGESIC LADDER"

Mild pain	paracetamol or an NSAID
Mild-to-moderate pain	combination analgesic ± an NSAID
Moderate pain	oral opioid or combination analgesic ± an NSAID
Moderate-to-severe pain	oral opioid + paracetamol ± an NSAID
Severe pain	parenteral opioid (IV, IM or SC) + paracetamol ± an NSAID

TREATMENT DETAILS

Simple Analgesic

- Paracetamol: Give by mouth, through a nasogastric tube, or as a suppository. The dose is 1g, 4-6 hourly (maximum 4g/day).

Non-Steroidal Anti Inflammatory Drugs (NSAIDs)

- Diclofenac: Give by mouth at a dose of 50mg, 8 hourly (maximum 150mg/day), or as a suppository (12.5mg, 25mg, 50mg, 100mg).
- Ibuprofen: Give by mouth (available as tablets or syrup), in a dose of 200-400mg, 4-6 hourly (maximum 2.4g).

Contraindications: Bleeding diathesis, peptic ulceration, renal dysfunction, allergy to NSAIDs (care in asthma), congestive cardiac failure.

Combination Analgesic

Co-dydramol: Each tablet contains 10mg dihydrocodeine + 500mg paracetamol. The dose is 1-2 tablets, 4-6 hourly (maximum 8 tablets/day).

Opioids – Oral

Dihydrocodeine: Give as tablets or syrup; 30mg dose, 4-6 hourly (maximum 240mg/day)

Codeine Phosphate: Give as tablets; 30mg, 4-6 hourly

Tramadol: Give as capsules; the dose is 50-100mg, 4-6hourly

Opioids – Parenteral

Morphine is the preferred opioid. It may be given IM, SC or IV (as a bolus, a continuous infusion or as patient-controlled analgesia; PCA). Pethidine may be used in patients with renal or biliary colic or when morphine has produced severe

generalised pruritis. Pethidine should not be used in patients taking MAOI drugs or given in large doses to patients with epilepsy as it has epileptogenic metabolite. All opioid solutions should be diluted with normal saline. If the patient is hypotensive or has signs of shock, treat these before starting an opioid as it may reduce blood pressure further.

- Injection: Severe acute pain often requires morphine to be given by injection to give adequate control**.** Either IV or IM administration are effective. Use the dosage regimens given in the following tables:

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

IM morphine	
Age (years)	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, and up to 1-2 hourly PRN after IV injection. Check for overdose post injection as below.
- *pain persists*, for IM administration immediately repeat injection but at a higher dose within the range; for IV administration immediately repeat same or higher dose in range. 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 the patient closely. A subcutaneous infusion may be used in patients without IV access.
- PCA: Patient Controlled Analgesia allows titration of the opioid to the patient's need with a higher degree of safety than a continuous infusion. Contact the Acute Pain Team for help with this regimen.

OPIOID OVERDOSE

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

1. stop the opioid
2. administer oxygen by face mask
3. give naloxone by IV injection 100mcg every 2-3 minutes until patient is rousable and respiratory drive returns
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.

ANAPHYLAXIS

(Link consultant: Dr Charlotte Rayner)

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 the doses of the drugs given 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. Repeat after 5 min 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 and 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-**300**mg (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 recurrence of symptoms and consider observation for a minimum of 8-24 hours.
- Write the name of the agent that caused the reaction prominently in the patient's notes and drug chart.

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 ± 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 (4 hours 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 the 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

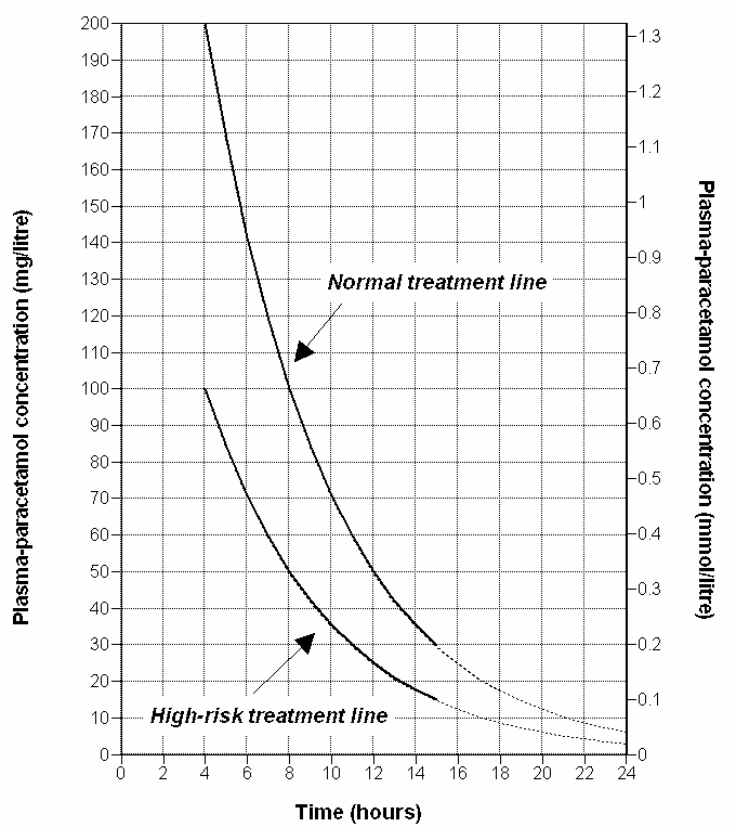
PARACETAMOL

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 44 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 regime:
  -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 the 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 4 hours 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^{o}C it is important to exclude infection, do:
1. blood cultures
2. MSU
3. sputum culture
4. ascitic tap – if the WBC is >250/mL (neutrophils) or >300/mL (lymphocytes**)** the patient is likely to have SBP. While awaiting culture results (send ascites inoculated in culture-medium bottles to increase diagnostic yield) start cefotaxime 2g bd or tds IV, or ciprofloxacin 750mg bd by mouth.

