Any protocols, guidelines, algorithms are subject to review and updating. Ensure you are using the current version.
Unresponsive? 

Open airway
Look for signs of life

Call 222
Resuscitation Team

CPR 30:2
Whilst defibrillator / monitor attached

Assess rhythm

Shockable
(VF / pulseless VT)

1 Shock
150-360 J biphasic
or 360 J monophasic

Immediately resume
CPR 30:2
for 2 min

During CPR:
• Correct reversible causes*
• Check electrode position and contact
• Establish / verify: IV access, airway and oxygen
• Give uninterrupted compressions when airway secure
• Give adrenaline 1mg IV every 3-5 min
• Consider: amiodarone, atropine, magnesium

Non-Shockable
(PEA / Asystole)

Immediately resume
CPR 30:2
for 2 min

* Reversible Causes
Hypoxia
Hypovolaemia
Hypo/hyperkalaemia/metabolic
Hypothermia

Tension pneumothorax
Tamponade, cardiac
Toxins
Thrombosis (coronary or pulmonary)
<table>
<thead>
<tr>
<th><strong>USEFUL TELEPHONE NUMBERS</strong></th>
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<tbody>
<tr>
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<td>&gt;6PM</td>
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<td>(THEATRE)</td>
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**LABORATORIES TELEPHONE AND BLEEP NUMBERS**

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<td>CEPOD Coordinator</td>
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USEFUL EXTERNAL NUMBERS

SHORT CODES
RIE SWITCHBOARD ......................................................7050
RIE NUMBERS ..........................................................705 plus extn
WGH SWITCHBOARD .....................................................7910
WGH NUMBERS ..........................................................791 plus extn
RIE LABORATORIES ENQUIRIES..............................extn 27777

PROCURATOR FISCAL ......................................................01506 844556
(Linlithgow for St John’s patients) ........................................short code 62174
Out of hours: via police

POLICE .............................................................short code 62148

TRANSPLANT COORDINATOR ........................................0131 536 3946

HSDU ..........................................................0131 242 6105
## IN CARDIAC ARREST & LIFE THREATENING EMERGENCY CALL 222

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<th>WGH .......................... 32496  Bleep: 8355/8113</th>
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<td>WARD 118 RIE.......................... 21187/21188</td>
<td>WARD 20 WGH.......................... 31664/31665</td>
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adult medical emergencies handbook | NHS LOTHIAN: UNIVERSITY HOSPITALS DIVISION | 2007/09
### OTHER USEFUL TELEPHONE NUMBERS

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EDITOR’S INTRODUCTION

The Adult Medical Emergencies Handbook was first developed for the Western General Hospital in Edinburgh in 1996 and first appeared in 1998. Over the following three years hospitals in Edinburgh became a Trust and a Lothian University Hospitals Trust edition was produced. This was greatly strengthened by the development of management plans agreed across the city by specialists in both the Royal Infirmary and the Western General and has been in use up until this edition. In this edition The Lothian Hospitals are working under the aegis of Lothian Health. So this edition of this evolving work (the 4th) is the Adult Medical Emergencies Handbook for Lothian Health and specifically for RIE, St John’s Hospital and WGH. I hope that it has improved with revision.

We have built on the strong foundations laid by all who have contributed to the previous editions and they are accredited in the intranet version. Many new colleagues have helped with this version and I hope that they have all been acknowledged, but if not I apologise to them and thank them for their contributions.

This kind of project is dynamic. I am extremely grateful to Nicky Greenhorn of the Medical Illustration Department, Learning Technology Section of the University of Edinburgh who has been a major partner in the production of this book. I also acknowledge the great support and help I have had from colleagues but wish to particularly thank Emma Williamson, Cathy Kelly, Simon Maxwell, Ben Shippey, Susan Nimmo, Neil McGowan and Robin Mitchell who have all contributed detailed reviews of the manuscript with multiple helpful comments and suggestions. I would also like to thank Jane Pearson and Morag Naysmith for their contributions and also for co-ordinating the proof reading by specialist pharmacists.

I would also like to encourage any “users” to feed back with comments, suggestions and criticisms so that we can continue to improve this work. On a lighter note copies of the handbook have been sighted around the world! Basra, Sydney and Kirkcaldy! The editor would be interested in receiving notice (or photos) of future sitings.

ACKNOWLEDGEMENT

We are grateful to the Resuscitation Council (UK) for permission to include algorithms from the Advanced Life Support teaching materials. We are also grateful to the American College of Surgeons for permission to reproduce a table from the ATLS manual.
LIST OF CONTRIBUTORS TO 4th EDITION

EDITORIAL COMMITTEE
B Chapman, C Kelly, S Maxwell, M McKechnie, G Nimmo, M Naysmith, E Olsen, J Pearson, B Shippey, E Williamson,

CONTRIBUTORS TO THIS EDITION OF THE HANDBOOK
Dr K Adamson, Dr N Amft, Mr D Anderson, Dr I Arnott, Prof J Bell, Dr C Blair, Dr B Chapman, Dr N Colledge, Dr P Dale, Dr J Dave, Dr R Davenport, Prof M Dennis, Dr A Dennison, Dr M Denvir, Dr V Dhillon, Dr A Elder, K Farrer, Dr M Ford, Dr A Gibb, Dr P Gibson, Miss T Gillies, Dr C Goddard, Dr I Grant, Prof A Greening, Dr E Halloran, Mr S Hartley, Prof P Hayes, Dr G Howard, Dr P Johnson, Mr M Johnstone, Dr P Kalima, Dr S Keir, Dr C Kelly, Dr C Leen, Dr M Logan, Dr V Macaulay, Dr S MacKenzie, Dr A MacLullich, Dr N McGowan, Dr M McKechnie, Dr J McKnight, Dr E McRorie, Dr M Mackie, Dr C Maguire, Dr L Manson, Dr S Maxwell, Dr S Midgley, Dr R Mitchell, Dr S Moultrie, Dr G Nimmo, Dr S Nimmo, Dr E Olson, Dr A Patrick, Dr P Padfield, Dr K Palmer, Prof S Ralston, Dr S Ramsay, Mr Z Raza, Dr P Reid, Dr R Reynolds, Dr P Riches, Dr Rustam Al-Shahi Salman, Dr J Spiller, Dr I Starkey, Dr M Strachan, Dr J Stone, Dr N Uren, Mrs L Waite, Dr J Walker, Dr S Waring, Dr W Whiteley, Dr D Wilks, Dr A Williams, Dr E Williamson

PHARMACISTS WHO PROOF READ SPECIALITY SECTIONS

ENTIRE TEXT PROOF READERS
Dr C Kelly, Dr S Maxwell, Dr B Shippey, Dr S Nimmo, Dr E Williamson, Dr N McGowan, Dr Karen Adamson

Grateful acknowledgement is made to authors of:
- CCU therapeutic schedule
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- Acute care algorithms
- Infusion devices guidelines
- Alcohol withdrawal guideline
- Agitation/confusion in the elderly guideline
- Major Haemorrhage protocol
- Malignant Hyperpyrexia protocol
- Acute pain guidelines
- Lothian DNAR group
Chapter 1

GOOD CLINICAL PRACTICE

INTRODUCTION

The purpose of this handbook is to provide management guidelines for adult medical emergencies in your hospital. The handbook has been written by specialists who deal with these emergencies on a daily basis. It has been edited to standardise the approach and has been reviewed and approved by colleagues. Contents are evidence and best-practice based as far as is possible and are aligned with National and International guidelines where appropriate.

It is designed to advise staff in training and in practice on the management of most common adult medical emergencies, and in the management of unusual but important clinical conditions. However, the book is intended as a guide and is not a substitute for immediate expert help when this is needed: if in doubt ask for senior advice or assistance.

The book is divided into three sections:

Section 1 General information
Section 2 Clinical Management
Section 3 Appendices

The handbook should be used in conjunction with

- Local and Divisional protocols and guidelines.
- The Lothian Joint Formulary,
- Divisional guidelines for anti-microbial therapy (Sepsis section Chapter 2)
- Divisional Acute Pain Guidelines offer detailed information which is complimentary to this text.

Medicine doses are being continually revised and novel adverse effects of drugs may be discovered over time. Every effort has been made to ensure that recommended dose ranges are appropriate and evidence based at the time of going to press but prescribers are advised to consult the BNF and where necessary the product data sheets.

Throughout the text useful clinical information is highlighted as ‘key points’ identified by the notation below.

KEY POINT these are useful, often practical, pieces of information.

Comments and suggestions relating to the book are welcomed and should be addressed to: Dr Graham Nimmo - Consultant Intensive Care and Clinical Education at WGH
E-mail: Graham.Nimmo@luht.scot.nhs.uk
GENERAL POINTS

Medicines

• Doses are for adults unless otherwise indicated.
• While every effort has been made to check doses, if doubt exists consult the BNF.
• The Lothian Joint Formulary should be consulted for local prescribing advice/guidance. All adverse events involving black triangle (recently marketed) and serious adverse events involving any drugs should be reported to the MHRA using yellow cards which are available in paper form in all BNF’s and also online via the Trust intranet or at http://www.mhra.gov.uk
• European law requires the use of the Recommended International Nonproprietary Name (rINN) for medicinal substances. In most cases the British Approved Name (BAN) and rINN were identical. Where the two differed, the BAN was modified to accord with the rINN with the important exceptions of adrenaline and noradrenaline. The new BANs are used in this text.

Infections

• The University Hospitals Division and St John’s antimicrobial guidelines offer excellent advice on treatment choice: these are updated regularly. (Sepsis section Chapter 2).
• Clinical Microbiological advice is available 24/7.

Specialist Referral

• Throughout the text advice on criteria for specialist referral is given, along with contact numbers for specific hospital sites.
• If the patient is pregnant discuss with the Obstetric registrar on call.
• Consider early referral for ICU or HDU care (see assessing illness severity Chapter 2) when appropriate.

Decisions to be made for every admission

• Medicines: consider which long term medicines should be continued and which with-held eg vasodilators in sepsis or in hypovolaemia, diuretics in dehydrated.
• Remember to assess the need for DVT prophylaxis in all admissions. Consult Divisional guideline for venous thromboembolism prophylaxis and treatment, or local protocol where appropriate.
• Consider the resuscitation status of each patient at admission: discuss with the consultant responsible. Document on the specific Do Not Attempt Resuscitation (DNAR) sheet and include in casenotes. Always discuss with next of kin.
• Incapacity: see below.

• **Diagnosis:** review the evidence. The diagnostic “label” may be inaccurate or incomplete. Don’t make assumptions and “don’t give up the search” for alternative explanations for the patient’s presentation especially if they do not respond to initial treatment.

---

**ADULTS WITH INCAPACITY ACT**

**General Principles**

• Under the Act an adult is defined as a person who has attained 16 years of age.

• All adults are considered capable of making their own medical decisions unless proven otherwise.

• Without consent for any procedure or treatment the health care professional carrying out the procedure or treatment could be liable for assault.

• It is the doctor primarily responsible for the patient’s medical treatment who is responsible for assessing the patient’s capacity to consent to treatment.

**Legal capacity requires that an individual be capable of:**

• understanding why treatment or a procedure is necessary.

• retaining information given before making a decision.

• being able to communicate a decision.

• understanding implications of refusing or allowing treatment and being able to retain this information.

Incapacity may be short lived e.g. acute confusional state or more longstanding e.g. dementia.

---

**MEDICAL TREATMENT**

**Medical treatment is defined as:**

• ‘any treatment that is designed to promote or safeguard physical or mental health’.

**The treatment must be clinically indicated and must:**

• promote or safeguard physical or mental health.

• take account of present and past wishes e.g. advance directive or living will.

• take account of the views of any relevant others including health care professionals involved in the patient’s care, relatives or carers as far as is reasonably possible.
minimise the restriction of the patient’s freedom - for example if it was considered unlikely that the patient’s clinical condition would be compromised by waiting until the patient regained the capacity to give consent, then the treatment should be delayed until such time.

respect the patient’s residual autonomy, thereby empowering them as much as possible.

Therefore any medical, surgical or nursing intervention, diagnostic study or physiotherapy is covered under the act.

How does this work in practice?

Every adult patient who is incapable of making decisions with regard to their medical treatment or care should have a completed Certificate of Incapacity filed in the front of their medical notes. The Certificate will replace consent forms unless the patient has a legally appointed proxy decision maker.

Any immediate treatment to save life or prevent serious deterioration in the patient’s medical condition is exempt from the procedures laid down in the act.

In all other cases the doctor primarily responsible for the patient can authorise the provision of medical treatment according to the general principles of the Act. However if a proxy decision maker has been nominated consent must be obtained from them prior to any procedure, other than emergency treatment.

Proxy decision makers include:

- **Welfare attorney.** The patient, in anticipation of their becoming incapacitated, nominates this power of attorney. The power of attorney must be registered with The Public Guardian who should issue a certificate in the prescribed form.

- **Welfare Guardian.** This proxy is appointed by a sheriff when an individual has become incapacitated or has never had the capacity to make decisions pertaining to their medical treatment.

- **Intervention Order.** Representation is provided by the courts on behalf of the incapacitated adult.
PROFESSIONAL RESPONSIBILITIES

• All staff and students must be **identifiable** by wearing an appropriate name badge at all times.
• In all interactions with patients, relatives and colleagues, courtesy and common sense should be exhibited.
• All staff and students should wear appropriate clothing at all times.

**Documentation:** quality notes are crucial for good patient management and as a lasting record of ward rounds, decisions, procedures and communication. Guidelines on following page.

• **Practical procedures:** from the simple and mundane to highly complex invasive procedures adherence to the guidelines below will optimise efficacy and minimise complications.
• Procedure for **Cardiac Arrest Management and Do Not Attempt Resuscitation Orders** for all sites are available in all clinical areas, from the Resuscitation Officers (see ‘phone list) and on the Intranet. Cardiac arrest audit forms are available widely and should be completed for every cardiac arrest call. These provide invaluable information on process and outcome and influence planning of resuscitation training and equipment acquisition.
• **Clinical Risk/Clinical Governance:** in order to minimise adverse events a Lothians wide system is in place to allow any staff member to report a near-miss or a critical incident occurring in patient care. This can be anonymous and is intended to alert senior staff to areas where improvements in the system will result in better outcomes for our patients. You should familiarise yourself with the Datix system on the hospital intranet home page. Dr A Arnstein chairs the Clinical Risk group and can be contacted via Department of Anaesthetics, St John’s Hospital.
• **Major Incident procedure:** in the event of a Major Incident being declared large numbers of casualties may be transported to the Royal Infirmary and St John’s Hospital. SJH and the RIE are DESIGNATED RECEIVING HOSPITALS for a major incident occurring in the Lothian Region. The RIE would act as the CONTROL HOSPITAL (responsible for co-ordinating all medical activity) and SJH would act in a SUPPORT capacity in all circumstances. SJH’s role will manage minor/intermediate injuries, medical cases, and isolated (i.e. without other major injuries) burns. Staff there should be familiar with the local policies available in the Medical Staff Handbook and...
RIE or SJH Major Incident Plans. At the time of the Major Incident the role of WGH will be to take all Medical and Surgical emergencies from both the North Zone (WGH) and South Zone (RIE). Details of WGH local policy can be found in the WGH Major Incident Plan.

**GOOD DOCUMENTATION**

- Write legibly, preferably in black ink (so it can be legible when photocopied).
- The medical case record is a legal document to which patients and relatives have right of access.
- All entries into case records should be legible, have a date and time assigned to them, and be signed in a way that allows the writer to be identified.
- Keep notes in chronological order: if writing in retrospect make this obvious.
- Keep clear progress notes both for inpatients and outpatients.
- Make clear your **reasoning** for clinical decisions.
- Any typed notes should be checked, corrected and signed.
- Cross out errors and write a corrected entry, dated and signed.
- Record details of discussions with patients or relatives.
- Record discussion of the patient’s condition or about risk/benefit of therapy.
- Any untoward or unexpected events and action taken in response to them should be adequately documented.
- At all times remember that the written record documents your thoughts for others to read.
- Write the patient’s name, date of birth, CHI number at the top of each page (each side).

**GOOD PRESCRIBING**

When a patient is admitted as an emergency consider which medicines may worsen the acute problem and omit until it is appropriate to restart.

**Good Practice in Writing Prescriptions on the Prescription and Administration Record (Drug Kardex)**

A clearly written prescription:
• saves everybody’s time.
• reduces the risk of medication errors.
• help ensures the right patient receives the right medicine in the right form and right dose by the right route at the right time.
• provides a clear record of the patient’s drug therapy.

GOLDEN RULES FOR PRESCRIPTION WRITING

1. Select the correct Prescription and Administration Record
   There are three versions available in RIE and WGH:
   • a standard 14 day record
   • a standard 14 day record with a warfarin chart
   • a 28 day record
   (SJH have 14 day, 28 day and 120 day records - all with separate warfarin chart)

2. Write clearly in block capitals, using a black ballpoint pen.

Note any allergies or adverse effects of medicines. Document what happens eg rash, anaphylaxis.

3. Complete all the required patient details on the front of the Record
   • Hospital/ward
   • consultant
   • weight
   • height
   • patient name*
   • patient number*
   • date of birth*
   *A printed label will suffice for these 3 details

Write the patient name and date of birth on each page of the record (ie each side). Include any previous adverse drug reactions, if known.

4. Use approved (generic) names of medicines
   Rare exceptions include drugs where a specific brand is necessary due to variation of response between brands e.g. theophylline, lithium, diltiazem, nifedipine, and verapamil, and combination products with no generic name e.g. Rifinah®.

5. Write the drug dose clearly
   • The dose of medicine must be specified. Prescribing a dose range e.g 10-20mg, is not acceptable.
   • The only acceptable abbreviations are
     \[ g - \text{gramme} \quad \text{mg} - \text{milligram} \quad \text{ml} - \text{millilitre} \]
     All other dose units must be written out in full e.g. micrograms
• Avoid decimal points write 100 micrograms (not 0.1 mg). If not avoidable, write zero in front of the decimal point e.g. 0.5 ml (not .5 ml).
• Prescribe liquids by writing the dose in milligrams, except where the strength is not expressed in weight e.g adrenaline 1 in 1000, where the dose should be written in millilitres (ml).
• For ‘as required’ medicines, include the symptoms to be relieved, the minimum time interval between doses, and the maximum daily dose. (Figure 1)

6. Route of administration
The only acceptable abbreviations are:

IV - intravenous         SL - sublingual         NG - nasogastric
IM - intramuscular       PR - per rectum        ID - intradermal
SC - subcutaneous        PV - per vagina        TOP - topical
ETT - endotracheal       INHAL - inhaled
NJ - nasojejunalostomy   NEB - nebulised
PEG - percutaneous endoscopic gastrostomy

Never abbreviate ORAL or INTRATHECAL.
Always specify RIGHT or LEFT for eye and ear preparations.

7. Enter the start date (Figure 2)
For courses of treatment, write only the dates therapy is required and discontinue as described below.
For alternate day treatment, put a horizontal line through the boxes in the administration section on the days the medicine is not given.

8. For once only prescriptions (Figure 3)
• Medicines intended to be given once only must be prescribed in the ‘once only’ section of the medicine chart (Figure 3).
• Medicines that are to be given once weekly must be prescribed in the regular section of the chart. A line must be drawn through the days that the medicine is not to be given and an instruction must be written in the notes section ‘Once a week on a ...day’.

9. Sign the prescription
Initials are not acceptable. Sign and print your name.

10. If the patient also has a supplementary chart in use (Figure 4)
Enter the details in the ‘other charts in use’ section on the Record.
11. *Never alter prescriptions*
   Cancel completely and rewrite.

12. **Discontinue medicines by** - *(Figure 5)*
   Drawing a diagonal line through the prescription box, but do not obliterate what has been written. Drawing a vertical line down the last administration time, then a double diagonal line, the signing and dating it.

13. **When the discharge prescription has been written**
   Enter date and initial on the box on the front page of the Record.

Prescription and Administration Charts must be re-written when required as follows:

- Any item no longer required must be cancelled, and a diagonal line drawn across each page of the old chart.
- The original start date for each medicine must be written in the new chart.
- The word ‘re-written’ and the date of re-writing, must be written at the top of the new chart.
- Ensure no medicines have been accidentally omitted from the new chart.

Good prescribing information can also be found in the **British National Formulary**
Specimen Drug Kardex Prescriptions

Figure 1

Figure 2

Figure 3

Figure 4

Figure 5
PRACTICAL PROCEDURES

Details of practical techniques for a number of emergency procedures are contained in this handbook. These notes are not meant to substitute for practical instruction in the correct method of carrying out these procedures. They will however be useful reminders and should ensure that details are not overlooked.

- Never undertake a procedure unsupervised when inexperienced.
- If difficulties are encountered, stop and call for help.
- Before starting consider the need for a coagulation screen and blood grouping.
- Explain the procedure to the patient and prepare the patient appropriately (see chest drain insertion as an example). Familiarise yourself with the patient’s anatomy and position the patient before scrubbing up.
- Always record details of the time of day and nature of the procedure in the notes together with the required monitoring asked of nursing staff.
- In the event of an unexpected change in the patient’s clinical condition remember possible complications especially hypoxia, vasovagal effects, haemorrhage, anaphylaxis and infection.

Peripheral IV access: antecubital cannulation is painful, irritant and potentially dangerous. Avoid unless no alternative.

TALKING WITH PATIENTS AND RELATIVES

- Talking with, and listening to, patients and their relatives is important.
- Be open and honest.
- Do not be afraid to say you don’t know the answer to a question.
- Seek advice from a senior member of staff when unsure.
- Record details of the interview in the case notes with written details of the information transmitted and the names of those present (doctor, nurse, relatives).
- Try to see relatives as soon as possible after admission to seek and document important information about the medical and social aspects of the acutely ill or confused patient.

Guidelines on the approach to breaking bad news can be found in Appendix 1.
Lothian Framework for Resuscitation Decisions

(See policy document for full details)

Can a cardiac or respiratory arrest be anticipated?

For example:
- Progressive cardiac or respiratory compromise
- Previous life-threatening event or condition in which cardiac arrest is likely
- Patient dying from irreversible condition e.g. advanced cancer

Are you as certain as you can be that CPR would realistically have a medically successful outcome?

Is there anything about the patient that makes you think they may not wish to be resuscitated in the event of an unexpected cardiac arrest?

For example:
- Severe incurable neurodegenerative condition

CPR should be carried out

- Do not burden the patient or relevant others with a CPR decision
- Continue to communicate and assess any concerns of the patient and relevant others. This may involve discussion on CPR and its outcome
- Review only when circumstances change
- In the event of cardio-pulmonary arrest, carry out CPR

Advanced Decision on CPR is possible

- Sensitive exploration of the patients wishes regarding resuscitation should be undertaken by the most experienced staff available
- **If the patient is competent** for this decision, discuss options of CPR and DNAR with patient. Involve relevant others* if appropriate (with patient’s permission).
- **If the patient is not competent** to understand the implications of this discussion, the medical team should make this decision based on available information regarding patient’s previous wishes (from relevant others*, other healthcare professionals or members of the multidisciplinary team). Relevant others* should never be asked to make the decision unless they are the legally appointed proxy/welfare guardian for the patient.
- Document the decision and any discussion around that process
- Continue to communicate and assess any concerns of the patient and relevant others*
- Ongoing review to assess any change in circumstances
- In the event of a cardio-pulmonary arrest, act in accordance with the documented decision

CPR inappropriate

- As CPR would not be successful it cannot be offered as a treatment option. A DNAR form should be completed and used to communicate this information to those involved in the patient’s care.
- Allow natural death with good palliative care and support for patient and relevant others
- Do not burden the patient or relevant others* with a CPR decision
- Document decision and review fortnightly or if the patient’s situation changes
- Continue to communicate and assess any concerns of the patient and relevant others (which may include discussion about why CPR is inappropriate)
- Ongoing review to assess any change in circumstances

*Relevant others refers to the patient’s relatives, carers, guardian etc
A decision about the appropriateness of CPR can only be made if the situation(s) where CPR might be required can be anticipated for the particular patient (e.g. recent MI, pneumonia, advanced cancer etc.). If such a situation can’t be thought through then there is no medical decision to make and there is no need to burden patients with resuscitation decisions.

**Advanced statements** - The exception to this would be where a patient has some chronic and/or irreversible condition such that they would not wish resuscitation in the event of any unexpected cardio-respiratory arrest from any unexpected cause. This would be a quality of life issue and therefore the patient’s decision rather than a medical decision. Such patients may have their wishes sensitively explored and a DNAR form +/- advanced statement completed if this is appropriate (see Lothian DNAR policy Appendix 2).

**MEDICAL DECISIONS ABOUT DNAR**

- The role of the medical team is to decide if CPR is realistically likely to have a medically successful outcome. Such decisions do not involve quality of life judgements.
- It may help in making a medical decision to decide whether the patient would be appropriate for Intensive Care treatment (likely outcome of a “successful” prolonged resuscitation).
- The consultant/GP responsible for the patient’s care has the authority to make the final decision, but it is wise to reach a consensus with the patient, staff and relevant others.
- It is not necessary to burden the patient with resuscitation decisions if the clinical team is as certain as it can be that CPR realistically will not have a medically successful outcome and the clinician is not obliged to offer CPR in this situation. This must never prevent continuing communication with the patient and relevant others about their illness, including information about CPR, if they wish this.

**PATIENTS DECISIONS ABOUT RESUSCITATION ISSUES**

- Where CPR is realistically likely to have a medically successful outcome consideration of a DNAR order for quality of life reasons must be discussed with the patient and their wishes must be given priority in this situation.
- Doctors cannot make a DNAR decision for a competent patient based on a quality of life judgement unless the patient specifically requests that they do this.
THE PATIENT WHO IS NOT COMPETENT TO MAKE A DECISION ABOUT RESUSCITATION

- Enquire about previous wishes from the relevant others to help the clinical team make the most appropriate decision. Continue to communicate progress to them.
- A Treatment Plan under Section 47 of the Adults with Incapacity (Scotland) Act must be completed prior to a DNAR decision being made.
- Continue to communicate progress to the relevant others.

THE ROLE OF THE RELATIVES/RELEVANT OTHERS

- A competent patient’s permission must be sought before any discussion takes place with the relevant others.
- Relatives should never be given the impression that their wishes override those of the patient. They can give information about the patient’s wishes but should not be burdened with the decision unless their status as proxy for the patient has been legally established.

PATIENTS WITH A DNAR ORDER AT HOME OR BEING DISCHARGED HOME

- It is the medical and nursing team’s responsibility to ensure that the family are aware of the existence of the DNAR form and know what to do in the event of the patient’s death.
- Where it is felt it may be harmful to the patient to have the DNAR form in the home the GP should keep the form in the front of the medical notes and ensure that all the healthcare professionals involved in the patient’s care are aware of this.
- The OOH service must be made aware of the existence of the DNAR order. Every effort must be made to ensure the emergency services are not called inappropriately where a patient’s death is expected.

PATIENTS WITH A DNAR ORDER BEING TRANSPORTED BY AMBULANCE

- The ambulance section of the DNAR form must be completed for any such patient form being transported in Lothian by the Scottish Ambulance Service.
- Ambulance control must be informed of the existence of the DNAR order at the time of booking the ambulance.
WHERE NO DNAR DECISION HAS BEEN MADE AND A PATIENT ARRESTS

- The presumption is that staff would attempt to resuscitate a patient in the event of a cardio-pulmonary arrest. However, it is unlikely to be considered reasonable for medical staff or senior nursing staff to attempt to resuscitate a patient who is in the terminal phase of an illness.
PATIENT DEATHS

- See patients without delay after being informed of their death.
- Inform the GP and the relevant consultant within 24 hours of the death of all patients.
- Guidelines for ‘When Death Occurs’ are found in Appendix 2.
- Sudden or unexpected deaths should be reported to the Procurator Fiscal. The Fiscal should be consulted in the event of any death associated with sudden unexplained ill health, occupational disease, medical accident or suspicion of foul play, drug overdose, suicide or neglect.

DEATH AND THE PROCURATOR FISCAL

If there is doubt about the cause of death discuss the patient with your seniors, and if necessary the Procurator Fiscal. In the following circumstances you MUST refer the case to the Fiscal. DO NOT issue any certificates without first talking to the Fiscal.

INDICATIONS FOR REFERRAL TO PROCURATOR FISCAL:

summarised from circular MEL (1996) 33 which gives full details

- Death due to an accident.
- Death resulting from an accident at work or industrial disease.
- Any death of a person while at work.
- Death due to poisoning.
- Death where suicide is a possibility.
- Death due to medical mishap.
- Deaths which occur unexpectedly having regard to the clinical condition of the deceased prior to his receiving medical care.
- Deaths which are clinically unexplained.
- Death seemingly attributable to therapeutic or diagnostic hazard.
- Deaths which are apparently associated with lack of medical care.
- Deaths which occur during the actual administration of general or local anaesthetic.
• Deaths which may be due to the anaesthetic.
• Death following abortion.
• Deaths where the circumstances indicate neglect on the part of another person.
• Deaths while under legal custody.
• Any death where the deceased’s residence is unknown.
• Death due to drowning.
• Death due to food poisoning or any notifiable infectious disease.
• Death due to fire or explosion.
• Death of a foster child.
• Any other death due to violent, suspicious or unexplained cause.
• Any death where a complaint is received from the next of kin about the medical treatment given to the deceased, and where the medical treatment may have contributed to the death.

THE DEATH CERTIFICATE

• Detailed advice on completion of the death certificate is contained in the certificate booklet.
• Ensure that both the counter foil and the death certificate proper have the patient’s name written legibly on them.
• Ensure the date is correct, and that it is neat, legible and signed.
• When speaking to the family explain what the technical terms mean, if this is appropriate.
• Document what has been written on the certificate in patient case record.
This checklist is used at ward level to ensure that all important steps are taken to document timing and responsible individuals.

<table>
<thead>
<tr>
<th>Initials</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient’s death confirmed by doctor?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Senior nurse in charge informed of patient’s death?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Next of kin notified of death? (see breaking bad news guidelines) Appendix 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procurator Fiscal Notification - <strong>inform as appropriate</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death certificate prepared? (see instructions in Deaths booklet)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death certificate given to family?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family returning later for death certificate? (if yes please record at the bottom of the page)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bereavement booklet given to family?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valuables/belongings returned to the family?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valuables held in cashier office?</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Post Mortem if required</strong> - Patient’s family must sign</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Copy of signed post mortem consent given to family?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cremation Form B <em>(if appropriate)</em> completed?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cremation Form B <em>(if appropriate)</em> sent to mortuary?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infection Certificate for undertaker sent to mortuary?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consultant informed - within 24 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GP contacted - within 24 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical records informed - within 24 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancel any follow-up appointments if already booked prior to death</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Arrangements to collect death certificate:** Date / / Time: 

**Other comments:**

Determine family’s wishes regarding jewellery? To remain on patient Yes/No

Comments:

Initials
Incomplete cremation forms can delay funeral arrangements, cause distress to bereaved relatives and cause major difficulty for mortuary staff. It is sensible to complete a Form B cremation form for every death unless it is known for certain a burial is planned.

- If there is no post mortem examination Form C is completed by a senior clinician (usually arranged by the mortuary). This is coordinated by Medical Unit secretary at SJH.
- If a post mortem examination is to be done, only Form B is required and should be completed (Q8a) after speaking to the pathologist.
- Cremation forms are not given to the family but are sent to the mortuary.

POST MORTEM EXAMINATION

- If a post mortem examination is considered desirable or is requested by the family, a clinician of F2/SHO grade or higher, or other suitably trained person, should explain to the family before requesting authorisation.
- Ensure the authorisation form is fully completed, signed and witnessed. One copy of this form is given to the family with the information booklet, one copy is filed in the medical notes and the third copy is for pathology.
- A post mortem request form is completed and countersigned by a clinician of SpR grade and upwards.
- Send the pathology copy of the authorisation form and the request form to the mortuary.
- The mortuary will invite clinical staff to view the post mortem findings.
Organ Donation

Transplants are one of the most miraculous achievements of modern medicine, but they depend entirely on the generosity of donors and their families who are willing to make this life-saving gift to others.

At present the number of people awaiting transplantation greatly exceeds the number of organs available. It is therefore essential to maximise the potential number of organs available from the existing potential donor pool.

Typically donation is from a variety of clinical settings:

- **Organ donation** - is from patients in the intensive care unit following confirmation of death by brain stem death tests or from non-heart beating donation.

- **Non-heart Beating Donation** - patients are certified dead following cardio-respiratory arrest within the intensive care unit.

- **Tissue** – can be donated following either the confirmation of death by brain stem tests or cardio-respiratory death. Tissue does not deteriorate immediately following cessation of the heartbeat due to its low metabolic requirements, allowing more time for retrieval and therefore may be offered in a variety of clinical settings.

All potential donors should be referred to the local donor transplant coordinator/tissue coordinator as early as possible for consideration for organ/tissue donation (Department of Health Working Party-Code of Practice for the Diagnosis of Brain Stem Death 1998). In cases of organ donation the donor transplant coordinator will be present throughout the organ donation process.

Donated organs/tissues and the families that donate them are a precious resource. The lives of hundreds of transplant patients are saved each year as a result of this gift. It is important that, where appropriate, the option of donating organs and/or tissue is offered to the next of kin/person closest in life.

To discuss organ or tissue donation call switchboard at RIE asking for the transplant coordinator on call.
GOOD PRACTICE FOR DISCHARGING PATIENTS

• Discharge is an extremely important part of patient care.
• Planning of discharges should begin **early in the patient’s admission.**

A patient’s suitability for discharge will depend on:

• **Medical Condition:** is the patient stable and can further investigation or treatment be completed as an outpatient?
• **Functional Ability:** is the patient independent or dependent on others?
• **Social Situation:** does the patient live alone or are there carers?

If there is any doubt about a patient’s ability to manage at home, a **MULTIDISCIPLINARY ASSESSMENT** should be performed. This will usually involve physiotherapy, occupational therapy and social work.

**Procedure**

• An expected date of discharge form is in use across Lothian.
• At least 6 hours before discharge of any patient you should use the patient’s **Prescription and Administration Record** and **Case Notes** to help complete the **Summary Form**.
• Fill in the **Patient Discharge Information Summary** with as much information as you can.
• Fill in an **OPD Appointment Card** with Clinic Name and approximate date of attendance.
• In some units the ward secretary or housekeeper will arrange appointments.
• Write in the Case Notes: Date of admission (DOA) Date of discharge (DOD) Diagnoses Any other relevant details Relevant Ix, Rx, changes to Rx Follow-up

• The **medication on Discharge** (see below) should be checked with a more senior member of medical staff. You **must** complete this accurately.
• Leave in the agreed place for checking by a Pharmacist where this service is available.
• Give the nurses the discharge letter to arrange supply of discharge medications where appropriate and place the notes with summary form in the appropriate place. Give appointment card to ward clerkess.
Always consider telephoning the G.P. This is ESSENTIAL for frail elderly patients and for other patients where further medical intervention will be required e.g. patients being commenced on warfarin.

If you are in any doubt, pick up the telephone.

DISCHARGE PRESCRIPTION WRITING

• Adhere to previous guidance on good prescription writing.
• The Patient Discharge Information Summary must be used to prescribe all current medicines. The information required must be accurately transcribed from the inpatient prescription chart and the patient’s medical notes.
• The doctor responsible for the patient’s care must ensure that the Patient Discharge Information Summary is completed in adequate time, taking account of the patient’s planned time and date of discharge.
• At least a seven day supply of medicines must be provided, unless a longer or shorter course of treatment is appropriate. The duration of therapy for antibiotic or steroid courses MUST be specified.
• Review all inpatient medicines and whether they need to be continued. Recommend review of changes to GP.
• Include (IN CAPITAL LETTERS)

Name - (Patient’s name, GP’s name, Consultant’s name)
Address - (Patient and GP’s address)
Ward / Department
Date
Signature - (name printed beside signature)

• If the patient already has his or her own supply of required medicines at home or stored in the ward an additional supply should not be issued from the hospital. However, the doctor who writes the prescription, or the pharmacist, nurse or other professional who checks the prescription, must satisfy himself or herself that the patient’s own supply is of an adequate quantity, quality and is correctly labelled with the current dosage instructions. The Patient Discharge Information Summary must be endorsed ‘patient’s own supply’.
• If the medicines are to be dispensed in the pharmacy, the Patient Discharge Information Summary must be delivered to the pharmacy at least 4 working hours before the patient is due to be discharged, to allow adequate time for dispensing and delivery to the ward.
Always review analgesic and sedative medication prior to discharge

Outpatient and discharge prescriptions for controlled drugs must comply with legal requirements. The prescription must:

- Include the name and address of the patient
- be written in indelible ink
- include the form (e.g. tablets) and if appropriate strength of the preparation e.g. morphine sulphate modified release tablets 10 mg
- include the total quantity of the preparation, or the number of dose units, in both words and figures e.g. 28 (twenty-eight) tablets, or 280 (two hundred and eighty) mg
- include the dose
- be signed and dated by the prescriber

For Warfarin: fully complete the yellow anticoagulation booklet and give to the patient.

Further information available on the Lothian Joint Formulary website: www.ljf.scot.nhs.uk

GUIDE TO GOOD DISCHARGE SUMMARIES

- The Discharge Summary is of vital importance.
- It is a summary of the inpatient stay.
- It is the main method of communication with the GP as well as being a summary for the case records.

Discharge summaries must include:

- The main diagnosis for that admission.
- Any important concurrent diagnoses.
- Important social factors e.g. living alone.
- Details of operations or major procedures e.g. central venous lines, endoscopy.
- Drugs on discharge including dietary advice.

Highlight any changes to treatment.

- Community Services arranged e.g. Home Help, District Nurse etc.
- Follow-up: arrangements at hospital or with GP.
- Information given/not given to patient and relatives.

How to do it

- Text should be relatively brief and detail changes in treatment and
review arrangements. Include:

a) drugs stopped (and reason why).
b) new treatments (which should be justified).

• Concentrate on selected details to indicate the disease and its severity.
• Unexpected findings, complications and **relevant** results should be included.
• **Check the format preferred in your unit.**
• The best summaries are **accurate, brief and timely.**
• The GP should have the full summary within **ten days of discharge,** thus it is essential you dictate promptly.

You should try to:

• Dictate summaries **daily**, or at most within 3 days of discharge.
• Give the date of dictation.
• Speak **clearly and slowly.**
• Include **all** the details listed above.
• Use **precise diagnoses** - ask your Consultant or Specialist Registrar if in doubt.
• Send **copies** of the summary where appropriate e.g. to other hospitals, to specialist units within the Division (e.g. Diabetes, Endocrine, Haematology, Neurosciences, Oncology, Orthopaedics, PAEP, Renal, Surgery), to other Consultants, and to Medical Officers of Nursing Homes.
• **Sign** your summaries (and other letters) promptly.

You should **NOT**:

• leave your summaries until a weekend on - this is unfair to the patient and secretary, and renders it much less effective as a means of communicating.
• use symptom diagnosis e.g. chest pain if a more precise one is available.
• give **all** clinical details and normal results - be selective.
• repeat all the past history - include relevant details under concurrent diagnoses.

**Dictation can seem a chore, but it is MUCH EASIER to do if the patient is fresh in your mind, and MUCH HARDER if you leave it and cannot remember who the patient was.**
RESUSCITATION & EMERGENCY MANAGEMENT

INITIAL ASSESSMENT AND MANAGEMENT OF THE ACUTELY ILL ADULT

GENERAL POINTS

• Acutely ill patients require rapid but careful assessment.
• Initiation of treatment often precedes definitive diagnosis but diagnosis should be actively pursued.
• Aim to prevent further deterioration and stabilise the patient.

Early involvement of experienced assistance is optimal i.e. GET HELP.

• The general principles of emergency management described here can be applied to the majority of acutely ill adults irrespective of underlying diagnosis or admitting speciality.
• Sepsis, shock and respiratory failure can occur in any clinical area. There may be life-threatening abnormalities of physiology present e.g. hypoxia or hypovolaemia, or the patient may have a specific condition which is at risk of rapid de-stabilisation e.g. acute coronary syndrome, GI bleed.

The approach to the acutely ill adult requires four elements to proceed almost in parallel:

THE FOUR KEY ELEMENTS OF EMERGENCY MANAGEMENT

1. Acute assessment & primary treatment with immediate targeted examination, investigations & support
2. Monitoring with frequent re-assessment
3. Illness severity assessment
4. Definitive diagnosis & treatment: full exam
Immediate investigations are those which will influence the **acute management** of the patient and include:

- Arterial blood gas
- Glucose
- Potassium
- Haemoglobin
- Clotting screen (where indicated).
- Twelve lead ECG.
- CXR (where indicated).
- Remember to take appropriate cultures including venous blood cultures before administering antibiotics (if practical).
- Consider sending blood for screen, group and save or cross-matching.

**1. ACUTE ASSESSMENT, PRIMARY TREATMENT & INVESTIGATIONS**

Acute assessment is designed to identify life-threatening physiological abnormalities and diagnoses so that immediate corrective treatment can be instituted (see algorithm). Patient observations and SEWS score are critical to the process. Within NHS Lothian an early warning scoring system (SEWS) has been developed to alert staff to severely ill patients. It is a decision support tool that compliments clinical judgement and provides a method for prioritising clinical care. An elevated SEWS score correlates with increased mortality and it is recommended that a patient with a SEWS of 4 or greater requires urgent review and appropriate interventions commenced. Think: Do they need specialist/critical care input **NOW**?
See explanatory notes below

**Approach:** Hello, how are you?

What is the main problem? Do you have any allergies? What medicines are you on? PMH?

Get a clear history to assist definitive diagnosis

<table>
<thead>
<tr>
<th>CLINICAL ASSESSMENT *GET HELP NOW</th>
<th>ACTION</th>
<th>INVESTIGATIONS IN ASSESSMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Airway and Conscious Level</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clear and coping? Stridor*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B Breathing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Look, listen and feel Rate and volume and symmetry WOB/ pattern RR &gt; 30* Paradoxical breathing*</td>
<td></td>
<td>High concentration 60-100% oxygen Monitor ECG, BP, SpO Ventilate if required</td>
</tr>
<tr>
<td>C Circulation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse Rhythm/character Skin colour and temp Capillary refill and warm/ cold interface Blood pressure (BP) HR &lt; 40 &gt;140* BP &lt; 90 SBP*</td>
<td></td>
<td>No pulse: cardiac massage IV access and fluids</td>
</tr>
<tr>
<td>D CNS and Conscious Level GCS/AVPU Fall in GCS 2 points* Pupils, focal neurological signs</td>
<td></td>
<td>ABC &amp; Consider the cause Management of coma Glucose</td>
</tr>
<tr>
<td>E Examine &amp; Assess Evidence &amp; Environment Temperature</td>
<td></td>
<td>Look at SEWS chart, results, drug &amp; fluid charts Standard bloods</td>
</tr>
</tbody>
</table>

1 If not breathing, get help and give two effective rescue breaths.
2 WOB: work of breathing.
3 Always record inspired oxygen concentration.
4 If collapsed carotid, if not start with radial.
5 Take blood for x-match and immediate tests (see text).
6 Should be <2 seconds.
NOTES ON INITIAL ASSESSMENT ALGORITHM

Airway and Breathing
- See BLS guidelines for cardiac arrest.
- By introducing yourself and saying hello you can rapidly assess the airway, breathing difficulties and the conscious level. If the patient is talking A is clear and B isn’t dire.
- AMPLE: ask about allergies, medicines, past history, last food/fluid, events at home or in ward e.g. drug administration.
- If any patient with known or suspected chronic respiratory disease arrives in A&E, CAA or ARAU on high concentration oxygen check ABG immediately and adjust oxygen accordingly.
- When assessing breathing think of it in the same way as you think of the pulse: rate, volume, rhythm, character (work of breathing), symmetry. Look for accessory muscle use, and the ominous sign of paradoxical chest/abdomen movement: “see-saw”.
- High concentration oxygen is best given using a mask with a reservoir bag and at 15l can provide nearly 90% oxygen.

The concentration of oxygen the patient breathes in is determined by the type of mask as well as the flow from the wall and the breathing pattern. By using a fixed performance system (Venturi) you can gauge the percentage much more accurately.
- The clinical state of the patient will determine how much oxygen to give, but the acutely ill should receive at least 60% oxygen initially.
- ABG should always be checked early to assess oxygenation, ventilation (PaCO₂) and metabolic state (HCO₃⁻ and base deficit). Always record the FiO₂ (oxygen concentration).
- Oxygen therapy should be adjusted in the light of ABGs: O₂ requirements may increase or decrease as time passes.

Circulation
- IV access is often difficult in sick patients.
- The gauge of cannula needed is dictated by the required use:
  - large bore cannulae are required for volume resuscitation. Ideally insert 2 large bore (at least 16G grey) cannulae, one in each arm in the severely hypovolaemic patient.
  - an 18 gauge green cannula is usually adequate for drug administration.
- The femoral vein offers an excellent route for large bore access and an 8.5F Swan-Ganz introducer is ideal.
• If there is major blood loss speak to the labs & BTS: you may need coagulation factors as well as blood. Consider activating the Major Haemorrhage protocol dial 222. Call Senior help.
• Use pressure infusors and blood warmers for rapid, high volume fluid resuscitation.

If the patient is very peripherally vasoconstricted and hypovolaemic don’t struggle to get a 14G (brown) cannula in. Put in two 18G cannulae (green) and start fluid resuscitation through both. CALL FOR HELP

• Machine derived cuff blood pressure is inaccurate at extremes of BP and in tachycardias (especially AF).
• Manual sphygmomanometer BP is more accurate in hypotension.
• In severe hypotension which is not readily corrected with fluid early consideration should be given to arterial line insertion and vasoactive drug therapy: GET HELP.

Disability
• Glasgow coma scale (GCS): document all three components accurately with best eye, best verbal and best motor responses.

Glasgow Coma Scale to record conscious level

<table>
<thead>
<tr>
<th>Eye Opening (E)</th>
<th>Verbal Response (V)</th>
<th>Motor Response (M)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4=Spontaneous</td>
<td>5=Normal conversation</td>
<td>6=Normal</td>
</tr>
<tr>
<td>3=To voice</td>
<td>4=Disoriented conversation</td>
<td>5=Localizes to pain</td>
</tr>
<tr>
<td>2=To pain</td>
<td>3=Words, but not coherent</td>
<td>4=Withdraws to pain</td>
</tr>
<tr>
<td>1=None</td>
<td>2=No words......only sounds</td>
<td>3=Decorticate posture</td>
</tr>
<tr>
<td></td>
<td>1=None</td>
<td>2=Decerebrate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1=None</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Total = E+V+M</td>
</tr>
</tbody>
</table>

• Check pupil size, symmetry and reaction to light.

Exposure, evidence and examination
• Targeted examination should be performed and information sought from any recent investigations, prescription or monitoring charts.
PREVENTING DETERIORATION & CARDIAC ARREST

- Around 80% of our in-hospital cardiac arrests are in non-shockable rhythms.
- In ventricular fibrillation/pulseless ventricular tachycardia the onset is abrupt, and an at-risk group with acute coronary syndromes can be identified and monitored. Early defibrillation results in optimal survival.
- In contrast, in-hospital cardiac arrest in asystole or pulseless electrical activity or PEA has a survival rate of around 10% and there is no specific treatment. There are usually documented deteriorations in physiology prior to the arrest. These are often treatable and reversible so the aim is to recognise decline early and to provide early corrective management in order to PREVENT CARDIAC ARREST. (See SEWS section).

Causes of preventable asystole and PEA can also cause VF.

- Hypoxaemia and hypovolaemia are common and often co-exist e.g. in sepsis, anaphylaxis, trauma or haemorrhage such as GI bleeding.
- Electrolyte abnormalities, notably hyperkalaemia, hypokalaemia or hypocalcaemia are easily detected and readily correctable.
- Drug therapy or poisoning/toxins may contribute to instability.

N.B. β-blockers and calcium channel blockers.
Physiological abnormalities | How to pick them up
--- | ---
Hypoxaemia, hypercarbia, acidosis | Do an early blood gas
Hypovolaemia, hypervolaemia | Assess circulation (see algorithm)
Hypokalaemia, hyperkalaemia | Early bloods
Hypothermia | Assess context, core temp
Tension pneumothorax | Clinical context and signs
Toxins | Clinical context, (chart in chapter 10)
Cardiac tamponade | Clinical context, early echo
Thromboembolic | Clinical context, PE/CTPA

- **Hypothermia, tension pneumothorax, cardiac tamponade** (particularly after thrombolysis, cardiac surgery or chest trauma) and **thrombo-embolic disease** must all be considered (in context).

### 2. MONITORING & REASSESSMENT

- Real-time continuous monitoring is invaluable in the acutely ill.
- Pulse oximetry, ECG and cuff BP monitoring should be instituted immediately in all patients.
- Monitoring is an integral part of the treatment/re-assessment/treatment/re-assessment loop.
- The place of urgent investigation is detailed previously.
- In order to make a definitive diagnosis other blood tests or imaging techniques may be required.

**Do not move unstable patients e.g. to x-ray until stabilised, and then only with adequate support, vascular access, monitoring and appropriate escort.**

**Assessment and re-assessment**

Assess response to treatment by constant clinical observation, repeated assessment of airway, breathing, circulation and disability (conscious level) as above with uninterrupted monitoring of ECG and oxygen saturation. Reassess regularly to see the effects of intervention, or to spot deterioration.

**If the patient is not improving consider:**

1. Is the diagnosis secure?
2. Are they so ill help is needed now?
3. Is there a new problem or diagnosis?
3. ILLNESS SEVERITY ASSESSMENT

- Working out how ill the patient is and what needs to happen to them next underpins the effective, safe management of all adult medical emergencies.

Specific scoring systems are included in specialist sections. The Scottish Early Warning Scoring System is being used in Lothian (QV).

Illness severity assessment informs four key decisions:

i) What level and speed of intervention is required? e.g. urgent ventilation, immediate surgery.

ii) Is senior help required immediately, and, if so, whom?

iii) Where should the patient be looked after? This is a decision about nursing care, monitoring and treatment level. The choices include:
   - General wards.
   - Intermediate care facility (Coronary Care Unit: CCU or High Dependency Unit: HDU).
   - Theatre
   - Intensive Care Unit (ICU).

   Placing the patient in a monitored HDU bed without increasing the level of appropriate medical input and definitive treatment will not improve outcome on it’s own. Senior advice should be sought early.

iv) What co-morbidity is present? (including drugs which blunt compensatory changes in physiology).

If you are called to a sick patient GO AND SEE THEM. Five seconds critically looking at the patient will tell you more than 10 minutes on the phone.

SEWS PARAMETERS AND SCORING SYSTEM

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory rate</td>
<td>0</td>
</tr>
<tr>
<td>≥ 36</td>
<td>1</td>
</tr>
<tr>
<td>31-35</td>
<td>2</td>
</tr>
<tr>
<td>21-30</td>
<td>3</td>
</tr>
<tr>
<td>9-20</td>
<td>4</td>
</tr>
<tr>
<td>≤ 8</td>
<td>5</td>
</tr>
<tr>
<td>SaO₂ (%)</td>
<td>0</td>
</tr>
<tr>
<td>&lt; 85</td>
<td>1</td>
</tr>
<tr>
<td>85-89</td>
<td>2</td>
</tr>
<tr>
<td>90-92</td>
<td>3</td>
</tr>
<tr>
<td>≥ 93</td>
<td>4</td>
</tr>
<tr>
<td>Temperature</td>
<td>0</td>
</tr>
<tr>
<td>≥ 39</td>
<td>1</td>
</tr>
<tr>
<td>38-38.9</td>
<td>2</td>
</tr>
<tr>
<td>36-37.9</td>
<td>3</td>
</tr>
<tr>
<td>35-35.9</td>
<td>4</td>
</tr>
<tr>
<td>34-34.9</td>
<td>5</td>
</tr>
<tr>
<td>≤ 33.9</td>
<td>6</td>
</tr>
<tr>
<td>BP (mm Hg)</td>
<td>0</td>
</tr>
<tr>
<td>≥ 200</td>
<td>1</td>
</tr>
<tr>
<td>100-199</td>
<td>2</td>
</tr>
<tr>
<td>80-99</td>
<td>3</td>
</tr>
<tr>
<td>70-79</td>
<td>4</td>
</tr>
<tr>
<td>≤ 69</td>
<td>5</td>
</tr>
<tr>
<td>HR</td>
<td>0</td>
</tr>
<tr>
<td>≥ 130</td>
<td>1</td>
</tr>
<tr>
<td>110-129</td>
<td>2</td>
</tr>
<tr>
<td>100-109</td>
<td>3</td>
</tr>
<tr>
<td>50-99</td>
<td>4</td>
</tr>
<tr>
<td>40-49</td>
<td>5</td>
</tr>
<tr>
<td>30-39</td>
<td>6</td>
</tr>
<tr>
<td>≤ 29</td>
<td>7</td>
</tr>
<tr>
<td>AVPU Response</td>
<td>Alert</td>
</tr>
<tr>
<td>Alert</td>
<td>0</td>
</tr>
<tr>
<td>Verbal</td>
<td>1</td>
</tr>
<tr>
<td>Pain</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>3</td>
</tr>
</tbody>
</table>

Case example

Patient presents in respiratory distress.
RR 32, SaO₂ 90%, T° 38.9, BP 160/70, HR 105, AVPU: Verbal
SEWS score = 6
Patient requires increased frequency of observations and urgent medical review.
<table>
<thead>
<tr>
<th>NAME</th>
<th>DATE</th>
<th>D.O.B.</th>
<th>RESPIRATION</th>
<th>TEMP</th>
<th>BLOOD PRESSURE</th>
<th>HEART RATE</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**SEWS SCORE** (with all obs): 9-10

*Severe*

*Moderate*

*Mild*

*None*

**PAIN**
An Early Warning Score (SEWS) must be calculated every time patient observations are recorded. If SEWS score 4 or more then call the appropriate doctor and nurse in charge using the guidelines below.

Increased frequency of observations (minimum hourly) should be commenced and a detailed report in the patient’s medical notes should be completed.

**Early Warning Score 4 or more**
- or concern with a patient's condition.

**Call Junior Doctor & Senior Nurse/Nurse Practitioner**
- If Dr cannot attend within 20 mins, they should arrange a Deputy.

**Practitioner/Dr unable to attend within 20 mins or SEWS increased by 2 or patient deteriorating.**

**Call appropriate SHO/Registrar & Senior Nurse/Nurse Practitioner**

**Dr unable to attend within 10 mins or SEWS increased by 2 or patient deteriorating.**

**Call appropriate Registrar/Consultant Consider ICU referral/review of treatment plan**

**Persistent Pain – 6 or above and unresponsive to guidelines**

**Call Medical Staff/Senior Nurse/Nurse Practitioner**

**For further advice contact:**

**ACUTE**
- Mon-Fri: Bleep Acute Pain Team
- Out of hours: On-call anaesthetist

**CANCER-RELATED**
- Mon-Fri: Bleep Palliative Care Team
- Out of hours: via switchboard
How to calculate SEWS Score

- Do not add pain score to SEWS Score.
- Record standard observations (RR, SpO₂, Temp, BP, HR, AVPU).
- Note whether observation falls in shaded “At Risk Zone”. Score as per SEWS key.
- Add points scored and record total “SEWS Score” in bottom row of chart.
- Action as per guidelines on front of chart.

<table>
<thead>
<tr>
<th>SEWS Score</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3</td>
<td>None</td>
</tr>
<tr>
<td>4-6</td>
<td>Mild</td>
</tr>
<tr>
<td>7-9</td>
<td>Moderate</td>
</tr>
<tr>
<td>≥10</td>
<td>Severe</td>
</tr>
</tbody>
</table>

**How to score pain:**

- **Cancer-related pain:** Always score worst pain in last 24 hours or since last assessment.
- **Acute pain:** Score current pain e.g. on movement/deep breathing.

<table>
<thead>
<tr>
<th>Pain Score</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None</td>
</tr>
<tr>
<td>1-3</td>
<td>Mild</td>
</tr>
<tr>
<td>4-5</td>
<td>Moderate</td>
</tr>
<tr>
<td>6-10</td>
<td>Severe</td>
</tr>
</tbody>
</table>

**Lothian Guidelines**

- **Cancer-related pain:** Initiate Edinburgh Pain Assessment Tool (EPAT™) for pain score of 4 or above.
- **Acute pain:** Use Palliative Care Guidelines.

**PERSISTENT MODERATE OR SEVERE PAIN, WHICH DISTRESSES PATIENT:**

REFER. SEE FLOW CHART OVER.

Illness Severity and Diagnosis (Risk of Deterioration)

- As the ABCD is secured a specific diagnosis is sought with the ‘Secondary Examination’ and specific treatment can then be instituted.

- Explanation, reassurance and analgesia are integral parts of acute care. Always keep the patient, family and/or relevant others informed about progress.

- Objective information on severity of illness may be obtained from blood tests e.g. acidosis and oxygenation, K⁺, renal dysfunction, liver failure and DIC.

- If acidosis is due to tissue hypoxia, base deficit can be followed as a guide to response to treatment (unless metabolic acidosis is due to e.g. renal failure).

**BASE DEFICIT is very important.**

| +3 to -3 | normal |
| -5 to -10 | moderately ill |
| -10 or worse | severely ill |

**Arterial blood lactate**

- If elevated has prognostic significance - the higher the worse.

  **N.B.** However, patient’s may have tissue hypoxia with a normal lactate.
IDENTIFICATION OF THE ACUTELY ILL PATIENT REQUIRING INTENSIVE CARE OR HIGH DEPENDENCY UNIT REFERRAL

CRITERIA FOR EARLY REFERRAL TO INTENSIVE CARE

<table>
<thead>
<tr>
<th>Condition</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Respiratory Failure</strong></td>
<td>Threatened or obstructed airway</td>
</tr>
<tr>
<td></td>
<td>Stridor</td>
</tr>
<tr>
<td></td>
<td>Respiratory arrest</td>
</tr>
<tr>
<td></td>
<td>Tachypnoea &gt;35/min, respiratory distress</td>
</tr>
<tr>
<td></td>
<td>SpO₂ &lt;90% on high concentration (&gt;60%) oxygen</td>
</tr>
<tr>
<td></td>
<td>Rising PaCO₂ (generally &gt;8 kPa or &gt;2 kPa above patient’s normal level, with respiratory acidosis)</td>
</tr>
<tr>
<td><strong>Shock</strong></td>
<td>Cardiac arrest (unless circulation restored rapidly by defibrillation and with return of consciousness)</td>
</tr>
<tr>
<td></td>
<td>Shock: tachycardia and/or hypotension not responsive to volume resuscitation</td>
</tr>
<tr>
<td></td>
<td>Evidence of tissue hypoperfusion/hypoxia</td>
</tr>
<tr>
<td></td>
<td>Clinically poor peripheral perfusion</td>
</tr>
<tr>
<td></td>
<td>Metabolic acidosis</td>
</tr>
<tr>
<td></td>
<td>Hyperlactataemia</td>
</tr>
<tr>
<td></td>
<td>Diminished conscious level</td>
</tr>
<tr>
<td></td>
<td>Poor urine output</td>
</tr>
<tr>
<td><strong>Renal Failure</strong></td>
<td>Oliguria</td>
</tr>
<tr>
<td></td>
<td>Hyperkalaemia</td>
</tr>
<tr>
<td></td>
<td>Uraemia</td>
</tr>
<tr>
<td><strong>↓ GCS</strong></td>
<td>Diminished conscious level</td>
</tr>
<tr>
<td></td>
<td>Threatened airway</td>
</tr>
<tr>
<td></td>
<td>Absent gag/cough</td>
</tr>
<tr>
<td></td>
<td>Failure to maintain normal PaO₂ and PaCO₂</td>
</tr>
<tr>
<td></td>
<td>Status epilepticus</td>
</tr>
<tr>
<td><strong>Gastro-intestinal/Liver</strong></td>
<td>Liver failure</td>
</tr>
<tr>
<td></td>
<td>GI bleeding</td>
</tr>
<tr>
<td><strong>Sepsis</strong></td>
<td>Severe sepsis and septic shock.</td>
</tr>
</tbody>
</table>

Even in the absence of a specific diagnosis of concern or greatly impaired physiology early ICU involvement may be appropriate: seek senior advice.
These guidelines are intended to facilitate referral of acutely ill patients for consideration of Intensive Care, High Dependency care and treatment of major organ system failure.

**Mechanism of referral:**

**ICU**
- **RIE** - Ward 118: 21187/21188
- **SJH** - 54063/54056 BLEEP 561
- **WGH** - Ward 20: Call ICU Consultant

**HDU (Level 2)**
- **RIE** - Ward 116 HDU: 21161  
  *(SHO 5198 for medical referrals or med.HDU consultant)*
- **SJH** - 54063/54056 BLEEP 561
- **WGH** - Ward 20: Call ICU Consultant

---

**EXAMPLES OF PATIENTS**

**Surgical problems**
- Perforated, ischaemic or infarcted bowel (both upper and lower).
- Acute pancreatitis.
- Sepsis from the gastro-intestinal, biliary or urinary tract.
- Respiratory or cardiorespiratory failure after any operation.
- Significant cardiovascular or respiratory disease in patients undergoing major surgery.

**Medical problems**
- Pneumonia, acute exacerbation of COPD, severe acute asthma.
- Sepsis.
- Cardiovascular failure e.g. severe LVF, post-MI.
- Post cardiac arrest (unless rapid return of circulation, ventilation and consciousness) usually go to CCU.
- GI bleed with haemodynamic instability.
- Severe diabetic ketoacidosis
- Poisoned patients at risk of airway or haemodynamic compromise.
Patients with Neurological disease with:
- Inability to breathe adequately.
- Inability to protect their airway.
- These include patients with reduced conscious level or brain-stem dysfunction.

4. DEFINITIVE DIAGNOSIS & TREATMENT

• Immediate life-saving treatment often prevents further decline or effects improvement while the diagnosis is made and specific therapy applied e.g. thrombolysis in MI, endoscopic treatment of an upper GI bleeding source. Outcome is better in patients where a definite diagnosis has been made and definitive therapy started.

FULL EXAMINATION & SPECIALIST INVESTIGATIONS

• Get a good history: useful information is always available.
• Relatives, GP, neighbours, ambulance staff may all be helpful.

If the patient is not improving consider:
1. Is the diagnosis secure?
2. Is the illness severity so great help is needed?
SHOCK

Definition & Classification

- Shock is an acute metabolic emergency where compromised oxygen transport leads to cellular oxygen utilisation which is insufficient to sustain normal aerobic metabolism.
- The common denominator in all types of shock is inadequate tissue oxygen availability.
- The aim of therapy in shock is to optimise tissue oxygen delivery in relation to oxygen requirements, whilst making a specific diagnosis and treating the underlying problem.
- Shock may result from inadequate oxygen delivery to the tissues (hypovolaemia, anaemia, low cardiac output), maldistribution of blood flow (sepsis, anaphylaxis) or the inability of the cells to utilise oxygen (sepsis).

TRADITIONAL CLASSIFICATION (aetiological):

- Hypovolaemic
- Septic
- Cardiogenic
- Anaphylactic
- Obstructive
- Neurogenic

FUNCTIONAL CLASSIFICATION (pathophysiological):

<table>
<thead>
<tr>
<th>Intact oxygen utilisation (low flow: low stroke volumes)</th>
<th>Abnormal (low oxygen utilisation low systemic vascular resistance)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Cardiogenic</td>
<td>- Septic</td>
</tr>
<tr>
<td>- Hypovolaemic</td>
<td>- Anaphylactic</td>
</tr>
<tr>
<td>- Obstructive</td>
<td>- Late low flow shock</td>
</tr>
<tr>
<td>e.g. pulmonary embolism, tension pneumothorax, tamponade</td>
<td></td>
</tr>
</tbody>
</table>

Clinical Presentation

Clinical evidence of organ dysfunction:

- Tachypnoea
- Tachycardia
- Hypotension
- Poor peripheral perfusion
- Abnormal mental state
- Oliguria
Hypotension is a sign of advanced shock in hypovolaemic/cardiogenic/low flow shock, implying decompensation of cardiovascular defence mechanisms.

Management
- Assess ABCDE and treat accordingly as detailed above.
- Get help.
- Correct hypoxaemia with high concentration oxygen by mask.
- Secure adequate IV access: this may be difficult.
- Correct hypovolaemia with colloid, crystalloid and blood as appropriate maintaining haemoglobin around 100g/l.
- Take blood and other samples for culture and give appropriate antibiotics. Early surgical intervention may be crucial e.g. laparotomy for perforated bowel, control of haemorrhage, abscess drainage.

SHOCK MANAGEMENT SUMMARY

In sepsis large volumes of fluid may be required and clinically important anaemia may result from haemodilution. Even if the patient is not bleeding blood transfusion may be necessary.

Monitoring
- Pulse oximetry, continuous ECG, non-invasive blood pressure (NIBP) should be used routinely.

Cuff BP by machine may be extremely inaccurate in sick patients.

Treatment
- The management of cardiogenic, hypovolaemic or septic shock unresponsive to the above measures can be very difficult.
- Tracheal intubation and ventilation, vasoactive drug therapy, invasive haemodynamic monitoring or mechanical circulatory support may be required.
- In acute coronary syndromes thrombolysis, PCI or other interventions may be needed.
- Persisting hypotension with impaired organ perfusion despite oxygenation and correction of volume status may necessitate vasoactive drug support.
- The drug of first choice is adrenaline (short term) as it has inotropic and vasoconstrictor effects, the latter predominating at higher doses.
• An arterial line should be used to monitor BP.
• Adrenaline should be infused via a central venous catheter.

Adrenaline 6mg is diluted in 100ml dextrose 5% and run initially at 3-10ml/hr.

)get expert help early: contact numbers for icu, cardiology, anaesthetics, respiratory, GI & other specialists in appropriate chapters of the book and in telephone lists.

blood and blood components

Comprehensive notes on the use of blood and its products can be found in the comprehensive Stationary Office Handbook of Transfusion Medicine 3rd Edition March 2001 and in the Divisional Blood Components Clinical Procedures Manual.

The major haemorrhage protocol is at the back of this book.

Blood
• Blood is usually supplied as red cell concentrates, unless otherwise requested (volume 280-350mls).
• In general fully cross-matched blood should be used.
• In an emergency this can be provided within 40 minutes of receipt of the sample.
• In an extreme emergency group specific blood can be used.
• ORhD negative uncrossmatched blood is available in A&E, Labour Ward and blood bank on RIE/Simpsons NRIE site and in WGH in Blood Bank and in blood bank (Haematology lab) at SJH.
• Please note that if a patient is found to have red cell antibodies there will be some delay in finding compatible blood.
• In the context of an acute bleed, blood may be transfused as quickly as required to attain haemodynamic stability.
• When transfusing anaemic patients with no acute bleed then it is given more slowly, in general 2 to 4 hourly. In these patients with poor cardiac reserve give blood 4 hourly and “cover” with oral furosemide (frusemide) e.g. 40 mg with alternate bags.
• Large transfusions may impair clotting and cause thrombocytopenia. After 5 units check FBC, PTR and APTT, and correct with fresh frozen plasma (FFP) and platelet transfusions if clinically appropriate i.e. PTR >1.5 x normal, APTT >2 x normal, platelets <50.
• If an additional transfusion is required more than three days later, then
a new sample must be sent for cross match (this is not necessary if more blood is requested within 72 hours of initial cross-match).

- If a reaction e.g. rigors, hypotension, loin pain occurs, STOP the transfusion, take down blood bag and giving set and send the blood bag, and a serum sample with EDTA and serum tubes to Blood Bank and citrate and EDTA tubes and urine specimen to the Haematology lab. Check coagulation screen, blood cultures, electrolytes.
- Contact the BTS or Haematologist for advice.
- Some patients e.g. bone marrow transplant, multiply transfused, renal patients require ‘special’ e.g. CMV -ve blood, irradiated, genotyped or filtered blood. These requests must be arranged in advance.
- Planned transfusion (top up or for surgery). Sample should be sent at least 24 hours ahead.
- If in doubt ask.

**Platelets**

- Check indication with a Haematologist.
- Given to correct a low platelet count (except when due to peripheral consumption e.g. ITP).
- In general transfuse if active bleeding and platelet count <50x10$^9$/l. If no bleeding and platelets <20x10$^9$/l consider transfusion. Transfuse if <10x10$^9$/l.
- 4 units of platelets are usually given over 30 to 60 minutes (a 250ml pooled or apheresis bag).
- Check platelet count 1 hour post transfusion for increment if pre-surgery or procedure.
- In general blood group specific platelets are given. If these are not available group compatible will be given. Rhesus negative platelet concentrates should be used for Rhesus negative patients.

**Fresh Frozen Plasma**

- Discuss with Haemotologist.
- Used to correct some coagulation defects e.g. over anticoagulation with warfarin, DIC.
- Usual dose is 10-15ml/kg i.e. 1 litre for 70kg adult.
- Must be compatible blood group i.e. AB universal, O only to O recipient.
- The units have a short half-life. Once defrosted use immediately if
possible and certainly within 4 hours.

- Infuse over <15mins.
- Use immediately pre procedure.
- Reactions may occur: contact the haematologist for advice.

**Reactions to blood products**

- Transfusion reactions with fever/rigors can be managed with paracetamol.
- Allergic reactions such as urticaria or bronchospasm may require hydrocortisone, chlorphenamine (chlorpheniramine), bronchodilators as detailed in anaphylaxis chapter.

**Sites with blood fridges**

<table>
<thead>
<tr>
<th>RIE</th>
<th>WGH</th>
<th>RHSC</th>
<th>SJH</th>
</tr>
</thead>
<tbody>
<tr>
<td>A&amp;E</td>
<td>Ward 1</td>
<td>Theatre</td>
<td>Haematology Laboratory</td>
</tr>
<tr>
<td>Orthopaedic recovery</td>
<td>Ward 8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI/Liver/Renal recovery</td>
<td>DCN theatre</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiothoracic recovery</td>
<td>Main theatre</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjacent to Obstetrics recovery</td>
<td>General HDU</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
SAFE PATIENT IDENTIFICATION

Mandatory Data Set Requirements for Blood Transfusion Requests

Dangerous or fatal transfusion errors are usually caused by failing to keep to standard procedures. Inadequate patient identification or sample labelling may lead to ABO-incompatible transfusions. As a result, the transfusion laboratory has to reject requests that arrive where the sample or request form do not comply with the mandatory data set.

Please remember to always seek positive identification of the patient before drawing the sample (‘Please tell me your name and date of birth’) and never write on the sample tube before drawing the sample.

Mandatory Data Set

Mandatory information required on the SAMPLE TUBE (handwritten at the bedside – patient identification sticky label must not be used):
1. Surname
2. First name
3. Patient identification number (hospital number or CHI number)
4. Date of birth
5. Sample date
6. Signature of person taking sample
7. If the patient identification number is unavailable, please include postcode

Mandatory information required on the REQUEST FORM (patient identification sticky label may be used):
1. Patient identification number (hospital number or CHI number)
2. Surname
3. First name
4. Date of birth
5. Gender
6. Location
7. Signature of requesting doctor (or appropriately trained nurse practitioner)
8. Name of person taking sample (if different from above)
9. If the patient identification number is unavailable, please include postcode
## Table 1: Guidelines for Recognition and Management of Acute Transfusion Reactions

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>SIGNS</th>
<th>SYMPTOMS</th>
<th>POSSIBLE CAUSE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Category 1: Localised</strong>&lt;br&gt;Mild cutaneous reactions:</td>
<td>Pruritis</td>
<td>Hypersensitivity&lt;br&gt;Febrile non-haemolytic transfusion reactions:</td>
<td>Antibodies to white blood cells, platelets&lt;br&gt;Antibodies to proteins, including IgA</td>
</tr>
<tr>
<td></td>
<td>• Urticaria</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Rash</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Mild Fever</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Category 2: Flushing</strong>&lt;br&gt;Urticaria&lt;br&gt;Rash&lt;br&gt;Mild Fever</td>
<td>Anxiety&lt;br&gt;Pruritis&lt;br&gt;Palpitations&lt;br&gt;Mild dyspnoea&lt;br&gt;Headache</td>
<td>Hypersensitivity&lt;br&gt;Febrile non-haemolytic transfusion reactions:</td>
<td>Antibodies to white blood cells, platelets&lt;br&gt;Antibodies to proteins, including IgA&lt;br&gt;Possible contamination with pyrogens and/or bacteria</td>
</tr>
<tr>
<td></td>
<td>• Febrile non-haemolytic transfusion reactions:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Antibodies to white blood cells, platelets</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Antibodies to proteins, including IgA</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Category 3: Rigors</strong>&lt;br&gt;Anxiety&lt;br&gt;Fever&lt;br&gt;Restlessness&lt;br&gt;Hypotension (fall of &gt;20% in systolic BP)&lt;br&gt;Tachypnoea +++&lt;br&gt;Tachycardia (rise of &gt;20% in heart rate)&lt;br&gt;Haemoglobinuria&lt;br&gt;Unexplained bleeding (DIC)</td>
<td>Anxiety&lt;br&gt;Chest pain&lt;br&gt;Pain near infusion site&lt;br&gt;Respiratory distress/shortness of breath&lt;br&gt;Loin/back pain&lt;br&gt;Headache</td>
<td>Acute intravascular haemolysis&lt;br&gt;Bacterial contamination and septic shock&lt;br&gt;Fluid overload&lt;br&gt;Anaphylaxis&lt;br&gt;Transfusion related acute lung injury (TRALI)&lt;br&gt;Transfusion associated Graft versus Host&lt;br&gt;Dyspnoea disease (TA-GvHD)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Febrile non-haemolytic transfusion reactions:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Antibodies to white blood cells, platelets</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Antibodies to proteins, including IgA</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** If an acute transfusion reaction occurs as you are treating the patient, as you are starting to treat the patient check the blood pack labels and the patient’s identity. These events should happen at the same time. If there is any discrepancy, stop the transfusion immediately and consult the hospital transfusion laboratory.

In an unconscious or anaesthetised patient, hypotension and uncontrolled bleeding may be the only signs of an incompatible transfusion.

In a conscious patient undergoing a severe haemolytic transfusion...
reaction, signs and symptoms may appear very quickly - within minutes of infusing only 5-10mls of blood. Close observation at the start of the transfusion of each unit is essential.

Table 2: Immediate Management of Acute Transfusion Reactions

<table>
<thead>
<tr>
<th>IMMEDIATE REACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CATEGORY 1: MILD</strong></td>
</tr>
<tr>
<td>1. Slow the transfusion.</td>
</tr>
<tr>
<td>2. If required, administer antipyretic/antihistamine.</td>
</tr>
<tr>
<td>3. If no clinical improvement within 30 minutes or if signs and symptoms worsen, <strong>treat as Category 2</strong>.</td>
</tr>
<tr>
<td><strong>CATEGORY 2: MODERATELY SEVERE</strong></td>
</tr>
<tr>
<td>1. Stop the transfusion. Replace the giving set and keep the IV line open with saline 0.9%.</td>
</tr>
<tr>
<td>2. Notify the doctor and the Hospital Transfusion Laboratory immediately.</td>
</tr>
<tr>
<td>3. Send the blood unit with the giving set, freshly collected blood samples (including blood cultures) with appropriate request form to the Hospital Transfusion Laboratory for investigations.</td>
</tr>
<tr>
<td>4. Administer antipyretic/antihistamine (avoid aspirin in thrombocytopenic patients).</td>
</tr>
<tr>
<td>5. Treat as per anaphylaxis protocol: stridor, wheeze and hypotension will require treatment with oxygen and im adrenaline. Call experienced help early: ICU/Anaesthetics.</td>
</tr>
<tr>
<td>6. Collect urine for next 24 hours for evidence of haemolysis and send to laboratory.</td>
</tr>
<tr>
<td>7. If clinical improvement, restart transfusion slowly with new blood unit and observe carefully.</td>
</tr>
<tr>
<td>8. If no clinical improvement within 5-10 minutes or if signs and symptoms worsen, <strong>treat as Category 3 and ensure help is coming</strong>.</td>
</tr>
</tbody>
</table>
### IMMEDIATE REACTION

**CATEGORY 3: LIFE-THREATENING**

1. Maintain airway and give high concentration (60-100%) oxygen by mask.
2. Stop the transfusion. Replace the giving set and keep the IV line open with 0.9% saline.
3. Manage as anaphylaxis protocol **and ensure help is coming:** stridor, wheeze and hypotension require treatment with oxygen and im adrenaline. Critical care admission will be necessary.
4. Notify the Consultant Haematologist and the Hospital Transfusion Laboratory immediately.
5. Send the blood unit with the giving set, freshly collected blood samples with appropriate request form to the Hospital Transfusion Laboratory for investigations.
6. Check a fresh urine sample visually for signs of haemoglobinuria.
7. Commence a 24 hour urine collection and fluid balance chart and record all intake and output. Maintain fluid balance.
8. Assess for bleeding from puncture sites or wounds, if DIC suspected seek expert advice.
9. Reassess:
   - Treat bronchospasm and shock as per protocol.
   - Acute renal failure or hyperkalaemia may require urgent renal replacement therapy.
10. If bacteraemia is suspected (rigors, fever, collapse, no evidence of a haemolytic reaction), take blood cultures and give broad spectrum antibiotics with Pseudomonas cover: Tazocin 4.5G tds IV plus gentamicin 7mg/kg od IV (ideal bodyweight). Discuss with haematologist on call.

---

Table 2 continued
<table>
<thead>
<tr>
<th>RELEVANT EFFECTS</th>
<th>DRUGS &amp; DOSES</th>
<th>NOTES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen</td>
<td>60-100%</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; line</td>
</tr>
<tr>
<td>Bronchodilator</td>
<td>Adrenaline 500 micrograms im repeated after 5 mins if no better, or worse</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; line</td>
</tr>
<tr>
<td>Vasopressor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Expand blood volume</td>
<td>0.9% - Saline, Gelofusine</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; line</td>
</tr>
<tr>
<td>Reduce fever and inflammatory response</td>
<td>Paracetamol Oral or rectal 10 mg/kg</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; line Avoid aspirin containing products if patient has low platelet count</td>
</tr>
<tr>
<td>Inhibits histamine mediated responses</td>
<td>Chlorphenamine (Chlorpheniramine) IV 0.1 mg/kg</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; line</td>
</tr>
<tr>
<td>Inhibits immune mediated bronchospasm</td>
<td>Salbutamol Aminophylline By 5mg nebuliser Use under expert guidance</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; line</td>
</tr>
<tr>
<td>Vasopressor Bronchodilator</td>
<td>Adrenaline 6mg in 100ml 5% dextrose (6%) 5-10ml/hr</td>
<td>Use only under expert guidance</td>
</tr>
</tbody>
</table>
Table 4: Investigating Acute Transfusion Reactions

<table>
<thead>
<tr>
<th>INVESTIGATING ACUTE TRANFUSION REACTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Immediately report all acute transfusion reactions with the exceptions of mild hypersensitivity and non-haemolytic febrile transfusion reactions, to the Consultant Haematologist and the Hospital Transfusion Laboratory.</td>
</tr>
<tr>
<td>2. Record the following information on the patient’s notes:</td>
</tr>
<tr>
<td>- Type of transfusion reaction</td>
</tr>
<tr>
<td>- Length of time after the start of the transfusion and when the reaction occurred</td>
</tr>
<tr>
<td>- Volume, type and pack numbers of the blood components transfused</td>
</tr>
<tr>
<td>3. Take the following samples and send them to the Hospital Transfusion Laboratory:</td>
</tr>
<tr>
<td>- Immediate post transfusion blood samples from a vein in the opposite arm:</td>
</tr>
<tr>
<td>- Group &amp; Antibody Screen</td>
</tr>
<tr>
<td>- Direct Antiglobulin Test</td>
</tr>
<tr>
<td>- Return blood unit and giving set containing residues of the transfused donor blood</td>
</tr>
<tr>
<td>4. Take the following samples and send them to the Haematology/Clinical Chemistry Laboratory for:</td>
</tr>
<tr>
<td>- Full blood count</td>
</tr>
<tr>
<td>- Coagulation screen</td>
</tr>
<tr>
<td>- Urea</td>
</tr>
<tr>
<td>- Creatinine</td>
</tr>
<tr>
<td>- Electrolytes</td>
</tr>
<tr>
<td>- Blood culture in an appropriate blood culture bottle</td>
</tr>
<tr>
<td>5. Complete a transfusion reaction report form.</td>
</tr>
<tr>
<td>6. Record the results of the investigations in the patient’s records for future follow-up, if required.</td>
</tr>
</tbody>
</table>
SEPSIS AND SEPTIC SHOCK

Sepsis is a systematic response to infection and a useful clinical definition allows early identification and treatment of patients before organ dysfunction or failure occurs.

For sepsis to be diagnosed two or more of the following should be present:

- Respiratory rate >20 breaths/min or PaCO$_2$ <4.3 kPa.
- Heart rate >90 beats/min.
- Temperature >38°C or <36°C.
- WBC>12,000 cells/mm$^3$, <4000 cells/mm$^3$, or >10 percent immature forms.

Plus suspected or confirmed infection.

A low diastolic and wide pulse pressure eg 110/40mmHg may indicate sepsis

Severe sepsis is present when organ dysfunction, hypoperfusion (e.g. lactic acidosis, oliguria, or an acute alteration in mental status) or hypotension (systolic BP <90mmHg) has supervened.

Septic shock is broadly defined as the development of hypotension and organ failure as a result of severe infection. Septic shock is a clinical diagnosis, confirmed by positive blood cultures in only a proportion of cases.

See Identifying Sepsis Early materials:
www.scottishintensivecare.org.uk education section

CLINICAL FEATURES

General clinical features

Fever and rigors.

Hypothermia is common and indicates poor prognosis.

Change in mental state: confusion or coma can occur.

Where is the source? Specific clinical features:

- Auscultation may reveal evidence of pneumonia or endocarditis.
- Abdomen - tenderness, peritonitis.
- Skin - rash, petechiae in meningoccalaemia.
- Skin: cellulitis, evidence of IVDA.
- CNS: Photophobia and neck stiffness in meningitis.
- Urinary tract symptoms? Loin pain?
- Lines - Intravascular
- Trauma
Assessment

• Airway: usually secure unless reduced conscious level.
• Breathing: tachypnoea is common - early.
• Circulation: tachycardia and hypotension may occur. In early shock there is peripheral vasodilatation and increased cardiac output. The patient is hypotensive, with warm peripheries. In advanced septic shock cardiac output falls due to hypovolaemia (+/- myocardial depression) and the skin becomes cold, cyanotic and mottled with increased capillary refill time. If unresponsive to volume resuscitation the patient is at high risk of death.
• Disability - GCS, pupils, focal neurological signs.

Organisms

• Community-acquired sepsis: Coliforms, Streptococcus pneumoniae, Neisseria meningitidis, Staphylococcus aureus. Group A Streptococcus.
• In hospital patients or recently discharged patients MRSA is increasingly encountered as are multi-resistant gram negatives.
• In patients with abdominal sepsis, mixed infection with coliforms, anaerobes.
• In patients with neutropenia, Pseudomonas aeruginosa must be covered.
• Splenectomised patients are at particular risk from capsulated organisms (Streptococcus pneumoniae, Haemophilus influenzae, Neisseria meningitidis) and severe malaria.
• Seek advice from ID or Microbiology if unusual features - travel history, animal contact, IVDU.

Investigations

Take blood cultures x2 before giving antibiotics. Administration of antibiotics should not be delayed in severely unwell patients until after other investigations including lumbar puncture.

• Blood cultures. Send at least 2 sets. At least 10ml of blood should be cultured per set
• Chest X-ray
• Urine: dipstick for WCC and nitrites (urgent laboratory microscopy is not usually necessary)
• Pus, wound swabs
• Sputum
• CSF
• Blood (EDTA or clotted) PCR if meningitis suspected
• Stool if diarrhoea
• FBC, CRP

**Neutropenia secondary to sepsis is an ominous finding indicating advanced sepsis.**

**ANTIBIOTIC MINI-GUIDE FOR ADULTS**

*Take cultures prior to start of antibiotics. INTRAVENOUS TREATMENT SHOULD BE REVIEWED DAILY.*

See Full Guidelines for duration of therapy and IV to oral switching.

**THERAPY SHOULD BE REVIEWED WHEN CULTURE RESULTS AVAILABLE**

1. **BACTERIAL MENINGITIS**

   CEFTRIAXONE 2 g bd IV
   
   • If patient is > 60 yo or immunocompromised, add AMOXICILLIN 2 g 4-hrly IV to cover Listeria.
   
   Consider DEXAMETHASONE 10 mg qds IV if pneumococcal meningitis likely and patient has not already received antibiotics.

2a. **COMMUNITY-ACQUIRED PNEUMONIA**

   **Not seriously ill:** AMOXICILLIN 500 mg tds ORAL
   
   • If atypical organism suspected:
     
     add CLARITHROMYCIN 500 mg bd ORAL
     
     **Penicillin allergy (not seriously ill):**
     
     CLARITHROMYCIN 500 mg bd ORAL
     
     **Severe illness:** CEFTRIAXONE 1-2 g od IV plus CLARITHROMYCIN 500 mg bd ORAL or IV

2b. **ASPIRATION PNEUMONIA**

   CO-AMOXICLAV 625 mg tds ORAL or 1.2 g tds IV
   
   **Penicillin allergy:** seek advice

2c. **ACUTE EXACERBATION OF COPD**

   1st line: AMOXICILLIN 500 mg tds ORAL
   
   2nd line: CO-AMOXICLAV 625 mg tds ORAL
   
   **Penicillin allergy:** CLARITHROMYCIN 500mg bd ORAL

2d. **HOSPITAL-ACQUIRED PNEUMONIA**

   Seek specialist advice: choice of antibiotic will depend on patient's previous antibiotic therapy, local prevalence of MRSA etc.

3a. **ENDOCARDITIS: native valve**

   BENZYL-PENICILLIN 1.2 g 4-hourly IV plus GENTAMICIN 80 mg bd IV
   
   • If patient is an IVDU or has skin sepsis, Staph aureus may be present, therefore add to the above:
     
     FLUCLOXACILLIN 2 g 4 hourly IV

3b. **ENDOCARDITIS: prosthetic valve**

   or if patient is known to be MRSA-colonised, or patient with penicillin allergy:
   
   Seek advice from Cardiology and Microbiology.

3c. **Antibiotic prophylaxis to prevent endocarditis is indicated for certain procedures: see full guidelines.**

4. **STAPH AUREUS BACTERAEMIA**

   Any S aureus bacteraemia is a life-threatening condition and should be treated with FLUCLOXACILLIN 1 – 2 g qds IV (or see 8a for MRSA) for at least TWO WEEKS.

5. **SOFT TISSUE INFECTION**

   Mild: FLUCLOXACILLIN 500 mg qds ORAL
   
   Moderate -severe: FLUCLOXACILLIN 1-2 g qds IV
   
   • plus BENZYL-PENICILLIN 1.2 – 2.4 g qds IV
   
   **Penicillin allergy:** CLINDAMYCIN 450 mg qds ORAL (mild infection)
   
   CLINDAMYCIN 600 mg – 1.2 g qds IV (severe infection)
   
   **Life- or limb- threatening infection: this is a surgical emergency. Seek urgent advice from Surgeon and Microbiology.**

6a. **INTRA-ABDOMINAL SEPSIS**

   including hepato-biliary
   
   CEFTRIAXONE 1-2 g od IV plus METRONIDAZOLE
   
   500mg 8-hrly IV

6b. **INFECTIVE DIARRHOEA (GASTROENTERITIS)**

   Antibiotics are usually contraindicated. If patient severely ill/ septicaemic, CIPROFLOXACIN 500 mg bd ORAL (or 400 mg bd IV only if oral not possible)

6c. **C DIFFICILE DIARRHOEA**

   Stop other antibiotics - this may lead to resolution of symptoms. If still symptomatic, then:
   
   METRONIDAZOLE 400 mg tds ORAL (or 500 mg tds IV only if oral not possible)

7a. **LOWER URINARY TRACT INFECTION (CYSTITIS)**

   TRIMETHOPRIM 200mg bd ORAL
   
   or CO-AMOXICLAV 625 mg tds ORAL

7b. **UPPER URINARY TRACT INFECTION (PYELONPHRITIS)**

   **Not seriously ill:** CO-AMOXICLAV 625 mg tds ORAL
   
   **Penicillin allergy:** CEFTRIAXONE 1-2 g tds IV
   
   **Severe illness:** CIPROFLOXACIN 500 mg bd ORAL

7c. **CATHETERISED PATIENTS WITH ?UTI**

   If no systemic symptoms, NO ANTIBIOTIC
   
   **Systemic illness/septicaemia:** GENTAMICIN
   
   160 mg IV once, with catheter change

8. **HOW TO TREAT MRSA**

   8a. **BACTERAEMIA or SEVERE PNEUMONIA**
   
   or **SEVERE SOFT TISSUE INFECTION**:
   
   VANCOMYCIN (at WGH and RIE, dose as local policy) or, SJH only, TEICOPLANIN 10mg/kg IV to nearest 200 mg (i.e. generally 600 mg or 800 mg), 3 loading doses 12 hours apart then once daily.
   