Jaundice
1. Exclude haemolysis, do conjugated bilirubin and blood film
2. Exclude biliary obstruction

Coagulopathy
1. Give vitamin K (phytomenadione) 10mg PO daily for 3 days. If severe coagulopathy, Vit K can be given IV 10mg over 10mins 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)
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 hypovolaemia
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 (cefotaxime+metronidazole) 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 200mg po od for 2 weeks)

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

Referral to Hepatology team
All patients with decompensated liver disease should be referred to the hepatology team. They should also be referred if they have:
1. Organ failure in addition to liver disease
2. Hepatocellular carcinoma
3. Variceal haemorrhage
4. Massive ascites and are likely to need total paracentesis
5. Recent-onset encephalopathy (<12 weeks of onset of jaundice)
6. Incipient renal failure
7. Alcoholic hepatitis

SUGGESTIONS FOR THE USE OF ANTIMICROBIAL DRUGS

(Link consultant: Dr Rick Holliman)

Take all necessary specimens before starting treatment. Get appropriate specimens, e.g. pus sample if possible rather than swabs, purulent sputum samples, stools rather than rectal swabs, etc. Rapid microscopic diagnosis is sometimes possible, especially from a CSF smear in meningitis, but also from pus or deep lung secretions. Send swabs in transport medium. Check with lab how to handle specimens collected out of working hours. Base initial antibiotic choice on your judgement of the most likely pathogen(s), picking the most suitable agent for this pathogen. When possible use a narrow-, rather than a broad-spectrum antibiotic. Seek advice on dosing of gentamicin and vancomycin in patients with renal impairment. Review the clinical and bacteriological data frequently. If the patient is hypersensitive to the suggested drug, seek advice on an alternative. Always consider the implications for cross-infection. These are set out in the Trust's infection control manual or can be obtained from the Infection Control Team (ext.5675).
If problems arise don't hesitate to seek advice from senior colleagues in the Clinical Infection Unit (CIU) or Paediatric Infectious Diseases Office (also see the Trust intranet site on the Management of Infection in Children), or from the Medical Microbiologist (Bleep 6480 in working hours or air-call). The same specialists, plus Genito-Urinary Medicine should be contacted for advice on management of patients with definite HIV, or potential HIV-related problems (see Appendix 8).

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

- temperature has been less than 38°C for 48 hours or more;
- oral foods/fluids are tolerated;
- there is no unexplained tachycardia;

and provided that:

- there is no evidence of impaired absorption;
- it is not a condition such as endocarditis or meningitis in which extra high tissue antibiotic concentrations are essential;
- a suitable oral formulation is available.

GASTRO-ENTERITIS

No antibiotic as a routine. If in doubt, or if patient is severely ill, seek advice. Specialists to consult will depend on the situation (paediatrician, microbiologist, Clinical Infection Unit, etc).

BACTERIAL ENDOCARDITIS

Refer to detailed guideline:
http://formulary/HTML/form2/DRUGS/PROTOCS/endo2006.pdf

Take x3 sets of blood cultures, a serum sample (send labelled for endocarditis screen) and seek advice which should inc lude that of a cardiologist (see cardiology 'red' book).

Clinical situation may allow one to wait. If confirmed, transfer the care of the patient to a consultant cardiologist and/or an infectious diseases physician.
Initial treatment: adults: benzyl-penicillin (1.2g six times daily) - + gentamicin (60-80mg bd); children: benzylpenicillin 50mg/kg qds + gentamicin 6mg/kg daily

If patient is in septic shock (or has abused IV drugs), in adults, add flucloxacillin (2g 4-hourly), in children, add flucloxacillin 50mg/kg qds (max 2g).
Seek advice if the patient is allergic to penicillin.

MENINGITIS

Whenever diagnosis of meningitis is suspected take blood cultures plus blood in EDTA for molecular studies and seek ***early*** advice on the need for a CT scan, the timing of a lumbar puncture, on the most appropriate antibiotic and the duration of therapy. The incidence of serious complications following meningitis increases with the delay in starting antibiotic treatment. The following are likely drugs of choice:

Pneumococcal	cefotaxime (2g qds) IV in adults; ceftriaxone (80mg/kg; max 4g) IV in children.
Meningococcal	cefotaxime IV (2g qds) in adults; ceftriaxone (80mg/kg; max 4g) IV in children. Seek advice on the treatment of contacts from the Consultant in Communicable Disease Control (phone 020 8682 6132 or Air-call through hospital switchboard).
Haemophilus	cefotaxime IV (2g qds) or (in children) ceftriaxone IV. Consult on contacts as for meningococcal above.
Listeria	amoxicillin IV (2g, 4 hourly) & gentamicin IV (120mg tds).
Unknown i.e: CSF smear negative for bacteria or culture result awaited	) neonate (<6weeks) amoxicillin 100mg/kg IV (give bd if <1wk;) tds if 1-3wks; qds if >3wks) + gentamicin (6mg/kg) IV +) cefotaxime (50mg/kg) IV, (bd if <1wk; tds >1wk)) child -ceftriaxone (80mg/kg) IV) adult -cefotaxime (2g qds) IV) immune-compromised -amoxicillin (2g 4-hourly) IV + cefotaxime (2g qds) IV

Seek advice if the patient is penicillin allergic.
In children with meningitis, or adults with suspected or proven pneumococcal meningitis, add a corticosteroid to the regimen. This should be given early in the course of treatment (for children refer to the Paediatric Infectious Diseases Unit manual for details; for adults discuss with CIU consultant).
Remember tuberculous and listeria meningitis – if you suspect either, seek advice. Tell the Consultant in Communicable Diseases about every suspected or confirmed case of meningitis and complete a notification form.

UPPER RESPIRATORY TRACT INFECTION

Remember that most throat infections are caused by viruses and so do not require an antibiotic.

Microbiologically confirmed streptococcal pharyngitis	oral penicillin V, for adults**:** 500mg qds; for children: 50mg/kg bd (max 1g), for 10 days (or erythromycin if allergic to penicillin)
Acute otitis media, and bacterial infection proven or strongly suspected	In adults: oral amoxicillin (500mg tds) for 3 days In children antibiotics are generally not indicated. If treatment is required, give oral amoxicillin (50mg/kg; max 1g) tds for 5 days.
Suspected acute epiglottitis	In adults, cefotaxime IV (1g tds); or in children ceftriaxone IV 80mg/kg (max 4g). Add flucloxacillin if tracheitis suspected. In children first call for ENT help to ensure patent airway.
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 in adults (50mg/1kg (max 1g) tds in children) If allergic to penicillin give oral erythromycin.

PNEUMONIA

***Refer to revised antibiotic policy during outbreaks of Clostridium Difficile infection*, http://formulary/HTML/form2/DRUGS/PROTOCS/eacap.doc**

ADULTS

a) Lobar pneumonia contracted *outside* hospital.
Treatment depends on the severity of the condition; consider 'severe' if two or more of the five "CURB65" score features are present (i.e a score of 2-5):

- respiratory rate > 30 per minute
- low blood pressure (systolic pressure <90mm Hg or diastolic pressure ≤ 60mm Hg)
- blood urea >7mmol/L
- confusion (Abbreviated Mental Test score ≤ 8)
- age ≥ 65 years

If severe, send blood cultures, sputum and urine (for antigen detection).