   Consider adding RIFAMPICIN 300 mg bd ORAL/IV

   **SEVERE PNEUMONIA**
   
   • If patient vomiting: CO-AMOXICLAV 1.2 g tds IV
   
   • or
   
   SEVERE PNEUMONIA
   
   **CONSIDER TREATMENT WITH ANTIMICROBIALS**, e.g.
   
   GENTAMICIN
   
   500mg 8-hrly IV
   
   • or
   
   CIPROFLOXACIN 400 mg bd IV
   
   **GENTAMICIN**
   
   500mg 8-hrly IV
   
   **GENTAMICIN**
   
   **DOXYCYCLINE**
   
   **100 mg bd ORAL**
   
   **400 mg bd ORAL**

8b. **MODERATE MRSA INFECTIONS:**

   DOXYCYCLINE 100 - 200 mg od ORAL
   
   Consider adding RIFAMPICIN 300 mg bd ORAL for more severe infection.
   
   • MRSA lower urinary tract infection only: TRIMETHOPRIM 200mg bd ORAL or NITROFURANTOIN 500 mg qds ORAL

9. **NEUTROPENIC SEPSIS**

   TAZOCIN 4.5g qds IV plus GENTAMICIN 7mg/kg od IV.
   
   Calculate dose using ideal body weight and monitor using Hartford nomogram.
   
   **Penicillin allergy:** seek Microbiology advice

10. **SEPSIS OF UNKNOWN ORIGIN**

    If patient too unstable to delay giving antibiotic pending investigation:
    
    CEFTRIAXONE 2 g od IV and seek senior opinion
Differential diagnosis
Remember other causes of hypotension and shock:

**Unexplained Hypotension - think of:**
- Sepsis (including Toxic Shock Syndrome)
- Myocardial infarct with no chest pain (early ECG).
- Occult blood loss
- Poisoning
- Pulmonary embolism
- Anaphylaxis
- Addison’s disease
- Autonomic dysfunction
- Cardiac tamponade

Supportive management

**Call ICU early**

- High concentration oxygen by face mask: 60-100% aiming for $\text{SpO}_2 > 96\%$.
- Secure adequate IV access and commence volume replacement. Insert a large bore peripheral venous line and administer saline 0.9% or colloid. If the patient is hypotensive give 250ml boluses of Gelofusine.
- Volume replacement is a priority, and should be monitored scrupulously.
- Take blood cultures x2, and other Microbiology samples, then choose and start appropriate IV antibiotics.
- Draw venous blood for FBC, U&Es, glucose, clotting.
- Check arterial blood gases and blood lactate.
- Make a full assessment of the patient’s condition and the likely aetiology as above.
- Insert a urinary catheter.
- Observe carefully for fluid overload and be aware of the possibility of acute renal failure.
- Remove or drain any obvious source of infection such as an abscess or infected IV line.
• Remember intra-abdominal sources, severe cellulitis, necrotising fasciitis or gangrene and if suspected seek urgent surgical opinion.

Septic shock unresponsive to oxygen therapy and initial volume loading has a high mortality. Invasive monitoring and vasopressor therapy are likely to be necessary.

CALL ICU EARLY.
Anaphylaxis is an acute allergic process where a substance to which the individual has been previously exposed results in mast cell degranulation and massive mediator release. Anaphylactic shock is twice as common in women and atopy is present in about a third of cases.

**Aetiology**
- Foods: nuts, fish.
- Drugs: NSAIDs, antibiotics, anaesthetics.
- Stings
- Idiopathic

**Presentation**
There is a spectrum of severity from mild to catastrophic, and treatment must be tailored to the individual situation.

**Clinical features**
- Airway compromise and breathing difficulties: stridor, wheeze, tachypnoea.
- Circulatory collapse: hypotension, tachycardia.
- Itch, skin rash, angio-oedema - may be completely absent.
- In about 20% abdominal or muscle pain or GI upset are major symptoms.

### ACUTE ANAPHYLAXIS

**Bronchospasm and/or cardiovascular collapse.**
Adrenaline should be given to all patients with respiratory difficulties and/or hypotension.

1. **Immediate action**

   - Discontinue administration of suspect drug, blood transfusion or IV fluid.
   - **GET HELP - call 222.**
   - ABC: maintain airway and give 100% oxygen by high flow with oxygen mask and reservoir bag or bag/mask/valve apparatus. Intubation may be required early, particularly if stridor is present.
• Commence basic life support (CPR) if no pulse present.
• Secure adequate IV access if not already.
• Monitor oxygen saturation and BP.
• ECG must be continuously monitored, and a defibrillator immediately available.
• Give adrenaline 500 micrograms intramuscular (0.5ml of 1 in 1000 solution). Repeat in 5-10 mins if no better or getting worse.
• Give IV fluids. Hartmann’s solution, 0.9% saline or Gelofusine 10ml/kg (about 500ml to 1 litre) can be used initially. Colloid may be more efficient at restoring blood volume especially in severe cases.

2. Supplementary action to damp down inflammation/prevent recurrence
• Give hydrocortisone 200mg IV (slowly).
• Give Antihistamines: chlorphenamine (chlorpheniramine) 10-20mg IV slowly.
• Give salbutamol 5mg nebuliser if wheeze present.
• Measure arterial blood gases and coagulation.

VERY SEVERE ANAPHYLAXIS

Most cases will resolve with the above treatment. However in the most severe cases with life-threatening shock or airway compromise, particularly in association with general anaesthesia, adrenaline should be given intravenously as described here.

• This is a rapidly life-threatening condition requiring experienced clinical management. Intravenous adrenaline boluses should only be given by, or under the direct supervision of, an appropriately experienced clinician.
• Give ADRENALINE INTRAVENOUSLY (especially in the presence of stridor or wheeze) starting with 50 to 100 micrograms (0.5-1 ml of 1 in 10,000 i.e. Minijet), with further 50 to 100 microgram aliquots as required.
• Adrenaline dose in cardiac arrest is 1 mg (10ml of 1 in 10,000).

SUBSEQUENT ACTION
Record allergy prominently in notes and explain to patient and family.

CONTINUING PROBLEMS (requiring ICU referral for:)
Severe and resistant bronchospasm
• **Salbutamol** 5mg nebulised in 100% oxygen, repeated as necessary.

**Always maintain oxygen therapy during administration of bronchodilators.**

• **Salbutamol** 250 micrograms slowly IV (4 micrograms/kg over at least 10 mins) as a loading dose followed by 5-20 micrograms/min infusion *(directed by Senior Clinicians).*

**N.B.** Can cause tachyarrhythmias, hypotension, hypokalaemia.

**Alternatively** *(as directed by Senior Clinicians)*

• **Adrenaline** by infusion 6mg diluted in 100ml of dextrose 5% at 3-10 ml per hour.

• **Aminophylline** 250mg IV over 20 mins by volumetric pump or syringe driver. This is usually sufficient but up to 6-8mg/kg can be used.

**N.B.** Can cause tachyarrhythmias, myocardial ischaemia and hypokalaemia. Caution in the elderly, IHD or if on oral theophylline. Half loading dose if on theophylline or level unavailable.

**Refractory hypotension and/or pulmonary oedema and/or bronchospasm requires ICU referral.**

**FURTHER MANAGEMENT**

Even if stabilised and improving:

• admit to ICU or HDU or appropriate monitored area.

• monitor respiratory rate, ECG, BP, SpO₂.

• continue steroids and anti-histamines orally or IV.

**Follow up is crucial: over 60% of patients will have repeated attacks.**

• Patients should be advised to wear a medic-alert type bracelet or talisman. Information on this is available from:

  **Anaphylaxis Campaign**
  01252 542029  info@anaphylaxis.org.uk

  **British Allergy Foundation**
  02083 038792  www.allergyfoundation.com
  email: allergybaf@compuserve.com

• In food, insect or unknown allergies provide an Epipen or Anapen adrenaline injector and training in use.

• Referral to allergist is ideal but in Lothian this service is not available.
1. An inhaled beta₂-agonist such as salbutamol may be used as an adjunctive measure if bronchospasm is severe and does not respond rapidly to other treatment.

2. If profound shock judged immediately life threatening give CPR/ALS if necessary. Consider slow IV adrenaline (epinephrine) 1:10,000 solution. This is hazardous and is recommended only for an experienced practitioner who can also obtain IV access without delay. Note the different strength of adrenaline (epinephrine) that may be required for IV use.

3. If adults are treated with an Epipen, the 300 micrograms will usually be sufficient. A second dose may be required. Half doses of adrenaline (epinephrine) may be safer for patients on amitriptyline, imipramine or a beta blocker.

4. A crystalloid may be safer than a colloid.

Updated May 2005
URTICARIA AND ANGIO-OEDEMA

- These conditions are sub-acute or chronic unless they accompany anaphylaxis or the airway is involved by swelling.
- Sudden total airway obstruction can result and is rapidly fatal unless oxygenation is maintained.

Management

- Airway compromise: if stridor is present and airway obstruction imminent endotracheal intubation is mandatory (GET HELP).
- Give high concentration oxygen. Intubation may be difficult: fast bleep (222) anaesthetics and ICU.
- In severe cases of urticaria/angio-oedema adrenaline should be given as for anaphylaxis: 500 micrograms im (0.5ml 1 in 1000 solution) or using the IV schedule detailed above.
- If total upper airway obstruction occurs oxygenation must be maintained via emergency cricothyrotomy. Kit in A&E, ARAU, Theatres and ICUs.
- May be more resistant to drug treatment than anaphylaxis and need early intubation. Often a very difficult procedure.
- Nebulised adrenaline may be effective.
- Antihistamines and steroids are used as for anaphylaxis.

LIFE-THREATENING UPPER-AIRWAY OBSTRUCTION

Inability to get gas in by patient or by attendants.

- Causes include foreign body, swelling (anaphylaxis, angio-oedema see above), trauma, burns and peri-anaesthetic (laryngospasm).
- Administer 100% oxygen via BMV and call for Anaesthetic/ICU HELP.
- May need to contact ENT surgeons for definitive airway.
Appropriate acute pain management is dependent on assessment of the severity of pain followed by appropriate prescription and administration of multimodal analgesia. This should be prescribed and given at a dose, route and frequency appropriate to the individual patients condition and the effect of each intervention reassessed. Assess pain in the context of the whole patient, remember to consider why they have lost control of their acute pain e.g. is there a new pathology?

<table>
<thead>
<tr>
<th>Pain score/ level of distress</th>
<th>Intervention</th>
<th>Prescription</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 to 3 mild undistressing pain</td>
<td>None required</td>
<td>Mild analgesics should be available as required if pain is anticipated Paracetamol, NSAID &amp; a weak opioid such as codeine or a combination e.g. cocodamol should be prescribed as appropriate</td>
</tr>
<tr>
<td>3 to 6 moderate pain &amp;/or distress</td>
<td>Rescue analgesia required</td>
<td>Usually an opioid titrated to effect orally or parenterally</td>
</tr>
<tr>
<td></td>
<td>Review of prescribed analgesia required</td>
<td>Is the patient on: • regular paracetamol? • regular NSAID? • regular opioid?</td>
</tr>
<tr>
<td>7 to 10 severe and distressing pain</td>
<td>Urgent rescue analgesia required</td>
<td>Intravenous morphine or equivalent titrated to effect.</td>
</tr>
<tr>
<td></td>
<td>Review of prescribed analgesia required</td>
<td>Is the patient on: • regular paracetamol? • regular NSAID? • regular opioid of appropriate strength?</td>
</tr>
</tbody>
</table>

PAIN MANAGEMENT BASIC PRINCIPLES

- Pain management regimens must be tailored to individual patient requirements. Where appropriate the combined use of different analgesics (multimodal analgesia) should be used. This is more effective, limits the dose of any one therapy and helps to minimise
serious side effects. It is necessary to review the patient’s response to therapy and then tailor ongoing analgesia to their needs.

- In acute pain it is anticipated that the worst pain will be present initially and steadily improve with time. It is therefore essential to have an appropriate level of maximum therapy instituted at the outset of treatment and gradually stepped down.

- Regular assessment of pain scores and the side effects of therapy is necessary to ensure effective and safe treatment.

- Pain management should aim to control pain to a tolerable level. Remember it should be possible with appropriate interventions as above to control acute pain for most hospital patients to a level with which the patient is comfortable. However, it is inappropriate to aim for complete analgesia in all patients since this is likely to lead to problems with treatment side effects.

- Pain relief from any analgesic regime is balanced against side effects. In some situations a compromise is necessary, where less effective analgesia is acceptable to avoid complications of therapy which may be distressing or which may lead to morbidity and even mortality.

- Regular analgesia is more effective than “as required” dosing. “As required” prescribing should only be used for the mildest pain or to relieve breakthrough pain in addition to regular analgesia.

- When converting from a more complex analgesic regime eg epidural, adequate step down analgesia must be prescribed.

- Analgesic prescriptions should be reviewed regularly, giving consideration to changing requirements and possible drug interactions.

### ACUTE PAIN GROUPS: CONTACT NUMBERS

**Western General** - 08.00-17.00: contact the Clinical Nurse Specialists for Pain on bleep 5292 or ext. 31670. Out of hours and at weekends contact the duty anaesthetist on bleep 5112.

**Royal Infirmary** - 08.00-17.00: contact the Clinical Nurse Specialists for Pain on bleep 5247 or ext. 23205. Out of hours and at weekends contact the duty anaesthetist on bleep 2140.

**St John’s Hospital** - 08.00-17.00: contact the Clinical Nurse Specialists for Pain on bleep 934 or ext. 53065. Out of hours and at weekends contact the duty anaesthetist on bleep 561

**Further Information:** Lothian Acute Pain Guidelines Site (Intranet)
SUMMARY OF PRINCIPLES OF ACUTE CARE

- Assess and treat simultaneously.
- Give enough oxygen to correct hypoxaemia.
- Establish adequate IV access. Take blood for urgent tests, including ABG and cross-match.
- Commence continuous monitoring.
- Perform Illness severity assesment: SEWS scoring and look at the patient!
  - ? risk of deterioration/cardiac arrest.
  - ? where to admit.
  - ? co-morbidity.
- Get help as indicated.
- Write in notes and prescribe drugs (including oxygen and fluids).
- Communicate with patient, family and significant others.
- Re-assess repeatedly and act on findings.
- Treat pain, nausea and other symptoms appropriately.
- Make a diagnosis, institute definitive treatment and assess response.
- Communicate the above and the plan with the patient, the ward team and the patient’s relatives.

Many of these elements should happen at the same time.

THE FOUR KEY ELEMENTS OF EMERGENCY MANAGEMENT

1. Acute assessment & primary treatment with immediate targeted examination investigations & support
2. Monitoring with frequent re-assessment
3. Illness severity assessment
4. Definitive diagnosis & treatment: full exam
You have been called to see the patient...

- Ensure P, BP, temp and RR are recorded while you are getting there.

On your way to the ward work through a differential diagnosis:

- Angina or MI
- Pleurisy or Pericarditis.
- Oesophageal/dyspepsia
- Musculoskeletal pain

On arrival use the initial assessment process described in Chapter 2.

- Give oxygen and establish IV access if appropriate.
- Make your clinical assessment and take an ECG.
- Draw bloods if appropriate.
- Make your own assessment of the need for analgesia and prescribe it/administer it as necessary.
- Pulse oximetry and ECG monitoring may be indicated.
- Decide on illness severity, the need for senior opinion and further treatment/investigations.
- Write in notes and prescribe drugs including oxygen.

A ROUGH GUIDE

- STEM I: call CCU and initiate treatment; see Cardiology section.
- Acute Coronary Syndrome: initiate treatment and consider need for monitoring, d/w Cardiology bleep #6816 RIE, 5689 at WGH, #630 at SJH (the on call medical middle grade).
- Angina (rate related): treat rate (will depend on cause and rhythm).
- Oesophageal reflux: gaviscon or mucogel.
- Musculoskeletal pain: prescribe appropriate analgesia.
- Pleurisy: treat cause (PE, pneumonia) and give analgesia.
ACUTE SHORTNESS OF BREATH

Use the initial assessment process previously described.

- Give oxygen and establish IV access if appropriate.
- Pulse oximetry is essential and ECG monitoring may be indicated.

Remember if RR > 30 &/or paradoxical breathing - it is serious. GET HELP EARLY.

- Ensure temp, P, BP, RR, PEFR are all done.
- Organise a CXR.
- Do ECG, take bloods and ABG’s on O₂ recording FiO₂.
- Based on your clinical judgement commence treatment e.g. nebulised bronchodilators.

Have a different diagnosis in mind, such as:
- Asthma
- LVF
- PE
- Pneumonia
- Pneumothorax
- Sepsis
- Metabolic acidosis

- Get bloods etc sorted.
- Write in notes and prescribe drugs including oxygen.
- Reassess when all the information is to hand, and consider response to initial therapy: better, the same or worse?

TREATMENT
Get help if necessary

Asthma
LVF see appropriate sections
PE
Pneumothorax
Pneumonia
Sepsis
Metabolic acidosis
PROTOCOLS FOR SPECIFIC PROBLEMS
(based on RIE protocols)

FIRST SEIZURE IN ADULTS

Was this a seizure?

NO Consider causes of non-epileptic attacks e.g. Syncope, panic attacks, pseudoseizures

YES

Has hypoglycaemia been excluded?

NO Treat hypoglycaemia, address underlying cause and then reassess

YES

Is this the first adult generalised seizure?

NO Consider poor compliance with medication, intercurrent illness or infection, alcohol or drug ingestion, or part of normal seizure pattern

YES

Does this patient require an urgent CT?

Emergency CT head. Is CT normal?

YES Refer to “first seizure clinic”

NO

Indications for CT scan prior to disposition:
- New focal neurological deficits
- Persistent altered mental status
- Fever or persistent headache
- Recent head trauma
- History of cancer or HIV infection
- Patients with new focal onset seizure
- Patients whose follow up cannot be ensured
- Anticoagulation or bleeding diathesis

NO

Are the ECG/blood results all normal? Consider: Uraemia, hyponatraemia, hypoglycaemia, hypercalcaemia, prolonged QT interval

NO ADMIT

YES

Does patient meet discharge criteria? (See Discharge Table)

YES

Give written advice about driving and lifestyle
Inform patient of their duty to notify the DVLA.
Discharge/arrange follow up at first fit clinic
File notes, investigations and assessment page with referral at reception.
Provide patient with a copy of “first seizure clinic” appointment letter.

NO

Produced by Protocol Group October 2003
DATE: 

Dear

We think it is possible that you have had an epileptic seizure (fit) to account for your recent symptoms. We are therefore referring you to a specialist for a further opinion regarding the diagnosis, possible investigations and treatment if necessary.

In order to make your appointment, you need to telephone the Medical Outpatient Department 2 on 0131 242 1368 or 1369. The department is open Monday-Thursday 0900-1700, and Fridays 0900 to 1600. You should ask the receptionist to book you an appointment in Dr Davenport’s First Seizure clinic and we recommend that you have a pen and piece of paper handy to note the date and time of your appointment. The clinic is currently held on a Monday morning in MOPD 2 in the Royal Infirmary of Edinburgh.

- If someone witnessed your collapse or funny turn, then please bring them with you to the clinic, or arrange for them to be contactable by phone, as the doctor may wish to speak to them.
- Please bring any prescription medications that you are taking with you, in their packaging, or a list of the medications you are taking and their doses.
- If you hold a driving licence, we would advise you that should not drive until you have been seen in the clinic.

Doctor’s name (printed):.................................

Responsible doctor should document in ED record that patient has been given this letter and forward the ED sheet together with completed first seizure protocol to Dr Davenport, Consultant Neurologist, MOPD 2, RIE

Doctor’s signature:.................................
**Inclusion Criteria**
- Patients >16yrs <60yrs
- Clear history of first generalised seizure
- Seizures related to drug or alcohol ingestion or withdrawal

**Exclusion Criteria**
- Patients with non-epileptic attacks (e.g. syncope, pseudoseizures)
- Patients with known seizure or metabolic disorder
- Seizures related to recent trauma
- Eclampsia

| History Table (please tick if relevant) | ✓ |
| Witness history | |
| Type of seizure (generalised, partial) | |
| Previous history of seizures, febrile fits, birth trauma, meningitis, head injuries | |
| Family history of seizures | |
| Possible precipitating events (alcohol, drugs, sleep deprivation) | |

**Clinical Findings (enter findings)**

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Pulse</th>
<th>BP</th>
<th>Resp Rate</th>
<th>BM</th>
<th>Breath Alcohol</th>
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<td>L Arm</td>
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<td>Reaction</td>
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<td>Alb</td>
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<tr>
<td>Glu</td>
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<tr>
<td>CT</td>
</tr>
</tbody>
</table>

**Discharge Table (all boxes MUST be ticked before discharge)**

- Patient fully recovered with no persistent neurological symptoms/signs (include headache)
- Normal observations and investigations (include temperature)
- Patient has been given written advice about driving and lifestyle changes and their duty to notify the DVLA
- Patient has a responsible adult to stay with following discharge
- Patient will attend follow up
- First fit clinic letter with copies of all notes and investigations forwarded to Dr Davenport's secretary at MOPD 2, RIE
**MANAGEMENT OF SYNCOPE**

**Identified cause for syncope**
- Manage appropriately

**Does the patient have:**
- Cardiac Failure
- Ischaemic Heart Disease
- History VT/VF
- Valvular Heart Disease
- ECG abnormality
- Exertional syncope
- Significant assoc injury

**Does the patient have:**
- Associated chest pain
- Associated palpitations
- Family history of sudden death
- Frequent syncopal episodes
- No presyncopal warning

**Age more than 60**
- CSM\(^8\) not contraindicated

**CSM for 5 sec on sequential sides results in:**
- Ventricular pause >3 sec
- Fall in systolic BP >50 mmHg

**ECG abnormalities @**
- Bifascicular block
- Second or complete heart block
- Sinoatrial block
- QRS > 120ms
- QTc > 450ms
- Bradycardia <50/min
- Previous ?MI
- RBBB with ST elevation V1-3 (Brugada syndrome)

**Review Collapse Algorithm**

**Admit to monitored bed**
- Organise Echo/24 hour tape +/- ETT

**Consider admission D/W senior colleague**
- Refer MOPD

**Frequent episodes**
- Refer cardiology

**Discharge GP**

\(^8\)Carotid Sinus Massage contraindicated if:
Carotid Bruit present; recent CVA, TIA or MI (6 months)

Produced by Protocol Group October 2003
### Inclusion Criteria
- Patients >16yrs
- Confirmed history of unexplained loss of consciousness

### Exclusion Criteria
- Any of the following after assessment for collapse (Fitz, hypoglycaemia, postural hypotension, arrhythmia, PE, CVA, vasovagal event, GTN syncope, situational syncope, structural cardiac cause)

### Indications for admission
- Congestive Cardiac Failure
- Ischaemic Heart Disease
- History of ventricular arrhythmia
- Significant Valvular heart disease
- ECG abnormality (see algorithm)
- Exertional syncope
- Significant injury associated with syncope

### Indications for MOPD follow up
- Syncope associated with chest pain or palpitations
- Family history of sudden death
- Frequent episodes
- No presyncopal warning
- Syncope while supine

### Medical History

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Pulse</th>
<th>O₂ saturations</th>
<th>BM</th>
<th>Breath Alcohol</th>
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### Investigations Table
- ECG
- Urea
- Creat
- Na
- K
- CO₂
- Ca
- Alb
- Glu
- Other

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<td>MCV</td>
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<tr>
<td>Na</td>
<td>WCC</td>
</tr>
<tr>
<td>K</td>
<td>PLT</td>
</tr>
<tr>
<td>CO₂</td>
<td>Bil</td>
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<td>Ca</td>
<td>GGT</td>
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<tr>
<td>Alb</td>
<td>ALT</td>
</tr>
<tr>
<td>Glu</td>
<td>Alk Ph</td>
</tr>
</tbody>
</table>

### Discharge Table
- Is the patient fully recovered and appropriate for discharge? (see admission criteria above)
- Is the patient fit for discharge i.e. consider social support, mobility and age
- Has MOPD referral form been completed and relevant documents included (see follow up criteria above)
- Patient has a responsible adult to stay with following discharge
- Arrange Echo and 24 hour tape for all patients attending MOPD. Other investigations e.g. ETT and Tilt table will be organised AFTER MOPD review
- Advice has been given regarding driving, hobbies and occupation
**MANAGEMENT OF COLLAPSE**

1. **Loss of consciousness**
   - Perform:
     - BM stix
     - ECG
     - Erect and supine BP
     - Oxygen saturation
   - **BM low**
     - Treat hypoglycaemia
     - Send lab glucose
   - **Witnessed seizure**
     - Manage and follow seizure algorithm
   - **Arrhythmia**
     - Structural cardiac cause
     - Pulmonary embolism
     - Cerebrovascular event
     - Vascular steal syndrome
   - **Postural drop in systolic BP**
     - >20 mm Hg or systolic BP < 90 mm Hg
     - Check FBC and U+E
   - **Vasovagal episode**
     - GTN syncope
     - Related to micturition, defaecation, coughing, emotional stress
   - Unexplained syncope
     - (Follow syncope algorithm)

**Consider:**
- Simple fall
- Intoxication
- Drop attacks
- Narcolepsy

**Check FBC and U+E**
- Consider:
  - Blood loss - inc occult
  - Dehydration
  - Drug therapy
  - Addison’s disease
  - Autonomic neuropathy
  - Sepsis

**Discharge if fully recovered and investigations normal**
- No follow up required

**Refer to appropriate specialty**
Out of hours if asymptomatic the patient can be discharged.

History meets inclusion/exclusion criteria for renal colic:
- IV access: Send FBC, U/Es, Ca, Urate
- Send MSU
- Check pregnancy test
- Provide analgesia (Morphine IV titrated +/- Diclofenac 50mg PO/100mg PR/75mg IM)
- Ensure hydration with oral/IV fluids

07.00 to 17.00
- Asymptomatic (discharge)
- Arrange CT

17.00 to 07.00
- Continued Symptoms
- Admit CAA

Asymptomatic
- Normal CT: Discharge GP
- Abnormal CT: Refer Lithotripsy

Continued Symptoms
- Normal CT: Reassess and manage appropriately
- Abnormal CT: Refer Urology

How to arrange a CT:
- Contact radiology directly 9-5.
- Patient should attend radiology with notes and return with report and film.
- Out of hours if asymptomatic the patient can be discharged and return to CAA base 1 the next day and CT organised.
- If symptomatic the patient will be admitted to CAA.
- If recent CT/IVU discuss with radiology regarding Ix.

MANAGEMENT OF RENAL COLIC
Based on RIE Procedure
### Inclusion Criteria
- Age <60y
- Typical pain with (microscopic) haematuria

### Exclusion Criteria
- Age >60y
- Temperature > 38C
- Known Vascular Disease

### Clinical Findings (enter findings)

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Pulse</th>
<th>BP</th>
<th>Resp Rate</th>
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### Investigations Table (enter result)

<table>
<thead>
<tr>
<th>Urea</th>
<th>Glu</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creat</td>
<td>Urate</td>
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<td>KUB</td>
<td>CT</td>
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<tr>
<td>IVU</td>
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</tbody>
</table>

### Indications for admission
- Intractable pain and vomiting
- Known single kidney
- Renal transplant
- Chronic renal failure
- CT reveals proximal stone or >8 mm stone and patient symptomatic (refer urology)

### Discharge from CAA (all boxes MUST be ticked before discharge)
- Patient fully recovered with controlled symptoms/signs
- Normal observations and investigations (include temperature), MSU sample sent
- Normal U+Es
- Regular analgesia prescribed
- If discharged at night patient able to attend RIE CAA base 1 or ARAU WGH 09.00 following morning for tx.
- If discharged during day CT normal

### Lithotripsy Unit follow up
- Abnormal CT
- Lithotripsy referral form completed
- Full length CT and KUB sent by courier to Lithotripsy Unit with referral form
- Patient aware Lithotripsy Unit will make contact within 3 working days
ADULT BASIC LIFE SUPPORT

**UNRESPONSIVE?**
- Shout for help
- Open airways

**NOT BREATHING NORMALLY?**
- Call 222
- 30 chest compressions
- 2 rescue breaths
- 30 compressions

- If he is breathing turn into recovery position, monitor for continued breathing and get/send for help.
- If he is not breathing send someone for help or, if you are on your own, leave the victim and telephone for help using 222.
- Return and immediately commence chest compressions at a ratio of 30 compressions to 2 ventilations at rate of 100/minute.
- Continue until the victim shows signs of life or the advanced life support techniques can be applied.
- Do not interrupt CPR unless the patient responds, help takes over or you are exhausted.
- In respiratory only arrest continue to ventilate the patient at a rate of 10-12 breaths per minute.

**www.resus.org.uk/siteindex.htm**

Training sessions can be arranged by contacting:
- In WGH/RVH Stephen Hartley RO or Dina Johnston RO on 32496.
- In RIE Marilyn Mathers RO or Denise Peden RO on 21670.
- In St John’s Stephen Short (Shorty) RO 53892 or bleep 909
Unresponsive?

Open Airway
Look for signs of life

Call 222: Cardiac Arrest team

CPR 30:2
Until defibrillator/monitor attached

Assess rhythm

Shockable VF/VT

Defibrillate x 1
360j monophasic
150j biphasic

Immediately resume CPR for 2 minutes

During CPR
Correct reversible causes

If not already:
- Check: electrode/paddle positions & contact
- Attempt/verify: airway & \(O_2\), IV access
- Give uninterrupted compressions when airway secured
- Give adrenaline 1mg every 3-5 minutes
- Consider: Amiodarone, (Magnesium)
  Atropine/pacing/buffers

Non Shockable PEA/Asystole

Immediately resume CPR
2 min cycles

- Hypoxia
- Hypovolaemia
- Hyper/hypokalaemia & metabolic disorders
- Hypothermia
- Tension pneumothorax
- Tamponade, Cardiac
- Toxic/therapeutic disturbances
- Thrombosis (coronary or pulmonary)

The ALS Algorithm for the management of cardiac arrests in adults.

NOTE that each successive step is based on the assumption that the one before has not resulted in restoration of circulation.
Basic life support is commenced in unmonitored situations while the monitor/defibrillator is obtained and attached.

In witnessed collapse a single precordial thump can be administered by trained personnel.

In monitored patients the clinical and ECG detection of cardiac arrest should be simultaneous.

In the presence of VF/pulseless VT defibrillation must occur as soon as possible.

Once the airway is secured (endo-tracheal tube) chest compressions are performed at 100/min and asynchronous ventilations at 10/min.

**VENTRICULAR FIBRILLATION/ PULSELESS VENTRICULAR TACHYCARDIA**

**Defibrillation:** The first shock is given at 150j biphasic or 360j monophasic ensuring good contact with the chest wall. Use of gel pads or hands free pads as applicable ensuring correct positioning and pressure application.

- The second and all subsequent shocks are delivered using the same energy level.
- A pulse check is no longer performed unless the patient responds to treatment.
- A two minute period of CPR immediately follows defibrillation.
- **Adrenaline 1mg should be given IV every 3-5 minutes (alternate cycles = every 4 minutes).** If no IV access give adrenaline 2mg via the ET Tube.
- **Adrenaline** should be administered just prior to shock.

**Drug therapy**

- In refractory cases amiodarone can be considered: prefilled 10ml syringe of 300mg or 300mg made up in 20 mls 5% dextrose over 2-5 mins in a large/central vein. A generous flush should be used if using a peripheral vein.
- Expert advice should be sought.
- The use of IV sodium bicarbonate should be limited to patients with a severe metabolic acidosis, hyperkalaemia or tricyclic antidepressant overdose.

The mainstay of the correction of acidosis in cardiac arrest is adequate ventilation and oxygenation with rapid restoration of a perfusing cardiac rhythm.
Unresponsive

Call for help

Open airway
Not breathing normally

Send or go for AED
Call 222

CPR 30:2
Until AED is attached

AED assesses rhythm

Shock advised

1 Shock
150 J biphasic or
360 J monophasic

Immediately resume
CPR 30:2
for 2 min

No shock advised

Immediately resume
CPR 30:2
for 2 min

Continue until the victim starts
to breathe normally
NON DEFIBRILLATABLE RHYTHMS

Cardiac arrest in asystole and PEA may result from a number of causes other than ischaemic heart disease (see table on previous page). These are potentially reversible causes of cardiac arrest. They should also be regarded as preventable causes of cardiac arrest, in that their recognition and treatment prior to cardiac arrest can prevent deterioration.

- On this side of the algorithm CPR is conducted for 2 minute periods whilst considering and treating any of the above.
- The airway should be secured, IV access obtained and adrenaline 1mg given IV every 3-5 minutes. (Alternate cycles every 4 minutes)

Drugs

- A single dose of Atropine 3mg IV is given asystole to block vagal activity, and in PEA with a ventricular rate under 60 beats per minute.
- Adrenaline is given 1mg IV at least every 3-5 minutes (as above).

Pacing

External or transvenous pacing is unsuccessful in asystole but may be effective in ventricular asystole where p waves are still evident. Percussion pacing may also be effective.

An external pacing defibrillator can be obtained from ARAU, CCU, ICU all HDUs, Wd 8 unit, wd 12, wd43 (ID), C.I.S in WGH and A&E or ICU in RIE and in SJH.

- After 3 minutes of CPR the ECG rhythm is re-assessed.
- If a rhythm compatible with cardiac output is present check the pulse.
- If VF/pulseless VT is present follow that side of the algorithm. Otherwise, continue with loops of the right hand path of the algorithm for as long as it is appropriate to continue active resuscitation.
## CONTACT NUMBERS

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<thead>
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<td>WGH fast bleep non cardiac arrest</td>
<td>222</td>
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<tr>
<td>WGH cardiac arrest</td>
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<tr>
<td>SJH cardiac arrest</td>
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## CARDIOLOGY: MEDICAL CONTACT NUMBERS

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<tr>
<td>Dr Nick Boon</td>
<td>21849</td>
<td>5090</td>
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<tr>
<td>Dr Peter Bloomfield</td>
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<td>Professor Keith Fox</td>
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<td>Professor David Newby</td>
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<td>#6537</td>
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<td>Dr David Northridge</td>
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<td>Dr Stuart Shaw</td>
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<td>Dr Ian Starkey</td>
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<td>Dr Neil Uren</td>
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<td>21046</td>
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<td>Registrars Room (WGH)</td>
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<tr>
<td>Registrars Rooms (RIE)</td>
<td>21910</td>
<td>#6816</td>
<td></td>
</tr>
<tr>
<td>Cardiology bed coordinator (RIE)</td>
<td>... 5606</td>
<td></td>
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</tr>
</tbody>
</table>
GENERAL ADMINISTRATIVE POLICY (RIE/WGH)

CCU services are changing in Lothian: seek up-to-date operational detail.

ADMISSIONS TO CCU

The following patients should be considered for admission to CCU:

- Patients with an acute coronary syndrome within the preceding 24 hours.
- Patients with life threatening, or haemodynamically unstable arrhythmias.
- Heart failure, pulmonary oedema or cardiogenic shock where intensive management/monitoring is required.
- Patients requiring monitoring after interventional cardiology procedures.
- Following cardiac arrest.

TRANSFERS/DISCHARGES

- After an uncomplicated ACS the patient may be transferred to a non-monitored cardiology or general medical bed after 24 hours. If there is a pressure on CCU beds transfer could take place sooner.
- Patients should be pain free and haemodynamically stable for at least 12 hours prior to transfer.
- Uncomplicated, stable infarct patients may be transferred from CCU to a monitored bed in the general cardiology ward within 24 hours of admission.
- The consultant with responsibility for CCU patients for the week will decide which patients are suitable for transfer out at each ward round in discussion with CCU charge nurse.
- Ideally all transfers should take place before 20.00hrs. Transfers should ideally be accompanied by a letter or written clinical summary when transferring out-with cardiology. Transfers within cardiology should at least involve a handover by verbal clinical summary for the team taking over care of the patient. This summary should include details of diagnosis, treatment at time of transfer/discharge and consultant responsible for the patient.
- Discharges home should be planned according to hospital discharge planning policy (for discharge pathway see the MI integrated care pathway).
CARDIOLOGY SUPPORT

There is an organised 24 hour rota for Consultant/Registrar cardiology opinion.
Out of hours the appropriate person can be contacted by bleep, radiopage (via switchboard), mobile phone or at home.
A copy of the rota is available in CCUs, cardiac catheter labs and switchboards.

HANDOVER OF CLINICAL CARE

When required, it is the responsibility of the medical and nursing team to ensure there is a hand-over to the team on the ward (letter or verbal).
Out with normal working hours (i.e.: from 5pm & at weekends) CCU nursing staff will inform the appropriate nursing and medical team of the transfer.
SUMMARY OF MANAGEMENT OF ACUTE CORONARY SYNDROME

Immediate Clinical Assessment
Electrocardiogram

- Intravenous access
- Morphine 2.5 - 10 mg iv + metoclopramide 10 mg iv
- Oxygen + cardiac rhythm monitoring
- Aspirin 300 mg po +
- Clopidogrel 300 mg po in unstable angina, clopidogrel 600 mg if for PCI
- Metoprolol 5-15 mg iv
- Blood sampling: FBC, U&E, lipid profile, glucose, troponin
- Transfer to a Specialist Cardiology Unit

Reperfusion Therapy

ST Segment Elevation ACS Presenting <12h From Onset?

- Yes
  - Thrombolysis contraindicated
    - Pentasaccharide sc

- No
  - Rapid Primary PCI Available?
    - Yes
      - GP IIb/IIa receptor antagonist iv + Emergency PCI
    - No
      - Thrombolysis iv + Pentasaccharide or LMW heparin sc in unstable angina, iv if for PCI
  - Yes
    - Successful Reperfusion?
      - Yes
        - Consider GP IIb/IIa receptor antagonist iv
      - No
        - Early coronary angiography with view to PCI or CABG
  - No

Pentasaccharide contraindicated and no primary PCI available

Medium To High Risk ACS?

- Yes
  - Consider GP IIb/IIa receptor antagonist iv

- No
  - Recurrent Symptoms?
    - Yes
      - GP IIb/IIa receptor antagonist iv
    - No

Maintenance In-hospital Medication Aspirin, clopidogrel, pentasaccharide/LMW heparin*, statin, beta-blocker and ACE inhibitor therapy

TIMI risk score
(Death, MI, recurrent ischaemia)
Low risk ≤ 2
Medium risk 3-4
High risk ≥ 5

GRACE score (Death)
Low risk ≤ 4.9%
Medium risk 5-9.9%
High risk ≥ 10%

1. Killip class 1 in the absence of bradycardia (heart rate <65/min) or hypotension (systolic blood pressure <105 mmHg).
2. Within 90 minutes of diagnosis or if thrombolysis is contra-indicated.
3. Patients presenting within six hours of symptom onset.
4. Continued for eight days, or until hospital discharge or coronary revascularisation.

Myocardial Ischaemia may be caused by anaemia, particularly with an acute bleed. In this case blood transfusion and cessation of bleeding are appropriate and most of the above therapy is contra-indicated ie heparin, GTN, antiplatelet agents and β-blockers.
In order to make a presumptive diagnosis of ACS the patient should exhibit symptoms consistent with acute myocardial ischaemia and have one of the following:

- electrocardiographic changes consistent with an ACS
- serial increases in biochemical markers of myocardial necrosis, and/or
- documentation of coronary artery disease.

**IMMEDIATE MANAGEMENT**

In combination with the clinical presentation, an ST segment elevation acute coronary syndrome is defined by the presence of ≥1mm ST elevation in at least two adjacent limb leads, ≥ 2 mm ST elevation in at least two contiguous precordial leads, or new onset bundle branch block. In absence of ST segment elevation (non-ST segment elevation acute coronary syndrome), patients are initially managed without emergency reperfusion therapy.

The categories of ACS, unstable angina or myocardial infarction, are defined by the serum concentration of cardiac enzymes and markers. The cardiac markers, troponin I and troponin T are extremely sensitive to myocardial injury and damage. Very small amounts of damage can be detected allowing identification of ‘micro-infarcts’ where there is an elevation in the troponin concentration without a significant rise in creatine kinase or other cardiac enzymes. One consequence of the availability of troponin measurement has been a blurring of the distinction between unstable angina and myocardial infarction.

<table>
<thead>
<tr>
<th>Cardiac Marker Concentrations</th>
<th>Troponin T (ng/mL), BCS</th>
<th>≥0.01 and &lt;1.0</th>
<th>≥1.0</th>
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<tbody>
<tr>
<td>Troponin I (ng/mL), RIE</td>
<td>&lt;0.2</td>
<td>≤0.2 and &lt;1.0</td>
<td>≥1.0</td>
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<tr>
<td>Troponin I (ng/mL), WGH</td>
<td>&lt;0.2</td>
<td>≤0.02 and &lt;2.0</td>
<td>≥2.0</td>
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<tr>
<td>Troponin I (ng/mL), SJH</td>
<td>&lt;0.034</td>
<td>≤ 0.034 and &lt;1.0</td>
<td>≥1.0</td>
</tr>
<tr>
<td>BCS definition</td>
<td>ACS with unstable angina</td>
<td>ACS with myocyte necrosis</td>
<td>ACS with clinical myocardial infarction</td>
</tr>
<tr>
<td>ESC/ACC definition</td>
<td>unstable angina</td>
<td>unstable angina</td>
<td>myocardial infarction</td>
</tr>
<tr>
<td>WHO definition</td>
<td>unstable angina</td>
<td>unstable angina</td>
<td>myocardial infarction</td>
</tr>
<tr>
<td>30-Day Mortality</td>
<td>4.5%</td>
<td>10.4%</td>
<td>12.9%</td>
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<tr>
<td>6-Month Mortality</td>
<td>8.6%</td>
<td>18.7%</td>
<td>19.2%</td>
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</table>
DIAGNOSIS AND RISK STRATIFICATION OF PATIENTS WITH ACUTE CORONARY SYNDROME

Many treatments, especially for ST elevation acute coronary syndrome, are critically time-dependent and the immediate clinical assessment of all patients with a suspected acute coronary syndrome is essential. The electrocardiogram should be repeated

- with recurrent or persistent pain
- the day after admission
- prior to discharge
- with any change in the patient’s symptoms

Patient’s with suspected acute coronary syndrome require immediate clinical assessment and 12 lead electrocardiogram.

To establish a diagnosis in patients with acute coronary syndrome, a serum troponin concentration should be measured 12 hours from the onset of symptoms. Troponin concentration provides one measure of risk that should not be relied upon in isolation. For example, patients with unstable angina and a troponin concentration within the reference range at 12 hours, can have a high risk of future cardiovascular events (30 day risk of death up to 4-5%). Elevated troponin concentrations are associated with adverse outcomes in many different clinical settings including congestive heart failure, sepsis, pulmonary disease, acute pulmonary embolism and chronic renal failure.

In patients presenting with chest pain, serum troponin concentration should be measured on arrival at hospital to guide appropriate management and treatment.

A fasting lipid profile should be determined within the first 24 hours of symptom onset. Investigations - electrolytes, urea, creatinine, liver function tests, glucose, full blood count and cholesterol.

Risk stratification using clinical scores should be conducted to identify those patients with an acute coronary syndrome who would benefit from early therapeutic intervention.
This section refers to all categories of ACS including those patients with ST segment elevation. In the event of unstable angina or acute MI including STEMI occurring in the wards, theatres or other clinical areas at WGH or SJH treatment should be initiated as described in this chapter and the Cardiology Registrar should be contacted.