Treatment:

Non-severe (i.e CURB65 score 0-1)
Oral therapy: amoxicillin 500mg tds plus erythromycin 500mg qds for 7 days.

Severe (i.e CURB65 score 2-5)
IV therapy: cefotaxime 1-2g 8 hourly (or co-amoxiclav 1.2g tds) + erythromycin 500mg qds.
Consider ITU referral (seek consultant advice). Most patients with community acquired pneumonia started on parenteral therapy can be switched to oral therapy after 48 hours unless they remain seriously ill (*follow guidelines on page 51*). Give for up to 10 days in total.

Note that: if urinary Legionella antigen positive add IV rifampicin 600mg bd;
if post-influenzal add IV flucloxacillin 1-2g qds;
if mycoplasma suspected use erythromycin (500mg qds IV) or oxytetracycline (500mg qds).

Seek advice about duration of therapy.

b) Pneumonia contracted *inside* hospital (treatment should be given for 10 days in total).
If patient is ventilated give ceftazidime IV (1g tds). Add metronidazole IV if aspiration suspected. Add gentamicin IV if patient in septic shock.
If patient is not ventilated give cefotaxime IV (1g tds). Add metronidazole 500mg IV tds if aspiration suspected. If no response, take advice.

CHILDREN

Neonates & 1-3months: ceftriaxone (50mg/kg, od) + amoxicillin** (20mg/kg, tds) **stop if –ve for listeria*

Mild <5 years: **amoxicillin (20mg/kg, po 8-hourly) for 7 days

Mild >5 years:	azithromycin (10mg/kg, daily) for 3 days
Severe <5yrs (or <5yrs, and unable to tolerate oral antibiotics):	**amoxicillin/clavulanate (Augmentin) 25-50mg/kg (max 1.2g) IV 8-hourly
Severe >5 years:	amoxicillin/clavulanate (Augmentin) 25-50mg/kg (max 1.2g) IV 8-hourly + azithromycin 10mg/kg (max 500mg) po daily
***if penicillin allergy*:	oral azithromycin preferred but consider ceftriaxone 50mg/kg

GENITO-URINARY INFECTION

Take specimen(s) for culture and sensitivities and then start treatment. Therapy can be changed later in the light of the results. In patients aged over 65 years, there is no need to treat asymptomatic bacteriuria. In this group the condition is common and not associated with increased morbidity.

CYSTITIS (in non-pregnant woman)		
Uncomplicated		
Community – first presentation		trimethoprim (200mg bd) or nitrofurantoin (50mg qds) } 3 days
- failed initial treatment		cefradine (500mg bd) or ciprofloxacin (250mg bd) } po 7 days
Complicated (including all male adult patients, and recurrent infections in adult females)		trimethoprim (200mg bd) or ciprofloxacin (250mg bd) } po 7days
Catheter-associated		Give a single, 120mg dose of IV gentamicin, then 30 mins later remove/replace catheter.
PROSTATITIS		trimethoprim (200 mg bd) or ciprofloxacin (250mg bd) or oxytetracycline (500mg qds) } 4 weeks
EPIDIDYMO-ORCHITIS	in adults	ciprofloxacin (500mg bd) plus, doxycycline 100mg bd } 7 days
	in children	Seek advice
PYELONEPHRITIS		
Initial therapy	in adults[1]	cefotaxime IV (1g) plus gentamicin IV (5mg/kg) stat, then oral ciprofloxacin (500mg bd) for 13 days
	in children	ceftriaxone IV 80mg/kg daily for 3-5 days, then seek advice

[1]***Refer to revised antibiotic policy during outbreaks of Clostridium Difficile infection*, http://formulary/HTML/form2/DRUGS/PROTOCS/eacap.doc**

MUSCULOSKELETAL INFECTION

Osteomyelitis: adults : flucloxacillin (1g qds) IV* + fusidic acid (1g tds) po*
children: flucloxacillin 50mg/kg (max 2g) IV six times daily + ceftriaxone 80mg/kg (max 4g) IV daily

Septic arthritis: adults: flucloxacillin (1g qds) IV* + fusidic acid (1g tds) po*
children: flucloxacillin 50mg/kg (max 2g) IV six times daily + ceftriaxone 80mg/kg (max 4g) IV daily

Prosthetic device infection: vancomycin (1g bd) IV* +fusidic acid (1g tds) po*

*Take advice on length of treatment, IV to oral switch, and for vancomycin, the dose in renal failure. Modify therapy according to microbiology findings.

SEPTICAEMIA

Take blood cultures. Treatment must often be started before a firm microbial diagnosis is available. Clinical features sometimes suggest the probable cause; in other patients the age or the immune status of the patient give the best clues.
Treatment advice, including duration of treatment, when early laboratory information gives likely cause, will be provided by a microbiologist.
Best-guess policies before laboratory results are available should be guided by the likely focus of the infection. *Seek advice in cases of penicillin allergy*:

a) Lower gut, pelvis — amoxicillin IV (1g tds) + gentamicin IV (5mg/kg od) +metronidazole IV (500 mg tds)
OR (in case of penicillin allergy) cefotaxime (1g tds) *or* ceftriaxone in children + metronidazole IV (500mg tds)

b) Urinary-tract — cefotaxime IV (1g tds) plus a single dose of gentamicin IV (5mg/kg) in severe cases

c) Intravenous catheter — flucloxacillin IV(1g qds) or vancomycin IV (1g bd) if at risk of MRSA infection (dose depends on renal function). Remove catheter and send tip for culture.

d) Cellulitis (severe) — benzyl penicillin IV (1.2g six times daily) + flucloxacillin IV (1g qds)
(mild/moderate)[1] — co-amoxiclav oral (375mg tds)
Seek advice in cases of penicillin allergy.

e) Neutropenia — see next section

f) Sepsis with pneumonia — cefotaxime IV (1g tds) + erythromycin IV(1g qds)

g) Septic shock (no obvious focus) [1]adults: cefotaxime IV (2g tds) + gentamicin IV(5mg/kg od) + metronidazole IV (500mg tds)
children <6wks: penicillin 50mg/kg IV 6-hourly + cefotaxime 80mg/kg 8-hourly
>6wks: ceftriaxone 80mg/kg (max 4g) IV daily

[1] ***Refer to revised antibiotic policy during outbreaks of Clostridium Difficile infection*, http://formulary/HTML/form2/DRUGS/PROTOCS/eacap.doc**

h) Sepsis with gangrene/ tissue necrosis	clindamycin IV (600mg qds) + cefotaxime IV (1g tds) + single dose of gentamicin IV (5mg/kg) (In severe cases seek *urgent* surgical advice)
i) Deep neck space infection	co-amoxiclav IV (1g qds)

ANTIMICROBIAL POLICY FOR PATIENTS WITH NEUTROPENIA

Neutropenic patients who develop fever (in adults, temperature greater than 37°C for 2 hours or more, or 38°C or more at any time; in children, greater than 38°C for **>2** hours or on two occasions at least 4 hours apart, or >38.5°C on any reading) should be given antibiotics. Take blood, urine etc for culture and start antibiotics immediately. In those patients who have had recent positive cultures start with the appropriate antibiotic. For those in whom treatment is blind the regimen will vary according to the number of febrile episodes. In children a suitable regimen would be: tazocin 90mg/kg IV qds + gentamicin 7mg/kg IV.

In adults:

- 1st or 2nd episodes – start with gentamicin IV (5mg/kg od) + tazocin IV (piperacillin plus tazobactam) 4.5g tds or ceftazidime IV (2g tds) if allergic to penicillin
- 3rd episode – take advice

If the patient responds, complete the original regimen. If no response is seen by 48 hours, seek advice.

PROPHYLACTIC USE OF ANTIBIOTICS IN SURGERY

The timing of the first dose is important. If the antibiotic is to be injected IM, the initial dose should be given with the pre-med but no more than 1 hour before the operation. If it is to be injected IV, and this is preferable, it should be given at the induction of anaesthesia. Alternative prophylaxis will be needed in patients known to be colonised with MRSA; for this seek specialist advice. Specific advice is also available on the Intranet.

GENERAL SURGERY

BILIARY/UPPER GI/BREAST/LOWER GI/ENDOCRINE SURGERY

Refer to Surgical Department Antibiotic Prophylaxis Policy, http://formulary/HTML/form2/DRUGS/PROTOCS/GISAP2006.doc

TRAUMA AND ORTHOPAEDICS

Drainage of abscess	Nil
Bites	For any bites (human or animal) clean the wound then give co-amoxiclav (625 mg tds) 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. In patients allergic to penicillin give clindamycin (300 mg qds) plus ciprofloxacin (500mg bd) by mouth for 3-5 days.
Lacerations	Nil
Compound major fractures	Debridement plus IV benzylpenicillin (1.2g qds) + flucloxacillin (1g qds) for 4 days or cefradine (1g qds) for 4 days.
Open reduction of fractures with internal fixation	)) cefradine 1g at induction) then two further doses at 8 hour intervals

Elective orthopaedic endoprosthetic surgery) (seek advice if MRSA colonised)

GENITO-URINARY SURGERY

VAGINAL APPROACH

a) Hysterectomy — metronidazole 500mg IV ± cefradine 1g IM/IV

b) Other — Nil

ABDOMINAL APPROACH

a) Hysterectomy — metronidazole 500mg IV ± cefradine 1g IM/IV. If patient is catheterised give a 3 day course of amoxicillin or trimethoprim or cefradine

TERMINATION OF PREGNANCY

a) Uncomplicated — Nil

b) History of P.I.D. — (1) amoxicillin 500mg IM/IV (or cefradine 1g IM/IV) + metronidazole 1g PR or 500mg IV

or — (2) doxycycline 100mg oral pre-operatively

CAESAREAN SECTION

Give cefradine (1g IM or IV) plus metronidazole 500mg (IV) as a stat dose, after the cord is clamped

UROLOGICAL SURGERY

Refer to Urology Department Antibiotic Prophylaxis Policy, http://formulary/HTML/form2/DRUGS/PROTOCS/anturol.htm

PULMONARY SURGERY

Cefradine 1g at induction, then, 2 further doses at 8 hour intervals

CARDIO-VASCULAR INTERVENTION

a) Permanent transvenous pacemaker — Nil (however, if infection is evident at revision of pacemaker insertion, treat using flucloxacillin [vancomycin if MRSA carrier] ± amoxicillin for 48 hours).

b) Valve replacement — gentamicin 120mg stat plus cefradine 1g at induction then 2 further doses cefradine at 8 hourly intervals.

If MRSA carrier, give vancomycin 1g by infusion starting 1 hour before induction then 1g 12 hours later, plus stat dose of gentamicin (120mg) at induction. Additional or alternative antibiotics to be given if treating culture-positive endocarditis – seek advice.

c) Vascular surgery with insertion of graft — co-amoxiclav 1.2gIV at induction then 2 further doses at 8 hourly intervals plus vancomycin 1g IV by infusion at induction.

AMPUTATION

High Amputation

Lower limb

Benzyl penicillin 600mg QDS IV, or metronidazole 500mg IV tds, for 5 days.

PLASTIC SURGERY

Seek advice from Plastic Surgery Department.

OPHTHALMIC SURGERY

a) 'Clean'	Nil
b) Traumatized eye	topical chloramphenicol

ENT SURGERY

Refer to ENT Department Antibiotic Prophylaxis Policy,
http://formulary/HTML/form2/DRUGS/PROTOCS/aent.doc

PROPHYLAXIS TO PREVENT ENDOCARDITIS

DENTAL

Under local anaesthetic

1) Known valvular disease including prosthetic valve or septal defect

adults: amoxicillin 3g
children: <5yrs 750mg
5-10 yrs 1.5g
>10 yrs 3g
or adults: clindamycin 600mg
children: <5yrs 150mg
5-10 yrs 300mg
>10 yrs 600mg
by mouth 1 hour before.