**Typical history of ACS**

Establish continuous ECG monitoring

- Oxygen to keep SpO$_2$ >97%
- IV access (2 for those receiving TNK)
- Anti-emetic: metoclopramide 10mg IV standard
- Morphine 2.5-10mg IV initially
- In the absence of bradycardia (<65 /min) or hypotension (systolic blood pressure <105 mmHg), patients with an acute coronary syndrome in Killip class I should be considered for immediate intravenous and oral beta-blockade.
- Aspirin 300mg to chew (unless already given by ambulance crew) and clopidogrel 300mg in unstable angina and 600mg if ECG ischaemic or elevation of cardiac markers or for PCI.
- Blood sampling for U+Es, lipid profile, glucose, FBC
- CK level if pain started >6 hours prior to presentation
- Tpi

**STEMI DEFINITIONS FULFILLED**

**IMMEDIATE REPERFUSION**

**TRANSFER TO CCU WITH CONTINUOUS MONITORING**
All patients with ST segment elevation acute coronary syndrome presenting within 12 hours of symptom onset should be considered for immediate reperfusion therapy.

If the ECG is normal, immediate reperfusion therapy should not be given, even if the history is suggestive of MI. ‘T’ wave inversion and widespread ST depression is not an indication for immediate reperfusion therapy. If there is diagnostic doubt then consider:

- Posterior ECG leads [ST elevation in 2 or more contiguous leads V7-V9]
- Repeat ECG after 10 minutes + nitrate administration
- Echocardiography
- Early Cardiology opinion

**Primary Percutaneous Coronary Intervention (PCI)**

When compared with thrombolysis, primary PCI reduces short and long-term mortality, stroke, re-infarction, recurrent ischaemia and the need for coronary artery bypass graft (CABG) surgery as well as the combined end points of death or non-fatal re-infarction. This benefit is consistent across all patient subgroups and is independent of the thrombolytic agent used. The greatest benefit is seen in those patients treated within 12 hours of symptom onset.

**Where available, patients with ST elevation acute coronary syndrome should be treated immediately with primary percutaneous coronary intervention.**

Patients undergoing primary percutaneous coronary intervention should be treated with a glycoprotein IIb/IIIa receptor antagonist and undergo intracoronary stent implantation.

**When primary percutaneous coronary intervention cannot be provided within 90 minutes of diagnosis, patients with ST elevation acute coronary syndrome should receive immediate thrombolytic therapy.**
Pt arrives in ARAU with CP

**ARAU**
- Assess, Cardiac Monitor, IV Access
- 12 lead ECG within 5 minutes
- Rx: O₂, 300mg Aspirin, GTN, Intravenous Opiate, 600mg Clopidogrel

12 lead ECG showing ST segment elevation of ≥ 2mm in 2 contiguous leads or new BBB and clinical suspicion of MI?

**CCU SHO on - duty? 0900 to 1630, Monday to Friday**

- Bleep CCU SHO (8102)/CP Nurse (8755), stating STEMI patient in ARAU possibly for PPCI

- CCU SHO & CP/CCU Senior Nurse go immediately to ARAU with STEMI kit-bag

- Patient eligible for immediate PPCI?

  - NO
    - EG unable to lie flat, refuses consent
    - **Discuss with Consultant Cardiologist**
    - Consider thrombolysis

  - YES
    - Phone Cath Lab (31855) and ascertain estimated time of availability

  - Cath Lab available immediately
    - Transfer patient to Cath Lab
  
  - Cath Lab available within 60 minutes
    - Cath Lab contact ARAU as soon as available
  
  - Cath Lab not available within 60 minutes eg equipment failure, ongoing complex case, on-call team at RIE
    - **Discuss with Consultant Cardiologist**
    - Consider thrombolysis and admit CCU

**Consider while awaiting Cath Lab availability**

- Repeat ECG. Chest X-Ray.
- Secure IV access, 2nd cannula if uncertain
- Discuss with family
- Routine biochemistry, haematology, **group & save**
- Document peripheral pulses

- 5,000 units IV Heparin
- High dose Tirofiban bolus + infusion
- 5-10mg IV Metoprolol
- IV Opiate

**CCU SHO not on - duty?**

- Out-of-hours 1630-0830, Mon-Sun

- Weekend daytime 0830-1630, Sat&Sun

- Phone Cardiology SpR (8689), requesting cascade of the Cath Lab on-call team

- Cath Lab contact ARAU as soon as prepared
Pt arrives in ED with CP

ED
- Assess, Cardiac Monitor, IV Access
- 12 lead ECG within 5 minutes
- Rx: O₂, 300mg Aspirin, GTN, Intravenous Opiate, 600mg Clopidogrel

12 lead ECG showing ST segment elevation of ≥ 2mm in 2 contiguous leads or new BBB and clinical suspicion of MI?

CP Nurse on-duty?
0730 to 1930
7 days per week unless otherwise stated

CP Nurse not on-duty?
1930 to 0730
or, CP Nurse not on duty

ED Bleep CP Nurse (5834) stating STEMI patient in ED possibly for PPCI

CP Nurse, CCU SHO or CCU Senior Nurse go immediately to ED with STEMI kit-bag

EG unable to lie flat, refuses consent
Discuss with Consultant Cardiologist, consider thrombolysis

Patient eligible for immediate PPCI

YES

Cath Lab staff hours
0830 to 1630, Mon – Fri

Phone CCU, remain on line while CCU contact Cath Lab via Primary PCI (red) phone and ascertain estimated time of availability

Cath Lab available immediately
Transfer patient to Cath Lab
Cath Lab available within 60 minutes
Cath Lab contact ED as soon as available

Cath Lab not available within 60 minutes
eg equipment failure, ongoing complex case, on-call team at WGH
Discuss with Consultant Cardiologist
Consider thrombolysis and admit CCU

NO

ED Bleep 1581 (either CCU Senior Nurse or CCU SHO on as part of HAN) stating STEMI patient in ED possibly for PPCI

Out-of-Cath Lab staff hours
Phone CCU, requesting cascade of the Cath Lab on-call team
Cath Lab contact ED as soon as prepared

Consider while awaiting Cath Lab availability

- Repeat ECG, Chest X-Ray.
- Secure IV access, 2nd cannula if uncertain
- Discuss with family
- Routine biochemistry, haematology, group & save
- Document peripheral pulses

- 5,000 units IV Heparin
- High dose Tirofiban bolus + infusion
- 5-10mg IV Metoprolol
- IV Opiate
Pt calls 999 with CP

SAS - Assess
- Rx: High flow O₂, 300mg Aspirin po, Sublingual GTN, Intravenous Opiate
- 12 lead ECG + telemetry

12 lead ECG showing ST segment elevation of ≥ 2mm in 2 contiguous leads and clinical suspicion of MI?
Telemetry transmission to receiving station, or (in event of transmission failure) SAS phonecall to RIE CCU?

Urgent call to receive a Primary PCI at ETA?

RIE CCU ask SAS
“Can you be at the nearest Cath Lab (RIE or WGH) within 60 of diagnostic ECG?”

YES

Advise SAS
- Give 5,000 unit bolus IV Heparin (unless contraindicated)
- Proceed directly to relevant Cath Lab
- If WGH, forward fax ECG to WGH CCU
- Cascade CCU SHO + CP nurse or CCU nurse to meet patient at Cath Lab

If WGH/STJH, pre-alert + forward ECG to CCU fax.

If not within JRCALC guidelines: RIE CCU advise admission to nearest appropriate Cath Lab hospital (RIE/WGH).

SAS administer TNK + Heparin (if within JRCALC guidelines).
RIE CCU advise direct admission to nearest appropriate CCU:
RIE(24/7), StJH(24/7) or WGH (0830 to 1630, Mon – Fri)
If WGH/STJH, pre-alert + forward ECG to CCU fax.

RIE CCU ask SAS
“Can you be at the nearest Cath Lab (RIE or WGH) within 60 minutes of diagnostic ECG?”

YES

Advise SAS
- Give 5,000 unit bolus IV Heparin (unless contraindicated)
- Proceed directly to relevant A&E/ARAU
- If WGH, forward fax ECG to WGH CCU
- Cascade CCU SHO + CP nurse or CCU nurse to meet patient at A&E/ARAU as soon as available

If the Cath Lab on-call team are on-site at either RIE/WGH performing a procedure, the Cath Lab Nurse will call RIE CCU to alert them both on arrival and departure. In this case the patient must be directed to the site where the team are at the time.

SAS transport to agreed destination unless the patient’s condition presents or deteriorates such that a crash-call to A&E/ARAU resus should be considered. Should this be the case, SAS will phone RIE CCU at the earliest opportunity.
Thrombolysis
Since the clinical benefits of thrombolysis are time-dependent with an increase of 1.6 deaths per hour of delay per 1,000 patients treated, various strategies have been employed to minimise the delay between diagnosis and administration of thrombolysis. Pre-hospital thrombolysis should be used where possible because it shortens the time between the call for help and the administration of thrombolysis. Significant improvements in door-to-needle times are achieved by administration of thrombolysis within the Emergency Department. This can be facilitated by an experienced cardiology nurse and accomplished without compromising the appropriateness of its administration. Door-to-needle time should be less than 30 minutes.

Contra-indications to thrombolysis:
- Known/suspected intracranial tumour, aortic dissection, pericarditis.
- Recent [within last 3 months] stroke of any type, GI or GU bleeding.
- Major surgery [including dental surgery], trauma, biopsy or head injury within 6 weeks.
- Severe hypertension – systolic > 180 mmHg and/or diastolic > 110 mmHg (see below)
- Bleeding diathesis, including uncontrolled anticoagulation.
- Puncture of a non-compressible vessel.
- Prolonged (> 10 minutes) cardiopulmonary resuscitation.
- Impaired consciousness following cardiac arrest.
- Pregnancy
- Neurosurgery (within last year).

Choice of thrombolytic:

Tenectplase is the thrombolytic agent of choice.

Ease of use favours a bolus fibrin-specific agent on practical grounds, particularly in the pre-hospital setting.
**Hypertension.** All patients should already have received intravenous opiate analgesia and beta-blockade. Repeated dosing should be given if appropriate. Further hypertension can be managed with intravenous nitrate infusion. Thrombolysis can be commenced once systolic blood pressure <180 mmHg and diastolic blood pressure <110 mmHg.

**Anticoagulation following thrombolysis:**
Patients with ST elevation acute coronary syndrome who receive thrombolytic therapy should be treated immediately with either a pentasaccharide (fondaparinux 2.5 mg iv then sc daily) or low molecular weight heparin (enoxaparin 1 mg/kg bd sc). This should be continued for eight days, or until hospital discharge or coronary revascularisation.

**Failure to reperfuse following thrombolysis:**
Rescue PCI is undertaken within 12 hours of thrombolysis administration when there is an apparent failure to reperfuse the infarct-related artery. Reperfusion is taken to have occurred when there is a >50% fall in ST segment elevation or new onset of idioventricular rhythm.

**Patients presenting with ST elevation acute coronary syndrome within six hours of symptom onset, who fail to reperfuse following thrombolysis, should undergo rescue percutaneous coronary intervention.**

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**Tenecteplase dosing: weight adjusted**

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<thead>
<tr>
<th>Weight Range</th>
<th>Dosage</th>
<th>Units</th>
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<tr>
<td>&lt;60kg</td>
<td>30mg</td>
<td>6000 units</td>
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<tr>
<td>60-69kg</td>
<td>35mg</td>
<td>7000 units</td>
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<tr>
<td>70-79kg</td>
<td>40mg</td>
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<tr>
<td>80-89kg</td>
<td>45mg</td>
<td>9000 units</td>
</tr>
<tr>
<td>&gt; 90kg</td>
<td>50mg</td>
<td>10000 units</td>
</tr>
</tbody>
</table>

**Heparin is commenced following this**

- <67kg 4000 units IV unfractionated (heparin bolus)  
  Then 800 iv/hr
- >67kg 5000 units IV unfractionated (heparin bolus)  
  Then 1000 iv/hr

**Check APTT at 6 hrs aiming for 1.5-2.5**

**From WGH CCU: check your local policy**

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*from adult medical emergencies handbook* | *NHS LOTHIAN: UNIVERSITY HOSPITALS DIVISION* | *2007/09* |
Reperfusion therapy not administered

Some patients may not reach the full electrocardiographic criteria for reperfusion therapy, have a delayed presentation (>12 hours from symptom onset) or have significant contra-indications or co-morbidity that limits the administration of reperfusion therapy.

Patients with ST elevation acute coronary syndrome who do not receive reperfusion therapy should be treated immediately with a pentasaccharide (Fondaparinux 2.5 mg sc). This should be continued for eight days, or until hospital discharge or coronary revascularisation.

IMMEDIATE MANAGEMENT OF NON-ST SEGMENT ELEVATION ACUTE CORONARY SYNDROMES

Patients with non-ST elevation ACS should be treated immediately with a pentasaccharide (fondaparinux 2.5 mg sc daily). This should be continued for eight days, or until hospital discharge or coronary revascularisation.

Patients with an ACS who have dynamic ST segment changes, haemodynamic compromise or acute heart failure are at particularly high risk. Such patients benefit from early invasive intervention. “Upstream” use of glycoprotein IIb/IIIa receptor antagonism reduces events and improves outcomes particularly where the patient has diabetes mellitus or an elevated troponin.

High-risk patients with non-ST elevation acute coronary syndrome should be treated with an intravenous glycoprotein IIb/IIIa receptor antagonist and considered for urgent PCI.
The following is suggested where there is persistent hypertension prior to administration of thrombolysis.

Assessment of pain and pain control

**MORPHINE**
Slow IV injection at a rate of 2mg/min. until pain relief achieved

Exclude the following:
- Bradycardia HR <60
- Heart block
- Cardiac failure
- Asthma
- Critical limb ischaemia

Excluded  Not excluded

**IV betablocker**
**IV metopropol**
- 1-2mg/min. up to 5mg
- Repeat after 5 min if required
  - Total dose 10-15mg
  - Give oral dose 25-50mg at same time

**Nitrate**
- IV GTN infusion
- GTN Spray
- Buccal nitrates

Commence thrombolysis once systolic BP <180 and diastolic <110mmHg
FURTHER MANAGEMENT OF ACUTE CORONARY SYNDROMES

Following ACS all patients should be considered for:
- aspirin
- clopidogrel for three months (for six months if drug-eluting stent implanted)
- statin
- ACE inhibition
- beta-blockade

PATIENTS WITH CLINICAL MYOCARDIAL INFARCTION

Patients with clinical myocardial infarction and diabetes mellitus or marked hyperglycaemia (>11.0 mmol/L) should have immediate intensive blood glucose control using intravenous insulin and glucose. This should be continued for at least 24 hours.

Where possible, patients with clinical myocardial infarction should be commenced on long-term angiotensin-converting enzyme inhibitor therapy within the first 36 hours.

Patients with clinical myocardial infarction complicated by left ventricular dysfunction or heart failure should be commenced on long-term angiotensin receptor blocker therapy if they are intolerant of angiotensin-converting enzyme inhibitor therapy.

Patients with clinical myocardial infarction complicated by left ventricular dysfunction (ejection fraction <35%) in the presence of either heart failure or diabetes mellitus should be commenced on long-term aldosterone receptor antagonist therapy.

RISK STRATIFICATION AND INVASIVE INVESTIGATION

- Risk stratification using clinical scores should be conducted to identify those patients with ACS who would benefit from early therapeutic intervention.
- Assess cardiac function to identify patients at high risk (those who benefit from selected therapeutic interventions, such as aldosterone receptor antagonism).

Patients with ST elevation ACS treated with thrombolytic therapy should be considered for coronary angiography and revascularisation during their index hospital admission.
MANAGEMENT OF COMPLICATIONS ASSOCIATED WITH MYOCARDIAL INFARCTION

RECURRENT ISCHAEMIC PAIN

- ECG should be recorded during pain if possible.

The following may be required, depending on the circumstances:
- Additional opiate.
- Buccal nitrates 2-5mg as required.
- Optimisation of beta blockade (heart rate<70 bpm).
- Consideration of IV glycoprotein IIb/IIa a receptor antagonist
- Consideration of urgent coronary angiography.

PERICARDIAL PAIN

A friction rub may or may not be heard. Mild pain can be controlled by paracetamol 1g qds or dihydrocodeine 30mg qds. Non-steroidal anti-inflammatory drugs such as ibuprofen may be considered but avoided in the presence of extensive infarction, renal failure or cardiac failure. More severe pain should be treated with IV opiate.

NAUSEA AND VOMITING

Metoclopramide 10mg IV 8 hourly is the first line drug of choice.

MILD-MODERATE LEFT VENTRICULAR FAILURE/PULMONARY OEDEMA

The following features either together or in isolation should raise clinical suspicion of heart failure in the setting of an acute coronary syndrome:
- Mild breathlessness at rest or on minimal exertion.
- Persistent tachycardia.
- Elevated JVP.
- Basal crepitations.
- Upper lobe diversion on CXR.
- Pulmonary oedema on CXR.

After a myocardial infarction an elevated JVP as an isolated feature may reflect right ventricular infarction particularly in the setting of an inferior or posterior infarction. Diuretic therapy may worsen the situation.
Management

- Oxygen (high concentration \( \geq 60\% \)).
- Blood gases are not always required but should be performed if shock or COPD also present.
- Monitor \( O_2 \) saturation and ECG.
- Consider reducing or stopping beta-blocker temporarily.
- Ensure the patient is on an ACE inhibitor such as lisinopril or ramipril unless contra-indicated.
- Consider oral diuretics such as furosemide or bumetanide. Assess the need for diuretics daily. Be careful not to over-treat.
- Assess cardiac structure and function by echocardiography prior to hospital discharge.
- Use intravenous glyceryl trinitrate if systolic blood pressure \( > 90 \text{mmHg} \).
- Once the patient is stable with no signs of pulmonary oedema or salt and water excess consider starting a low dose beta-blocker such as bisoprolol 1.25mg od or Carvedilol 3.125mg bd.

SEVERE LEFT VENTRICULAR FAILURE (LVF)

Patient breathless at rest. Sinus tachycardia is usual, or possibly rapid atrial fibrillation (BP often high). A gallop rhythm and widespread crepitations are often present. CXR shows features of pulmonary oedema.

If a patient looks “shocked”: tachypnoeic and tachycardic but with a high BP LVF is the likely problem

Management

- Give high concentration oxygen 60% or greater via a venturi mask or non-rebreathing mask/reservoir system.
- Give sublingual GTN, 2 puffs immediately if SBP \( \geq 100 \text{mm Hg} \).
- All patients with severe LVF should receive an intravenous infusion of nitrates (GTN 0.3-10mg/hr starting dose depending on baseline BP between 0.3mg/hr and 1mg/hr).
- Consider titrating morphine -1-5mg IV over 5-10 minutes preceded by prophylactic anti-emetic (metoclopramide 10mg IV). Reduce morphine dose in frail, elderly, chronic respiratory disease: give 1mg increments.
- If bronchospasm is a major component nebulised salbutamol 2.5mg or 5mg may be beneficially improving oxygenation and reducing work of breathing.
Morphine 10mg made up to 10ml with water for injection titrated slowly IV in 1mg increments, 2mg/minute.

- IV diuretic: If normal renal function and diuretic naive use furosemide 20mg: if currently on diuretic or in renal failure may require higher doses e.g. 50-100mg. Slow IV ≤ 4mg/minute.
- Consider arterial blood gases (caution with thrombolysis).
- Consider CPAP early for refractory hypoxia and respiratory fatigue.
- Consider early HDU/ICU referral.
- Monitor urine output: insert a urinary catheter.

Investigations

- ECG
- CXR
- Early Echocardiography
- Review the cardiac rhythm and blood pressure, treating tachy/bradyarrhythmias and hypertension appropriately.
- Consider and exclude mechanical causes e.g. acquired VSD, mitral regurgitation, left ventricular aneurysm, cardiac tamponade.

Echocardiography is of particular value in this situation and should be obtained as early as possible.

If patient becomes drowsy or obtunded, or if CO₂ retention is present, give an opiate antagonist and, if there is no immediate response, consider ventilatory support. Maintain high concentration oxygen therapy throughout.

Do not hesitate to seek a Cardiology opinion if there is no improvement and refer to ICU early.

- In severe or resistant cases support with intra-aortic balloon pump maybe life saving.
- Consider insertion of an arterial line and pulmonary artery catheter to measure cardiac output, guide the administration of inotropes, and assess response. Particularly useful in hypotension.
- Vasoactive drugs may be required and should be administered under expert guidance.
- Digitalisation, for its inotropic effect, may be beneficial but 3-6 hours may elapse before there is any appreciable effect. Loading doses as per AF (caution in renal impairment).
RIGHT VENTRICULAR INFARCTION/FAILURE

Diagnosis
- Right Ventricular Failure (Hypotension and elevated JVP/hepatic congestion) in the absence of clinical /radiological evidence of pulmonary congestion suggests the possibility of right ventricular infarction. This is more likely in association with acute inferior/infero-posterior infarctions.
- The right ventricular leads on the 12 lead ECG (V3R & V4R placed in the equivalent positions but to the right of the sternum as V3 & V4) may show ST elevation, confirming RV Infarction.
- Echocardiogram and/or the insertion of a pulmonary artery catheter will confirm the diagnosis.

Management
- **Diuretics or vasodilators/GTN should be avoided** as right ventricular function is dependent upon high filling pressures.
- If hypotension/oliguria persists, administer IV fluids and consider haemodynamic monitoring using a PA catheter.
- In the event of persistent hypotension/low cardiac output inotropic therapy may be required. Seek expert advice.

CARDIOGENIC SHOCK

Combined Cardiology and ICU referral early is appropriate.

Diagnosis
Cardiogenic shock should be considered if the following features are present:
- Hypotension
- Tachycardia or profound bradycardia.
- Poor peripheral perfusion.
- Oliguria
- Absence of haemorrhage or hypovolaemia.

Management
- High concentration oxygen, 60-100% humidified (35% initially in patients with severe COPD).
- Seek expert help (above).
- Treat any arrhythmias appropriately, wherever possible restoring sinus rhythm. Many anti arrhythmics are myocardial depressant.
In the setting of acute myocardial infarction consider the option of immediate PCI possibly with support from the intra aortic balloon pump.

- A urinary catheter should be inserted to monitor urine output.
- Check electrolytes and blood gases.
- Consider early insertion of a pulmonary artery catheter and arterial line.
- Vasoactive therapy may be required and is titrated to effect.
- An immediate echocardiogram should be performed to assess LV and RV function and to exclude cardiac tamponade, acute mitral regurgitation, ventriculo-septal defect.
- Consider other causes e.g. haemorrhage (especially retroperitoneal in anticoagulated patients), volume depletion or RV infarction.

1. Seek cardiology advice regardless of the time of day or night.
2. Make ICU referral early.

DVT

There is a policy for the prevention and treatment of DVT in ambulant patients. See the Prescribing Bulletin (on Intranet).

HYPOKALAEMIA

Potassium supplementation should be instituted in the following circumstances:

- Potassium <3.5mmol/l associated with any acute coronary syndrome.
- Any arrhythmia associated with hypokalaemia or low normal potassium.
- During insulin infusion in a diabetic patient.
- Take care to review drug therapy on a daily basis taking into account any subsequently prescribed potassium-sparing diuretics or ACE-inhibitors.
- For intravenous replacement of potassium always use a pre-prepared bag for infusion.

DIGAMI Protocol:
- All patients admitted with an acute myocardial infarction within the preceding 24 hours who are known to have diabetes mellitus or, although not known to have diabetes, who have a random blood glucose concentration >11 mmol/l should be started on an IV insulin/dextrose regimen for 24-48 hours.
- The diabetes team should be consulted regarding advice on management of diabetes/impaired glucose tolerance thereafter.

Infusion regime (RIE/WGH)
- Dextrose: start a drip infusion of 5% dextrose and run at 500mls per 12 hours.
- Insulin: use 50 i.u. of human actrapid or humulin S in 50mls of saline (0.9% NaCl) equivalent to 1u/ml via a syringe driver at a rate determined by the table below.
- Blood glucose levels monitored hourly by BM stix until stable, then 4-6 hourly during infusion.
- Potassium levels may fall rapidly and should be monitored closely during insulin infusion.

<table>
<thead>
<tr>
<th>Blood glucose (mmol/l)</th>
<th>Insulin infusion rate (iu/hr)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-4</td>
<td>0 (0.5 if known diabetic)</td>
</tr>
<tr>
<td>4.1-6.9</td>
<td>1</td>
</tr>
<tr>
<td>7.0-10.9</td>
<td>2</td>
</tr>
<tr>
<td>11.0-15.0</td>
<td>3</td>
</tr>
<tr>
<td>&gt;15</td>
<td>6 (check pump + connections) + IV access</td>
</tr>
</tbody>
</table>

- This scale is flexible and should be adjusted according to individual patient response.
LATE MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION

DRUG THERAPY AT THE TIME OF DISCHARGE

All patients with acute coronary syndrome should be discharged on:

- Aspirin 75 mg od
  Clopidogrel 75 mg od for 3 months (6 months where drug-eluting stent has been implanted)
- Statin (see below), such as simvastatin 40 mg daily
- Beta-blocker, titrated to achieve HR <70 /min
- ACE Inhibitor, up titrated to evidence based dose, such as ramipril 10 mg od or lisinopril 10 mg od. Patients intolerant of ACE inhibitor therapy should be considered for an angiotensin receptor blocker, such as valsartan 40-160mg bd or candesartan 4-16 mg od.
- All patients should be given a GTN spray to use as required for chest pain.

If any of these drugs are not prescribed, reasons for this should be clearly documented in the casenotes.

CARDIAC REHABILITATION

Additional secondary prevention strategies addressed as part of the rehabilitation program include:

- Smoking cessation.
- Dietary advice.
- Exercise
- Alcohol intake.

These are incorporated in the HEART MANUAL that is received by all patients with clinical myocardial infarction.

- Cardiac Rehabilitation Programme should be considered for all patients suffering clinical myocardial infarction. There is no age limit for referral. The cardiac rehabilitation co-ordinators are contactable by page and should be informed of all patients admitted with clinical MI regardless of the perceived need for rehab. Frail and infirm patients will be offered information and support.
- The Lothian Hospitals offer lifestyle education, smoking cessation advice and, for those who are suitable, group exercise programs. The majority of patients are given the ‘Heart Manual’: a six week home based program with support during the time by the hospital rehab team or the BHF nurse, and/or the community heart manual.
facilitators. As a rough guide, patients referred for group exercise should be able to complete stage 1 (3 minutes) of full Bruce protocol. Treadmill testing.

Phase III programmes are offered at Astley Ainslie Hospital, Western General Hospital and St John’s Hospital.

OTHER POTENTIAL PROBLEMS IN THE PERI-INFARCT PERIOD

ALCOHOL WITHDRAWAL

There is an alcohol withdrawal policy (see Lothian Joint Formulary) and chapter 12.

NICOTINE WITHDRAWAL

Evidence suggests that transdermal nicotine, and nicotine gum should not be withheld from patients who suffer an MI unless there is evidence of ongoing ischaemia (Goldstein, Niaura, 2000). The safety of NRT in those with unstable angina or post MI within 2 weeks has not yet been studied but cardiac complications should be lower than with smoking.

A risk/benefit assessment should be made with each individual patient. Patients struggling with the withdrawal effects of nicotine should be offered treatment for the first 48 hours, or until haemodynamically stable, using benzodiazepines, as for alcohol withdrawal. The risks of NRT should be explained using written information about the specific product they will use. They should sign a statement in the case notes indicating that they have read this information and accept responsibility for its use. They should also agree not to smoke whilst on NRT.

Patients should be seen by the smoking cessation nurse when considering starting NRT in the setting of a recent acute coronary syndrome.
Acute myocardial infarction is the underlying cause in 40% of cases and the general principles of its management should be followed. Emergency coronary angiography and reperfusion therapy should be considered if the patient has recovered consciousness. Reperfusion therapy may also be considered in patients with impaired consciousness who show signs of awakening provided there is no evidence of head injury arising from the collapse.

In cases without definite evidence of acute myocardial infarction investigations should be directed towards other causes of ventricular arrhythmia:

- Electrolyte disturbance.
- Underlying bradycardia.
- Pro-arrhythmic effect of medication.
- Drug overdose (tricyclic antidepressants, amphetamines, cocaine).

Coma is present in approximately half of cases admitted to CCU with an out-of-hospital cardiac arrest. Coma is compatible with meaningful survival even if it persists for up to 72 hours. Management should be directed at maintaining oxygenation, circulation and renal function during this period. Patients should be considered for therapeutic hypothermia and this is indicated in the presence of:

- coma (unresponsive to voice, GCS <9)
- intubation and ventilation
- no other cause of coma
- negative pregnancy test
- haemodynamically stability

Mechanical ventilation should be considered for:

- Maintenance of oxygenation
- Normalisation of CO₂
- Shock
- Seizures
- Worsening acidosis

Intubation with spontaneous breathing is not ideal for optimisation of coronary and cerebral oxygen delivery. Mechanical ventilation is often appropriate.
Referral to ICU should follow discussion with the duty cardiologist. Late management should include assessment of left ventricular function and exercise testing. In those without acute myocardial infarction the indications for an ICD should be discussed.

**MANAGEMENT OF ARRHYTHMIAS**

The following are guidelines only. Any complex problems or arrhythmia unresponsive to treatment should be discussed with the Cardiologist on call at any time.

**TACHYARRHYTHMIAS**

**NARROW COMPLEX TACHYCARDIAS**

**Sinus Tachycardia**
Characterised by a narrow QRS (3 small squares or less, unless bundle branch block present) and normal ‘P’ waves. Rate >100bpm at rest. Consider other supraventricular tachycardias for any rate above 140bpm. Carotid sinus massage may help to differentiate.

Sinus tachycardia commonly results from underlying pathology outwith the heart e.g. sepsis, hypovolaemia, pain.

**Management**
- Treat possible causes (e.g. pain, anxiety, fever, cardiac failure, pericarditis).
- In acute coronary syndromes, consider beta-blocker unless contraindicated.
- Consider echocardiography to assess LV function.

**ATRIAL FIBRILLATION**
Characterised by irregular, narrow QRS complexes with no discernable P-waves.

AF may result from underlying pathology outwith the heart e.g. sepsis, hypovolaemia, pneumonia, pulmonary embolism.

**Management**
- For management refer to UK Resuscitation Council guidelines (see algorithm).
- Atrial fibrillation often accompanies left ventricular failure. Therapy may be ineffective unless it is given in conjunction with effective treatment of cardiac failure.
- Check TFTs.
Is Patient stable?

Signs of instability include:
1. Reduced conscious level
2. Chest pain
3. Systolic BP < 90 mmHg
4. Heart failure
   (Rate related symptoms uncommon at less than 150 beats min⁻¹)

- Support ABCs: give oxygen; cannulate a vein
- Monitor ECG, BP, SpO₂
- Record 12-lead ECG
- Identify and treat reversible causes (e.g., electrolyte abnormalities)

Is QRS narrow (<0.12 sec)?

- Is rhythm regular?
  - Probable atrial fibrillation
  - Control rate with:
    - β-Blocker IV or digoxin IV
    - Amiodarone 300 mg IV 20-60 min; then 900 mg over 24 h
    - Continuous infusion of amiodarone should be administered via a central venous catheter using an appropriate infusion device

- Probable re-entry PSVT:
  - Record 12-lead ECG in sinus rhythm
  - If recurs, give adenosine again & consider choice of anti-arrhythmic prophylaxis

- Probable atrial flutter
  - Control rate (e.g., β-Blocker)

If QRS irregular:
- Use vagal manoeuvres
- Adenosine 6 mg rapid IV bolus; if unsuccessful give 12 mg; if unsuccessful give further 12 mg
- Monitor ECG continuously

TACHYCARDIA ALGORITHM (with pulse)

- Synchronised DC Shock
  - Up to 3 attempts
  - Amiodarone 300mg IV over 10-20 min and repeat shock; followed by:
  - Amiodarone 900mg over 24h

- Broad QRS
  - Is QRS regular?
    - Seek expert help
  - Is rhythm regular?
    - Normal sinus rhythm restored?
      - Yes
        - Broad QRS
        - Irregular
        - Probable atrial fibrillation
        - Control rate with:
          - β-Blocker IV or digoxin IV
          - Amiodarone 300 mg IV 20-60 min; then 900 mg over 24 h
          - Continuous infusion of amiodarone should be administered via a central venous catheter using an appropriate infusion device
        - Probable re-entry PSVT:
          - Record 12-lead ECG in sinus rhythm
          - If recurs, give adenosine again & consider choice of anti-arrhythmic prophylaxis
        - Possible atrial flutter
          - Control rate (e.g., β-Blocker)
      - No
        - Seek expert help

- Narrow QRS
  - Is rhythm regular?
    - Yes
      - Narrow QRS
      - Regular
      - Probable atrial fibrillation
      - Control rate with:
        - β-Blocker IV or digoxin IV
        - Amiodarone 300 mg IV 20-60 min; then 900 mg over 24 h
        - Continuous infusion of amiodarone should be administered via a central venous catheter using an appropriate infusion device
      - Probable re-entry PSVT:
        - Record 12-lead ECG in sinus rhythm
        - If recurs, give adenosine again & consider choice of anti-arrhythmic prophylaxis
      - Possible atrial flutter
        - Control rate (e.g., β-Blocker)
  - No
    - Seek expert help

*Attempted electrical cardioversion is always undertaken with sedation or general anaesthesia
ATRIAL FLUTTER

Characterised by rapid regular or irregular narrow QRS complexes with a saw-tooth appearance to the baseline. A regular, narrow complex (unless BBB pattern present) tachycardia of 150 bpm should be suspected to be atrial flutter with a 2:1 block irrespective of whether or not flutter waves are obvious on the ECG.

Management

- Carotid sinus massage/vagal manoeuvres may slow the ventricular response revealing underlying flutter waves and assisting the diagnosis.
- **Adenosine** may also be used to help assist the diagnosis by slowing AV conduction and revealing flutter waves. Rapid IV bolus of 6mg followed by saline flush, up to 12mg IV a total of twice at 1-2 minute intervals may be given if tolerated. **Do not use in asthmatics** (bronchoconstriction) or those taking dipyridamole, carbamazepine or with denervated (transplanted) hearts (effects prolonged and potentiated). Administration may be accompanied by flushing and/or chest tightness but the half life is short (20 seconds) with clinical effects resolving in about 2 minutes. **WARN THE PATIENT.** Always run an ECG rhythm strip during administration. Adenosine is contra-indicated in 2nd or 3rd degree AV block.
- Atrial flutter tends to be sustained and does not respond readily to AV node blocking drugs. Therefore, every patient with persistent atrial flutter should be considered for early cardioversion.
- For immediate management consider using the management guideline for atrial fibrillation.
- Note that IV Flecainide cardioversion should **NOT** be used for atrial flutter. It can slow the flutter rate and cause a paradoxical rise in the heart rate to >200bpm. (Increased rate of conduction through a-v node).

SUPRAVENTRICULAR TACHYCARDIA

Characterised by regular narrow QRS complexes.

Three types exist:

2. **AV re-entry tachycardia** (the tachycardia associated with WPW). Also presents in young adults. Inverted ‘P’ waves may be seen after the QRS and a pseudo-RBBB pattern in V1.
3. Atrial tachycardia due to enhanced automaticity in an atrial focus. ‘P’ waves visible before the QRS but with abnormal P wave morphology.
BROAD COMPLEX ARRHYTHMIAS

VENTRICULAR PREMATURE BEATS - VPBs
Ventricular premature beats occurring in the early phase of acute myocardial infarction, are common and are not in themselves predictors of serious ventricular arrhythmias. However, in the presence of frequent VPBs combined with significant left ventricular impairment (ejection fraction <35%) consider a 24 hour ECG recording prior to discharge to exclude non-sustained VT.

IDIOVENTRICULAR RHYTHM
Characterised by regular, broad complex arrhythmia at a rate <120 bpm (a rate >120 bpm indicates VT). It is common during reperfusion after thrombolysis.
- Idioventricular rhythms are usually self-terminating and do not require anti-arrhythmic therapy.

BROAD COMPLEX TACHYCARDIA
Treat as ventricular tachycardia until proven otherwise. Characterised by regular broad QRS complexes >120 bpm. Differentiation between VT and SVT with a bundle branch block is aided by the diagnostic algorithm.

Diagnosis
- Where possible compare previous ECGs in sinus or previous arrhythmia.
- In a patient with previous myocardial infarction, IHD, cardiomyopathy, age >60 years, a broad complex tachycardia is nearly always ventricular in origin.
- Adenosine may be used in an effort to assist diagnosis.

Do not use verapamil if VT is not excluded. It can cause haemodynamic collapse or asystole.

Management
- See algorithms
- Treatable factors should be identified e.g. persistent cardiac failure, hypokalaemia, hypomagnesaemia.
- Pro-arrhythmic effect of anti-arrhythmic drugs or inotropic agents may necessitate their reduction or cessation.
- Occasionally mechanical causes are responsible e.g. central lines or pacing wires.

Seek senior cardiology advice regardless of the time of the day or night and before proceeding further through the algorithm.
DIAGNOSIS OF BROAD-COMPLEX TACHYCARDIA

Broad complex Tachyarrhythmia

Regular

Clinical evidence of AV dissociation

Yes

No

ECG shows AV dissociation, blocked VA conduction, capture or fusion beats?

Yes

No

Is there an ECG showing sinus rhythm available?

Yes

No

Assume patient does not have bundle branch block when in sinus rhythm

Remember to reapply algorithm after restoring sinus rhythm

Narrow QRS <120ms?

Pre-excitation

Bundle branch block?

Ventricular Tachycardia

Yes

No

Is QRS >140ms during tachycardia?

Consider Intracardiac study

Is QRS morphology unchanged during tachycardia?

Yes

No

Ventricular Tachycardia

Yes

No

Is axis <-30° during tachycardia?

Has axis altered to <-45° during tachycardia?

Yes

No

Ventricular Tachycardia

Yes

No

Is QRS >180ms during tachycardia?

Yes

No

Ventricular Tachycardia

Are there previous records of broad complex tachycardia with different morphologies?

Yes

No

Is there ventricular concordance?

Is there Rsr' in V1 or QS or rS in V6 in patients with right bundle branch block configuration?

Yes

No

1. Detailed analysis of morphology
2. Oesophageal/right atrial electrode
3. Intracardiac study
• **Maintenance anti-arrhythmic therapy** following restoration of sinus rhythm depends on the arrhythmia substrate for SVT prophylaxis. VT prophylaxis centres around the use of beta-blockers and/or amiodarone. Some patients may require ICD implantation. Long-term management of these patients should always be discussed with a consultant cardiologist.

• Most anti-arrhythmic drugs can cause sinus bradycardia or AV block. For patients with impaired LV function, beta-blockers should be introduced at a low dose (e.g. bisoprolol 1.25-2.5mg daily) and titrated gradually. Amiodarone is effective for VT treatment and prophylaxis in these patients. **Class Ic drugs such as flecainide and propafenone are contraindicated in heart failure/LV impairment.**

• Overdrive pacing may be considered for resistant or recurrent arrhythmias.

VT or VF are commonly triggered within the first 48 hours of acute MI. In this situation recurrence after the acute event is uncommon and no specific prophylaxis is needed. VT or VF occurring more than 48 hours after acute MI is more sinister; this may indicate the development of a chronic arrhythmia substrate. These patients need assessment with a view to revascularisation and either anti-arrhythmic drug treatment or an ICD.

**Do not hesitate to seek a senior cardiology opinion in the case of troublesome dysrhythmias - ‘cocktails’ of anti-arrhythmics cause more problems than they solve.**

### TORSADE-DE-POINTES TACHYCARDIA

Characterised by rapid, broad QRS complexes twisting around the baseline giving the appearance of changing QRS morphology and axis. It is a form of polymorphic VT and **can be mistaken for VF. It is often self terminating and recurrent.** Its recognition is important because the aetiology and treatment differs from monomorphic VT.

**Diagnosis**

Consider Torsade when the following are present:

• Polymorphic VT.

• Prolonged QT interval.

• Initiation of tachycardia with long-short coupling intervals.
Causes

- Bradycardia
- Electrolyte disturbances - hypokalaemia/hypomagnesaemia/hypocalcaemia.
- Tricyclic antidepressants.
- Certain anti-psychotics (e.g. thioridazine).
- IV erythromycin.
- Antihistamines (e.g. terfenadine).
- Anti-arrhythmic drugs - amiodarone, sotalol, disopyramide, procainamide etc.
- Myocardial ischaemia.
- Inherited long QT syndrome (may be family history of syncope, sudden death or “epilepsy” in association with any of the above).

Management

- The primary treatment of drug induced Torsade is intravenous magnesium infusion
- Withdraw any drug known to prolong QT interval
- Consider the use of temporary atrial or ventricular pacing.
- Intravenous isoprenaline (2.25 mg isoprenaline sulphate in 500 mL 5% dextrose infused at 10-30ml per hour) is an effective short-term treatment. Use with caution in patients with angina or heart failure, and discuss management with cardiologist.

VENTRICULAR FIBRILLATION

Characterised by a chaotic electrical pattern with no discernible cardiac rhythm.
Follow cardiac arrest algorithm.
MANAGEMENT OF BRADYARRHYTHMIAS

SINUS BRADYCARDIA, JUNCTIONAL RHYTHM

Characterised by rate <60 bpm, ‘P’ wave present (sinus brady). ‘P’ wave inverted or buried within or after QRS in the case of junctional rhythm.

Management - see bradycardia algorithm. (Always consider and treat the cause e.g. hypothermia, drugs, ↑ intracranial pressure, hypothyroidism etc).

FIRST DEGREE ATRIOVENTRICULAR (AV) BLOCK

Characterised by one ‘P’ wave per QRS but with a PR interval >0.2 secs, is not uncommon especially after inferior MI. Those on beta-blockers may have an acceptable, marginal first degree AV block.

If a prolonged PR interval is associated with either new bifascicular block (RBBB +LAD or RBBB +RAD) or with complete LBBB DISCUSS WITH A CARDIOLOGIST, as a prophylactic pacing electrode may be required.

SECOND & THIRD DEGREE (COMPLETE) AV BLOCK

Second degree heart block

- Type I (Wenckebach) - characterised by lengthening PR interval with each successive beat until failure of conduction of the atrial impulse through the AV node occurs; this tends to occur in a cyclical pattern.
- Type 2 (usually 2:1 block) - characterised by a constant PR interval and the sudden failure of conduction of an atrial impulse through the AV node; this tends to occur in a cyclical pattern.

Third degree heart block

- Characterised by complete dissociation of atrial and ventricular activity with all atrial impulses blocked within the conducting system.

In inferior infarction only requires treatment if associated with hypotension, syncope, cardiac failure or ‘escape’ ventricular rhythms. Initially, IV atropine 500 micrograms should be given and repeated, if necessary, up to a maximum of 3mg. If AV block recurs a temporary pacing electrode should be inserted. AV block associated with inferior MI usually resolves within 10 days, therefore permanent pacing is not normally necessary.
BRADYCARDIA ALGORITHM
(includes rates inappropriately slow for haemodynamic state)

Adverse signs?
- Systolic BP <90mm Hg
- Heart rate <40 beats min⁻¹
- Ventricular arrhythmias compromising BP
- Heart failure

Yes

Atropine
500 mcg IV

Satisfactory Response?

Yes

Interim measures:
- Atropine 500 mcg IV repeat to maximum of 3 mg
- Adrenaline 2-10 mcg min⁻¹
- Alternative drugs*
- Transcutaneous pacing

No

Risk of asystole?
- Recent asystole
- Mobitz II AV block
- Complete heart block with broad QRS
- Ventricular pause>3s

Yes

Seek expert help
Arrange transvenous pacing

No

Observe and Monitor

*Alternatives include:
- Aminophylline
- Isoprenaline
- Dopamine
- Glucagon (if beta-blocker or calcium-channel blocker overdose)
- Glycopyrronium can be used instead of atropine

In anterior infarction the development of second degree or complete heart block is an indication for insertion of a temporary pacing electrode. A permanent DDD system may be required before discharge.

- Transcutaneous pacing can be used as a “safety net” until a pacing wire has been inserted.

If bradycardia is a “secondary” phenomenon treat the cause hypothermia, hypothyroidism
EXTERNAL CARDIAC PACING

GET HELP

Main Indications (as below pending transvenous pacing)

- Complete heart block.
- Ventricular standstill.
- Symptomatic bradycardia unresponsive to atropine.
- Risk of asystole: see algorithm.