2) Previous endocarditis — As for GA below

Under general anaesthetic

1) Previous endocarditis - adults: amoxicillin 1g **IV** or vancomycin* 1g IV +gentamicin 120mg IM) 15-30mins before, then after 6hrs: 500mg amoxicillin po

children: 1-5yrs: IV amoxicillin 250mg + IV 2mg/kg
Then after 6hrs, 125mg amoxicillin po

5-10yrs: IV amoxicillin 500mg + IV gentamicin 2mg/kg. Then after 6hrs, 250mg amoxicillin po

>10yrs: as for adults

2) Valve or septal defect but at no special risk

adults: amoxicillin 1g **IV ±** gentamicin 120mg IM at induction, then amoxicillin 500 mg orally after 6 hrs.

children: amoxicillin 20mg/kg (max 2g) IV + gentamicin 1.5mg/kgIV

INVESTIGATION/SURGERY (damaged or prosthetic valve or septal defect)
UNDER GENERAL ANAESTHETIC

Tonsillectomy	)1 hr before give amoxicillin 1g
Bronchoscopy	)IM (or vancomycin 1g IV*)
	)+ gentamicin 120mg IM
Sigmoidoscopy	)If amoxicillin given, repeat in a dose of
Barium enema	)500mg after 6 hours
Cystoscopy	)
Catheterisation	)If urine infected prophylaxis
D & C	)should cover the infective organism
Labour	)
IUCD insertion/removal	)

* Use if patient is allergic to penicillin or if has had 2 or more penicillin courses in previous month.

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 penicillin and 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 penicillin together with appropriate tetanus prophylaxis.

MALARIA IN RETURNING TRAVELLERS

Any traveller returning from an endemic area should be considered as at risk of having malaria. If it is suspected, seek specialist advice from an adult or paediatric infectious disease physician. Other conditions, such as typhoid, should also be considered.

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 500mcg/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). Also give cefotaxime in elderly patients and those with predisposing disease such as rheumatoid arthritis. 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 RENAL FAILURE

(Link consultant: Dr Iain MacPhee)

Acute renal failure (ARF), 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 haemoglobinaemia). Other less common causes of ARF are obstruction, acute interstitial nephritis, as seen with drug hypersensitivity, and rapidly progressive glomerulonephritis occurring as a primary event or complicating multi-system disease.

ARF 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 up to a maximum dose of 40mL.
   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) 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.
   e) Check serum K^+ levels at least twice daily.
   f) 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 3400, or via switch-board), should be considered in any patient with –

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

Remember ARF 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. Patients receiving contrast probably benefit from being given N-acetylcysteine 600mg twice daily orally on the day before, and on the day of, administration of the contrast agent. They should also be given IV fluids (sodium bicarbonate 1.4% or sodium chloride 0.9%) at 125mL/hr, 12 hours before, and 12 hours after, the procedure. Hypovolaemia due to blood or fluid loss should be avoidable or rapidly reversible. Be very cautious when using drugs such as aminoglycosides and NSAIDs that might cause renal damage.

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.

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 Renal Failure on page 61.

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, i.e 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 (e.g. 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 1-5 micrograms 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 i.e. 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 hyper-glycaemia or renal failure and treat as such
b) if osmolality less than 275 mosmol/kg, then treatment will depend on whether the patient is
   - hypovolaemic (causes: diuretics, vomiting, diarrhoea, cortisol deficiency)
   - 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 the volume of 0.9% saline (150mmol/L) to be given over 24 h from the above 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 ather 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 David Bevan)

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 over 100 crises every year. Many patients have a personal management protocol which is kept in a file in their name in Majors in A&E. This 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 100mcg naloxone every 2 mins as necessary.
- Do not delay starting analgesia while awaiting the results of investigations, or 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 unless specifically indicated on the patient's personal protocol.
- 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. 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 specialist nurse counsellors and let him/her know of the attendance and assessment. This may be done by telephoning Balham Health Centre on 0208 700 0615.
- 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 and 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 oxygen tensions – a falling PaO_2 will require exchange transfusion which needs expert advice. Patients with chest pain should be encouraged 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 the patient may develop abdominal pain with rigidity, loss of bowel sounds and increasing icterus. IV fluids are vital. A surgeon should be consulted to exclude other abdominal events, but surgery should be with-held 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 presenting 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 a simple transfusion for anaemia (except in those sequestrating), without reducing the HbS level by exchange. If this precaution is not taken the blood viscosity will be increased and the patient made 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 PSYCHOSIS

(Link consultant: Professor Hamid Ghodse)

Acute psychotic states may present in casualty or develop unexpectedly in a general ward. Possibilities include acute organic reactions, severe depression, acute paranoid psychosis, schizophrenia and mania. Restless hallucinated patients are easily terrified

or bewildered. For some patients the situation can be managed by using a calm approach and interviewing in a well-lit side ward or cubicle; if this fails seek help from the duty psychiatrist.
Acute organic reactions are the commonest psychoses in general wards and may be caused by drugs used for treatment (cimetidine, anti-cholinergics), drug withdrawal (alcohol, barbiturates), some underlying systemic disease (cardiac, renal, hepatic or respiratory failure), a local cerebral lesion such as meningitis or a tumour or dementia plus infection. In recent admissions (or in casualty) common causes are drug abuse (alcohol, amphetamine, cocaine, ecstasy, cannabis), head injury, epilepsy or meningeal irritation. Management involves treatment of the underlying cause and withdrawal or reduction of as many psychotropic drugs as possible. Avoid hypnotics. If sedation is required, small doses of haloperidol (1.5-3mg) or chlorpromazine (25-50mg) may be given by mouth up to four times daily, although in those with cocaine intoxication a benzodiazepine is the drug of choice as it does not lower the seizure threshold. Be sure to check the blood sugar concentration to rule out hypoglycaemia in any patient in whom the level of consciousness is altered.

Treatment of alcohol withdrawal involves vitamin supplements, water and electrolyte control and the administration of sedative anticonvulsants. For sedation and control of withdrawal symptoms use a benzodiazepine in decreasing doses over 6-7 days; doses will vary depending on the patient's gender, size, symptom severity, co-morbidity and history of withdrawal seizures. Reasonable benzodiazepine regimes for severely dependent patients are:

	diazepam	chlordiazepoxide
day 1:	15mg QDS	30mg QDS
day 2:	10mg QDS;	30mg TDS
day 3:	10mg TDS;	20mg TDS
day 4:	5mg QDS;	20mg BD
day 5:	5mg TDS;	10mg BD
day 6:	5mg BD.	10mg nocte
day 7:	5mg nocte	

An alternative (second choice) sedative is clomethiazole (starting dose 9-12 capsules per day in 3-4 divided doses), but this is difficult to use. Do not discharge patients with clomethiazole. For further information contact the Drug and Alcohol Liaison Service (SGH Ext 0601).
If symptoms of withdrawal are severe, or if the patient is delirious, it may be necessary to increase the dose temporarily. Heavy drinkers should receive Vitamin B_1 (thiamine), 100mg orally two or three times daily for three weeks. Patients with severe thiamine depletion or who have Wernicke's encephalopathy will require IV administration of Pabrinex High Potency (vitamin B + C) for 5 days followed by oral administration. Intravenous injections should be administered slowly (over 10min) to avoid serious allergic reactions. *Remember not to mix drugs in the same syringe*. If fits persist following withdrawal, either phenytoin or carbamezepine would be suitable anti-convulsants.
Severe depression. Early psychiatric referral is important. Anti-depressant drugs are not always necessary.

Acute paranoid psychosis also needs psychiatric referral. While waiting, give oral lorazepam (1-2mg) or chlorpromazine (50-100mg) or haloperidol (5-10mg) 4-6 hourly, the dose depending on the size of the patient and the degree of disturbance.

Manic, violent or aggressive psychotic patients can usually be sedated with lorazepam 1mg IM or IV (up to a dose of 4mg/day); or haloperidol by IM injection up to 5-10mg, 4-8 hourly, up to a dose of 18mg/day or as syrup orally 5-10mg every 4 hours, up to a maximum of 30mg/day. If an oculogyric crisis or an acute torsion dystonic reaction develops, give procyclidine 5-10mg IM or 5mg IV, repeating after 20 minutes if reactions not controlled, up to a maximum (daily) dose of 20mg, or benzatropine 1-2mg IM or IV.

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 T (cTnT). Diagnosis of AMI requires elevation of a cardiac troponin. Measurements can be particularly helpful in providing an accurate diagnosis in patients with musculoskeletal injury causing rises in CK and CK-MB. Moreover, levels remain elevated for at least 7 days following acute myocardial infarction, so can be used in diagnosis when the patient presents late. It should be noted that cTnT also rises in other conditions where there is cardiac damage, such as myocarditis.

In most patients, measurement of CK and cTnT on admission and 12 hours later will be adequate for diagnosis of AMI. Measurement at 4 hours may be used if diagnosis is uncertain and an intervention is being considered. Measurement at 24-48 hours after admission can also help if uncertainty persists.

Measurement of cTnT is particularly helpful when making decisions about patients:

- presenting more than 12 hours after the onset of symptoms
- in whom the CK elevation may be of musculoskeletal origin (as in trauma or after surgery)
- without ST segment elevation but who are being considered for angiography and subsequent intervention.

CK and cTnT should be requested on admission and 12 hours from admission in all patients with chest pain with the possible diagnosis of acute MI. Re-infarction may be detected by repeat measurement.

APPENDIX 2

WHEN TO ATTEMPT CARDIO-PULMONARY RESUSCITATION

(Link consultant: Dr Jeremy Cashman)

All patients should receive cardio-pulmonary resuscitation (CPR) in the event of cardiac arrest unless specific ("Do Not Resuscitate"; DNR) instructions are written in the notes. Anyone initiating CPR in such circumstances should be supported by their medical and nursing colleagues.

The Trust takes the position that it is appropriate to consider a patient for a "Do Not Resuscitate" (DNR) order in any of the following circumstances:

- where the patient's condition indicates that attempts at CPR would not be successful.
- where CPR is not in accord with the recorded, sustained wishes of the patient who is mentally competent (see final note in this appendix).
- where not providing CPR is in accordance with a valid applicable advanced directive ("living will"). A patient's informed and competently made refusal, which relates to the circumstances that have arisen, is legally binding upon doctors.
- where successful CPR is likely to be followed by a length and quality of life, which would not be in the best interests of the patient to sustain.

What does "Not for CPR" mean?

- The instruction "Not for CPR" means that full CPR is inappropriate for that particular patient and hence the "Crash" team should not be called automatically in the event of cardiac or respiratory arrest. If such a patient were to collapse or arrest, an immediate assessment must be made as to the cause, and appropriate simple measures taken with regard to airway patency, patient position etc., and a relevant doctor from the appropriate firm informed immediately. No other attempts at basic or advanced life support should be commenced unless specifically instructed by that firm.
- "Not for CPR" does not affect the patient's routine therapy (antibiotics, surgery, dialysis), which should continue to be provided as normal.

Who should make the decision not to resuscitate?

- The decision not to resuscitate is always a medical one, i.e. made by a doctor. The final responsibility for the decision rests with the consultant in charge of the patient's care, who is ultimately responsible for the essential documentation and communication of the patient's resuscitation status. However, the decision should be discussed with all other members of the multidisciplinary team involved in the patient's care.
- If feasible, the views of the patient, particularly if he/she is mentally competent (see below), should be sought and taken into consideration, as should the views of the immediate relatives, but with due regard to patient confidentiality. Nursing staff should be present when the decision is discussed with the patient and/or relatives.
- Under the Access of Medical Records Act 1990, a patient (and perhaps relatives) has the right to receive copies of their Medical Record. Thus, soundly-based decisions and full documentation are necessary.
- The decision not to resuscitate should be made as early as possible by a senior member of the medical team in charge of the patient's care, usually the consultant. In certain circumstances a DNR order may *initially* be made by the most senior doctor on-call(usually a specialist registrar) after seeing the patient and discussing the situation as detailed above. *Any decision made by a junior doctor must be communicated to the consultant "on call" within 24 hours and then subsequently communicated to the patient's own consultant, at the first available opportunity*.
- The decision not to attempt CPR in children must only be taken by a consultant together with the parents. The decision must be documented and communicated, as detailed here.

Writing the instructions "not for CPR " in the notes

- The instruction "not for cardiopulmonary resuscitation" should be entered in writing on the inside front cover of the medical notes, together with the reason for the decision, whether or not the decision has been discussed with the patient and/or relatives, dated and signed legibly;

- the instruction should also be entered in the nursing notes. This should be done by the primary nurse or the senior nurse responsible for informing all other members of staff.

The decision not to resuscitate can be rescinded

- The decision "Not for CPR" should *never* be regarded as final, since its appropriateness may vary with the patient's clinical condition. The decision should be reconsidered regularly as part of the patient's management plan. This should be undertaken at intervals appropriate to the patient's clinical condition.
- Nurses and Professions Allied to Medicine must be alert to any alteration in the patient's underlying condition which might affect the patient's resuscitation status. Such concerns must be brought to the attention of senior medical staff.
- If a DNR order is rescinded, the "CPR status" entry in the medical notes should be struck out with a single line, signed legibly and dated. A new dated and signed entry should then be made at the correct chronological point in the patient's notes. In addition, nursing documentation will need to be updated accordingly and subsequent changes communicated to all staff.