Equipment

- External pacing defibrillators are located in:
  WGH:- ARAU, HDU’s Ward 21, Ward 26 and some other wards.
  RIE:- A&E, ICU, CCU Ward 114 and some other wards.
  SJH:- A&E, CCU (spare machines are located in the resuscitation department and Medical Physics).

See current cardiac arrest trolley list for full location list.

Method

- Appropriate gel pads are applied to the chest in the defibrillator paddle sites on front and back. There is a diagram on the outside of the bag in which they are provided.
- The ECG electrodes from the defibrillator monitor must be attached to the patient or it will not pace.
- The starting default settings which appear when you press the on button are below.
- Mode is demand: don’t change this.
- Rate 70bpm: can be increased or decreased to achieve the optimal haemodynamic condition.
- Power: 30mA: can be increased to gain capture. The lowest level which is effective should be selected.
- External pacing can be uncomfortable, painful and distressing. Titrate IV morphine for comfort and add 0.5-1mg midazolam if distressed. Be careful with this potentially destabilising combination. Monitor continuously (ECG, SpO$_2$ and BP) and re-assess frequently.

INTRAVENTRICULAR OR BIFASCULAR BLOCK

Right bundle branch block plus left or right axis deviation (-30° or > +90°) constitutes bifascicular block. Following anterior myocardial infarction, unless known to be long-standing, it is an indication for considering insertion of a temporary pacing electrode. If more severe conduction abnormalities develop i.e.: second or third degree AV block a permanent
pacemaker is indicated prior to discharge. New left bundle branch block associated with first degree heart block should be treated similarly.

INTRA-AORTIC BALLOON PUMP (IABP)

May be useful in severe acute valvular disease, in severe unstable angina or in cardiogenic shock. Prior to insertion there should be a clearly agreed clinical management strategy. Discuss with senior cardiologist.

SPECIFIC DRUG POINTS

A full account of all drugs mentioned in the schedule is available in the BNF, which should be consulted. Further detail in CCU Therapeutic Schedule.

ACE-INHIBITORS

Reduce mortality after AMI by approximately 20-30%.
Most frequently used drugs: ramipril, lisinopril, enalapril.

- Following AMI, therapy is normally commenced when the patient is stable, within 24-36 hours after the acute event.
- Where significant hypotension might occur (e.g. pre-existing hypotension, reno-vascular disease), a test dose of captopril 6.25mg is normally used. The blood pressure and pulse should be monitored every 30 minutes for 2 hours following this.
- Where hypotension is unlikely to be a problem, low dose ramipril, lisinopril or enalapril are equally appropriate as initial therapy.
- It is important to titrate ACE inhibitors to appropriate doses as used in clinical trials - lisinopril 10mg od, ramipril 5mg bd (especially if signs of heart failure present), enalapril 10-20mg bd.
- Effects of potassium supplements or potassium sparing diuretics should be monitored closely by checking plasma potassium and adjusting prescription accordingly.

BETA BLOCKERS

Reduce mortality after AMI by ~25%

- Contra-indicated in asthma.
- Prescribe with caution in COPD.
- Stable chronic peripheral vascular disease is NOT a contra-indication.
- Beta blockers should also be considered in patients with heart failure associated with AMI once stabilised.
DIGITALIS TOXICITY

Digitalis toxicity is most often associated with bradycardia, ventricular bigeminy, paroxysmal atrial tachycardia (often with variable AV block), accelerated idioventricular rhythm and AV block, but almost any arrhythmia can be provoked. Visual disturbance, anorexia, nausea and vomiting are also common features - level should be measured.

CONTACT THE POISONS BUREAU AT RIE FOR ADVICE: 0131 242 1389.

ANTI-PLATELET THERAPIES

Clopidogrel

The CURE (Clopidogrel in Unstable Angina to Prevent Recurrent Ischaemic Events) study, demonstrated that initiating oral therapy with clopidogrel early, and continuing its use long-term, reduces the relative risk of heart attack, stroke and cardiovascular death by 20% in patients with acute coronary syndromes (unstable angina and non-Q wave myocardial infarction).

Indications: Patients with non-ST segment elevation myocardial injury (including non-Q MI) and unstable angina with ↑ troponin or ECG changes - 300mg loading followed by 75mg daily for 12 months + aspirin 75-300mg.

Following insertion of intra-coronary stent in elective PCI (300mg loading dose followed by 75mg daily for 1 month combined with aspirin 75-300mg daily thereafter).

Cautions: Check FBC 7 days after initiation of combination therapy.

N.B. STOP treatment at least 7 days prior to major surgery including coronary by-pass.

Integrilin (Eptifibatide)

Indications: acute coronary syndromes (i.e. unstable angina or non Q-wave MI) regardless of whether they ultimately undergo PCI.

Tirofiban

Tirofiban is a non-peptide inhibitor of the platelet glycoprotein (GP) IIb/IIIa receptor, the final common pathway for platelet aggregation.

Abciximab (REOPRO)

• Indications: in association with high risk percutaneous coronary intervention.

• Abciximab: (c7E3) is a monoclonal antibody directed against the GP IIb/IIIa receptor.

For details of pharmacology, dosing and administration please refer to the Coronary Care Unit Therapeutic Schedule.
Acute aortic dissection is a medical emergency and should be investigated and treated with the same urgency as acute MI. Always discuss management with a senior cardiologist at first opportunity. Risk factors are hypertension, Marfans syndrome, pregnancy, aortitis, coarctation of the aorta.

Presenting features include:
- Very severe anterior chest pain.
- Very severe posterior chest pain.
- Ischaemic syndromes - coronary, cerebral, upper limbs, renal, lower limbs.
- Syncope
- Cardiac tamponade.
- Acute aortic regurgitation.
- Unequal limb pulses and blood pressures.

Management
- Oxygen 60-100%
- IV access
- IV opiate analgesia and anti-emetic therapy
- Transfer to CCU if imaging assessment is delayed
- Urinary catheter
- Transfer immediately to cardiac surgery if diagnosis of Type A dissection is confirmed. Mortality is as high as 5% per hour.
- Intensive BP control with IV beta-blocker, such as labetolol. In patients with contraindication to beta-blocker, IV verapamil should be used. Additional vasodilators may need to be considered such as IV GTN or sodium nitroprusside. Target blood pressure should be to maintain systolic blood pressure <120 mmHg.

Get expert help.

Investigations
- CXR - look for widened mediastinum, pleural effusions.
- ECG - may show ischaemia
- Transthoracic echocardiography as a rapid initial evaluation for type A dissections but has low sensitivity and cannot be relied upon.
• Emergency multislice computed tomography, magnetic resonance imaging or transoesophageal echocardiography should be considered to diagnose and define the extent of the dissection.

**Early mortality in acute aortic dissection of the ascending aorta is 10% per hour. Surgery can be life saving and should not be delayed. Never transfer a patient for surgery without adequate BP control with IV therapy.**

---

**INVESTIGATION ALGORITHM FOR ACUTE AORTIC DISSECTION**

- **Suspected acute aortic dissection**
  - Transthoracic echocardiography
    - **Aortic dissection confirmed**
      - Involves ascending aorta (type A)
        - Urgent surgical opinion
    - Dissection limited to arch and/or descending aorta (type B)
      - **Conservative management**
    - Echo normal or inconclusive
      - Further non-invasive imaging TOE, CT or MRI
Stroke is an acute stroke integrated care pathway should be initiated immediately on arrival at hospital. It will guide you through the initial management of the patient. If there is a known time of onset of symptoms, the patient presents within **2.5 hours** of onset and there are no contraindications to thrombolysis, ring the stroke team immediately for consideration for immediate thrombolysis. If onset within 5.5 hours, ring stroke team immediately for consideration for randomisation into IST 3 (thrombolysis trial).

During day, bleep the WGH Stroke SpR page 8699, out of hours the Neurology registrar.

Service at RIE from 9-5.

At SJH page Stroke Liaison Nurse on 986.

In any patient presenting with an apparent stroke, your management should centre on answering the following questions:

- Where is the brain lesion?
- Has this patient had a vascular event or not?
- If this is a vascular event is it a haemorrhage or an infarct?
- Why has this patient had a stroke?
- What are this patient’s problems?

**MANAGEMENT**

- If possible, all patients admitted with an acute stroke should be directed to the Acute Stroke Unit in Ward 55 WGH or Ward 101 in RIE and at SJH to the Medical Admissions Unit in wards 23 & 24 or to CCU if thrombolysis is being administered.
- Perform standard acute initial assessment to ensure that the patient is maintaining an adequate airway, is breathing and has an adequate circulation.
- Check swallowing prior to allowing free fluids. Nursing staff on the acute stroke units have a protocol for swallowing assessment. A formal swallowing assessment may be organised within normal working hours by contacting a speech and language therapist on bleep 5221 WGH, ext. 21967 RIE or at SJH SALT on ext 54191.
- If there are doubts about the patient’s ability to swallow safely the patient should be placed nil by mouth and given intravenous fluids.
• Urinary catheterisation should be avoided in the acute phase unless there is urinary retention, a high risk of pressure sores or unless the urine output needs to be monitored. Discuss with Nursing staff.
• DVT Prophylaxis: avoid Heparin.

INVESTIGATION
• Immediate BM to exclude hypoglycaemia
• U&E’s, and glucose
• LFTs, cholesterol
• FBC, ESR
• ECG
• CXR

CT scan should be requested immediately in all patients. These can normally wait until the next day.

• If clinical evidence of a cardiac source of embolism e.g. atrial fibrillation or significant cardiac murmur, request an echocardiogram.
• If the patients has had a minor stroke or TIA affecting the carotid territory arrange a carotid duplex to screen for significant carotid stenosis.

INDICATIONS FOR IMMEDIATE CT SCANNING
• Coma or reducing conscious level.
• Likelihood of important non-stroke diagnosis (e.g. subdural haematoma, subarachnoid haemorrhage).
• Patient on or requiring anticoagulants.
• Patient eligible for thrombolysis.
• Unusual presentation? Basilar artery thrombosis.

STROKE DIFFERENTIAL DIAGNOSIS: CONDITIONS WHICH MAY MIMIC OR BE MIS-DIAGNOSED AS STROKE

Toxi-metabolic derangements
• Hypoglycaemia
• Alcohol intoxication
• Drugs e.g. tricyclic antidepressants
• Hyponatraemia

Other CNS disease
• Meningitis/encephalitis
• Subarachnoid haemorrhage
• Sub-dural haematoma
• Todd’s paresis
• Tumour
• Cerebral vasculitis
• All patients with stroke who have not been admitted to the stroke units should be notified to Professor Dennis or Dr Keir (WGH) or Drs Hart, Mead, Chapman or Coull ext 26927 (RIE) or the stoke unit on ext 54104 or stoke liaison nurse on bleep 986 (SJH) as early as possible.
• Once a cerebral haemorrhage has been excluded (by CT brain), a single dose of aspirin should be prescribed at a dose of 300 mg oral od. If patient cannot swallow give by suppository. After that 75mg per day should be given (minimise GI side effects).
• Thrombolysis is available 24/7 at WGH and 9-5 at RIE and SJH. Contact Stroke Registrar or Consultant via appropriate switchboard.
• If already on aspirin leave on aspirin until CT result known.
• For patients with ischaemic stroke dipyridamole MR 200mg daily should be given in addition to aspirin.
• If the total cholesterol is greater than 3.5mmol/l start patient on simvastatin 40 mg nocte (consider pravastatin if on warfarin and digoxin). See Lothian lipid guidelines.
• Antihypertensive medication may be continued if the patient is able to swallow.
• New antihypertensive medication should not be initiated within the first week of an acute stroke unless there is accelerated phase hypertension.
• After the first week blood pressure lowering with an ACE inhibitor and thiazide diuretic should be considered even if blood pressure is “normal”.
• Patients with a proven ischaemic stroke who are in atrial fibrillation should be considered for anticoagulation after 2 weeks.
• If AF is symptomatic (e.g. palpitations or breathlessness) consideration should also be given to subsequent chemical or electrical cardioversion.
• All patients with ischaemic stroke who are shown to have a severe stenosis (>70%) of the ipsilateral internal carotid artery on the carotid duplex should be referred to Professor Dennis or Dr Keir (WGH), Dr Chapman or Dr Hart (RIE) or Dr S Ramsay (SJH) on ext 53846 for further consideration of carotid endarterectomy.
ICP for Acute Admission following **SUSPECTED STROKE** v3.5, post mdt 25/08/06

**CRITERIA:**

**Initiate for ALL PATIENTS ATTENDING with a SUSPECTED STROKE**

| Date initiated: | / / |

**Site:** St. John’s, RIE, WGH

| Unit number: | CHI |

**INSTRUCTIONS:** Insert information into appropriate spaces as required.

Circle Y or N to indicate status of patient or your actions. Do not initial until actually done!

This ICP is an immediate action checklist & a clinical record, & also requires a Kardex & SEWS chart.

### PHASE 1 - IMMEDIATE PATIENT ASSESSMENT – complete with Y or N

**FAST criteria:**

- **Facial weakness:** [ ] Can the person smile?
- **Arm weakness:** [ ] Can the person raise both arms?
- **Speech problems:** [ ] Can the person speak clearly & understand what you say?

Test all 3: If NO to any question, a Stroke is probable, continue with ICP. If YES to all three, continue with full Medical Clerking & devise a Medical management Plan.

**GLASGOW COMA SCALE:** on arrival: EYES ........, MOTOR ........, SPEECH ........ : total ........

- Airway: Is airway compromised & / or GCS < 9
  - N Y IF YES, d/w HDU/ICU time: .................

- Blood Pressure: SBP >210 or <90
  - N Y IF YES, control hyper- / hypo-tension

- Cardiac rhythm: In Atrial Fibrillation?
  - N Y IF YES, control heart rate

- Oxygenation: Is $O_2$ sats. < 95%
  - N Y IF YES, prescribe $O_2$ & check ABG

**CT BRAIN SCAN:**

- Date: / / 
- Time: _ : ___ hrs

**RESULT:**

- Date: / / 
- Time: _ : ___ hrs

Arrange scan NOW (completed a request card & sent to X-Ray dept. Y / N )

- Intracerebral Haemorrhage N Y IF YES, if INR>1.4 - reverse anticoagulation NOW (NHSL VTE Guideline) & STOP all antithrombotics

- Infarction N Y IF YES, give Aspirin 300mg stat oral/pr NOW (must be given < 48 hrs of presenting to hospital) & continue with 75mg daily Clopidogrel only if Aspirin allergic

- Posterior Fossa bleed, Hydrocephalus, or Malignant MCA infarct?
  - N Y d/w Stroke consultant [bl ......] NOW

- SAH N Y IF YES, d/w Neurology Sp.Reg in DCN give Nimodipine 60mg oral 4hrly [iv if no swallow]

☐ once REQUESTED these ROUTINE INVESTIGATIONS (if in bold to be performed in all patients)

**FBC**

- If > 50mmHg N Y: IF YES, consider endocarditis or arteritis

**ESR**

- If urea raised N Y: IF YES, adjust fluid regime

**U & E’s, LFT’s**

- If LFT > x N Y: IF YES, do NOT prescribe a statin

**Random Glucose**

- If >7.0mmol/l N Y: IF YES, arrange fasting glucose

**ECG**

- If in AF N Y: IF YES, control HR

**Chest X-ray**

- Initial
ICP for Acute Admission following **SUSPECTED STROKE** v3.5, post mdt 25/08/06  
NHS Lothian

**Phase 2 – planning for transfer to ward**

### IF YES TO ANY QUESTION

<table>
<thead>
<tr>
<th>Question</th>
<th>N</th>
<th>Y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drowsy or unsafe swallow</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Prescribe IV 0.9% Saline depending on state of hydration. Avoid Dextrose in first 48hrs: 2/4/6 rule</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temp &gt; 37.5°C</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Prescribe Paracetamol 1g 4-6 hrly oral / IV / PR</td>
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<tr>
<td>Take blood cultures, look for &amp; treat infection</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood Sugar (4hrly BM’s)</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Glucose &gt;11mmol/L</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Prescribe dextrose / potassium / insulin infusion (see GKI ICP)</td>
<td></td>
<td></td>
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<tr>
<td><strong>DVT prophylaxis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not mobilising independently</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Consider CLOTS trial enrolment (unless PVD, neuropathy or ulcers). Avoid Heparin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swallow screen</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Swallow screen failed</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Referral to S &amp; L T., consider need for medications, fluid &amp; food</td>
<td></td>
<td></td>
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<tr>
<td>Contineneca</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Incontinent of urine</td>
<td>N</td>
<td>Y</td>
</tr>
<tr>
<td>Avoid urinary catheterisation unless renal failure, skin broken or acute urinary retention</td>
<td></td>
<td></td>
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<tr>
<td>Positioning</td>
<td></td>
<td></td>
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<tr>
<td>Perform Moving &amp; Handling Assessment</td>
<td></td>
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</tr>
<tr>
<td>Nurse 30º Head-up if drowsy or NG fed, &amp; Physio referral prior to transfer (if not for transfer on bed)</td>
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<td></td>
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<tr>
<td>Nutritional screen</td>
<td></td>
<td></td>
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<tr>
<td>Complete nutrition screen</td>
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<td></td>
</tr>
<tr>
<td>Document weight</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consider Modified diet or NG feeding at 24 hrs and referral to Dietitian</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clinical Trials</td>
<td></td>
<td></td>
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<tr>
<td>consider: CLOTS - Y / N/A</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trial C - Y / N/A</td>
<td></td>
<td></td>
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<tr>
<td>Trial B - Y / N/A</td>
<td></td>
<td></td>
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<tr>
<td>Trial D - Y / N/A</td>
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</tbody>
</table>

### IF ISCHAEMIC STROKE

- Carotid Duplex scan  
  - If TIA or minor non-disabling stroke & considering endarterectomy

- Transthoracic Echocardiogram  
  - If in AF, recent MI, cardiac murmur or bilateral infarcts  
  - Bubble contrast echo if <55 yrs

- 24 Hour ECG  
  - If arrythmia suspected

- Secondary prevention  
  - 75mg Aspirin, once daily + 200mg Dipyridamole, twice daily
  - If ischaemic & total chol>3.5mmol/l  
  - prescribe Simvastatin* 40mg first choice

* (Pravastatin if already stabilised on warfarin, as Simvastatin interacts)

### IF <55yrs old consider the following investigations

- Lupus anticoagulant  
  - 3 Green & 1 Red tubes to Haematology RIE
  - sent , initial

- Auto-antibody screen  
  - 1 White tube to Immunology RIE
  - sent , initial

- Syphilis serology  
  - 1 White tube to Microbiology
  - sent , initial

- Fasting Homocysteine  
  - 1 Red tube to Royal Hospital for Sick Children Biochemistry
  - sent , initial

- Trans-oesophageal Echo  
  - d/w stroke consultant
  - sent , initial

**TRANSFER** to ward & initiation of ‘Continuing Stroke care’ ICP: date ....../ ........./ ......... time .... : ......

<table>
<thead>
<tr>
<th>Print name</th>
<th>Designation</th>
<th>Initials</th>
<th>Signature</th>
<th>date</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>5</td>
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</tr>
</tbody>
</table>
Acute Stroke Patient

**FAST Protocol**

- **Page Acute Stroke Team**
  - Stroke Nurse
  - SGR
  - KJ

- **Time of Onset <3hrs**
  - Yes
    - Suitable for Thrombolysis?
      - Yes
        - Urgent CT Request
          - Alert CCU / Bed Manager
      - No
    - No
  - No
    - Probable TIA
      - If TIA:
        - Refer to Neurovascular OPC
        - Discharge with TIA Pack

- **CT Scan Suitable for Thrombolysis?**
  - Yes
    - Final Decision to give rt-PA
      - Consent / Assent from Patient / Relatives
        - Provide Information Leaflet
  - No

- **Commence rt-PA Bolus**
  - Admit to CCU

- **Acute Stroke Monitoring Protocol**
  - rt-PA Infusion
  - Avoid Aspirin / Heparin / NG Tube / Urinary Catheter
THROMBOEMBOLIC DISEASE

DVT and pulmonary embolism are a spectrum of the same disease and often co-exist. There are about 20,000 deaths per year from thromboembolic disease in the UK. The clinical diagnosis is difficult. It is therefore helpful to follow a process to assess clinical probability and have an agreed investigative pathway which includes:

Recognition of the symptom complex
- Breathlessness
- Pleuritic chest pain
- Cough
- Haemoptysis
- Syncope (usually indicates major PE).
- The symptoms in isolation are not diagnostic and merely help support the diagnosis or differential diagnosis.

Determination of the risk factors for thrombosis (risk increases with age)

<table>
<thead>
<tr>
<th>Major (5-20)</th>
<th>Minor (2-4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery</td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>Oral contraceptive pill</td>
</tr>
<tr>
<td>Orthopaedic</td>
<td>Hormone replacement therapy</td>
</tr>
<tr>
<td>Malignancy</td>
<td>Obesity</td>
</tr>
<tr>
<td>Immobility, e.g. hospital</td>
<td>Travel (&gt;5-6hrs)</td>
</tr>
<tr>
<td>Previous VTE</td>
<td></td>
</tr>
<tr>
<td>FH of VTE</td>
<td></td>
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</tbody>
</table>

Baseline investigations
All patients with suspected pulmonary embolism should have standard bloods, chest X-ray, ECG and arterial blood gases on admission.

The clinical probability of PE can then be determined:

High probability patients (>80% likelihood of PE)
- Risk factor present.
- Unexplained dyspnoea, tachypnoea or pleurisy.
- Unexplained radiographic changes or gas exchange abnormality.

Low probability (<20%)
- No risk factors.
- Dyspnoea, tachypnoea or pleurisy with possible alternative cause.
- **Alternative explanation** for radiographic changes or gas exchange.
**Role of D-dimer**

D-Dimer is helpful if used according to protocol.
It is not a routine screening test and is best used when there is suspicion of PE but this is low probability.
Only a negative result is of value and a Vidas D-dimer test < 500 is negative.

D-dimer tests should not be performed if there is a history of:
- Malignancy
- Recent trauma or surgery
- Active infection
- Pregnancy
- Bleeding

In patients with low probability of PE and a negative D-dimer then PE can be excluded and an alternative diagnosis determined.

**DIAGNOSTIC ALGORITHM**

The commonest tests undertaken are CT pulmonary angiography and perfusion lung scan (occasionally plus ventilation lung scan). In patients with equivocal results particularly from perfusion lung scanning then leg imaging or CTPA should be considered to confirm or refute the diagnosis. CTPA has the advantage of providing an alternative cause for symptoms in a proportion of patients.

Echocardiography in massive PE may help confirm the diagnosis and support treatment stratification to thrombolysis if there is evidence of right ventricular strain.

Pulmonary angiography is now rarely performed and only after consultation with radiology and the Consultant involved with patient care.
The current recommended approach in uncomplicated PE is initiation of LMWH - currently weight adjusted S/C Enoxaparin 1.5 mg/kg once daily and oral warfarin (Fennerty regime). LMWH Heparin should be started immediately.

For patients with a high risk of bleeding eg post major surgery or in renal failure then unfractionated heparin should be considered because of the shorter half life. When unfractionated heparin is used then APTT must be measured regularly according to protocol.

Oral warfarin can be initiated on the first day and heparin should be overlapped for a minimum of 5-6 days and until INR has been therapeutic (>2.0) for 2 days.

Warfarin should be continued for 3 months in patients with PE with a precipitating factor eg surgery and for 6 months in those with idiopathic PE or extensive thromboembolic disease. Target INR is 2.5.

Patients with thrombophilia or recurrent disease should be referred to Haematology for ongoing management.

Thrombolysis should be considered in patients with massive pulmonary embolism after consultation with a Consultant and the patient should be managed in a critical care environment.
Thrombolysis bolus alteplase (rtPA) 10mg IV over 1-2 minutes followed by an infusion of 90mg over 2 hours (max dose 1.5 mg/kg if weight is <65kg). Start IV unfractionated heparin once APTT <2.0 at rate if 1000u/hour. Check APTT after 6 hours and aim for ratio of 1.5-2.5.

In the cardiac arrest or peri arrest scenario 50mg of alteplase can be administered while resuscitation is on-going and attempts to confirm or refute the diagnosis are arranged.

Outlook is bleak but there are individual patients who have survived.

**RUPTURED OR LEAKING ABDOMINAL AORTIC ANEURYSM**

**Presentation**
- Severe back pain or abdominal pain.
- History of collapse (often with brief recovery).
- Hypotension.
- Large pulsatile mass in the abdomen.

**Misdiagnosis**
- Renal colic.
- Acute Pancreatitis.
- Perforated intra-abdominal viscus.

*In older patients presenting with renal colic for the first time or after many years disease free think of AAA.*

**Risk factors**
- Elderly.
- Male sex.
- History of hypertension.

**Initial Management**
- Radiopage the Vascular Registrar (#6440).
- High concentration oxygen; analgesia - titrate IV morphine in 1mg increments.
- IV access in the upper limb (femoral lines should be avoided).
- Low volume resuscitation; aim for a systolic pressure of between 60 to 80mmHg (or enough to maintain consciousness).
- Monitoring ECG, BP and pulse oximetry.
- Bloods including x-match (The Vascular Registrar shall initiate the Major Haemorrhage Protocol).
WGH blood bank phone is 32419.  
RIE blood issue phone is #27501/27502/27503. 
SJH bloodbank phone is 53354

Investigations
- Plain CXR and AXR (loss of psoas shadow, calcified aneurysm).
- Portable ultrasound scan if there is any element of doubt regarding the diagnosis.

(The use of CT scanning to establish the diagnosis of a ruptured/leaking AAA is both time-consuming and unhelpful.)

Prognosis
Overall >75% mortality and 50% operative mortality.

Hardmans Criteria (guide to overall prognosis)
- Age >76
- Loss of consciousness
- Haemoglobin <9
- Creatinine >180
- ECG ischaemia

3 or more of the above on admission indicates a very poor outcome.

Transfer from WGH or SGH
- The patient should be transferred by the most senior middle-grade doctor of the receiving speciality. For patients referred to the waiting Surgeons this will be the SHO or SpR on call for General Surgery. For patients referred to Medicine (e.g. as renal colic) this will be the SHO or Specialist Registrar on call for Acute Medicine.
- If a patient requires transfer to the Royal Infirmary, there should be no delay and all transfers should be ‘blue lighted’ with an appropriate SHO/Registrar.

The only definitive management of these patients is early surgery. It is our aim to maintain cardiovascular stability using low volume resuscitation to allow transfer, but speed is pivotal to good outcome.
MANAGEMENT OF THE ACUTELY ISCHAEMIC LIMB

Presentation
• Pain
• Paraesthesia
• Pulseless
• Power reduced
• Pallor
• Perishing with cold
(all of the above may not be present simultaneously)

Initial Management
• Assessment of the affected and contralateral limb and pattern of pulses.
• IV access and analgesia - morphine titrated.
• Doppler assessment.
• Discuss with Vascular Registrar regarding heparinisation and further investigation.
  (In the absence of paraesthesia, significant pain and loss of power to the limb, many patients may be heparinised and avoid surgical embolectomy).

Investigations
• FBC, U & E’s, CK, Clotting screen and G & S.
• Duplex examination.
• Angiogram.
  (Imaging is avoided if the site of the embolus can be determined clinically)

Further Management
• Correction of cause (if appropriate) e.g. atrial fibrillation

Prognosis
• Depends on ischaemia time and aetiology.

The diagnosis of an acutely ischaemic limb is a surgical emergency. A favourable outcome depends on the speed of limb reperfusion. Call Vascular Registrar #6440.
HYPERTENSION

Hypertension is common and most patients do not require admission. The key to its correct management is a slow reduction, even in hypertensive emergencies. Rapid reduction may result in cerebral infarction. Hypertension immediately following intracerebral events is not uncommon, and may aid cerebral perfusion. It does not usually warrant intervention. Seek expert advice on treatment. See the Lothian Guidelines for the treatment of hypertension.

**Moderate Hypertension: diastolic BP 105-115mmHg**
- Secondary hypertension is rare and should not be pursued unless there are clear clinical or biochemical clues.
- Check fundi, creatinine, ECG for end organ damage.
- Urine dipstick.
- Check for radiofemoral delay, and renal bruits.
- Identify cardiovascular risk factors and treat if necessary e.g. diabetes, hypercholesterolaemia smoking.
- Follow Lothian Hypertension guidelines.
- Most patients will require more than one agent.

**SEVERE: diastolic BP >115mmHg**
- Management is similar to that for moderate hypertension although repeat measurement is less important.
- Patients with accelerated phase (‘malignant’) hypertension should be admitted to hospital.
- They have evidence of end organ damage.
- Atenolol 50mg oral od if no contraindication, an alternative would be Nifedipine LA 30mg orally od. The second drug could be added after 24 hours.
- Ix and Rx as above.

**ENCEPHALOPATHY OR INTRACEREBRAL BLEED**
- Seek expert opinion.
Presentation
Breathlessness, wheeze, chest tightness and cough. Often in a patient known to have asthma, but first episode can occur at any age and may be severe. Onset may be rapid (minutes/hours) or gradual over a few days.

Severe Episode
• Too breathless to complete sentences in one breath.
• Respiratory rate of 25 or more.
• Heart rate of 110 or more.
• PEF <50% of predicted normal or best known (see table in ARAU, A&E and BTS guidelines).
• Oxygen saturation <92% (depends on FiO₂).

Life-threatening Attack
• Unable to talk.
• Sweaty, pale or cyanosed.
• Silent chest on auscultation.
• Feeble respiratory efforts.
• Bradycardia
• Hypotension
• Confusion
• Exhaustion

Management
Immediate treatment with oxygen, salbutamol nebulised with 8l/min oxygen and systemic corticosteroids (see below) should be given during assessment with ABGs etc. Aim for SpO₂ at least 92%. Contact Respiratory Registrar/Consultant IMMEDIATELY.

• High concentration humidified oxygen 60% via mask initially and adjust according to ABG.
• IV access.
• Arterial blood gas on oxygen in all patients with severe asthma (record inspired oxygen concentration).
• Management decisions depend upon clinical state and ABGs.
• Check peak flow and compare to predicted or previous best PEFR: may be too ill to do this.

<table>
<thead>
<tr>
<th>PaCO₂</th>
<th>PaO₂</th>
<th>[H⁺]</th>
<th>SEVERITY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
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<td>Low</td>
<td>Moderate</td>
</tr>
<tr>
<td>Normal</td>
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<td>Severe</td>
</tr>
<tr>
<td>High</td>
<td>Low</td>
<td>High</td>
<td>Life-threatening</td>
</tr>
</tbody>
</table>

Immediately contact Respiratory Registrar/Consultant and alert ICU if ABGs indicate life-threatening attack.

• Salbutamol 5mg nebulised in oxygen 8l/minute, repeated every 10-15 mins if necessary.
• Prednisolone 40mg orally or
• Hydrocortisone succinate 200mg IV (slowly) if unable to take orally.
• If no response give salbutamol 5mg plus ipratropium bromide 500micrograms nebulised in oxygen at 8l/minute.
• If no response to repeated nebulised bronchodilators, Respiratory/Medical Registrar or ICU staff could consider IV magnesium sulphate 2.0g over 20 minutes or IV aminophylline 250mg (maximum 5mg/kg) by controlled infusion over 20 mins followed by a continuous infusion.

Do not use aminophylline without the advice of Respiratory or Intensive Care specialists.

Do not give loading dose of aminophylline to patients on oral therapy. Check the theophylline blood concentration.

Caution: Magnesium is a powerful vasodilator and may cause dangerous hypotension in the hypovolaemic or septic patient.

• Chest x-ray - all severely ill patients. Urgent if clinical signs suggest pneumothorax.
• Calm reassurance throughout is highly beneficial.
• U&Es, FBC, 12 lead ECG should be performed.

ASSESSMENT OF RESPONSE

Clinical Improvement
• Less distressed.
• Decreased respiratory and heart rates.
• Able to talk in sentences.
• Louder breath sounds on auscultation (may be more wheezes).
• Arterial blood gases must be repeated within 30 minutes if no or poor response to treatment.

ABGs should be regarded as a monitor and repeated early and again if required.

• Pulse oximetry may be used to assess response in patients who have clinically improved and did not have a high PaCO₂ initially. Aim for SpO₂ >92%.
• PEF: repeat 15 and 30 minutes after starting treatment.
• Monitor heart rate and oxygen saturation continuously and measure blood pressure frequently.

Contact ICU if:
• Deteriorating or not improving.
• ABG worsening.
• Exhaustion
• Confusion, drowsiness, coma.

Transfer to ICU if respiratory acidosis worsens or develops in spite of treatment.

Further Management if Improved
• Continue oxygen titrating concentration against saturation/PaO₂. Aim for SpO₂ >92%.
• Continue prednisolone 40mg daily orally.
• Regular nebulised salbutamol e.g. 2.5mg-5mg 4 hourly +/- ipratropium 6 hrly.
• If immobile thromboprophylaxis with subcutaneous heparin should be given: see local protocols.

COMMUNITY-ACQUIRED PNEUMONIA

IMMEDIATE MANAGEMENT GUIDELINES

Definition - Acute lower respiratory infection with recently developed radiological signs.

Diagnosis

Symptoms
• Specific: dyspnoea, chest pain (peripheral/pleuritic or dull central), cough, sputum (often absent early), wheeze.
• General: malaise, fever, rigors, myalgia.
Signs

- Tachypnoea
- Tachycardia
- Focal signs: dullness, crackles, bronchial breathing, pleural rub.
- Cough
- Sputum (mucopurulent, rusty or bloodstained).
- Cyanosis

### Likely Underlying Causes

<table>
<thead>
<tr>
<th>Type</th>
<th>Organism</th>
<th>Approx. % cases</th>
</tr>
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<tbody>
<tr>
<td>Typical</td>
<td><em>Streptococcus pneumoniae</em></td>
<td>31</td>
</tr>
<tr>
<td></td>
<td><em>Haemophilus influenzae</em></td>
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<tr>
<td></td>
<td>Influenza virus</td>
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<td><em>Staphylococcus aureus</em></td>
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<td></td>
<td>Gram negative eg Klebsiella</td>
<td>2</td>
</tr>
<tr>
<td>Atypical</td>
<td><em>Mycoplasma pneumonia</em></td>
<td>10</td>
</tr>
<tr>
<td></td>
<td><em>Chlamydia psittica</em></td>
<td>4</td>
</tr>
<tr>
<td></td>
<td><em>Legionella pneumophila</em></td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No organism found</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(most probably pneumococcal)</td>
<td>35</td>
</tr>
</tbody>
</table>

Travel history and animal contact may point to less common pathogens: seek advice from Respiratory/ID/Microbiology.

### Assessment of Severity: CURB 65

Prognostic indicators (on admission) of high mortality:

- **C** - new onset confusion.
- **U** - Urea over 7 mmol/l.
- **R** - Respiratory rate 30/minute or above.
- **B** - BP systolic <90mmHg and diastolic <60mmHg.
- **Age ≥65**

Two or more of these gives 36 x risk of death: predicts requirement for Intensive Care or High Dependency Care.

- Co-morbidity.
- Multilobar involvement.
- Atrial fibrillation.

If none of these, consider outpatient treatment.
IMMEDIATE INVESTIGATIONS

- ABG (record inspired oxygen concentration).
- CXR
- FBC, urea & electrolytes.
- Blood cultures.
- Sputum culture.
- If atypical pneumonia suspected, serum for antibodies, urine for Legionella antigen.
- Throat swab (for virology in viral transport medium)

INITIAL TREATMENT

- **Oxygen** - high concentration is normally safe in pneumonia; use enough to relieve hypoxaemia. (monitor arterial $pCO_2$ in patients with pre-existing COPD and in those worsening).
- **IV fluids** to correct hypovolaemia and total body fluid deficits and prevent renal dysfunction.

**Antibiotics: should be started immediately and related to likely organism and severity of illness.**

- Amoxicillin 500mg orally tds suitable for those with CURB-65 score 0-2 who would have been sent home but for social or other reasons and for elderly patients (as atypical organisms uncommon).
- If not seriously ill but unable to tolerate oral medication: amoxicillin 500 mg tds IV.
- If penicillin allergy: Clarithromycin 500 mg bd oral or IV.
- If severely unwell Ceftriaxone 2g od IV plus Clarithromycin 500mg bd IV.
- If Staphylococcus is cultured or suspected Flucloxacillin 1-2g 6-hourly IV plus Clarithromycin 500mg 12-hourly IV.
- If Mycoplasma or Legionella is suspected Clarithromycin 500mg 12-hourly orally or IV or
- If Legionella is suspected Rifampicin 600mg 12-hourly IV in severe cases.

Severe CAP

- Clarithromycin 500mg 12-hourly IV or
  - Co-amoxiclav 1.2g 8-hourly IV or
  - Ceftriaxone 1-2g daily IV or
  - Cefuroxime 1.5g 8-hourly IV or
  - Amoxicillin 1g 6-hourly IV plus flucloxacillin 2g 6-hourly IV
Clarithromycin must be diluted to 250ml in 5% dextrose or 0.9% saline and given IV over 1 hour.

Signs and symptoms do not reliably distinguish pneumococcal pneumonia from “atypical” pathogens Legionella or Mycoplasma. Therefore patients with severe pneumonia CURB 65>3 should receive dual therapy. Dual therapy is not necessary in mild illness.

- If the patient has already had antibiotics from GP find out what and for how long. May need to modify treatment accordingly.
- If patient has unusual travel or animal contact history, seek advice from microbiology or ID.

Primary resistance to amoxicillin (amoxicillin) in S pneumoniae is very rare in Lothian (<1%). H.Influenzae causes relatively few cases and beta-lactamase resistance in this organism remains uncommon (around 7%), so the routine initial use of antibiotics stable to beta lactamase (e.g. Co-amoxiclav) is not justified.

- Intravenous therapy is expensive in materials, nursing and medical time and should only be used when oral therapy cannot be taken and in severely ill.

Consult Respiratory Registrar regarding appropriate further management and transfer to Respiratory Unit care.

**REASON FOR FAILURE OF TREATMENT**

- Incorrect diagnosis.
- Incorrect antibiotic, or too low a dose.
  
  Not treated for long enough: it takes 48-72h for antibiotics to start to improve pneumococcal pneumonia.
- Unusual or resistant pathogen: check laboratory report.
- Immunocompromised patient: have less common pathogens, eg has pneumocystis been considered?
- Complication:
  - empyema
  - lung abscess
  - pulmonary embolism
  - cardiac failure (LVF, RVF, both)
HOSPITAL - ACQUIRED PNEUMONIA

Likely pathogen will depend on previous antibiotic therapy, whether the patient is MRSA - colonised and other factors. Consult local antibiotic guidelines or microbiologist for advice.

ACUTE EXACERBATIONS OF COPD

Chronic obstructive pulmonary disease (COPD) is the preferred term. Acute exacerbations of COPD present as a worsening of the previous stable situation.

Symptoms

- Increased breathlessness.
- New or increased sputum purulence.
- Increased sputum volume.
- Increased wheeze.
- Chest tightness.
- New or increased ankle oedema.

Important features in the history

- Previous exercise tolerance.
- Social circumstances and quality of life, especially whether living alone/alone with support/with family; whether housebound.
- Current treatments, including home nebulisers and oxygen therapy.
- Number of previous admissions in past five years.
- Number of admissions to ICU.
- Previously ventilated?
- Time course of current exacerbation.
- Smoking history.

IMPORTANT SIGNS

- Frankly purulent sputum
- Tachypnoea, wheeze and use of accessory muscles with increased work of breathing.
- Pyrexia
- Cyanosis
- Confusion
- Peripheral oedema
Initial Investigations

Priority
- ABGs (record inspired $O_2$ concentration).
- CXR (exclude pneumothorax).

Less urgent investigations
- PEFR and start PEFR chart.
- FBC, U&Es.
- Sputum and blood cultures.
- 12 lead ECG.

INITIAL TREATMENT

- Oxygen: do not give an inspired $O_2$ of more than 28% via Venturi mask or 2l/min via nasal prongs until arterial blood gases are known.
- Check ABG within 20 mins of starting $O_2$ and within 20 mins of changing inspired $O_2$. Aim to achieve a $PaO_2$ of $>6.6$ kPa and $H^+$ of $<55$. If the $PaO_2$ is responding and the effect on $H^+$ is modest increase the inspired $O_2$ to achieve a $PaO_2 >7.5$ kPa.

This applies to this particular group of COPD patients and must not be extrapolated to other acute conditions such as asthma, pneumonia, LVF, sepsis and so on.

Bronchodilators
- Nebulised salbutamol 5mg and ipratropium bromide 500 microgrammes should be given on arrival and repeated 4-6 hourly.
- Consider using air compressor and 2l nasal $O_2$.
- For distressed patients more frequent salbutamol nebulisers may be given.
- For the improving patient the ipratropium may be discontinued.
- If the patient is not responding to repeated nebulised bronchodilators IV aminophylline may be considered by the Respiratory/Intensive Care Specialist. Controlled IV infusion of 250mg (maximum 5mg/kg) aminophylline over 20 mins only if patient NOT receiving oral theophyllines).
- NIV - non-invasive positive pressure ventilation via face mask - should be considered for decompensated patients with hypercapnoea and acidosis $H^+ >55$nmol/l: discuss with Respiratory/ Intensive Care Specialist.
**Antibiotics**

- Amoxicillin (amoxycillin) 500mg oral tds (clarithromycin 500mg oral bd in penicillin allergic subjects).
  
  If patient has severe infection, or has bronchiectasis: seek specialist advice.

**Other measures**

- Prednisolone 40mg oral daily should be given unless there are contraindications to steroids.
- Hydrocortisone 200mg IV may be given initially if the oral route is not appropriate.
- Diuretics are indicated if there is peripheral oedema and/or raised JVP.
- Atrial fibrillation with an uncontrolled ventricular rate should be treated with digoxin.
- If immobile thromboprophylaxis with subcutaneous heparin should be given: see local protocols.
PNEUMOTHORAX

Tension pneumothorax is an EMERGENCY and requires immediate treatment by inserting a 14G cannula in the 2nd intercostal space in the mid-clavicular line on the affected side. A formal chest drain can then be sited. Otherwise treatment is based on the symptoms, degree of pneumothorax, and underlying pathology.

SPONTANEOUS PNEUMOTHORAX

If the lungs are normal:
- **Aspirate if complete collapse** - aspirate in 2nd intercostal space, mid-clavicular line with an 18G cannula, 50ml syringe and 3 way tap. This should follow explanation, skin cleansing and infiltration of the site with adequate 2% lidocaine (lignocaine). If the pneumothorax resolves or is rendered small the procedure has been successful. Repeat once only.
- **Moderate collapse** (degree of collapse: small = a rim of air; <2 cm air moderate = 2cm rim; complete = airless lung).
- Admit to Respiratory Unit for observation. May be discharged within 24 hours.
- **If small**, uncompromised and a sensible patient discharge and review with CXR within one week. **Must** be advised to return immediately if less well or more breathless.

If the lungs are abnormal:
- Aspirate if moderate/complete collapse, or if smaller pneumothorax but breathless or compromised. Compromise includes tachypnoea, hypoxaemia/low SpO₂ and/or signs of tension.
- If <50% collapse and patient not dyspnoeic or compromised observe as an inpatient (refer to Respiratory Unit).

**STOP aspiration if:**
- More than 2.5 litres aspirated.
- Resistance is felt.
- Excessive coughing.

**Chest drains are required for the following:**
- Tension pneumothorax.
- Symptomatic patient with underlying lung disease.
- Failed aspiration (unsatisfactory resolution of pneumothorax or breathlessness).
- History of chest trauma (includes CVP line and CPR related pneumothorax).
This is a clinical diagnosis associated with:

- Chest trauma including CPR.
- Positive pressure ventilation, especially with poorly compliant lungs.
- Rib fractures.
- CVP line insertion (and attempts) and insertion of pacemaker.
- Severe airways obstruction e.g. asthma.

**Signs**

- Cyanosis/low SpO\textsubscript{2}/low PaO\textsubscript{2}.
- Hypotension with raised JVP or CVP.
- Raised airway pressures (if ventilated).

Diagnosis is **NOT** radiological but is clinical

- Tracheal deviation away from side of other signs.
- Reduced air entry, reduced expansion and hyper-resonance to percussion on side of pneumothorax.