Communicating the policy

- All members of the medical team must be aware of the decision that a particular patient is "Not for CPR".
- The fact that a patient is "Not for CPR" must be communicated at every nursing hand-over, such that the resuscitation status of every patient is known to every nurse on the ward.
- The fact that the patient is "Not for CPR" must be communicated to all Healthcare Staff with whom the patient may come into contact.

Assessing competence in adults

Patients are considered to be mentally competent if:

- they can comprehend and retain information about procedure
- they believe that information
- they can weigh information and balance risks to arrive at a choice

APPENDIX 3

ENTERAL/PARENTERAL FEEDING

(Link consultant: Dr Penny Neild)

Malnutrition – overt or covert – delays recovery and increases the risk of clinical complications. Any patient at risk of malnutrition by virtue of disease or complications should be referred to the ward dietician via the patient's notes. 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.

The Nutrition 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. PN should not be seen as an emergency intervention and will not be instigated outside weekday working hours or at weekends (on Fridays referrals

should be made before midday). The team may be contacted by bleeping any of the members: Dr Penny Neild, Consultant Gastroenterologist (ext.3429; bleep SG 151); the Gastroenterology SpR (bleep 6590); Vin Kumar, PN Pharmacist (bleep 6269); Catherine Collins, Chief Dietician, Claire Hanika, Senior Dietician (bleep 7007), Alison Green, Senior Dietician (bleep 6623) or Tanya Robinson, Macmillan Dietician (bleep 6383).

APPENDIX 4

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^+(> 43mmol/L) and a low plasma HCO_3^-.

Mechanisms: a net gain of acid (increase in endogenous production or exogenous administration) eg. diabetic ketoacidosis, aspirin poisoning;

a net loss of alkali eg. loss from intestine (diarrhoea) or renal tract (renal tubular acidosis)

a 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 therapeutic goal is to raise the arterial pH to about 7.20, a level at which arrhythmias are less likely and cardiac contractility is restored. Do not attempt to fully correct the pH as continuing hyperventilation will make the patient alkalotic and may precipitate tetany.

- In patients with renal failure who are acidotic and volume *deplete*, give $NaHCO_3$ 1.4% (regime 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 30.
- 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 5

CONSIDERATIONS BEFORE ATTEMPTING LUMBAR PUNCTURE

(Link consultant: Dr Tim Von Oertzen)

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 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 it is an absolute emergency, and this includes suspected meningitis (see page 52), lumbar puncture is best done during normal working hours. Make sure that samples reach the lab(s) in good time. Remember, most indications for lumbar

puncture 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 tests (bio-chemistry, microbiology) and for tests that might need to be done later (cytology, virology). Volumes greater than 10ml may be needed. At the same time as 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 lumbar puncture 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 or coagulation defect or low platelet count (see Appendix 6).

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 or 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.

PROTECTING ANTICOAGULATED PATIENTS FROM BLEEDING

(Link consultant: Dr Muriel Shannon)

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 in:

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

Initially, weekly measurement is advised, and the anti Xa assay (measured 4 hours 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.

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
2. Give a 5000u loading dose as a bolus injection IV
3. Start IV infusion, giving 15-25u/kg/hr (dilute the 2mL ampoule containing 25,000u in 48mL normal saline and start at 2mL/hour; each mL contains 500u)
4. The target therapeutic range for APTTR (using synthetic reagent) 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 500u/hr (1mL/hr)
5.3-5.9	reduce by 300u/hr (0.6mL/hr)
4.7-5.2	reduce by 200u/hr (0.4mL/hr)
4.1-4.6	reduce by 100u/hr (0.2mL/hr)
3.6-4.1	reduce by 50u/hr (0.1mL/hr)
1.5-3.5	NO CHANGE
1.2-1.4	increase by 200u/hr (0.4mL/hr)
<1.2	increase by 400u/hr (0.8mL/hr)

5. Repeat APTTR daily while on unfractionated heparin
6. Check platelet count 5 days after starting therapy

Bleeding in a patient on heparin. Older patients on heparin for >4 days are most at risk but bleeding can occur in anyone, from any source. Bleeding can be silent into a "third space" such as the retroperitoneum. A falling haematocrit, back pain or even severe anxiety on the part of 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 intramuscular injection.

Action – if the patient is on continuous infusion of heparin (as for UFH) via a pump the heparin should be STOPPED (heparin activity will be lost from the plasma within 2 to 4 hrs). For rapid reversal, protamine should be given at the dose of lmg per 100U heparin infused per hour. 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). This affects up to 1 in 10 patients on IV or SC heparin. An interaction with an antibody in the plasma causes clumping and loss of platelets. It usually shows as a progressive fall in platelet counts. This is gradual in most patients but in some there will be explosive thrombotic disease due to platelet emboli. Attempts at surgical removal or thrombolysis fail if heparin is continued. It is crucial to recognise this syndrome and immediately stop heparin by ALL routes and ALL doses including that in IV fluids and cannulae. An alternative anti-thrombotic agents such as danaparoid should be substituted. Seek urgent advice from the Haematology Department on-call service.

Invasive procedures in patients on heparin. Intravenous UFH should be stopped at least 2 hours before undertaking an invasive procedure. In a patient on LMWH, or subcutaneous UFH, the time should be extended to 12 hours.

WARFARIN - Tackling excessive warfarin-induced anticoagulation
Minor haemorrhage eg haematuria, epistaxis. Reduce dose or, if INR >4.5, withhold warfarin for one or more days. If INR >8 consider giving phytomenadione 0.5 to 2.0 mg orally (the IV preparation can be given by mouth). Vitamin K administration is, however, not appropriate for a patient with an artificial heart valve as in this instance it may induce prolonged 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 cross-matching. Stop warfarin and immediately give Vitamin K 5mg by slow IV injection. Prothrombin complex concentrate (PCC), which is held in the Haemophilia centre, should be given. Involvement of the Haematology Team will be required. If no concentrate is available then FFP should be infused (15mL/kg). Do not re-start warfarin until bleeding is controlled.

INR >4.5 with no bleeding. Reduce dosage or withdraw warfarin for one or two days and review. Stopping for 1-2 days is likely to be necessary if INR > 5.0.

APPENDIX 7

HIV POST-EXPOSURE PROPHYLAXIS

(Link consultants: Dr Phillip Hay & Dr Nita Mitchell-Heggs)

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 is terminally ill with HIV infection, 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 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 the three drugs; either the combination tablet Combivir (zidovudine 300mg plus lamivudine 150mg) and nelfinavir (1.25g) both twice daily. 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 3-day packs containing Combivir plus nelfinavir are kept by Staff Health, McEntee ward, A&E, Courtyard Clinic and the Pharmacy at AMH.

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.

APPENDIX 8

WORKING WITH AIDS PATIENTS

(Link consultant: Dr Phillip Hay)

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 in Appendix 9.

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 Daniel Wallis)

St George's must always be prepared and ready 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 hospital may be warned by the London Ambulance Service (LAS) that such an incident has arisen, 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 (A & E consultant), Nursing Coordinator (senior nurse on call/Director of Nursing), and Incident Coordinator (senior 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 (other entrances may be locked). All *patients* involved in the incident should enter via the ambulance entrance to A & E. If there is a *suspected chemical, biological or radiation hazard*, the hospital and A & E Department will be made 'secure'. In these circumstances patients coming from the incident will be corralled outside the A & E Department; a decontamination area with 'clean' and 'dirty' areas established; and decontamination facilities set up by A & E staff with appropriate personal protective equipment. No patient who has been exposed to a chemical or biological hazard will be allowed into the A & E department until after they have been decontaminated. If patients potentially contaminated with radioactive material require admission to the A & E department or theatres for life-saving treatment, special precautions will be taken.

All patients who leave the A & E 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 who have been discharged from A & E will be located in the Rehab Gym in the Physiotherapy Department, ground floor St James' Wing (ext 3013), and a Relatives' Information Centre (for friends and relatives) will be located in the Main Gym in the Physio Department (ext 1357).

- **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-callshould continue with their normal work unless contacted and asked to do otherwise.

Outside normal working hours, all on-call medical staff should report 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-callwho wish to come in should go in the first instance to the Anaesthetic Department, first floor Grosvenor Wing.

- The assembly area for registered volunteers and medical students will be the Monckton Theatre, Grosvenor 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 Specialist Registrar 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 Specialist Registrar in 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 3196) 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 IV/IM dose; therapeutic range: trough <1μg/mL.
Conventional regimen - sampling time between 3rd and 4th dose: trough immediately prior to next dose, peaks 60min post IV dose, 60min post IM dose. Therapeutic range: trough <2μg/mL, peak 5-10μg/mL (4-6μg/mL may be acceptable when used with another antibiotic), for strepto-coccal or enterococcal endocarditis trough <1μg/mL, peak 3-5μg/mL; time to steady state 12-40 hours (longer in renal failure).
Vancomycin[1] In patients receiving vancomycin by intermittent infusion - sampling time between 3rd and 4th dose: trough immediately prior to next dose; peak 60min after end of infusion; therapeutic range: trough 5-10μg/mL, peak 25-40μg/mL; time to steady state: 30-40 hours (longer in renal failure).
In patients receiving vancomycin by continuous infusion, sample during the infusion; therapeutic range 15-25μg/mL.
Phenytoin[2] Sampling time: immediately prior to next dose; therapeutic range: 10-20mg/L (interpretation difficult in renal failure, low albumen, raised bilirubin); time to steady state: 7 days or longer.
Carbamazepine[2] Sampling time: immediately prior to next dose; therapeutic range: single therapy 8-12mg/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: 0.9-2.2μg/L; time to steady state: 7 days (longer in renal failure).
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.
Lithium[2] Sampling time 12 hours post dose; therapeutic range: as treatment - 0.4 –1.2 mmol/L, as prophylaxis - 0.5-0.8mmol/L; time to steady state: 3-7 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, ext 5843 or pager SG 138 or via switchboard out of hours.**

BLEEP & TELEPHONE NUMBERS

PRHOs

Medicine	**Bleep**	**Surgery**	**Bleep**
Drs Clark & Forton	6424	Mr Fiennes & Mr Reddy	6364
	6576	Mr Fiennes & Mr Reddy	6300
Dr Bourke & Dr Kiely		Mr Fiennes & Mr Reddy	6367
Dr Bourke & Dr Kiely	6335	Mr Melville & Mr Hagger	6581
Dr Pollok & Dr Poulis	6577	Mr Melville & Mr Hagger	7161
Dr Earle	6322	Professor Kumar	6362
Dr Panahloo & Dr Seal	6559	Mr Loftus & Prof Thompson	6536
Dr Panahloo & Dr Seal	6578	Mr Loftus & Prof Thompson	6555
Dr Eastwood & Dr Banerjee	6225/6579	Mr Mokbel	6361
Dr Baker, Dr Rayner & Dr	6326	Mr Sharma & Mr Banerjee	6582
McGrath		Mr Sharma & Mr Banerjee	7352
Dr Ong & Dr Draper	6563	Mr Bircher (orthopaedics)	6583
Dr Ong & Dr Draper	6580	Miss Daly (orthopaedics)	6556
Dr Antonios	6135/6324	Mr Powell (plastics)	6279
Geriatrics			
Dr Coles	6554		
Dr Hastie	6539		
Dr Cottee & Dr Cloud	6221	**Intensive Care**	
		Dr Newman	
Ophthalmology (1)		**Paediatrics**	
Mr Thompson	6252	Professor Walters	7011

DEPARTMENTS

Audiological Medicine
Chief receptionist 1880
Enquiries 5264

Cardiothoracic Unit 5288

ECG 1385/6/7
Bleeps: 6641 St James
6403 Lanesborough
6436 Knightsbridge

Casualty X-ray 1296

Chest Medicine
Receptionist 3318/1273
Lung function tech. 1667

EEG/EMG 4632

Clinical Biochemistry
Enquiries/results 5862/3/4
Urgent requests 5871

Dermatology
Secretary 1997

Diabetes/Endocrinology
Appointments 1429

Genito-urinary medicine
Reception 3353/4

Haematology
Blood bank 5471/5477
Enquiries 5468/5470
Haemostasis 5479

Maxillofacial Unit
Reception 1244

Medical Microbiology
Consultant/Registrar 1970
Enquiries/results 5693
Mobile X-ray bleep 6345/6284

Neurology Department
Secretary 1796

Norman Tanner Unit 1564
Nursing 1491

Histopathology
Enquiries 5264
PM's 5240
Frozen section bookings 5257/5264
Cytology 5266/5268

ITU 1307
Nurses station 3294

Patient Affairs 3410/3411

Palliative Care Team 3311
bleep: 6796/6508

Pharmacy Drug Info 1759

Psychiatry Emergency Clinic 5288

Switchboard -
SGH 1000
SGHMS 5000
Bolingbroke 43000
Springfield 42000