**Action**

- 100% oxygen.
- 14G cannula inserted perpendicular to skin in 2nd intercostal space, mid-clavicular line.
- Give analgesia.
- Formal intercostal drain insertion.
- CXR to check position and re-expansion. Adjust position of chest tube if required.

---

**INTERCOSTAL DRAINAGE TUBE INSERTION**

**Respiratory Unit staff can be contacted for insertion of intercostal drain.**

- Oxygen and IV access. Atropine should be available as profound vagal stimulation, with resulting bradycardia, can occur during pleural manipulation.
- Explain the procedure to the patient.
- Premedicate anxious patients with midazolam 1mg to 2.5mg IV or diazepam 5mg to 10mg sublingually (ordinary tablets dissolve) unless the patient is in respiratory failure. Flumazenil should be immediately available.
- Analgesia including morphine should be considered.
• Look at the CXR and mark intubation site (4th or 5th ICS in mid-axillary line) with pen on patient’s chest.
• Place the patient supine (20-30 degrees) with the patient’s ipsilateral arm behind head.
• Wash hands and put on gown and gloves.
• Clean the lateral chest wall, drape it and infiltrate skin then down to parietal pleura with 1% lidocaine (lignocaine) 10-20ml, using a blue then subsequently a green needle aspirating intermittently (look for air in syringe to confirm that the pleural space has been entered).
• In many patients insertion of a small chest drain by the Seldinger technique using, for example, the Sims-Portex system may be appropriate: see product inserts and video for details of technique.
• Select a 20-24 French guage chest drain tube (adult) using a smaller size for pneumothoraces and larger for fluid.
• Check all drain connections and make sure the underwater bottle is prepared with fluid before inserting the chest drain.
• Make a small transverse incision into skin and subcutaneous fat at the intercostal space below that selected for insertion of the tube.
• Using blunt dissection afforded by spreading forceps between the skin and chest wall, a subcutaneous track is developed to the level of the selected intercostal space. The intercostal muscles are separated over the edge of the rib below (remember the neurovascular bundle runs in a groove on the inferior surface of the rib) and the parietal pleura is gently penetrated (Fig 1).
• The tip of the finger is then inserted through the skin incision and pleural penetration is verified, and a gentle finger sweep ensures that the lung is not adherent to the chest wall.
• Insert the clamped chest drainage tube (with the trocar removed) through the prepared track using forceps to guide it apically (Fig 2).
• Never force the chest drain in.
• Connect drain to underwater seal bottle, release clamp (look for bubbling and swinging of water column confirming position in the pleural space) and secure drain with 2/0 suture (one loop through skin and at least four ties on tube).
• Apply dressing (swabs and elastoplast), loop tube and secure with further plaster (Fig 2).
• CXR
• Instruct the patient to keep water bottle below waist level, and prescribe adequate analgesia.
Reassure and prepare the patient (note position of suture)

Form dissection with forceps

Ensure that there is a space between the visceral and parietal pleura

See next page

'Load' chest drain onto forceps

Incision

Lower end of suture

Lung

Visceral pleura

Rib

Intercostal muscle

Parietal pleura

See next page
Apply dressing along the line the line of the ribs

Use the forceps to guide the chest drain through the dissection into the pleural space
MANAGEMENT OF CHEST DRAINS

- CXR post-insertion to check position and ensure re-expansion.
- Once bubbling has stopped for 24hrs **AND** CXR shows lung re-expansion remove the drain.

### NEVER routinely clamp a chest drain: only clamp a chest drain if the bottle breaks or the tube becomes disconnected.

- Repeat CXR after removal and if lung collapsed again discuss with Respiratory Registrar (if not already).
- If lung not re-expanded repeat CXR next morning. If still not re-expanded and drain bubbling discuss with Respiratory team.
- If drain not swinging or bubbling then it has either come out or is blocked: check the drain. If drain has come out and is still needed the drain tube should be removed and a new drain inserted with full aseptic technique.

### Never replace the drain through a previous insertion site.

- If still bubbling at 24hrs consult Respiratory Registrar. Consider low-grade suction (5-10cm H$_2$O). Do not use a standard wall kPa suction pump.
- If subcutaneous emphysema check the drain is not blocked.
- Great care should be taken to insure that tubing between the chest drain and the underwater seal bottle does not disconnect.

## TROUBLESHOOTING

### Fails to swing

- Check connections.
- Check CXR and if drain blocked or outside pleural cavity remove.
- Replace only if lung not up.

### Bubbling at 24hrs

- Check position **(as above)**.
- Check connections.
- Check entry wound is not sucking in air.

If a patient with a chest drain in situ requires transfer by ambulance a trained nurse with experience in the management of chest drains must be part of the escort.

**Discuss with Respiratory team if requiring suction or any problems.**
Chapter 5

GASTROINTESTINAL EMERGENCIES

ACUTE UPPER GASTROINTESTINAL BLEEDING

- Common emergency.
- 10% mortality in the UK.
- Presentation with haematemesis and/or melaena, and with shock or collapse.
- Syncopal symptoms such as dizziness or weakness may be present.

AETIOLOGY

<table>
<thead>
<tr>
<th>AETIOLOGY</th>
<th>FREQUENCY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peptic ulcer</td>
<td>50%</td>
</tr>
<tr>
<td>Varices</td>
<td>5-10%</td>
</tr>
<tr>
<td>Oesophagitis</td>
<td>10% *</td>
</tr>
<tr>
<td>Mallory-Weiss tear</td>
<td>5%*</td>
</tr>
<tr>
<td>Vascular malformation</td>
<td>5%*</td>
</tr>
<tr>
<td>Gastritis</td>
<td>15%*</td>
</tr>
</tbody>
</table>

* usually respond to conservative therapy and are not life-threatening.

MANAGEMENT OF HAEMATEMESIS AND MELAENA

Standard initial assessment and management of the ill patient as described in Chapter 2.

Immediate action for all

- Oxygen
- Secure adequate IV access.
- IV fluids: 0.9% saline or colloid.
- Avoid saline in liver disease.
- Send bloods (below) including cross-match.
- 12 lead ECG in elderly/history of cardiac disease.
- Keep NBM. Consent for endoscopy will be obtained by endoscopist or other GI staff. Note any previous history of DU or GU, NSAID, anticoagulants, liver disease or dyspeptic symptoms.
- Look for evidence of chronic liver disease such as jaundice or spider naevi. If present refer to the GI Registrar and commence resuscitation (below).

In all patients ascertain the severity of the bleed and at risk factors: risk stratify.
Features of a major bleed?

- Tachypnoea
- Tachycardia >100 bpm.
- Hypotension SBP <100mmHg supine or postural drop at any stage. Relate BP to the patient’s normal e.g. hypertensive.
- Clammy, cold and peripherally shutdown.
- Conscious level reduced/confusion.
- History of syncope.

Is the patient at Significant Risk of Death?

- Hypotensive after initial resuscitation.
- Variceal bleed likely.
- Obvious signs of chronic liver disease or deranged clotting indicative of liver disease.
- Continuing melaena, haematemesis, or rebleed.
- Existing co-morbidity e.g. IHD, renal failure, disseminated malignancy.
- Age >60 years.

Complicating factors

- Co-morbid disease e.g. cardiovascular, respiratory, renal, malignancy.
- Rate limiting drugs prevent compensatory tachycardia e.g. β-blockers, verapamil.
- Vasodilators prevent compensatory vasoconstriction, e.g. ACE inhibitors.

TREATMENT AND ASSESSMENT

SHOCKED PATIENT

- High concentration oxygen, at least 60%.
- IV access with two large bore cannulae 16G or bigger.
- Draw blood for FBC, PTR/clotting, U&E, LFTs, blood for CROSS MATCH at least 4 units of red cells. Alert BTS: consult Major Haemorrhage protocol (Appendix 3).
- Commence IV fluids: 0.9% saline or Gelofusine 10-20 ml/kg (500-1000ml).
- Use O negative blood if patient exsanguinating or unable to keep BP above 100mmHg systolic, and more than 1 litre of colloid given (see next page).
• Monitor closely: ideally ECG and pulse oximeter for continuous heart rate and oxygen saturation readings with frequent BP measurement e.g. every 5 mins. Consider need for HDU/ICU referral and invasive monitoring: elderly, co-morbid disease and severe bleed are indications.

A large bore femoral venous line can be invaluable for rapid fluid infusion.

• Refer to GI Registrar.
• ABG for oxygenation and base deficit (i.e. the severity of bleed).
• Get Hb and K⁺ results early.
• A urinary catheter should be inserted.
• Nasogastric intubation is not necessary.

If features of circulatory compromise persist after the initial bolus of fluid commence blood transfusion. If available use type-specific or cross-matched blood. If not, use O Negative blood: this is kept in the blood fridge in clinical chemistry, WGH and in A&E in RIE and in Haematology laboratory at SJH. Inform Blood Transfusion that it is a significant bleed: consider triggering Major Haemorrhage protocol.
• Use a blood warmer if large volumes are to be given. A ‘Level One’ blood warmer is available from main theatre WGH, and A&E and Theatres, RIE; A+E and Theatres at SJH.
• Coagulopathy should be corrected using FFP.
• Early endoscopy should be performed for all large bleeds and suspected varices but the patient must be adequately resuscitated first. Guidelines for endoscopy in high risk patients are available in theatre.

Rebleeding is a major predictor of death.

**Modified Rockall Score** is a means of assessing risk of rebleed and mortality following non-variceal upper GI bleeding. Total (pre-endoscopic + endoscopic) score of 0 or 1 implies 0% mortality and therefore discharge should be safe. Calculation of the pre-endoscopic score is as follows: (add scores for each line).
VARICEAL BLEEDING

Contact GI Registrar.

- A Rockall score of 1-2 suggests a mild bleed and a score of greater than 2 a major or severe bleed. There is 50% mortality with a Rockall score of 7.
- In SJH if Rockall score is 0 or 1 the patient is admitted there and OGD performed at the latest the next day. If the Rockall score is 2 or greater the patient is transferred to RIE (following appropriate assessment and resuscitation).

<table>
<thead>
<tr>
<th>Age</th>
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<th>3</th>
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<td>Systolic&gt;100 Pulse&lt;100</td>
<td>Systolic&gt;100 Pulse&gt;100</td>
<td>Systolic&lt;100</td>
<td></td>
</tr>
<tr>
<td>Co-morbidity</td>
<td>None</td>
<td>Heart failure IHD Major Cormorbidity</td>
<td>Renal failure Liver failure Disseminated Malignancy</td>
<td></td>
</tr>
</tbody>
</table>

- Resuscitate as above: avoid saline, use colloid and IV dextrose 5% and FFP as required.
- Monitor cardiac rate and rhythm, BP and oxygen saturation.
- Give terlipressin 2mg IV then 1-2mg IV every 6 hours until bleeding is controlled, for up to 72 hours. Caution in ischaemic heart disease, peripheral vascular disease and unresuscitated patients.
- A Sengstaken-Blakemore tube may be necessary for massive or ongoing bleeding. It is available in the Resuscitation room in A&E and ARAU.

Sengstaken-Blakemore tube is only for use in dire situations. It should be placed only by the GI team or other senior staff experienced in its use, therefore get help. The patient should be discussed with anaesthetics re intubation prior to placement of tube if possible. In general patients with Sengstaken tubes in situ, should not be transferred between hospitals intubated.

- The gastric balloon is inflated with 300mls of air and the tube held in place with two tongue depressors taped together and padded (to avoid pressure on lips). CXR should be performed to confirm correct position.
- Inadequate placement of Sengstaken tube confers no benefit but has risk of major complications e.g. oesophagael perforation.
- Prophylactic antibiotic: Ceftriaxone 1g od IV
- Discuss further interventions e.g. TIPSS with GI Registrar/Consultant if not already referred.

**MANAGEMENT OF UPPER GI HAEMORRHAGE: SUMMARY**

### MAJOR BLEED: HIGH RISK

- Pulse > 100
- Systolic BP < 100mm Hg
- Hb < 100 g/L

**EMERGENCY (OUT OF HOURS) ENDOSCOPY**
Contact GI Registrar

**RESUSCITATE**
As above

### LIKELY VARICEAL: HIGH RISK

- Stigmata of liver disease
- Abnormal LFTs, clotting

**EMERGENCY (OUT OF HOURS) ENDOSCOPY**
Contact GI Registrar

**RESUSCITATE**
As above

### MINOR BLEED: LOW RISK

- Pulse < 100
- Systolic BP > 100mm Hg
- Hb > 100 g/L

**ENDOSCOPY ON NEXT ELECTIVE LIST**
Contact GI Registrar

**OBSERVE**
2 hrly observations

---

An intravenous proton pump inhibitor is only indicated in patients who have active bleeding or stigmata of recent haemorrhage at endoscopy.

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### ACUTE ON CHRONIC LIVER FAILURE

**General points**

- Avoid hypoxaemia, hypotension, and hypoglycaemia (2-4hrly BM measurement).
- Lactulose 30ml oral tds is beneficial in early encephalopathy. Titrate to produce 2 to 3 bowel movements daily. Use phosphate enemas
in more severe cases or if unable to take lactulose.

- Blood, urine and ascitic fluid cell count and culture if encephalopathic and in all cases on admission. Cell count: WBC>250/microlitre is suggestive of spontaneous bacterial peritonitis.
- Establish if there is a history of drug misuse, blood product transfusions, hepatotoxic drug ingestion, alcohol abuse or foreign travel.
- Clinical assessment: note the presence of jaundice, ascites, encephalopathy, spider naevi, pruritis, bruising, splenomegaly, rashes or arthritis.
- Try to ascertain what has precipitated this episode.
  Consider: bleeding
  infection
  drugs, particularly diuretics or sedatives
  electrolyte disturbances e.g. hyponatraemia, hypokalaemia

**Investigations to consider**

- FBC, U&E, glucose, phosphate, LFT, PT, AFP.
- Venous blood cultures, MSSU.
- Viral hepatitis screen.
- Autoantibody profile.
- Paracetamol level.
- Ascitic tap for bacteriology (Gram stain, cell count and culture), protein.
- USS of abdomen.

**FULMINANT HEPATIC FAILURE**

Always check for PARACETAMOL OVERDOSE in patients presenting with acute liver failure or unexplained metabolic acidosis.

The commonest causes of acute liver failure are paracetamol poisoning and viral hepatitis. Patients with liver failure may present in a variety of ways. They may show non-specific features such as confusion, sepsis, or shock. Specific modes of presentation are with ascites or encephalopathy, and the management of these is detailed below.

Early ICU involvement for airway protection, ventilation, haemodynamic monitoring and resuscitation may be necessary. At the same time early referral to the Scottish Liver Transplantation Unit is crucial (22068 in RIE). GI/Liver registrar on bleep #6361 or via switchboard RIE. Referral criteria in ARAU/WGH, A&E and Combined Assessment Units.
Hepatocellular failure is the result of impairment of hepatocyte function, which manifests itself in a variety of ways, either encephalopathy, in acute liver failure, or encephalopathy or ascites in chronic liver failure.

**Acute liver failure**
- Presents as hepatocellular jaundice, elevated transaminases, and prolongation of the INR, in the context of acute liver injury, e.g. acute viral hepatitis.
- This is complicated by hepatic encephalopathy.
- Encephalopathy occurs within 8 weeks of onset of jaundice (first illness).
- Risk of hypoglycaemia and raised intra-cranial pressure (ICP).

**Chronic hepatocellular failure** occurs when there is decompensation in a chronic liver disease, presenting either with ascites or encephalopathy.

**ENCEPHALOPATHY: Grading of Hepatic Encephalopathy**
- **Grade 1** Mildly drowsy with impaired concentration/number connection test.
- **Grade 2** Confused but able to answer questions.
- **Grade 3** Very drowsy and able to respond only to simple commands.
- **Grade 4** Unrousable.

**DECOMPENSATED CHRONIC LIVER DISEASE**
- Hypoglycaemia and raised ICP uncommon.
- Causes and management differ.
- Repeated assessment and documentation of GCS is very useful.
- Identify and treat infection or bleeding, and stop precipitant drugs.
- Paracentesis or diuretics can precipitate encephalopathy (hypokalaemia, hyponatraemia).
- Diagnostic paracentesis to exclude spontaneous bacterial peritonitis (i.e. >250 polymorphs per microlitre) of ascites.
- In patients with encephalopathy treat with lactulose 30ml oral tds titrated to produce 2 to 3 bowel movements daily, and use phosphate enemas if patient unable to take oral lactulose.
- Ensure good nutrition, correction of hypoxaemia and electrolyte abnormalities (including hypophosphataemia).
For alcoholic liver disease: thiamine 300mg oral daily is given. In acute/imminent Wernicke’s encephalopathy or Korsakoff’s psychosis, and in patients unable to take orally, IV vitamins are given as Pabrinex IV two pairs (No1 and No2 mixed) by IV infusion in 100ml 5% dextrose over 15-30 mins 8 hourly.

**N.B. Risk of anaphylaxis.**

- In patients with altered consciousness exclude focal neurology e.g. subdural haematoma.
- Sedation should be avoided if possible. Small doses of haloperidol e.g. 1-2mg IV may be used in severe agitation.
- Monitor renal function closely as there is a high risk of renal failure.

### TENSE ASCITES

- Large volume paracentesis can be performed with IV 20% Albumin ‘cover’, 6g per litre drained or 400ml 4.5% Human Albumin solution every 3 litres ascites drained.
- Correct intravascular volume before paracentesis: stop diuretics.
- Close monitoring of renal function is required: hourly urine volumes, daily U&Es.
- Exclude spontaneous bacterial peritonitis by diagnostic paracentesis cell count, Gram stain, culture and protein.

### ACUTE BLOODY DIARRHOEA

Bloody diarrhoea tends to occur in two groups of patients: those with known inflammatory bowel disease and those in whom bleeding arises de novo.

- In severe cases hypovolaemia and/or sepsis may result in shock which should be managed as described in Chapter 2.
- A history of inflammatory bowel disease should be sought. If this is positive refer to the GI registrar having secured adequate IV access and sent blood for FBC, ESR, U&E, glucose, albumin, CRP and LFTs.
- Duration of symptoms: this is an important point as a history of less than 7-10 days suggests an infective aetiology. Ascertain the frequency of motions/amount of blood, any recent foreign travel, a similar history amongst friends and family, any recent antibiotic or NSAID use (in last 2 weeks) or abdominal pain. Take a sexual history.
- Examine for abdominal tenderness or distension, arthritis, erythema nodosum and iritis.
INITIAL MANAGEMENT
- IV access.
- FBC, ESR, U&Es, glucose, albumin, CRP, LFTs. Group, screen and save or cross match if appropriate.
- Stool culture
- C. difficile toxin if
  a. any antibiotic in last eight weeks or
  b. hospitalised within last eight weeks.

SPECIFIC MANAGEMENT

Infective
- Short history, no antibiotic usage.
- Isolate patient.
- FBC and culture stool (and C. diff toxin).
- Maintain “hydration” and observe.
- Ciprofloxacin 500mg oral bd if unwell. Use antibiotics with caution in probable infective diarrhoea (may worsen outcome in E Coli 0157 infection).

Pseudomembranous Colitis (likely if antibiotic therapy in last eight weeks)
- FBC and bloods as above.
- Isolate patient.
- Sigmoidoscopy and biopsy.
- Stool culture and C. difficile toxin.
- Monitor pulse, BP, temperature oxygen saturation.
- If ‘toxic’ do AXR looking for toxic dilatation.
- Consider empirical metronidazole 400mg oral tds or 500mg tds IV if not tolerated orally (alternative is vancomycin 125mg oral qds only if metronidazole not tolerated or if recently treated with metronidazole).
- Stop other antibiotics if possible: seek microbiology advice if antibiotics need to be continued.
- Seek early surgical review if very unwell.

Known IBD
- Exclude infection.

Mild colitis (<4 stools per 24 hours)
- Apyrexial, pulse <90/min, Hb >120g/l, ESR <10mm/hr, small amount of blood in motion.
ACUTE DIARRHOEA

CAUSES

- Infective
- Inflammatory bowel disease
- Irritable bowel syndrome
- Overflow
- Antibiotic associated
- *Clostridium difficile*
- Malabsorption
- Thyrotoxicosis
- Laxative abuse
- Alcohol
- Other drugs e.g. NSAIDs, PPIs

INVESTIGATIONS

- FBC, U&E’s, LFT’s, ESR, CRP; endomysial antibodies, and haematinics if coeliac disease suspected.
- Stool culture
- CI difficile toxin if recent antibiotics or hospitalisation. Microscopy for amoebae and other Gl pathogens, (if at risk 3 samples).
- Blood cultures if febrile or features of sepsis.

Severe colitis - Refer to GI Unit( >8 stools per 24 hours)

- Febrile >37.5 C, pulse >100/min, Hb <110g/l, ESR >30mm/hr, blood in motion +++.
- Inform GI Registrar.
- AXR
- Sigmoidoscopy
- Stool cultures.
- IV methylprednisolone 60mg/24hr, given by continuous IV infusion (can cause dysrhythmias) in WGH or hydrocortisone 100mg IV QDS in RIE.
- DVT prophylaxis as per local policy.
• Plain abdominal x-ray
• Rectal examination
• Sigmoidoscopy and rectal biopsy
• Thyroid function tests

**Travel history and suspected food sources must be stated on lab request form - some pathogens eg Vibrio cholerae are only looked for if appropriate clinical information**

**MANAGEMENT**

• Rehydrate, replacing sodium, potassium, and chloride loss using oral rehydration fluids.
• Isolate if infection is suspected.
• Notify suspected food poisoning cases to Lothian Public Health: 7720.
• In general infective diarrhoea is not treated with antibiotics.
• Consider metronidazole or vancomycin for pseudomembranous colitis: see above.
• If inflammatory bowel disease - refer to GI.

**CONSTIPATION**

• A rectal examination must be performed
• Evidence of obstruction? Clinical examination
• Plain abdominal X-ray only if suggestion of intestinal obstruction.
• Is this acute/recent or chronic?
• Check U&E’s, calcium, TFT’s.
• Are any drugs implicated e.g. opiates?
• Refer to surgeons if obstruction (clinical evidence or on abdominal X-ray). AVOID stimulants. Use lactulose.
• Barium enema to exclude megacolon.
• Refer to GI team for advice.
• Disimpact with Glycerine suppositories
  Phosphate enema
  Arachis oil enema
  Picolax
  Manual disimpaction (with care)
• Maintain with Fybogel or lactulose
• Stimulate with Senna (Stimulants are best avoided except in terminal care)

• In refractory cases PEG-based agents Movicol 1/2-1 sachet/day titrated

Codanthrusate (Potentially carcinogenic. So only use long-term in the elderly, or terminally ill)
Definitions vary but a general one would be disabling abdominal pain of less than two weeks duration.

Take a careful history about the onset and progression of pain, site and radiation, exacerbating and relieving factors, associated symptoms.

In general visceral pain is ill-localised and felt in the area corresponding to the organ’s origin—foregut, midgut or hindgut.

Irritation of the parietal peritoneum is well localised and responsible for features of peritonism found on examination—guarding and rebound or percussion tenderness.

Any inflammatory pathology will give rise to symptoms of peritoneal irritation e.g. pain exacerbated by movement; obstruction of a tubular structure gives rise to colic type symptoms.

Examine carefully for signs of shock—increased respiratory rate is a useful early indicator.

Lie the patient flat and expose the whole abdomen, watching for excursion with respiration.

Remember to include examination of hernial orifices and genitalia and PR.

If the patient is shocked, begin resuscitation at the same time as undertaking investigations—O₂, fluids and iv access, analgesia, catheter as necessary and call for senior help. See chapter 2.

**Diagnosis and resuscitation are simultaneous processes in the shocked patient**

**Baseline investigations :**
- Fbc, U&E, glucose, LFTs, amylase, G&S
- ABG, lactate— if unwell
- Urinalysis
- Exclude pregnancy if relevant
- Erect CXR— if possibility of perforation (only positive in 50%)
- AXR— if obstructed
- CRP— remember can lag behind clinical features

**Decisions to be made**
- Does the patient require a laparotomy?
- What is the timescale of this?
• Is more resuscitation or investigation required?

If the patient is bleeding this will usually need surgical control, but if the patient is obstructed with metabolic derangement there is normally time for fluid replacement. This process of preoptimisation needs to be actively managed and will benefit from management in HDU/ICU.

PITFALLS

• Medical causes of abdominal pain including DKA, pneumonia and Herpes Zoster.
• Much of the abdominal cavity is not easily accessible to palpation – the pelvis and much of the supracolic compartment

URGENT SURGERY

• Ensure blood is cross matched if required.
• Operating surgeon should liaise with theatre and anaesthetist and obtain consent from patient if appropriate.
• Make plans for post op care early- will the patient need management in ICU or HDU.

ICU and Anaesthetics opinions should be sought early to allow planning of and delivery of optimal perioperative care.

ACUTE PANCREATITIS

Determine severity of pancreatitis on all patients using modified Glasgow Score* (see below).

The following investigations should be undertaken daily on all patients with SEVERE PANCREATITIS for at least 3 days:

• FBC
• U&E’s, creatinine
• Blood glucose
• Serum calcium
• LFT’s (LDH must be specified)
• Serum albumin
• Arterial blood gas (on air initially) only if well enough to tolerate this.
• CRP

Initial management in all patients involves:

• nil by mouth
• IV fluids
• urinary catheter and measurement of hourly urine volumes. Patients with severe pancreatitis may need to be managed in HDU/ICU and often warrant invasive haemodynamic monitoring.

**FURTHER INVESTIGATIONS**

• U/S Scan as soon as possible after admission.
• CT Scan is usually needed in all patients with severe pancreatitis within 10 days of admission and must be a **dynamic contrast-enhanced scan**.
• **ERCP** should be considered in all patients with severe pancreatitis thought to be due to the gallstones who do not settle promptly on conservative management and **have evidence of cholangitis**.

| **AGE** | > 55 years |
| **arterial PO<sub>2</sub>** | <8.0 kPa (on air) |
| **albumin** | <32 g/L |
| **calcium** | <2.0 mmol/l |
| **WBC** | >15 x 10<sup>9</sup>/l |
| **LDH** | >600 U/l |
| **ALT** | >100 U/l |
| **glucose** | >10 mmol/l (in absence of diabetes) |
| **urea** | >16 mmol/l |

*A modified GLASGOW criteria: a severe attack is predicted if 3 or more criteria are positive.*

(A C-reactive protein level over 100 mg/l may also reflect the presence of a severe attack and can be used to monitor progress and the need for CT scan).

**INTER HOSPITAL TRANSFER BETWEEN UPPER GI (RIE) AND LOWER GI (WGH) UNITS IN EDINBURGH**

• Both units receive “General Surgery” where the diagnosis is either uncertain or out with the GI tract. Allocation will depend on bed availability and patient location. Bed Bureau will usually decide destination.
• Patients assessed in either hospital should have appropriate first line investigations carried out in that hospital, if feasible, to confirm diagnosis before transfer, e.g. abdominal ultrasound for suspected biliary colic and CT abdo/pelvis for acute diverticular disease etc.
Discussion must take place at Registrar or Consultant level only.

• Patients must be stable before transfer and the “transfer form” (attached) must be completed in all cases. Between arranging transfer and the patient leaving, the referring team must continue to ensure resuscitation and ongoing monitoring is taking place, with regular review and reassessment.

• Patients who are unstable and therefore unsuitable for transfer should be discussed between consultants.

• In-patient emergencies arising in St John’s Hospital outside those hours where there are surgeons on site (8am – 6 pm Monday – Thursday and 8 am – 2 pm Friday) should be discussed initially with the consultant on-call at the Western General Hospital. Clearly if the problem is upper gastrointestinal/biliary pancreatic then it would be more appropriate to contact the consultant at the Royal Infirmary. If the patient is considered unsuitable for transfer then the consultant at the Western General Hospital will arrange for that patient to be seen and assessed at St John’s Hospital. At times discussion may need to take place between the consultant at the Western General Hospital and the consultant at the Royal Infirmary to ascertain who would be best to go to St John’s and what cover we would put in place in Edinburgh while that consultant is at St John’s.

• Stable patients should not be transferred (where possible) overnight.

GUIDELINES FOR THE ASSESSMENT OF SURGICAL PATIENT SUITABILITY FOR TRANSFER BETWEEN RIE AND WGH

Patients present to RIE and WGH. To ensure optimal management and avoid morbidity some of these should not be transferred but should be resuscitated, analgedesed and operated on where they present e.g perforated intra-abdominal viscus.

Remember: Identification of the sickest patient is usually straightforward, but the patient who is ‘compensating’ physiologically may appear much better than he/she really is.

TRANSFER GUIDELINES FOR ILL PATIENTS

Patient Assessment
• Respiratory rate
- Pulse
- BP and peripheral perfusion
- SpO₂
- Metabolic state: potassium, base excess/deficit, lactate
- Haemoglobin, presence of active bleeding
- Pain

**ASSESS USING CRITERIA IN BOX BELOW:**

IF ALL ACHIEVED OK TO TRANSFER

- RR >10 and <25/min
- Pulse <110/min BP > 110mm Hg systolic Hg (systolic no more than 30 mmHg lower than normal for patient), not peripherally shutdown
- SpO₂>96% (on <60% oxygen)
- K⁺ 3 to 5.5mmol/l
- Base deficit better than -7 (if unwell arterial blood gases should be done)
- Pain controlled adequately
- Appropriate IV access
- Hb>100g/l, not actively bleeding
- The patient should be cardiovascularly stable

If not achieved not ok to transfer

Can correct to figures in box
Do so then Senior opinion about safety of transfer (qv)

Cannot correct easily to figures in box. Senior assessment and keep in that hospital, resuscitate and operate there as appropriate.

**Minimum treatment and monitoring**

- Oxygen to achieve sats >96%
- IV access and fluids to restore perfusion; x-match and transfuse as required
- Adequate analgesia with iv opioids and iv anti-emetic
- Correction of potassium imbalance
- Monitor ECG, pulse oximetry, cuff BP, urinary catheter and output.
• If the patient is considered unstable enough to require the above then it is questionable that they should be transferred.
• If diagnosis is unclear transfer is unwise.
• A senior surgical opinion should always be sought before transfer.
• If you are not 100% happy don’t transfer the patient.
• A specific protocol for management of AAA presenting to WGH is in chapter 3.

Guideline developed by Dr Graham Nimmo with surgical and anaesthetic cross site group RIE/WGH 1998.
Must be completed by clinician arranging transfer

**PATIENT ID**

**DATE:**
Assessing hospital: RIE / WGH

### DIAGNOSIS

**Criteria to be achieved for transfer**

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Patient</th>
<th>Criteria fulfilled?</th>
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</thead>
<tbody>
<tr>
<td>Temp &lt;40C, no rigors</td>
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<td></td>
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<tr>
<td>Pulse pulse &lt;110</td>
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<td></td>
</tr>
<tr>
<td>BP systolic &gt;110</td>
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<td></td>
</tr>
<tr>
<td>Peripheral perfusion warm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resp Rate RR&gt;10 or &lt;20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SpO₂ SpO₂&gt;96% on &lt;60% O₂</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hb &gt;100g/l</td>
<td></td>
<td></td>
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<tr>
<td>K 3-5.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BM 4-10</td>
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<td></td>
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<tr>
<td>BHCG Negative</td>
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<tr>
<td>H⁺ &gt;35 or &lt;45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pO₂ &gt;9 on air</td>
<td></td>
<td></td>
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<tr>
<td>pCO₂ &lt;6 on air</td>
<td></td>
<td></td>
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<tr>
<td>BE -2 to +2</td>
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<td></td>
</tr>
<tr>
<td>Adequate IV access? Yes (18G or greater)</td>
<td>Y/N</td>
<td></td>
</tr>
<tr>
<td>Fluids running? Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain controlled? Yes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Active bleeding? No</td>
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<td></td>
</tr>
<tr>
<td>Anticoagulated? INR&lt;4</td>
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<td></td>
</tr>
<tr>
<td>Perforation? No</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time of transfer</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### GUIDELINES FOR TRANSFER

**IF CRITERIA ACHIEVED**, for transfer after discussion specialist registrars at each hospital or consultants.

**IF CRITERIA NOT ACHIEVED**, **NOT FOR TRANSFER**

Can correct parameters to acceptable range. Then **Consultant Opinion** about safety of transfer

Cannot correct parameters easily

**Senior** assessment

**Keep** in original hospital

**Resuscitate** & operate as appropriate

**Clinician accepting transfer:**

Name  Designation

**Clinician arranging transfer:**

Name  Designation  Signature
ACUTE RENAL FAILURE

Acute renal failure (ARF) is an abrupt decline in renal function which is usually reversible. It is recognised by the accumulation of waste products (urea, creatinine), with the development of electrolyte disturbance (hyperkalaemia) and metabolic acidosis. A decline in urine output (oliguria) is usually found. ARF is commonly caused by acute cardiovascular failure.

CLASSIFICATION OF ARF

PRERENAL: compromise to renal perfusion and oxygen supply commonly due to hypovolaemia or hypotension. Reversible with early resuscitation. Effects can be potentiated by a number of medicines including non-steroidal agents and ACE inhibitors or AII antagonists which should be stopped. If strong indication for ACE consider restarting following recovery with close monitoring of renal function.

OBSTRUCTIVE: blockage to urinary flow.
- Ureteric/urethral as in prostatic hypertrophy or bladder/ureter blockage as a result of tumour, stone, clot or stricture.
- Renal tubules/pelvis, ureters with myoglobinuria, haemoglobinuria, crystal formation, myeloma and papillary sloughing (e.g. diabetes). Uncommon.

INTRINSIC: damage to the renal parenchyma.

Intrinsic Causes of ARF

Nephrotoxins
- Drugs: NSAIDs, aminoglycosides, paracetamol in overdose.
- Poisons: methanol, ethylene glycol.
- Contrast media.

Specific conditions
- Vasculitis/glomerulonephritis: is there a history of skin rash, arthralgia/arthritis, rigors?
- Accelerated phase hypertension.
- Interstitial nephritis: follows a period of drug exposure in most instances.
- Infections: legionella, leptospirosis, malaria.
VASCULAR: renal artery disease and aortic atheroma/cholesterol emboli. Cholesterol emboli commonly follow intervention e.g. angiography or on commencement of anticoagulation.

**MANAGEMENT**

**Approach to ARF**
- Stabilise the patient whilst trying to improve or protect renal function by identifying potentially reversible factors.
- Seek underlying cause of ARF.
- Immediate concerns are hypoxaemia, blood volume abnormalities (hypovolaemia or fluid overload), hyperkalaemia, metabolic acidosis.

**IMMEDIATE TREATMENT**
- Correct hypoxaemia.
- IV access. Remember sites in upper limbs may be required for fistulae and consider using only one arm for cannulae and blood sampling (remembering potential pitfalls of blood dilution).
- Treat hyperkalaemia (see below).
- Correct volume status. If shocked commence resuscitation and refer to ICU.

> The combination of shock and acute renal failure has a high mortality.

- Insert a urinary catheter and measure hourly volumes.
- CVP measurement may help in monitoring volume replacement.

> Do not give loop diuretics unless there is a positive reason such as severe fluid overload.

- Stop nephrotoxins including drugs which may be a factor in ARF and hyperkalaemia.
- Treat metabolic acidosis: discuss with senior medical staff.
- In WGH/SJH if hyperkalaemia requires urgent renal replacement therapy (haemodialysis/haemofiltration) the first treatment should be performed in ICU before transfer to Renal Unit RIE. Contact ICU.

**Indications for urgent dialysis or haemofiltration: the clinical state of the patient should be taken into account before commencing renal replacement therapy.**
- Refractory and severe hyperkalaemia.
• Fluid overload with pulmonary oedema, refractory to diuretics.
• Severe metabolic acidosis.
• Pericarditis
• Renal replacement also indicated if urea and/or creatinine are markedly elevated. Discuss with the renal registrar.

Call Renal Registrar page #6394 in RIE, ICU in WGH or SJH - seek advice early.

INVESTIGATIONS

• U&E (including total CO₂), creatinine, glucose, FBC, clotting screen, group, screen and save, blood cultures.
• Plasma CK and urinary myoglobin (if available).
• ABGs
• Blood film for red cell fragments.
• Ca, PO₄, LFTs, albumin.
• Urate
• Glomerulonephritis screen where appropriate.
• Viral screen.
• Urinalysis
• Urine sodium and osmolality: interpretation is complicated by prior administration of IV fluids or diuretics.
• Urgent ultrasound of kidneys: size, number, obstruction, aorta.

All patients with acute renal failure should have USS of renal tract. Timing will depend on clinical presentation.

FURTHER MANAGEMENT

• If oliguria persists or biochemistry worsens renal replacement therapy (haemodialysis or haemofiltration) may be required: discuss with the Renal Registrar RIE or ICU in WGH/SJH.
• Scrutinise the notes, drug charts and review the history.
• Fully examine the patient.
• Look for infection and treat it.

Remember rhabdomyolysis. Muscle signs and symptoms are only seen in 50%, and myoglobin is absent from urine in about 30%. Causes include trauma, burns, compartment syndrome, epilepsy, drugs (including self-poisoning), coma with hypotension, falls and ischaemic limbs.
• Examine the urine: proteinuria and haematuria may indicate glomerulonephritis and urgent renal referral is obligatory. Look for skin rash, nail changes, arthralgia and history of rigors.

• Don’t delay referral as early diagnosis and appropriate treatment such as immunosuppression/plasma exchange may save renal function. GN bloods include anti-nuclear factor, anti neutrophil cytoplasmic antibody, anti-glomerular basement membrane antibody, rheumatoid factor.

• Fluid balance: once volume depletion corrected, and in the absence of fluid overload, give previous hour’s output (urine and other losses) plus insensible (about 20-40ml/hr).
DANGEROUS HYPERKALAEMIA

May cause sudden death with no warning features. Symptoms include paraesthesiae, circumoral tingling, muscle weakness, malaise. There may be no clinical signs.

**Diagnosis:** elevated potassium: absolute level and rate of rise are important. An abrupt rise of 2 mmol e.g. from 4 mmol/l to 6 mmol/l may cause arrhythmias whilst some patients with chronic renal failure tolerate higher levels. Consider level >6mmol/l as potentially dangerous.

ECG changes may provide the first clue to hyperkalaemia and its severity. ECG may be NORMAL in presence of dangerous hyperkalaemia.

CAUSES OF HYPERKALAEMIA

1. **Reduced excretion**
   - Renal failure
   **Drugs:**
   - Potassium sparing diuretics: Spironolactone, Triamterene, Amiloride
   - ACE inhibitors, angiotensin II antagonists
   - NSAIDs
   - Hypoaldosteronism: adrenal insufficiency

2. **Shift of K⁺ from cells**
   - Tissue damage: rhabdomyolysis, trauma, burns, haemolysis, internal bleeding
   - Drugs: suxamethonium, digoxin, β-blockers
   - Acidosis
   - Others: hyperosmolality, insulin lack, periodic paralysis

3. **Excessive intake**

4. **Pseudohyperkalaemia**
   - Thrombocytosis, leukocytosis
   - Haemolysis: in vitro or sampling
   - Delayed analysis

ECG CHANGES OF HYPERKALAEMIA

- Prolonged PR interval.
- Peaked T waves.
- Widening of QRS interval and flattening/loss of P waves.
- Sine wave proceeding to ventricular fibrillation or asystole.
1. IMMEDIATE ACTION: STABILISATION

- Assess ABCDE and treat accordingly.
- Correct hypoxaemia.
- IV access.
- Continuous ECG monitoring is mandatory.
- Monitor oxygen saturation.
- Specific treatment depends on ECG changes and potassium concentration.
- If ECG shows peaked T waves or more severe changes titrate IV calcium gluconate 10% or calcium chloride 10% in 1 ml aliquots watching the ECG. The trace will normalise as the calcium takes effect. If too much IV calcium is given it can result in cardiac arrest in asystole. The required amount varies from 2 or 3 mls to 20mls. This simply stabilises the myocardium giving time to institute therapy to reduce the potassium. This may need to be repeated.
- In cardiac arrest follow ALS algorithm and give 10mls 10% calcium chloride IV. VF will be resistant to defibrillation if calcium not given.

2. REDUCING THE POTASSIUM

- Bolus IV dextrose 50ml 50% solution with 5-10iu Actrapid (or equivalent e.g. Humulin S). Takes 20-30 mins to work.
- This can be followed with a slow infusion of 10% or 20% dextrose running at between 10ml/hr and 50ml/hr. Monitor blood sugar regularly and add insulin as required.
- Nebulised salbutamol 5mg and repeated.
- Sodium bicarbonate 1.26% IV infusion. Start at 100ml/hr and titrate to HCO₃ and K⁺ levels. Not for routine use. May help: discuss with renal registrar RIE or ICU, WGH/SJH.

3. ELIMINATING THE POTASSIUM

- The best way of removing potassium is to restore urine output and recover renal function.
- Failing this potassium removal by haemodialysis or haemofiltration may be required.

Stop dextrose and insulin infusions to allow potassium to re-enter the blood, thus making it available for removal in the dialyser.
• In WGH or SJH haemofiltration should be arranged with ICU pre-transfer to Renal Unit RIE if the level is high or the patient at risk.
• In potassium poisoning with normal renal function give IV fluids and furosemide (frusemide) to secure renal potassium loss.
• Ion exchange resins are difficult to administer orally or pr in the ill patient and take several hours to work. Most useful in chronic situations or if the patient needs to be transferred a long distance. Calcium resonium 15g stat oral, then 15g 2 to 3 times daily. An oral laxative should be prescribed at the same time.

Use the femoral vein for insertion of dialysis access as cardiac arrest in VF can be precipitated by the guidewire when using the internal jugular or subclavian routes.
METABOLIC ACIDOSIS

CAUSES OF METABOLIC ACIDOSIS (MA)

- Tissue hypoxia: shock with lactic acidosis.
- No tissue hypoxia: loss of or impaired generation of bicarbonate.
- Anion gap: acid accumulation (other than lactate).

Anion gap: \[ \text{Na}^+ + \text{K}^+ - (\text{Cl}^- + \text{HCO}_3^-) \]: normal up to 18mmol.

RAISED ANION GAP MA: ‘KUSSMALE’

- Keto-acidosis
- Uræmia
- Salicylate poisoning, paracetamol poisoning
- Severe losses of bicarbonate e.g. diarrhoea, GI fistulae
- Starvation
- Methanol poisoning
- Alcohol i.e. ethanol
- Lactic acidosis
- Ethylene glycol poisoning

Severe elevation of anion gap >35mmol is usually due to:

- Toxin ingestion e.g. methanol, ethylene glycol.
- Severe shock or cardiac arrest (lactic acidosis).

NORMAL ANION GAP MA

- Subsiding DKA.
- Renal tubular abnormalities (renal tubular acidosis).
- Hypoaldosteronism.
- Acute diarrhoea.
- Ureterosigmoidostomy.
- Acetazolamide.

CLINICAL FEATURES

- Hyperventilation of Kussmaul type.
- Circulatory insufficiency: may be a late feature e.g. in DKA.
- Confusion, stupor, coma.
- Signs and symptoms of underlying cause.
## Diagnosis

$H^+ > 45$ pH $< 7.35$, standard base deficit $> -5$mmol/l.

### Diagnostic investigations: will depend on circumstances

- Glucose
- U&Es
- Blood for ketones.
- Blood lactate.
- Toxins: ethylene glycol, methanol, paracetamol, salicylate, ethanol.

## MANAGEMENT

- ABCDE
- Correct hypoxia.
- Correct circulatory abnormalities: see Chapter 2.
- Treat specific causes (see below) e.g. infection, DKA.
- Poisoning: methanol, ethylene glycol, salicylate, paracetamol seek expert advice.
- IV sodium bicarbonate is seldom indicated unless renal failure or specific poisoning.
- Bicarbonate loss from gut or in renal tubular acidosis: correct cause, replace fluid and electrolyte losses (especially potassium) and infuse sodium bicarbonate 1.26% titrated.

> Sodium bicarbonate use should be limited to patients WITHOUT tissue hypoxia as it has many detrimental effects in anaerobic lactic acidosis.
MANAGEMENT OF DIABETIC KETOACIDOSIS

DIAGNOSIS

- Elevated plasma and/or urinary ketones.
- Metabolic acidosis (raised $H^+$/low serum bicarbonate).

Remember that hyperglycaemia, although usually marked, is not a reliable guide to the severity of acidosis, and in children, pregnant women, malnourished or alcoholic patients, blood glucose may not be very raised.

The degree of hyperglycaemia is not a reliable guide to the severity of the metabolic disturbance in DKA.

The presence of the following features should alert you to the possibility of DKA:
- Intra- and extra-vascular volume depletion with reduced skin turgor, tachycardia and hypotension (late feature).
- Rapid and deep sighing respirations, smell of ketones.
- Ketonuria
- Vomiting/abdominal pain.
- Drowsiness/reduced conscious level.

Remember:
- Consider DKA in any unconscious or hyperventilating patient.
- Patients with adverse clinical signs (on the SEWS chart) or signs of cerebral oedema (see below) should be discussed immediately with senior medical staff.
- These guidelines refer to adult patients. All patients under the age of 16 should be discussed with the paediatric diabetes team at the Sick Children’s hospital and arrangements made for transfer when clinically appropriate.

RIE/WGH/SJH have an integrated care pathway which should be adhered to. The following is the RIE/WGH protocol. The SJH protocol differs slightly.

IMMEDIATE MANAGEMENT - WITHIN THE FIRST HOUR

Initial Assessment and Treatment
- Airway and breathing - correct hypoxaemia.
- IV access.
• Monitor respiratory rate, ECG, O₂ saturations, pulse rate, BP, respiratory rate, conscious level and fluid balance.
• Perform laboratory blood glucose, bedside BM, urea and electrolytes, serum bicarbonate, arterial blood gases.

**Fluid Replacement**
• Commence fluid therapy with 0.9% saline 1 litre over 1 hour. A specimen IV fluid regime is shown below.

**Intravenous Insulin**
• Prepare intravenous insulin infusion (see below) and commence at 6 units/hr.

**Other Interventions/Actions**
• 12 lead ECG
• NG tube if impaired consciousness or protracted vomiting.
• Urinary catheter if oliguric.
• Admit patient to a high dependency area.
• Consider central line if clinically indicated.
• Call the diabetes registrar and/or senior medical staff.

---

**ONGOING MANAGEMENT - HOURS 2-4**

**Reassess patient regularly and monitor vital signs**

**Intravenous fluids**
• Aim to rapidly restore circulating volume and then gradually correct interstitial and intracellular fluid deficits.
• Use isotonic saline (see example below) - infusion rates will vary between patients, remember risk of cardiac failure in elderly patients.
• If hypotension (SBP <100mmHg) or signs of poor organ perfusion are present, use colloid to restore circulating volume.

- 1000mls 0.9% NaCl over 2nd hour
- 500 mls 0.9% NaCl over 3rd hour
- 500 mls 0.9% NaCl over 4th hour

• Add in 10% dextrose once blood glucose ≤14mmol/l. Infuse at 100 mls/hr. **Do not alternate saline and dextrose.**
• Measure U&Es and *venous* bicarbonate at the end of hour 2 and hour 4.
Electrolyte replacement

- Despite a considerable total body potassium deficit (300 - 1000 mmol/l), plasma potassium levels are usually normal or high at presentation because of acidosis, insulin deficiency and renal impairment.
- Potassium concentration will fall following commencement of treatment; expect to have to give plenty of potassium.
- Target potassium concentration is 4.0-5.0mmol/l.

Severe hypokalaemia complicating treatment of DKA is potentially fatal and is avoidable.

Potassium Replacement

No potassium in the first litre unless known to be < 3.0 mmol/l. Thereafter, replace potassium as below:

<table>
<thead>
<tr>
<th>Plasma Potassium</th>
<th>Potassium Added</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3.5 mmol/l</td>
<td>40* mmol/l</td>
</tr>
<tr>
<td>3.5 – 5.0 mmol/l</td>
<td>20 mmol/l</td>
</tr>
<tr>
<td>&gt;5.0 mmol/l, or anuric</td>
<td>No supplements</td>
</tr>
</tbody>
</table>

* must be given in one litre of fluid; avoid infusion rates of KCL >10mmol/hr

- Occasionally Infusion rates of over 10mmol/hr may be required. If so senior medical staff should decide this and ECG monitoring is mandatory.
- 40mmol of potassium should be diluted in 1 litre of fluid if given by peripheral cannula. Use pre-prepared bags with KCl.

Blood Glucose and Insulin

- Hourly laboratory glucose.
- Aim to ensure a gradual reduction in blood glucose over the first 12-24 hours. There is no specific evidence to avoid rapid rates of fall (e.g. >5mmol/hr), but there are some observational data to suggest that excessive rates of fall may be associated with cerebral oedema.
- The target blood glucose concentration for the end of the first day is 9-14 mmol/l.
- Make up an infusion of 50 units of soluble insulin (e.g. Humulin S or Actrapid) in 50 mls 0.9% saline (1 unit/ml) and infuse using a syringe driver.
Rate of Insulin Infusion

- 6 units/hr initially.
- 3 units/hr when blood glucose ≤14 mmol/l.

If plasma glucose does not fall in the first hour, the rate of infusion needs increased - phone the diabetes registrar and/or senior medical staff for advice.

- If blood glucose falls below target (i.e. <9 mmol/l) on 3 units/hr, reduce insulin infusion to 2 units/hr. Do not reduce the insulin infusion rate below this. If glucose continues to fall, increase the infusion rate of dextrose or the concentration. Discuss with the diabetes registrar and/or senior medical staff.
- Remember that intravenous insulin has a half-life of 2.5 minutes. It is important that the insulin infusion is not interrupted.

Consider Precipitating Factors:

If indicated check:

- FBC
- CXR
- ECG
- urine gram stain and culture
- blood cultures and other infection screen

Correction of acidosis

- Volume resuscitation and insulin infusion will correct metabolic acidosis in the majority.
- Ketonaemia typically takes longer to clear than hyperglycaemia.

Intravenous sodium bicarbonate should not be used routinely and certainly not without discussing with a senior doctor (no evidence that it is effective).

Other measures

- Urinary catheter: if cardiac failure, persistent hypotension, renal failure or no urine passed after 2 hours.
- CVP line: consider if elderly with concomitant illness, cardiac failure or renal failure.
- Give standard venous thromboembolism prophylaxis according to local protocols: exclude coagulopathy.
- Antibiotics: only if infection is proven or strongly suspected. Remember that raised WBC and fever occur with metabolic acidosis.
- Screen for myocardial infarction if > 40 years old.
**Fluids and Electrolytes**
- Allow oral intake if swallowing safe and bowel sounds present.
- Measure U&Es and venous bicarbonate twice daily, until bicarbonate within the normal reference range.
- Continue with 0.9% saline ≤250ml/hour until bicarbonate is in the reference range and the patient is eating.
- Continue potassium infusion until target is maintained.

**Insulin and Dextrose**
- A blood glucose meter can be used to monitor blood glucose concentration if the previous laboratory blood glucose is <20 mmol/l.
- Pre-meal subcutaneous soluble insulin should be administered to patients who are eating, even when on intravenous insulin. Discuss the doses with the diabetes team.
- Maintain IV insulin (minimum rate 2 units/hr) and 10% dextrose infusion (100ml/hr) until biochemically stable and patient has eaten at least two meals. In such circumstances, stop IV insulin 30 minutes after subcutaneous insulin.

**CONTINUING CARE**
- Ensure patient is reviewed by the diabetes team on the day following admission (at the very latest), so that the cause of the DKA can be elucidated, appropriate education be given and follow up arranged.
- Ensure patient is reviewed on the day following admission, so that the cause of the DKA can be elucidated, appropriate education be given and follow up arranged.
- Ensure that a copy of the discharge summary is sent to the diabetes team.

**ACUTE COMPLICATIONS OF DKA**
- Hypokalaemia: due to inadequate potassium replacement and predictable due to insulin and fluid administration and resolution of acidosis. Avoid by regular monitoring of electrolytes and appropriate potassium replacement.
- Hypoglycaemia: due to over treatment with insulin.
- Hyperglycaemia: due to interruption or discontinuation of intravenous insulin after recovery without subsequent coverage by subcutaneous insulin - always ask advice of diabetes team.
• Cerebral oedema: rare but potentially fatal. More common in children, but is seen in young adults. Characteristically, the patient has initially responded well to treatment prior to the development of severe headache and neurological deterioration. **Get urgent senior help: call ICU.** Treatment depends on clinical state and includes mannitol 0.5 - 2 g/kg body weight.

• ARDS: suspect if dyspnoea, tachypnoea, central cyanosis and non-specific chest signs. Manage ABCDE and call ICU.

• Thromboembolism - prevention and management as standard.

**MANAGEMENT OF DIABETIC HYPEROSMOLAR NON-KETOTIC SYNDROME**

• Common in frail elderly.

• High mortality (30%).

• May be previously undiagnosed diabetes, but can also develop in people with known type 2 diabetes.

• Significant hyperglycaemia: ketonuria and acidosis are usually absent.

• Acute intercurrent illness is common.

**DIAGNOSIS**

Typical features include:

• Severe hyperglycaemia (>50 mmol/l).

• Hyperosmolality (>320 mosmol/kg) with profound dehydration and prerenal uraemia.

• Depression of the level of consciousness; coma is well recognised.

**Plasma osmolality**

\[
2 \times (Na + K) + \text{urea} + \text{glucose (all mmol/l)}
\]

normal range is 280 – 300 mosmol/kg

**IMMEDIATE MANAGEMENT - WITHIN THE FIRST HOUR**

**Initial Assessment**

• Airway and breathing ensure airway and correct hypoxaemia.

• IV access.

• Monitor respiratory rate, ECG, \(O_2\) saturations, pulse rate, BP, conscious level and fluid balance.
• Laboratory blood glucose, bedside BM, urea and electrolytes, serum bicarbonate, arterial blood gases.

**Fluid Replacement**
• Commence rehydration with 0.9% saline 1000 ml over one hour.

**Intravenous Insulin**
• Prepare intravenous insulin infusion (see below) and commence at 3 units/hr.

**Other Interventions/Actions**
• Admit patient to a high dependency area.
• **Call the diabetes registrar/senior medical staff.**
• NG tube if impaired consciousness or protracted vomiting.
• Catheter if oliguric.
• Consider central line if clinically indicated.

---

**ONGOING MANAGEMENT - HOURS 2-4**

**Reassess patient regularly and monitor vital signs**

**Intravenous fluids**
• Aim to rapidly restore circulating volume and then gradually correct interstitial and intracellular fluid deficits.
• Use isotonic saline (see example below) - infusion rates will vary between patients, remember risk of cardiac failure in elderly patients.
• If serum sodium exceeds 155mmol/l, use 0.45% saline instead of isotonic. **Discuss with diabetes registrar/senior medical staff.**

- 500 mls saline over 2nd hour
- 500 mls saline over 3rd hour
- 500 mls saline over 4th hour

• If hypotension (SBP <100 mmHg) or signs of poor organ perfusion are present, use colloid to restore circulating volume.
• Add in 10% dextrose once blood glucose ≤15mmol/l. Infuse at 125-250 mls/hr. **Do not alternate saline and dextrose.**
• Measure U&Es and serum osmolality at the end of hour 2 and hour 4.

**Electrolyte Replacement**
• Target potassium concentration is 4.0-5.0mmol/l.
Potassium Replacement

No potassium in the first litre unless known to be < 3.0 mmol/l.
Thereafter, replace potassium as below:

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* must be given in one litre of fluid; avoid infusion rates of KCL >10 mmol/hr

Occasionally infusion rates of >10 mmol/l are required if so ECG monitoring is mandatory.

Blood Glucose and Insulin

- Hourly laboratory glucose
- Aim to ensure a gradual reduction in blood glucose over the first 12-24 hours. There is no specific evidence to avoid rapid rates of fall (e.g. >5 mmol/hr), but there are some observational data to suggest that excessive rates of fall may be associated with cerebral oedema.
- The target blood glucose concentration for the end of the first day is 10-20 mmol/l.
- Make up an infusion of 50 units of soluble insulin (e.g. Humulin S or Actrapid) in 50 mls 0.9% saline (1 unit/ml) and infuse using a syringe driver.
- 3 units/hr initially

If plasma glucose does not fall in the first hour, the rate of infusion needs increased - phone the metabolic registrar for advice.

- If blood glucose falls below target (i.e.<10 mmol/l) on 3 units/hr, the insulin infusion can be reduced to a minimum of 1 unit/hr. **Do not reduce the insulin infusion rate below this.** If glucose continues to fall, increase the infusion rate of dextrose or the concentration. Discuss with the metabolic/diabetes registrar.
- Remember that intravenous insulin has a half-life of 2.5 minutes. It is important that the insulin infusion is not interrupted.

Consider Precipitating Factors:
- FBC
• CXR
• ECG/MI screen
• Urine gram stain and culture.
• Blood cultures and other infection screen.

Other measures
• Urinary catheter: if cardiac failure, persistent hypotension, renal failure, no urine passed after 4 hours or impaired consciousness.
• CVP line: consider if elderly with concomitant illness, cardiac failure or renal failure.
• Thromboembolic complications are common, however full anticoagulation has been associated with a high risk of GI bleeding. Patients should receive DVT prophylaxis with LMWH, rather than unfractionated heparin (unless renal impairment) and should have TED stockings (unless contra-indicated).
• Nasogastric tube: if consciousness is impaired, to avoid aspiration of gastric contents.
• Antibiotics: low threshold for use.

SUBSEQUENT MANAGEMENT - 4 HOURS+

Fluids and Electrolytes
• Allow oral intake if swallowing safe and bowel sounds present.
• Measure U&Es twice daily, until within the normal reference range (or back to usual baseline for that patient).
• Continue with isotonic saline \( \leq 250\text{ml/hour} \) until U&Es back to baseline and the patient is eating.
• Continue potassium infusion until target is maintained.

Insulin and Dextrose
• A blood glucose meter can be used to monitor blood glucose concentration if the previous laboratory blood glucose is \(<20 \text{ mmol/l}.\)
• Maintain IV insulin (minimum rate 2 units/hr) and 10% dextrose infusion (250ml/hr) until biochemically stable and patient has eaten at least two meals. It is not necessarily the case that the patient will require subcutaneous insulin; the need for sc insulin or oral hypoglycaemic therapy should be discussed with the diabetes team.

Continuing Care
• Ensure patient is reviewed by the diabetes team prior to discharge,
so that the cause of the HONK can be elucidated, appropriate education be given and follow up arranged.

- Patient should not be discharged until biochemically normal, eating normally and established on appropriate therapy.
- Ensure that a copy of the discharge summary is sent to the diabetes team.

HYPOGLYCAEMIA

PERI-OPERATIVE MANAGEMENT OF DIABETIC PATIENTS

General Principles

Plan Ahead
Admit 1 day before elective surgery for:
- full assessment of risk factors, baseline biochemistry, glucose profile, ECG.
- optimisation of metabolic control
- formulation of peri-operative management plan with Diabetic Registrar
- Schedule the patient for surgery (whenever possible) early in the morning and first on the list.
- Discuss all patients with the anaesthetist and remember that the Diabetes Team are ALWAYS available to give you help/advice (Page #6800 RIE, WGH via switchboard, SJH Diabetes consultants).
- If patients have poor metabolic control but require emergency surgery, discuss with the Diabetes Team.

WHICH PATIENTS NEED PERI-OPERATIVE INSULIN?

- All outpatients being treated with insulin
- All patients having major surgery (most abdominal and thoracic procedures)
- Any traumatic procedure especially in poorly controlled patients
- All patients undergoing emergency surgery
- All who are acutely ill

HOW SHOULD THE INSULIN BE ADMINISTERED?

GKI or Sliding Scale
The precise method should be discussed with the anaesthetist but is likely to be either GKI or sliding scale.
Continuous infusion of glucose (G), potassium (K), and insulin (I) i.e. a GKI regimen - according to the guidelines set out below.

**Proforma for GKI regimen**

1. On the morning of operation, omit breakfast and do not give subcutaneous insulin.

2. Before 0800hr measure blood glucose on the ward and send an urgent blood sample to Clinical Chemistry for plasma urea, electrolytes and glucose determinations.

3. If blood glucose < 10 mmol/l commence infusion with
   - 16 units soluble insulin e.g. Human ACTRAPID
   - 10 mmol/l KCl
   - in 500 ml 10% Dextrose at 100 ml/hr

4. Less insulin (i.e. start with 12 units) is required in
   - thin elderly patients
   - those on less than 30 units/day at home
   - those who have had previous total pancreatectomy

5. More insulin (i.e. begin with 20 units) is required in
   - patients requiring high insulin doses previously ( > 1 unit/kg/day)
   - patients with intercurrent infection
   - some endocrine (e.g. acromegaly) and metabolic disorders

6. If the blood glucose is > 12 mmol/l and rising, the insulin in the infusion should be increased by 4 units (**THIS REQUIRES A NEW BAG**)  

7. If the blood glucose is 6 mmol/l and falling the insulin in the infusion should be reduced by 4 units (**THIS REQUIRES A NEW BAG**).

**If GKI is continued beyond 18 hours**

- monitor Urea and Electrolyte levels daily
- adjust K supplement to maintain normokalaemia
- watch for water overload causing dilutional hyponatraemia
- less fluid can be given if 20% dextrose is used with double the insulin dose but local phlebitis may occur.
- Long term GKI is therefore best given through a central venous catheter.

**Stopping the GKI**

- The infusion should be continued until one hour after patient’s first post-operative meal.
- Subcutaneous insulin is given with this meal - with at least as intensive a regimen as pre-operatively.
Remember, intravenous insulin has a half-life of only 2.5 minutes, so if intravenous insulin is disconnected for any appreciable length of time, hyperglycaemia will quickly ensue (unless subcutaneous insulin has been given).

The insulin infusion is prepared by adding 50 Units of Actrapid insulin to 0.9% saline in a syringe to a total volume of 50ml. Thus, 1ml of the solution contains 1 unit of insulin. The doses of insulin are adjusted according to a sliding scale, which is prescribed as below.

<table>
<thead>
<tr>
<th>BG (mmol/l)</th>
<th>Insulin infusion (Units actrapid/hour = ml/hour)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;16</td>
<td>6 (test urine for ketones, call Dr as the sliding scale may need revision)</td>
</tr>
<tr>
<td>13-15.9</td>
<td>4</td>
</tr>
<tr>
<td>10-12.9</td>
<td>3</td>
</tr>
<tr>
<td>7.0-9.9</td>
<td>2</td>
</tr>
<tr>
<td>5.0-6.9</td>
<td>1</td>
</tr>
<tr>
<td>4.0-4.9</td>
<td>0.5</td>
</tr>
<tr>
<td>&lt;4</td>
<td>off (call Dr, the sliding scale may need revision)</td>
</tr>
</tbody>
</table>

- Capillary blood glucose should be tested every hour. It is crucial that medical staff monitor the pattern of blood glucose every 2-4 hours as the sliding scale may require modifications to ensure that blood glucose concentrations remain between 5 and 10 mmol/l.
- Commence glucose infusion with 20 mmol/l of KCl - infusion should run at 50ml/hr. Usually this will be 5% or 10% glucose, but in some special circumstances (e.g neurosurgery) an infusion of 5% glucose/0.45% saline is preferred. The anaesthetist will advise which glucose infusion should be used. If the patient is very hyperglycaemic, the glucose infusion should be deferred until the intravenous insulin has lowered the blood glucose to <14 mmol/l.
- The insulin and glucose infusions should both be given through the same IV cannula, rather than separate cannulae, to avoid the danger of a blocked cannula resulting in only one of the two being given.
- The insulin syringe should be attached to a ‘PCA giving set’, incorporating a Y-connector with a one-way valve for attaching the glucose infusion. The one-way valve prevents insulin being pumped backwards into the glucose giving set.
- Inform anaesthetist if blood glucose is less than 4 mmol/l or greater than 16 mmol/l.
• If capillary blood glucose is greater than 20 mmol/l, take blood for urgent laboratory glucose and U&E’s (including venous bicarbonate).

• If blood glucose concentrations are stable and in the desired range, the frequency of monitoring may be reduced, e.g.to 2 hrly.

• U&E’s and a laboratory glucose should be checked daily while the patient is on intravenous insulin/glucose.

• Be prepared to vary the KCl content of the intravenous fluids according to plasma K⁺ levels. Be especially careful in patients with renal impairment.

• If patient is on intravenous insulin and glucose for greater than 24hrs, ensure that Hartman’s solution is also given to avoid hyponatraemia. Remember 10% glucose is hypertonic. This glucose infusion is not designed for volume replacement, but glucose control. Extra fluids such as Hartmann’s will invariably be required and these can be piggy-backed in through a separate IV infusion line. However, if patients are volume overloaded, discuss management with the anaesthetist and/or diabetes registrar.

DIABETIC PATIENTS NOT REQUIRING INSULIN

Patients undergoing relatively minor procedures (e.g. hernia repair, laparoscopic cholecystectomy)

• Treatment is simpler if insulin is not required but frequent blood glucose monitoring is still essential.

• Check blood glucose pre-operatively to confirm that level < 10 mmol/L (lab analysis).

• Omit usual oral hypoglycaemic agent(s).

• Avoid IV glucose infusion

• On return from theatre repeat blood glucose. If > 15 mmol/l, insulin may be required.

CAUTION -

Diabetic patients on metformin are at risk of acute renal failure from radiological investigations where intravascular contrast material is given. (CT scan, IVP, angiogram, CTPA etc). Metformin should be omitted before the procedure and for 48 hours after. Careful watch is to be kept on the renal function and it should be ensured that patients remain well hydrated.
HYPOGLYCAEMIA

INTRODUCTION

• Complication of diabetes most feared by patients.
• Mild hypoglycaemia common in diabetic patients on insulin and is usually managed by themselves.
• Severe hypoglycaemia is that requiring help from another person to treat it.
• Unconsciousness caused by hypoglycaemia in hospital setting requires parenteral treatment.
• Hypoglycaemia is implicated in 4% of deaths in diabetics under the age of 50 years.

RECOGNITION AND DIAGNOSIS

• Defined arbitrarily as laboratory blood glucose < 3.5 mmol/l.
• Always confirm hypoglycaemia with a laboratory measurement, but treat on basis of BM while awaiting lab result.
• Symptoms of hypoglycaemia are age specific, with behavioural change being common in children and neurological symptoms prominent in the elderly - always check blood glucose in patients with suspected stroke or altered conscious level (including confusion).
• Most patients presenting with hypoglycaemia will be on insulin or sulphonylurea drugs, e.g. gliclazide.

AETIOLOGY

In patients with diabetes mellitus on insulin or sulphonylureas (not biguanides i.e. metformin or thiazolidindione) common causes include:

• Lack of food.
• Unaccustomed exercise.
• Alcohol
• Excess insulin.
• May be more than one of these factors. About 25% of Type 1 diabetic patients have reduced/lost awareness which increases the risk of severe hypoglycaemia.
## COMMON SYMPTOMS OF HYPOGLYCAEMIA

<table>
<thead>
<tr>
<th>Autonomic</th>
<th>Neuroglycopenic</th>
<th>Non-specific</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sweating</td>
<td>Weakness</td>
<td>Headache</td>
</tr>
<tr>
<td>Trembling</td>
<td>Visual disturbance</td>
<td>Nausea</td>
</tr>
<tr>
<td>Pounding heart</td>
<td>Difficulty speaking</td>
<td></td>
</tr>
<tr>
<td>Anxiety</td>
<td>Tingling</td>
<td></td>
</tr>
<tr>
<td>Hunger</td>
<td>Dizziness</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Difficulty concentrating</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tiredness</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Drowsiness</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Confusion</td>
<td></td>
</tr>
</tbody>
</table>

## UNUSUAL ASSOCIATIONS AND PRESENTATION OF HYPOGLYCAEMIA

<table>
<thead>
<tr>
<th>Cardiovascular</th>
<th>Neuropsychological</th>
<th>General</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolongation of QT-interval</td>
<td>Focal/generalised convulsions</td>
<td>Fracture of long bones/vertebrae</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>Coma</td>
<td>Joint dislocation</td>
</tr>
<tr>
<td>Non-sustained ventricular tachycardia</td>
<td>Stroke; TIA's</td>
<td>Soft tissue injury</td>
</tr>
<tr>
<td>Silent myocardial ischaemia</td>
<td>ataxia, choreoathetosis</td>
<td>Head injury</td>
</tr>
<tr>
<td>Angina</td>
<td>Focal neurological deficits</td>
<td>Burns</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>Decortication</td>
<td>Hypothermia</td>
</tr>
<tr>
<td>Sudden death</td>
<td>Cognitive impairment</td>
<td>Road traffic accidents</td>
</tr>
</tbody>
</table>

## MANAGEMENT

- Maintain ABCDE and oxygenation whilst correcting hypoglycaemia (especially airway).
- **ALWAYS** confirm hypoglycaemia with a laboratory measurement, but treat on basis of BM whilst awaiting lab result.
• If patient not known to have diabetes, or deliberate overdose of insulin/sulphonylurea suspected, take blood for assay for insulin and c-peptide concentrations.

**Mild or severe but conscious**

- 2-4 dextrose tablets or
- small glass of carbonated sugar-containing drink
- If no improvement within 5-10 mins, repeat
- If next meal not imminent, longer acting carbohydrate should be administered, e.g. biscuit, sandwich, fruit

**Severe and unconscious**

- IV dextrose
  - 125mls 20% dextrose, 250mls 10% dextrose or 500mls 5% dextrose*
  - OR
- Glucagon 1 mg IM (not if liver disease/alcoholism)

*Glucagon is effective almost as quickly as dextrose but may not work in alcohol related hypoglycaemia, in liver disease or in prolonged hypoglycaemia. Occasionally causes vomiting, abdominal pain, diarrhoea. Dextrose infusion 10-20% IV may be needed especially when a long acting insulin or oral hypoglycaemic agent is responsible.

Give oral starchy carbohydrate within 10-30 mins of glucagon to replenish liver glycogen stores and prevent recurrent hypoglycaemia.

• In the situation of a massive insulin overdose with a long acting preparation the injection site can (occasionally) be surgically removed.

Recovery from hypoglycaemia may be delayed if:

- hypoglycaemia has been prolonged or severe.
- an alternative cause for impairment of consciousness co-exists, e.g. stroke or drug overdose.
- patient is post-ictal (convulsion caused by hypoglycaemia).

**Follow up**

- Think of the causes of hypoglycaemia.
- Why has it occurred?
- If patient recovers quickly then admission is rarely indicated (unless sulphphonylurea induced hypoglycaemia) but ensure that adequate follow up is arranged through the diabetes team.
Always discuss with the metabolic registrar the management and follow up of patients admitted with hypoglycaemia. However, it is important to elucidate the reason for hypoglycaemia. The most common cause for hypoglycaemia is patient error, i.e. too much insulin or not enough carbohydrate. Others include:

- Excessive exercise (hypoglycaemia can be early or occur the following day).
- Excess alcohol (inhibits hepatic gluconeogenesis).
- Renal failure (insulin and sulphonylureas undergo renal clearance).
- Development of coincidental endocrine disease, e.g. Addison’s disease (weight loss, anorexia, skin pigmentation, postural hypotension, hyponatraemia, hyperkalaemia etc), hypopituitarism, hypothyroidism.
- Malabsorption and gastroparesis, e.g. coeliac disease (weight loss, abdominal pain, bloating, loose stools, glossitis, aphthous ulceration, anaemia, hypoalbuminaemia etc).

### Risk factors for severe hypoglycaemia

- Intensive insulin therapy
- Low HbA1c
- Previous history of severe hypoglycaemia
- Long duration of diabetes
- Impaired awareness of hypoglycaemia
- Irregular lifestyle
- Alcoholism or binge drinking

### Risk factors for sulphonylurea-induced hypoglycaemia

- Age (not dose of drug)
- Impaired renal function
- Previous history of cardiovascular disease or stroke
- Reduced food intake; diarrhoea
- Alcohol
- Adverse drug interactions
- Use of long-acting sulphonylureas
- Recent hospital admission
**SULPHONYLUREA-INDUCED HYPOGLYCAEMIA (SIH)**

- Mild SIH is treated in a similar way to insulin-induced hypoglycaemia (see above).
- Sulphonylurea-induced hypoglycaemic coma requires intravenous dextrose and treatment in hospital because relapse after initial treatment is well recognised. An intravenous bolus of glucose stimulates insulin secretion, especially in individuals who have retained pancreatic beta-cell function, and many people will require an ongoing intravenous infusion of 10% dextrose to sustain the blood glucose concentration above 5.0 mmol/l. Inform diabetes team.

> All of these patients should be admitted.

**HYPERCALCAEMIA**

Severe hypercalcaemia (corrected calcium >3.0mmol/L) is uncommon, and usually due to hyperparathyroidism, or malignancy (e.g. myeloma). Symptoms may be masked by underlying malignancy. In any unwell patient with known malignancy check the serum calcium, and albumin.

**CAUSES OF HYPERCALCAEMIA**

- Primary hyperparathyroidism.
- Malignancy: solid tumours with metastases to bone; tumours secreting PTH or PTHRP (usually squamous carcinomas); haematological malignancy.
- These two causes account for >80% of cases.
- Familial hypocalciuric hypercalcaemia.
- Sarcoidosis, granulomatous disease.
- Endocrine: thyrotoxicosis; Addison’s disease; phaeochromocytoma.
- Milk-alkali syndrome.
- Immobilisation (<16 yr old).
- Meds: Vit D analogues, anti-oestrogens, lithium, thiazides.

**SYMPTOMS**

- Thirst
- Polyuria
- Constipation
- Nausea and anorexia
- Abdominal pain
• Depression
• Confusion

COMPLICATIONS

• Peptic ulceration
• Acute pancreatitis
• Muscle weakness
• Psychosis, drowsiness, coma
• Corneal calcification
• Short QT interval on ECG

INVESTIGATIONS

All patients
• FBC, ESR
• U&Es
• Ca++, PO₄, Mg++, ionised Ca++, albumin
• ALP, LFT’s
• ECG
• CXR
• Parathyroid hormone

Specific depending on the history.
• Myeloma screen and skeletal survey.
• Bone scintigraphy.
• Thyroid function tests.
• Serum ACE.
• 24hr urine for calcium and creatinine.
• Short Synacthen test.

TREATMENT

Calculate corrected calcium or refer to ionised value. Emergency treatment is required if corrected calcium >3.5 mmol/l (ionised>1.8 mmol/l). Between 3 and 3.5mmol/L may not require emergency treatment, but this depends on signs and symptoms.

For each 1g the albumin is below 40g/L add 0.02mmol/L to the uncorrected calcium e.g. calcium 2.62mmol/L with an albumin of 30g/L gives a corrected calcium of 2.62 + (10 x 0.02)= 2.82mmol/L.
Fluid
- Urgent fluid replacement with 0.9% saline (add potassium chloride as required) will lower calcium, and enhance renal clearance.
- Check U&E’s and calcium twice daily.

Diuretics
- Loop diuretics (e.g. furosemide 40mg IV bd) will enhance calcium loss in the urine. DO NOT start until fluid deficits rectified.
- NEVER use thiazides as they cause calcium retention.

Bisphosphonates
- Ensure fluid deficit corrected first.
- A single infusion of pamidronate (see table) will lower calcium levels within 2 to 4 days (but not acutely).
- Maximal effect is at about 1 week.
- Recurrent hypercalcaemia may be treated with repeated IV infusions of pamidronate.

Other
- If patient is on digoxin, discontinue.
- Steroids should not be used routinely. May be helpful in sarcoidosis, myeloma and hypervitaminosis-D (prednisolone 60-80mg oral daily).

**PAMIDRONATE DOSE TABLE**

<table>
<thead>
<tr>
<th>Serum calcium (mmol/L)</th>
<th>Dose of pamidronate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3.0</td>
<td>15mg</td>
</tr>
<tr>
<td>3.0-3.5</td>
<td>30mg</td>
</tr>
<tr>
<td>3.6-4.0</td>
<td>60mg</td>
</tr>
<tr>
<td>&gt;4.0</td>
<td>90mg</td>
</tr>
</tbody>
</table>

If creatinine clearance >30ml/min infuse at rates up to 60mg/hr. If creatinine clearance <30ml/min administer maximum rate of 20mg/hour (4 hr 30mins for 90mg) but *do not reduce dose*.

**HYPOCALCAEMIA - THE 5 COMMON CAUSES**

- Spurious hypocalcaemia, that is failure to correct for low albumin (check ionised calcium). Add 0.02 mmol/l to the total calcium for each g/l albumin is below 40 g/l.
- Hypoparathyroidism, surgical.
• Renal failure.
• Vitamin D deficiency.
• Hypomagnesaemia.

**CLINICAL FEATURES**

Mild hypocalcaemia may be asymptomatic.

**Early features**
• Anxiety and nervousness.
• Paraesthesiae around the mouth, in toes and fingers.

**Late features (esp. if total Ca$^{++} < 1.9$ mmol/l)**
• Convulsions
• Prolonged QT interval on ECG.
• Papilloedema
• Muscle cramps
• Muscle twitches
• Chvostek’s sign
• Trousseau’s sign (carpal spasm).

**INVESTIGATIONS**

• Total and ionised calcium, albumin, phosphate, magnesium.
• U&Es
• 25(OH)$_2$D$_3$ and 1,25(OH)$_2$D$_3$, PTH and alkaline phosphatase may help establish aetiology.

**EMERGENCY TREATMENT**

• Required for severe complications e.g. fits, dysrrhythmias, tetany.
• Monitor ECG.
• 5-10mls calcium chloride 10% or calcium gluconate 10% IV over 15 minutes will reverse tetany. Calcium chloride is immediately available in minijet form, but ampoules of calcium gluconate are available for injection and the preparation of infusions.
• Follow up: slow IV infusion at 0.5-2mg Ca/kg/hr (0.06 -0.22mls/kg/hr) as calcium gluconate 10%; dilute 60mls calcium gluconate in 1 litre 5% dextrose. (10% calcium gluconate contains 8.9 mg elemental Ca$^{++}$/ml).
• Oral calcium: introduce as below +/- vitamin D as soon as possible.
Hypomagnesaemia may be the cause. Emergency treatment is with IV magnesium. Hypomagnesemia is caused by chronic alcoholism, malabsorption, cyclosporin treatment, prolonged parenteral nutrition or diuretic therapy.

Treat convulsions and arrhythmias with magnesium sulphate IV: 8 mmols magnesium sulphate diluted in 100 ml 0.9% NaCl and infused over 20 minutes. Monitor ECG. May cause hypotension.

TREATMENT OF MILD AND MODERATE CASES

Mildly symptomatic or asymptomatic patients with chronic hypomagnesaemia, oral replacement can be tried but may be unsuccessful due to diarrhoea. Magnesium glycerophosphate is used most commonly, however this is an unlicensed medicine and should be discussed with the appropriate consultant first.

Primary hypocalcaemia: oral or IV calcium +/- vitamin D will be required. Therapeutic target is low normal calcium. Oral calcium is administered as calcichew (2-3 tablets daily) or sandocal 400 (1-4 tablets daily) or sandocal 1000 (1-2 tablet daily). Vitamin D as Alfa calcidol usual dose is 0.25 - 1 microgram per day.

Chronic asymptomatic hypocalcaemia: may need larger doses of oral calcium up to 7g per day in multiple divided doses. Vitamin D usually needed (0.25-1 microgram per day). Use shorter-acting vitamin D analogues as that will make reversal easier if any toxicity/hypercalcaemia.

HYPOKALAEMIA

Potassium <3.5mmol/l

GENERAL

- Common
- Rarely an emergency, except in diabetic ketoacidosis, or arrhythmias.
- Common causes are GI losses, vomiting and diuretics.
- Oral replacement is preferred.
- IV treatment required if patient vomiting, NBM, or with cardiac arrhythmia.
**TREATMENT**

**Oral**
- Sando K 2-3 tablets oral bd, or tds. Avoid Slow K, especially in the elderly as it can cause oesophageal erosions and ulcers.
- Add a potassium sparing diuretic if diuretic induced.
- Monitor potassium levels daily and review dose regularly.

**Intravenous**

NEVER administer stat or undiluted.

- Maximum concentration is 40 mmol/L usually over 4hrs.
- Higher concentrations (e.g. 80 mmol/L) may be given centrally, but the rate must not exceed 20 mmol/hr (with continuous ECG monitoring).
- **Caution:** monitor serum potassium levels to ensure hyperkalaemia does not occur, especially in patients with renal impairment.
- Use pre-prepared bags to minimise risk of error (wherever possible).
- Serum potassium concentration is a poor reflection of total body potassium (frequently much lower). Seemingly large quantities may be required e.g. in DKA.
- If difficulty replacing potassium is experienced, check serum magnesium. May be low, especially in alcoholics, and patients on diuretics.
- Hypomagnesaemia impairs potassium retention by the kidney.

**ADDISON’S DISEASE**

**SYMPTOMS AND SIGNS**

- Weight loss
- Pigmentation
- Abdominal pain
- Vomiting, diarrhoea
- Fatigue
- Postural hypotension
- Shock
LABORATORY INVESTIGATIONS

- Low Na⁺
- High K⁺
- Metabolic acidosis
- High urea
- Hypoglycaemia
- Hypercalcaemia
- These changes occur late in the disease.

TESTING FOR ADDISON’S DISEASE

A short Synacthen test is useful to confirm the diagnosis and only takes 30 mins. However do not do short Synacthen test in the very ill. Take blood to check cortisol and ACTH then start hydrocortisone treatment.

SHORT SYNACTHEN TEST

- Venous blood sample for baseline cortisol and ACTH.
- Give 250 micrograms Synacthen im (IV if peripherally shutdown).
- Recheck cortisol at 30 minutes.
- A normal response is a 30min cortisol >460nmol/L.

TREATMENT

- Correct hypoxaemia.
- Establish IV access.
- Take a sample for serum cortisol/ACTH.
- 0.9% saline IV with 10% dextrose IV if hypoglycaemic.
- 200mg hydrocortisone IV.
- Then 100mg hydrocortisone IV qds for 48hrs.
- Decrease hydrocortisone dose to 50mg qds the following day and continue to decrease at daily intervals as follows
  - Then 50mg bd iv or oral if well enough
  - Then 25mg bd oral
  - Then 20mg am and 10mg pm (no later than 6pm) oral
  - Then 10mg am and 5mg pm (usual maintenance dose)
- Fludrocortisone 50-100 micrograms oral per day may be required (when total daily hydrocortisone dose is <30mg), as determined by plasma electrolytes, and blood pressure.
• Measurement of plasma renin activity can also be helpful in assessing mineralocorticoid deficiency.
• Discuss with Endocrinology registrar.
• Patients should be advised to wear a medic-alert type bracelet or talisman and should be given a steroid card. Further details can be found in the BNF.
• Advise not to stop steroids unless told to by a doctor. If become unwell steroid dose should be doubled. If vomiting, or diarrhoea contact GP at once.

Management of intercurrent illness in patients requiring glucocorticoid replacement

• Patients with Addison’s disease or ACTH deficiency secondary to pituitary failure are unable to mount an increased cortisol response to stress.
• In mild or moderate illness, patients should double or triple their glucocorticoid replacement for the duration of the illness.
• In severe illness or if vomiting/diarrhoea, iv hydrocortisone is required. 100mg iv qds. NB replace fluid deficit with iv saline as appropriate.
• Decrease back down to usual dose gradually as outlined above.

HYPONATRAEMIA

Seen in 1.5% of hospital admissions.

Mechanism
Dilutional (impaired renal water clearance) or depletional. Often a combination.

Dilutional commoner and seen in:
• Cirrhosis
• Cardiac failure
• Nephrotic syndrome
• Hypothyroidism
• ACTH deficiency
• SIADH: check plasma and urine osmolality urinary sodium, TFT, synacthen test, CXR

Depletional causes:
• Vomiting/ diarrhoea
• Diuretics
- Addison’s disease.
- Renal salt wasting.

**HISTORY, EXAMINATION & INVESTIGATIONS**

- Accurate history to establish rate of onset of symptoms, any obvious cause eg diuretics.
- Assess hydration status.
- Check urine sodium concentration before giving any IV therapy.

### Patients with urinary sodium >30mmol/l are more likely to have dilutional hyponatraemia (exceptions are Addison’s disease and renal salt-wasting and patients receiving diuretic therapy).

- Symptoms and signs relate to the rate of onset more than the degree of fall in sodium.
- Na⁺ <130 mmol/l may give headache with nausea and vomiting and lead to fits, coma and respiratory arrest. This is more likely in women of child-bearing age (16-45 yrs).
- Na⁺ <120 mmol/l is associated with 50% mortality. Chronic development has much lower morbidity and mortality. If chronic even severe hyponatraemia may be asymptomatic.

### Chronic fall to 110mmol/l can be well tolerated, acute fall to 127mmol/l has been fatal.
Flow Chart for the assessment and management of a patient with hyponatraemia*

- *Most difficulty arises in differentiating mild hypovolaemia from euvolaemic, dilutional, hyponatraemia. In both hypovolaemic and euvolaemic hyponatraemia plasma osmolality will be low and the urine will be less than maximally dilute (inappropriately concentrated). Posm/Uosm will rarely help clinical management.
- Monitor the sodium concentrations carefully (every hour if necessary during iv therapy).
- In a sick individual consider Addison’s disease and give parenteral hydrocortisone (100mg) after taking blood for plasma cortisol as glucocorticoids are anticipated to have little toxicity in this acute setting and may be life-saving.
MANAGEMENT

- Depends on degree and rate of fall: in patients with a severe abrupt fall in sodium associated with symptoms rapid correction is well tolerated and beneficial.
- In more chronic onset and without symptoms correction should be much slower.
- Depends on body salt and water status.

EMERGENCY TREATMENT

Prompt treatment is required for patients with neurological signs, and sodium less than 120mmol/L. EMERGENCY treatment is required for seizures. Discuss all such cases with Endocrine Registrar. Consider high dependency or intensive care management early.

- Hyponatraemic encephalopathy: symptomatic and Na <120mmol/l use hypertonic saline aiming to raise the sodium by 3-5mmol in the first instance over 4-6 hours: get senior advice. Sodium should not rise more than 12mmol in 24 hours. In chronic cases, the Na+ increment should be no greater than 8mmol in 24 hours. Hypertonic saline should only be given on advice of endocrine or ICU Reg/Cons. Caution in renal and cardiac disease.
- Treat fits as standard with diazemuls and refer early to ICU.
- Remove cause.
- Fluid restriction ± Demeclocycline where appropriate. Cardiac failure, cirrhosis and nephrotic syndrome: water restrict, diuretics, no improvement with hypertonic saline (can make worse).
- In volume depleted patient give 0.9% saline, and correct hypokalaemia (may benefit from hypertonic saline, but usually respond to isotonic).
- Treat hypothyroidism and Addison’s disease with appropriate hormones.

CRITERIA FOR SIADH

- Plasma sodium <130 mmol/L, urine sodium >30 mmol/L.
- No oedema or hypovolaemia.
- Normal renal, thyroid, and adrenal function.
- No diuretic usage.
This guideline relates to the unrousable, unresponsive patient. There are many causes of coma, (GCS<8) but the initial approach is similar for all of them.

**IMMEDIATE MANAGEMENT**

**Assess the airway and breathing**
- Open airway and stabilise the cervical spine if there is a history of head or neck trauma.
- Give high concentration oxygen and if no breathing ventilate with a bag-valve-mask and 100% oxygen.
- Intubation will often be necessary - seek expert help from an anaesthetist early.

**Assess the circulation**
- Check pulse, perfusion, oxygen saturation and blood pressure.
- Correct hypovolaemia or arrhythmias.
- Obtain large bore IV access.
- Immediate investigation: take blood for BM, full blood count, glucose, electrolytes and toxicology screen.

**Look for evidence of hypoglycaemia**
- Measure glucose rapidly.
- If you believe hypoglycaemia is present give 200-500ml 5% dextrose.
- Give Pabrinex IV HP 1+2 (20ml in 100ml 5% glucose or 0.9% sodium chloride over 30mins, as per LUHD guidelines) to alcoholics or malnourished patients at the same time as glucose.

**IMMEDIATE ASSESSMENT**
- Obtain history from ambulance crew, relatives, partner, friends or GP.
- Record the level of consciousness using the Glasgow Coma Scale, and reassess it frequently.
- Examine pupils, look at eye movements, and look for unilateral weakness (suggesting an intracranial cause such as stroke) by giving a painful stimulus to each limb.
- Examine the rest of the patient carefully looking for pointers to a diagnosis.
FURTHER TREATMENT TO CONSIDER

If you suspect an OPIATE OVERDOSE (drug paraphernalia at scene, track marks, pinpoint pupils, reduced respiratory rate) give NALOXONE as per Toxicology chapter.

If you suspect a BENZODIAZEPINE OVERDOSE (previous prescriptions of benzodiazepines, empty drug boxes, reduced respiratory rate). manage as per Toxicology chapter.

If you suspect MENINGITIS (neck stiffness, rash or fever) give INTRAVENOUS ANTIBIOTICS: Ceftriaxone 2g IV. See meningitis section.

FURTHER INVESTIGATIONS TO CONSIDER

If the cause of the coma is not immediately evident, further investigation is usually required. Consider;

- CT brain: once circulation is stable and the airway is secure
- Drug levels: toxicology screen
- Lumbar puncture: cell count, protein, glucose, culture.

Early advice from a neurologist and/or neurosurgeon and/or intensive care physician may be crucial.

ONGOING CARE OF THE UNCONSCIOUS PATIENT

- Monitoring of conscious level, blood pressure, pulse, ECG and oxygen saturations
- DVT prophylaxis: heparin may be contraindicated
- pressure sore prevention
- nutrition
AETIOLOGY OF COMA

Primary neurological disease
- Trauma
- Intra-cranial haemorrhage: SAH, intra-cerebral, sub/extradural
- Arterial/venous infarction
- Infection: meningitis, encephalitis, cerebral abscess
- Other structural: tumours
- Epilepsy: postictal non-convulsive status epilepticus
- Psychogenic

Secondary to systemic disease
- Toxic (drugs/alcohol)
- Hypoxia/hypercarbia
- Liver or renal failure
- Wernicke’s encephalopathy
- Hypertensive encephalopathy

Metabolic
- Hypoglycaemia
- Hyperglycaemia
- Hyponatraemia
- Hypocalcaemia

GLASGOW COMA SCALE

<table>
<thead>
<tr>
<th>Eye Opening</th>
<th>Best verbal response</th>
<th>Best motor response</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>None</td>
<td>1 None</td>
</tr>
<tr>
<td>2</td>
<td>To pain</td>
<td>2 Sounds only</td>
</tr>
<tr>
<td>3</td>
<td>To voice</td>
<td>3 Incoherent Words</td>
</tr>
<tr>
<td>4</td>
<td>Spontaneous</td>
<td>4 Confused speech</td>
</tr>
<tr>
<td>5</td>
<td>Normal conversation</td>
<td>5 Localises a painful stimulus</td>
</tr>
<tr>
<td>6</td>
<td>Normal</td>
<td></td>
</tr>
</tbody>
</table>

Add up the score for each component of the scale, and report them separately.
Localisation to pain is defined as reaching above the clavicle to a painful stimulus given above the neck.
CLINICAL ASSESSMENT

General medical assessment

Raised intracranial pressure leads to bradycardia and hypertension (Cushing response). Once haemodynamically stable, look for:

- Neck stiffness in flexion of neck: may be absent in deep coma despite meningeal irritation.
- Skin rash: remember conjunctivae, hands and feet (soles).
- Pyrexia
- Medic-alert bracelets.
- Evidence drug abuse (needle tracks).
- Cranial trauma (feel over whole head).

Neurological assessment

- Use sternal and nail bed pressure to elicit response, if no response to verbal stimuli.
- If asymmetrical, score best side, but note asymmetry (lesion localisation).

Pupil size/reactions:

- Pinpoint pupils = opioid OD or pontine structural lesion.
- Asymmetry pupil size: lesion localisation, especially emerging third nerve palsy.

Reflex asymmetry:

- Lesion localisation.
- Bilateral extensor plantar response common in coma.

Further management

If diagnosis obvious from initial assessment/blood tests (e.g. metabolic), treat appropriately and reassess (should improve after correction, if not, why not?).

If primary neurological cause suspected:

- Brain imaging (CT usually), but ensure patient stable first. Airway protection, correction of hypoxaemia and abnormal CO₂ may necessitate intubation. Get help early.
- Consult neurological advice (Neurology SpR via WGH switchboard).

Further investigation will depend on the above clinical assessment.
Epilepsy is a syndrome characterised by two or more unprovoked epileptic seizures.

Epileptic seizures may be:
- Generalised (most commonly tonic-clonic).
- Focal.

## CAUSES

### First seizure:
- Patients presenting with suspected first ever seizure should be managed as per the ‘First seizure in adults’ protocol. See Chapter 2.
- Further investigations/treatment should only be undertaken after consultation with a neurologist.

### Symptomatic seizures in a person known to have epilepsy:
- Subtherapeutic drug concentration (poor compliance, drug interaction).
- Primary CNS disease (infection, stroke, trauma etc.).
- Encephalopathy due to toxic/metabolic disturbances.
- Intercurrent illness, infection, fatigue, stress.

### Isolated presentation:
Patients presenting with suspected first ever seizure must have:
- ECG, FBC, glucose, U&Es, (toxicology if indicated) LFTs, calcium, magnesium.
- If recovered may be discharged, and referred to “first seizure” clinic (Dr Davenport, Consultant Neurologist, RIE): see referral sheet in Chapter 2.
- Inform patient and document advice regarding DVLA (patients have legal obligation to inform DVLA regarding any suspected epileptic seizures or episode of disturbed consciousness not explained by vasovagal syncope). The patient should not drive until further assessment.
- Further investigations/treatment should only be undertaken after consultation with neurologist.

## STATUS EPILEPTICUS
Defined as more than 30 minutes of:
- continuous seizure activity or;
two or more sequential seizures without full recovery of consciousness between seizures.

This summary is for tonic/clonic status.

In 50% of patients it is the first seizure. The longer status goes on the harder it is to control and the greater the cerebral damage and systemic effects.

**COMPLICATIONS OF STATUS EPILEPTICUS**

- Systemic and cerebral hypoxia
- Neurogenic pulmonary oedema
- Rhabdomyolysis, acute renal failure, hyperkalaemia
- Lactic acidosis
- Hepatic necrosis
- DIC
- Death

**MANAGEMENT**

- **Airway:** assess, open and maintain, high concentration oxygen. Naso-pharyngeal airway may be helpful.
- **Breathing:** assess and support.
- **Circulation:** assess, IV access (check blood glucose), IV fluids. Use 0.9% sodium chloride and avoid 5% dextrose.
- **Drugs:** abolish seizure activity (below).
- **Monitoring:** pulse oximeter, ECG, BP, GCS, pupils.

**Urgent investigations**

- Blood glucose.
- U&Es, Ca**, Mg**, CK.
- ABG
- LFTs
- FBC and coagulation screen.
- The specific cause may be crucial e.g. meningitis, subarachnoid haemorrhage and so on. See below for details of these.
- Discuss with Neurology Registrar: contact via switchboard WGH.
DRUGS

Initial treatment is with **DIAZEPAM** emulsion (Diazemuls).

- 2mg increments IV initially up to 10mg over 5 minutes.
- Alternative is IV lorazepam 4mg slow IV into a large vein.
- Benzodiazepines may cause respiratory depression and hypotension.
- Repeat Diazepam once 15 minutes later up to total 20mg if required.
- Repeat lorazepam once 15 mins later up to a total of 8mg, if required.
- Usually effective but wears off allowing recurrent seizures in many.

Second line therapy for seizures persisting despite benzodiazepines is **PHENYTOIN**.

For patients NOT already on phenytoin:

- Give by IV infusion diluted in 100ml 0.9% sodium chloride.
  - Recommended maximum concentration is 10mg/ml. Sodium chloride is the ONLY suitable diluent.
- For otherwise fit adults a loading dose of 15mg/kg given no faster than 50mg/min is used.
- The solution is liable to precipitation and a 0.2µm filter should be used in the line.
- To avoid local venous irritation flush cannula with 0.9% sodium chloride before and after infusion.
- Monitor ECG continuously as heart block may occur.
- Measure BP frequently as phenytoin causes hypotension.
- Maintenance: IV 100mg phenytoin 8 hourly (or 300mg phenytoin od orally/NG) until the need for ongoing anti-epileptic treatment is reviewed by a neurologist.

In the elderly or in patients with cardiac disease a lower loading dose should be used e.g. 10mg/kg and can be divided into two separate doses.

- Refractory status, continuing for >30 mins despite the above therapy requires expert involvement from Intensive Care and Neurology.
- **Call the duty anaesthetist and inform the ICU Consultant on call.**
  - The next line of therapy involves the use of IV general anaesthetic drugs, tracheal intubation and assisted ventilation.
- Remember specific causes especially meningitis, encephalitis and other intra-cranial pathology.
If cranial CT scan is required secure ABCD first. This will involve invasive monitoring and ventilation.

SUBARACHNOID HAEMORRHAGE

Acute bleed into the subarachnoid space, may also have intracerebral component.
- 80%: aneurysmal
- 10%: no known vascular cause (perimesencephalic)
- 5%: avms, tumours etc.

PRESENTATION

- Acute onset (severe) headache (usually maximal instantly or within few minutes).
- Transient or persisting loss of consciousness.
- Epileptic seizures.
- Vomiting
- Focal neurological signs.
- Meningism is uncommon in the early stages; irritability common.
- Fever is uncommon in the early stages.
- Limb and cranial nerve signs; subhyaloid retinal haemorrhages.
- Hypertension and tachycardia.
- Pulmonary oedema may occur early.
- 20% SAH present with headache alone.

Contact the Neurology Registrar on-call via switchboard WGH.

MANAGEMENT

- Airway: assess, maintain, give high concentration oxygen if hypoxic.
- Breathing: assess and support. Laryngoscopy and intubation cause severe hypertension and may precipitate rebleeding. Unless the patient has arrested or cannot be ventilated intubation should not be attempted except with an appropriate anaesthetic technique by an experienced clinician.
- Circulation: assess, support, gain IV access. Most patients will be hypertensive: no attempt should be made to reduce blood pressure as it is critical to maintenance of cerebral perfusion pressure.
INDICATIONS FOR INTUBATION AND VENTILATION IN SAH

- Airway or breathing compromised.
- Hypoxaemia not corrected by high concentration oxygen.
- GCS 8 or less.
- Hypoventilation and PaCO$_2$ > 6kPa.
- Hyperventilation and PaCO$_2$ < 3.5kPa.

Disability

Neurological assessment: grading of subarachnoid haemorrhage is by the World Federation of Neurological Surgeons Classification and is based on Glasgow Coma Scale.

WFNS GRADING SAH

Grade 1: GCS 15
Grade 2: GCS 13-14
Grade 3: GCS 13-14 with deficit
Grade 4: GCS 7-12
Grade 5: GCS 3-6

- Lower grades may be due to convulsions or hydrocephalus as well as the magnitude of the bleed.
- Management depends on grade: CT brain scanning should be performed early. If negative, lumbar puncture must be performed (unless contra-indicated). Timing is crucial (not before 6-12 hours since symptom onset) and xanthochromia is sought biochemically (bilirubin on spectrophotometry).
- Discuss Grade I-2 with Neurology Registrar WGH and discuss Grades 3-5 with Neurosurgery Registrar WGH.

PRIORITIES

- Resuscitation as previous.
- Analgesia: oral paracetamol 1g 6 hourly, oral/IM codeine phosphate 30mg 6 hourly, and lactulose 10ml bd. Subcutaneous/intravenous morphine 10mg 2 hourly can be used with care, preceded by an anti-emetic.
- Investigation: CT scan head. Transport only after appropriate stabilisation and with adequate monitoring and escort.
**PREVENTION & TREATMENT OF COMPLICATIONS OF SAH**

- **Standard preventive measures for delayed ischaemic neurological deficit/vasospasm:** usually occurs day 4-12. Good fluid intake and oral nimodipine 60mg 4hrly for 21 days. Nimodipine may cause hypotension necessitating halving of dose to 30mg, or omission of doses until BP recovers. Do not treat hypertension.

- **Rebleeding:** peak of up to 20% in first 24 hours, and 40% in 1st month if left untreated. Definitive treatment is to occlude the aneurysm by endovascular ‘coiling’, or sometimes neurosurgical ‘clipping’.

- **Raised intracranial pressure:** haematoma and hydrocephalus may be treatable surgically.

- **Epileptic seizures.**

- **Neurogenic Pulmonary Oedema (NPO):** in the patient with SAH if BP is normal or low, they are poorly perfused and oxygenation is poor with crackles in the chest NPO is likely. Involve ICU early for specific treatment.

**MENINGITIS**

Suspect meningitis in every patient with a fever, headache, meningism, or neurological signs. Optimal management requires a rapid assessment, diagnosis, and treatment.

**FEATURES OF MENINGITIS**

- Meningism- photophobia, neck stiffness in 70%, headache, Kernig’s sign.
- Fever
- Decreased level of consciousness.
- Seizures in about 20%.
- Focal neurological signs, especially cranial nerve palsies in about 20%.
- Petechial rashes in meningococcal septicaemia. However, a similar rash can occur in staphylococcal and pneumococcal septicaemia.
**LIKELY ORGANISMS**

- Depend on age, and a large number of other factors e.g. immunological state.

The commonest bacterial pathogens are:

- <60yrs *Streptococcus pneumoniae, Neisseria meningitidis*.
- >60yrs *Streptococcus pneumoniae, Neisseria meningitidis, Listeria monocytogenes*.

**INITIAL MANAGEMENT**

- Full ABCDE assessment and treatment: see Chapter 2.
- Take blood cultures and start antibiotics immediately. Do not delay while awaiting a CT scan or LP.
- Careful examination for neurological signs and rashes.
- Check vital signs: if shocked treat as for septic shock at once with high concentration oxygen and IV fluids.
- Document GCS.
- Signs of raised ICP give mannitol 20% 200ml, furosemide 20mg and Alba 200ml IV stat and call ICU and Neurosurgery.
- CT scan with appropriate escort, resuscitation and monitoring.

**ANTIBIOTICS**

- If <60 yrs ceftriaxone 2g IV bd.
- If >60 yrs or immunocompromised ceftriaxone 2g IV bd and amoxicillin 2g IV qds to cover *Listeria*.
- Seek urgent microbiological advice re antibiotics and duration of treatment, once organism is known.
- Contact ID middle grader.
- Consider IV aciclovir (10mg/kg tds) if LP is delayed (see encephalitis).
- Discuss use of dexamethasone 10mg 6 hourly IV or oral for 4 days if pneumococcal meningitis likely (eg no purpuric rash) and it can be started before or at same time as antibiotics. There is no benefit in giving steroids after antibiotics.

**INVESTIGATIONS**

- FBC
- U&E’s and glucose.
- Blood cultures.
• EDTA blood sample for PCR (pink tube, as FBC).
• Coagulation screen.
• Throat swabs. One for bacteria, and one in viral transport medium for viral culture. State clearly on request form “meningitis”.
• Stool for viral culture.
• If clinical features suggest recent mumps, parotid duct swab in viral transport medium.
• Lumbar puncture: see below re CT scanning. A CT scan may be required first if a mass lesion, or abscess is suspected, i.e. focal neurological signs, papilloedema, middle ear pathology, or a history suggestive of a neoplasm or if profoundly immunosuppressed eg HIV positive. Check opening pressure, if >35 cmH₂O, remove only the fluid in the manometer and refer to ICU urgently (see next page). Otherwise try and send at least 5ml to Microbiology (greatly increases diagnostic yield). One sample to microbiology for MC&S, one to biochemistry for glucose, protein, and xanthochromia if subarachnoid haemorrhage is a possibility, and one to Virology.
• Contemporaneous blood glucose.

Contraindications to lumbar puncture include signs of raised intracranial pressure, (including reduction in conscious level, focal neurological signs) or major coagulopathy.

CXR

Normal CSF is gin-clear. Any haze/turbidity is an indication for immediate antibiotic if not already given.

Cerebro-spinal Fluid Findings

<table>
<thead>
<tr>
<th></th>
<th>BACTERIAL</th>
<th>VIRAL</th>
<th>TUBERCULOUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell count</td>
<td>↑↑↑</td>
<td>↑↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Cell type</td>
<td>Polymorphs</td>
<td>Lymphocytes</td>
<td>Lymphocytes</td>
</tr>
<tr>
<td>(normal up to 5 lymphocytes)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>↑↑ (0.5-2.0)</td>
<td>↑ (0.4-0.8)</td>
<td>↑↑↑ (1.0-3.0)</td>
</tr>
<tr>
<td>(normal &lt;0.4g/L)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>&lt;40% serum</td>
<td>&gt;50% serum</td>
<td>&lt;40% serum</td>
</tr>
</tbody>
</table>
TENTORIAL HERNIATION AND CONING

• Raised ICP can cause this.
• Rapidly fatal but it is potentially reversible if identified and treated early.
• May occur post lumbar puncture but this is rare in patients with no focal neurology or raised ICP

Diagnosis

• Intra-cranial pathology e.g. recent lumbar puncture in meningitis or SAH, haematoma.
• Pupil(s) dilate abruptly, and fix.
• Respiration periodic or stertorous.
• Bradycardia and hypertension.
• Coma

Action

• Call 222 then ICU and Neurosurgeon.
• Bag, mask, valve hyper-ventilate with high concentration oxygen.
• IV access.
• Mannitol 20% 200ml IV, furosemide 20mg IV, ALBA 200ml all stat.
• Require intubation and ventilation with anaesthetic.
• Further management will be decided by ICU and Neurosurgical specialists.

FURTHER MANAGEMENT OF MENINGITIS

• Analgesia.
• IV fluids if dehydrated.
• Infection control for suspected meningococcal disease, isolate patient for first 48h.
• Notify the on-call consultant in Public Health of all meningococcal and *Haemophilus influenzae* infections. They will arrange prophylaxis for all contacts, including the patient’s immediate household contacts and any significantly exposed staff contacts (mouth-to-mouth resuscitation or other close prolonged contact; prophylaxis rarely necessary for staff).

ICU referral if:

• Shock unresponsive to initial fluid resuscitation.
• Respiratory failure.
• GCS <11.

Prophylaxis Don’t forget to give the patient prophylaxis before discharge (if they have not received ceftriaxone during admission).
ENCEPHALITIS

VIRAL ENCEPHALITIS

Viral encephalitis is inflammation of the brain due to viral infection, with Herpes simplex being the most destructive but potentially treatable causative agent. Currently, Herpes simplex encephalitis is estimated to occur in approximately 1 in 250,000 to 500,000 individuals a year. It occurs throughout the year and in patients of all ages, 1/3 in those aged less than 20 years and approximately one half in those aged over 50 years. In pre aciclovir (acyclovir) days, the mortality was over 70%.

Other viral causes of acute encephalitis include:
- Enterovirus, mumps, influenza, EBV, VZV, CMV.
- In patients with travel history: arboviruses, rabies.

Presentation
- Signs of meningeal inflammation: e.g. fever, headache, neck stiffness.
- Altered mentation/personality change.
- Decreasing conscious level.
- Focal neurology.
- Seizures

INITIAL MANAGEMENT

- Careful clinical examination including full neurological examination and Glasgow Coma Score.
- ABCDE as Chapter 2.
- Ask for travel history.
- If patient needs a CT head scan, do not delay antibiotics (see Meningitis chapter) and aciclovir (10mg/kg tds IV).
- Ensure the patient is stable enough for transfer to CT scan.

INVESTIGATIONS

- FBC, U+E’s, glucose, LFT, Ca, clotting screen.
- Blood cultures X3.
- Blood for serology.
- Throat swabs in viral transport medium.
- Stool for Virology
• Parotid duct swab (for mumps) in viral transport medium.
• Swab any lesion suggestive of *Herpes simplex*.
• CT scan if focal neurology, raised intracranial pressure, mass lesion. Lumbar puncture if CT shows no features to contraindicate this. CSF to microbiology for microscopy, cell count and gram stain. CSF also to biochemistry for sugar and protein (a contemporaneous plasma sugar is also required). CSF should be sent to Virology for culture and PCR (min. volume 1ml).
• CXR
• EEG
• CT/MRI

## FURTHER MANAGEMENT

• Analgesia
• Glasgow Coma Scale and other vital signs should be carefully monitored. Seizures should be treated with anticonvulsants.
• IV fluids to maintain euvoilaemia.
• Notify Infectious Diseases and Neurology SpR on call via switchboard WGH.

## SPECIFIC ANTIMICROBIAL THERAPY

• Antibiotics as per meningitis of unknown aetiology (see meningitis).
• IV aciclovir (acyclovir) 10mg/kg tds depending on renal function.

## DIFFERENTIAL DIAGNOSIS

There is a wide spectrum of conditions that may mimic viral encephalitis, including brain abscess/empyema, partially treated bacterial meningitis, tuberculous meningitis, tumour, vasculitis, connective tissue disorders and toxic/metabolic causes. Consult Neurology or Infectious Diseases.

## SUPPORTIVE CARE

If raised ICP, depressed conscious level, shock or respiratory failure early ICU referral is appropriate. See Chapter 2.
Delirium is a medical emergency and needs prompt assessment and treatment.

Delirium (‘acute confusional state’) is an acute deterioration in cognition, often with altered arousal (drowsiness, stupor, or hyperactivity) and psychotic features (eg. paranoia). The main cognitive deficit in delirium is ‘inattention’, eg. the patient is distractable, cannot consistently follow commands, and loses the thread during a conversation. Delirium is different from dementia, where there is a much slower decline in cognition and inattention is much less prominent, but the two conditions commonly co-exist.

Delirium affects 1 in 5 of older patients in hospital. It is important because it frequently indicates serious illness – NB ‘confusion’ in the CURB-65 score. The outcome is frequently poor.

CAUSES OF DELIRIUM

- Three main groups:
  1. physical and psychological stress: any acute illness, trauma, surgery, etc.
  2. drugs: drugs with anticholinergic activity (eg. amitriptyline, oxybutinin), opiates, benzodiazepines, steroids; also drug withdrawal (eg. benzodiazepines, alcohol)
  3. metabolic, eg. hyponatraemia, hypercalcaemia, hypoglycaemia

- Note that a higher number predisposing factors (old age, baseline cognitive impairment, multiple comorbidities) mean that an apparently minor insult, eg. a UTI or a change of drugs, can precipitate delirium.

INITIAL ASSESSMENT

- Delirium should be suspected in any patient with (a) cognitive impairment and/or altered arousal and (b) evidence that the altered mental status is of recent onset (hours, days, weeks).
- Therefore, to screen for delirium you need to assess cognition and arousal, and seek a third party history regarding the patient’s baseline state.
• Assessment of cognition can be done formally using the Abbreviated Mental Test, and through clinical observation (eg. inability to converse normally, distractibility, inability to follow commands, etc.)
• Note other features, such as irritability, paranoia, lability of mood, apathy, etc.
• Agitation is not necessary to make the diagnosis: more than 50% of patients will not show this.
• Once you have made the diagnosis you need to consider the predisposing and precipitating factors.
• In older patients delirium may be the presenting feature of acute illness, for example pneumonia, UTI, cholecystitis, etc. Often patients will lack other obvious features of the illness. Thus, initial examination is directed at looking for an acute cause.
• Do not neglect examination of the nervous system (stroke can cause delirium), joints, and skin.

**ABBREVIATED MENTAL TEST SCORE (AMT)**

<table>
<thead>
<tr>
<th>SCORE OUT OF 10</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. What is your present age (± 1 year)?</td>
<td></td>
</tr>
<tr>
<td>2. What is the time just now (± 1 hour)?</td>
<td></td>
</tr>
<tr>
<td>3. What year is it?</td>
<td></td>
</tr>
<tr>
<td>4. What is the name of this place? Please memorise this address - 42 West St</td>
<td></td>
</tr>
<tr>
<td>5. When is your birthday (date and month)?</td>
<td></td>
</tr>
<tr>
<td>6. When did the First World War begin?</td>
<td></td>
</tr>
<tr>
<td>7. What is the Queen’s name?</td>
<td></td>
</tr>
<tr>
<td>8. Can you recognise 2 people?</td>
<td></td>
</tr>
<tr>
<td>9. Count backwards from 20 to 1?</td>
<td></td>
</tr>
<tr>
<td>10. Can you remember the address I just gave you?</td>
<td></td>
</tr>
</tbody>
</table>

**INVESTIGATIONS**

• Exclude hypoglycaemia and hypoxia at the bedside.
• U&Es, Ca
• LFTs
• FBC
• ESR & CRP
• Troponin
• Glucose
• Blood cultures if any evidence of infection
• ABGs if tachyapnoeic, low O₂ sats (<96%), possibility of CO₂ retention or metabolic acidosis
• Urinalysis +/- MSU
• CXR
• ECG
• Abdo USS if LFTs deranged – eg. to investigate possible cholecystitis
• Consider CT brain +/- LP if delirium persists without known precipitant. Further investigations should be under the supervision of a specialist.

MANAGEMENT

• Because delirium is usually due an interaction between multiple predisposing factors and precipitating factors, management should be aimed at not just finding and treating the assumed cause, but also optimising all aspects of care:

  1. optimise physiology: correct hypoxia and hypoglycemia, treat anaemia, dehydration, hyponatraemia, malnourishment, etc.
  2. treat any possible precipitants
  3. stop or reduce deliriumogenic drugs (amitriptyline, etc.) – consult pharmacist if unsure
  4. minimise mental stress – provide repeated re-orientation, involve family/carers, and provide care in as quiet and stable an environment as possible (eg. side room)
  5. avoid prolonged bedrest: mobilisation can help recovery

• Management is best carried out on specialist units: transfer to Acute Medicine of the Elderly ward early. Appropriate nursing care can often avoid sedation (quiet, well lit environment).

• If agitation causes severe distress or immediate danger of injury consider using drug treatment. The first line drug is haloperidol 0.5mg oral or im, at intervals of 20 min – 1 hr until agitation is reduced to acceptable levels. If in any doubt contact a senior colleague for advice or seek specialist help. See below for further details

ADDITIONAL POINTS

• Benzodiazepines prolong delirium and may worsen outcome. Do not use unless under specialist supervision, alcohol withdrawal is suspected, or the patient has Parkinson’s disease or dementia with Lewy Bodies.

• Delirium is very common in dying patients – treat cause(s) if possible and consider antipsychotics

• Differentiation between depression, dementia and delirium can be difficult, and where the delirium persists seek specialist advice.
ACUTE AGITATED CONFUSION IN AN OLDER PATIENT

PRESCRIBING GUIDELINE

Look for possible precipitants

**Metabolic problems** - sodium, calcium, hypoxia, hypoglycaemia?
Is your patient in pain?

Is there **infection** in chest, urine, skin, joints, or meninges?
Is alcohol withdrawal a possibility?

Is **benzodiazepine withdrawal** a possibility?

**Drugs** - be suspicious of all prescribed drugs and check that none have been suddenly stopped.

Can you modify the environment?

One to one nursing - discuss **extra staff** with the directorate manager.
Try to find a quiet, well lit, **side room**.

Can **family** stay with the patient for some of the time?
Provide an understanding **nurse**.

Is your patient too hot, too cold, or hungry?

**Drug Treatment**

*N.B. Only use drugs if your patient is at risk of causing harm to themselves or others.*

If alcohol or benzodiazepine withdrawal is a possibility refer to the alcohol withdrawal guideline.

In other cases use:

1. **Haloperidol** 0.5-1mg orally if possible **Wait 20 mins at least**
2. If no response 0.5-2mg orally or IM **Wait 20 mins at least**
   repeat **Haloperidol**
3. If no response discuss with a **senior member** of your **team**
4. If agitation remains an acute problem discuss with **on-call psychiatric staff**. (Out of hours contact via REH switchboard on Ext.7600)

An alternative to haloperidol in patients in whom this is unsuitable (eg. Parkinson’s disease, dementia with Lewy Bodies) is lorazepam 0.5mg orally or im, using same regime as for haloperidol. Use as little as possible: benzodiazepines prolong delirium and may be associated with a worse outcome.

**REMEMBER**

This is a general guideline - your patients have **individual** problems

Seek and treat participants
Try to modify the environment
Give drugs time to work
FALLS AND IMMOBILITY

Falls and unsteadiness are very common in older people. Although only 10-15% of falls result in serious injury, they are the cause of 92% of hip fractures in older women. There is now a good evidence base for falls and fracture prevention.

PROBLEMS THAT MAY PRESENT AS A “FALL” OR “OFF LEGS”

Bear in mind that many patients will be in more than one of these categories:

• Loss of consciousness: syncope or seizure.
• Acute illness e.g. infection, stroke, metabolic disturbance.
• Simple trip.
• Chronic neurological and locomotor disease (see below).

ASSESSMENT

Full history and examination are required:

• Ask about the circumstances of the fall, and frequency if they are recurrent
• Try to establish if the patient lost consciousness e.g. “do you remember hitting the ground?”. A witnesses account is best.
• Check for symptoms or signs of acute illness, especially infection.
• Find out the past history - if necessary ask the relatives and GP.

Conditions associated with falls:
- Stroke and vascular dementia.
- Parkinson’s disease.
- Alzheimer type dementia.
- Disease of weight bearing joints e.g. OA, joint replacement or previous fracture.
- Depression.

• Look for the known risk factors for falls (many patients will have several):
  - Impaired cognitive function: check the AMT.
  - Poor balance: ask and examine the patient’s gait.
  - Reduced strength: grade 1-5 and look for wasting.
  - Poor vision: ask and check eyesight.
  - Postural hypotension: ask about dizziness on standing up and check erect and supine blood pressure and drug treatment.
Drugs:
- polypharmacy (>4 drugs).
- sedatives and anti-psychotics.
- antidepressants including SSRI s.
- hypnotics

Exclude serious injury e.g. hip or vertebral fracture, head injury.
Assess osteoporosis risk e.g. previous low trauma fracture, low BMI, steroid use, smoker and consider DEXA scan.

Admission is required in those who:
- cannot walk without help
- are acutely unwell
- are falling so frequently that they can’t manage at home.

INVESTIGATION

- FBC
- U&E, glucose, LFTs, CRP, Ca & PO₄
- Digoxin level if on this.
- Urinalysis and MSU if features of sepsis, pyrexial or raised WBC and CRP.
- ECG (24 hour ambulatory ECG is not helpful unless the patient is having recurrent syncopal episodes).
- Chest X-ray.
- If the patient has impaired cognitive function unexplained by known pathology:
  - CT brain, B₁₂ and folate, TFTs.
- If the patient has impaired balance or strength unexplained by known pathology, consider CT brain if focal neurological signs, X ray if abnormal joints. Occasionally other investigations are required such as nerve conduction studies to confirm a peripheral neuropathy, or Vitamin D levels in proximal myopathy. (Osteomalacia).

MANAGEMENT

This has to be tailored to the individual patient’s problems and requires input from a multidisciplinary team.

- Treat any acute illness.
- Optimise the management of any chronic pathology e.g.:
  - Pain control and physiotherapy for degenerative joint disease.
  - Adjust anti-Parkinsonian medication to achieve best control.
- Ensure 2\(^0\) stroke prevention and treatment measures are in place.

- Multifactorial intervention for falls prevention in all:
  - Exercise and balance training by physiotherapy.
  - Reduce medication as far as possible and reduce or stop any contributing drugs e.g. diuretics, antihypertensives, SSRI or anti-anginals.
  - Reduce postural hypotension:
    - ensure not anaemic
    - reduce or stop any contributing drugs
    - teach the patient to rise carefully from bed or chair
    - consider TED stockings
    - in extreme cases, seek expert advice regarding drug treatment to maintain or raise BP
  - Safety education and home hazard assessment by OT
  - Correct visual impairment

- Refer to Lothian Joint Formulary guidelines on osteoporosis.
- Commence treatment with a weekly bisphosphonate and Adcal D3 in those with proven osteoporosis and those with 2 or more previous fragility fractures.
1. NEUTROPENIC SEPSIS

Definitions
Neutropenia: neutrophil count of less than $1.0 \times 10^9/l$.
Fever: isolated temperature greater than 38.5°C or 2 recordings greater than 38.0°C two hours apart.

Presenting features
- Generalised constitutional symptoms are common (lethargy, rigors, confusion). Patients can go from being well to being in life threatening septic shock in just a few hours. Neutropenia markedly alters the host’s immune response and makes infection more difficult to detect.
- Ask about respiratory, urinary, oropharyngeal and lower GI symptoms. Enquire about recent instrumentation/dental work.
- Does the patient have a Hickman Line? Ask about recent line use and whether there is pain around the line.

Patients with febrile neutropenia MUST receive antibiotics even if there are no localising signs of infection.

Assessment
Look for:
- Signs of shock e.g. tachypnoea, tachycardia, hypotension, altered mental state.
- Fever
- Detailed examination for any localising signs of infection.

Management
- Assess ABCDE. Treat as in Chapter 2.
- Give high concentration oxygen by mask.
- Gain IV access and resuscitate with colloid if hypotensive.
- Check full blood count, electrolytes, renal function, LFT’s, calcium, lactate, ABG & CRP.
- Take blood cultures, peripheral and from all line lumens.
- Urine for culture (even if dipstick - negative).
- Stool for culture and $C$ difficile toxin if patient has diarrhoea.
• Sputum if available.
• Other microbiology samples depending on symptoms and signs e.g. throat swab, mouth swab, ear swab.
• Viral throat swab and serology.
• Perform chest x-ray.
• Monitor respiratory rate, \(\text{SpO}_2\), pulse/BP every 15 mins until stable.
• Monitor urine output.

Anti-microbial therapy

Information available: WGH Haematology handbook on LUHD intranet - microsites - haematology-WGH

• Tazocin 4.5g IV QDS & IV gentamicin 7mg/kg OD (ideal bodyweight) (see guideline for administration and monitoring).
• Add clarithromycin if chest infection.
• Add metronidazole if lower GI symptoms.
  Replace gentamicin with vancomycin if a) patient MRSA colonised; b) long term central line. In latter consider removing the line.

2. SPINAL CORD COMPRESSION

• 20% of patients with vertebral metastases develop spinal cord compression.
• Early recognition is vital.
• Pre-treatment neurological status is the most powerful predictor of functional outcome.

Presenting features

• Back pain (local or radicular) in 90% of cases.
• Weakness (80%) which is usually progressive over days/weeks.
• Sensory loss or paraesthesia, which may ascend to the level of compression.
• Autonomic dysfunction (urinary retention, constipation) occurs late & is associated with a worse prognosis.

Assessment

Careful neurological examination. There are two broad syndromes.

1. Spinal cord compression (above L2) - increased tone (reduced if acute syndrome), weakness, hyper-reflexia & extensor plantors. Sensory level to pinprick (spinothalamic tracts) & proprioceptive
loss (posterior columns). The bladder may be palpable. Local spinal tenderness.

2. Cauda equina syndrome (L2 down) weakness, variable sensory loss, characteristically perianal sensory loss.

Management

- Early identification and treatment are vital.
- Give adequate pain relief.
- Give dexamethasone 16mg IV on clinical suspicion (helps pain & reduces pressure on cord).
- **Contact on call Clinical Oncology registrar via WGH switchboard. Neurosurgical referral may be appropriate.**
- X-ray spine (85% are abnormal). Look for pedicular erosion (“winking owl” sign) on AP films & collapse on lateral films.
- Urgent MRI spine (discuss with on-call Neuroradiologist).
- Catheterise, if in urinary retention.
- Anti-thrombotic measures (heparin prophylaxis as per local policy, TEDS).
- Bedrest until further advice.

Definitive Treatment

Steroids followed by neurosurgical decompression/stabilisation or palliative radiotherapy.

3. SUPERIOR VENA CAVA OBSTRUCTION

90% of cases have a malignant cause. The commonest is lung cancer (65%) followed by lymphoma, metastatic lymphadenopathy, germ cell tumours & thymoma.

Presenting features

Usually insidious onset with progressive dyspnoea, facial swelling, head fullness, arm swelling & cough. Symptoms may be present only on waking, and may be aggravated by lying down.

Assessment

- Distended neck veins, distended chest wall veins & facial oedema are the commonest signs. Cyanosis, facial plethora and arm oedema can also be seen.
- Examine for neck nodes.
Management

- Sit upright.
- Give high concentration oxygen by mask.
- Arrange chest x-ray (60% have superior mediastinal widening).
- **Discuss with on-call Clinical Oncology registrar via WGH switchboard.**
- 60% of patients have no known malignant diagnosis. Consider sputum cytology, biopsy of neck nodes or bronchoscopy.
- **Where at all possible histological diagnosis should be made prior to treatment:**
  - Discuss with Chest Physician, Thoracic Surgeon.

Treatment

Depends on:

1. Aetiology of obstruction (extrinsic compression vs. SVC thrombus).
2. Histology of tumour.
   - CT thorax and/or venography add more information.
   - Dexamethasone 4mg qds.
   - SVC stent + or - thrombectomy (discuss with interventional Radiologists at RIE).
   - Radiotherapy (if no histology or non small cell lung carcinoma).
   - Chemotherapy (lymphoma, small cell lung cancer, germ cell tumours).
National Poisons Information Service (NPIS)

NPIS (Edinburgh) is one of four UK centres commissioned by the Health Protection Agency that contribute to the TOXBASE® website. This provides guidance on the management of poisoning by any one of a large number of different drugs, chemicals and plants. TOXBASE® is a standard reference for advice on the features and treatment of poisoning cases, and is updated on a daily basis. It can be accessed from the Combined Assessment Area (Base 6) and A&E departments at the Royal Infirmary, ARAU at the WGH, the A&E department of St. John’s Hospital, and the critical care areas on all three sites. Additional information related to unusual or severe poisoning can also be obtained by telephone.

NPIS : 0870 600 6266
24-hour information service for more severe and complex poisoning cases

ADMISSION POLICY

• Patients who present after drug overdose or deliberate self-harm (e.g. self-cutting) normally require admission to hospital. In some cases this may be for psychiatric assessment alone, rather than ongoing medical care. The preferred site for admission in Edinburgh is CAA Base 6, Royal Infirmary (0131-242-1443).
• Patients who are unconscious or at high risk of airway or haemodynamic compromise should normally be admitted to a critical care area (e.g. HDU, ICU).
• Patients expressing suicidal thoughts but who have not actually harmed themselves or taken a drug overdose do not usually need admission to a medical unit, and should be discussed with the on-call duty Psychiatrist.
• At SJH patients are managed in A&E.

IMMEDIATE MANAGEMENT

• Maintain airway using nasopharyngeal or oropharyngeal airway if conscious level is reduced
• Endotracheal intubation is required if unresponsive and loss of protective airway reflexes. Make ICU referral early
• Give oxygen if drowsy aiming to maintain SpO$_2$ > 92%
• Ensure adequate ventilation. Treat underlying cause if applicable or consider need for intubation and ventilation. **NB. Opiate overdose may lead to respiratory depression with hypoventilation**

• Consult TOXBASE®; contact NPIS if further advice needed 0870 6006266 (24hr)

• Consider need for activated charcoal or gastric lavage

• Record pulse, blood pressure, oxygen saturation, respiratory rate and temperature

• Monitor cardiac rhythm if drug likely to have haemodynamic effects or cause arrhythmia

**GENERAL MANAGEMENT**

• Manage in an appropriate care area eg HDU/ICU

• Correct underlying hypoxia to reduce risk of seizures or arrhythmias

• If hypotensive administer IV fluids to ensure adequate hydration and elevate the legs. If blood pressure remains low then inotropes may be required.

• Metabolic acidosis increases the risk of seizures and arrhythmia after overdose with certain drugs. If acidosis persists despite correction of hypoxia and hydration status then IV sodium bicarbonate may be administered. 1.26% sodium bicarbonate 250 ml can be administered and repeated as necessary. Seek expert advice.

• Control agitation with oral or IV diazepam (0.1–0.3mg/kg body weight). Repeated administration may be needed. Large doses may be needed in patients using recreational drug such as cocaine or amphetamines.

• Treat seizures with IV lorazepam 2–4 mg or diazepam 5–10 mg; repeated doses might be needed. Management of persistent seizures should be discussed with the NPIS; some anticonvulsants can increase toxicity of a number of drugs.

• If cardiac arrhythmias occur ensure that hypoxia and metabolic acidosis are corrected. Arrhythmias due to tricyclic antidepressant overdose should be treated with IV sodium bicarbonate, which should be given as 50 ml 8.4% sodium bicarbonate or 250ml 1.26% sodium bicarbonate via a central or large peripheral vein.

• Torsade de pointes arrhythmia (polymorphic ventricular tachycardia) can be caused by some drugs that prolong the QTc interval. This should be treated with IV magnesium sulphate 8–10 mmol, given over 1–2 minutes. This may be repeated after 5–10 minutes if necessary.
• Use antidotes where indicated in TOXBASE®.

GUT DECONTAMINATION/DRUG ELIMINATION

• Induced vomiting is of no benefit, is potentially hazardous, and should be avoided.
• Absorption of many drugs may be reduced by oral activated charcoal (50 g) within the first hour post ingestion. Activated charcoal must not be given without adequate airway protection.
• Some substances including iron, lithium, methanol and ethylene glycol are not bound to charcoal.
• Repeated doses of activated charcoal enhance elimination of certain drugs, and can be beneficial beyond 1 hour post-ingestion: carbamazepine, phenobarbitone, quinine and theophylline.
• Gastric lavage is rarely necessary, and should be considered only if a life-threatening dose of chemical or drug have been ingested within 1 hour.
• Gastric lavage should NOT be undertaken in patients with reduced conscious level or inadequate airway protection, or after ingestion of petroleum distillates or corrosives due to the risk of aspiration. If in doubt discuss with the NPIS.
• Whole bowel irrigation with osmotic laxatives may reduce absorption of some drugs that are not adsorbed by charcoal. It is occasionally necessary for patients who have ingested packages of illicit drugs (e.g. ‘body-stuffers’).
• Urinary alkalinisation may increase elimination of some drugs (e.g. salicylate), and can protect against renal impairment in patients who have rhabdomyolysis.
• Haemodialysis can improve outcome in some cases of severe toxicity, e.g. digoxin, ethylene glycol, lithium, methanol and salicylates. Further information is available from TOXBASE® and NPIS.

EMERGENCY INVESTIGATIONS

• See table for suggested investigations
• Perform arterial blood gas if airway is compromised, hypoventilation or metabolic acidosis is suspected. Carboxyhaemoglobin should also be measured in cases of suspected carbon monoxide poisoning.
• Chest X-ray should be performed if the patient is persistently...
hypoxic or after inhalational exposure.

- Paracetamol and salicylate concentrations should be measured if there is suspected ingestion of either, or the ingested drugs are unknown. The timing of sample collection is important.

- Plasma concentrations of certain other drugs can be helpful, e.g. carbamazepine, digoxin, iron, lithium, phenytoin, theophylline and thyroxine.

- In cases of severe unexplained metabolic acidosis, consider measurement of ethanol, methanol and ethylene glycol concentrations (discuss with local laboratory).
<table>
<thead>
<tr>
<th>Drug</th>
<th>At risk of</th>
<th>U&amp;Es</th>
<th>LFTs</th>
<th>INR</th>
<th>CK</th>
<th>ABG</th>
<th>Drug level</th>
<th>Cardiac monitor</th>
<th>Detected in TOX screen?</th>
<th>Comment</th>
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<td>✓</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>Level at 2 and/or 4 hours</td>
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<td>Beta-blocker</td>
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<td>✓</td>
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<td>Level not urgent</td>
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<td>Level at 4 hours</td>
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<td>Level if on regularly</td>
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</table>
**Paracetamol**

**Early features:** usually none  
**Late features:** nausea, vomiting  
**Toxicity:** Ingestion of >150 mg/kg (or >12g) or >75 mg/kg in a high-risk patient may be fatal or cause severe toxicity

**PRESENTATION WITHIN 8 HOURS OF INGESTION**

- Give activated charcoal if >150 mg/kg ingested within 1 hour  
- Measure paracetamol concentration at 4 hours. There is no point in measuring the concentration before this.  
- Use paracetamol nomogram (shown below) to determine need for treatment. Remember to check if patients have any risk factors.  
- If paracetamol level is above treatment line, give N-acetylcysteine (NAC). Normally, N-acetylcysteine should not be given until paracetamol levels known.

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**Diagram:**

- **TREATMENT LINES**
- **Plasma Paracetamol (mg/l)**
- **Plasma Paracetamol (mmol/l)**
- **Hours after ingestion**

**Prognostic accuracy after 15 hours uncertain**
HIGH RISK FACTORS

A number of factors increase the risk of toxicity after paracetamol ingestion, either because they are associated with hepatic enzyme induction (more rapid formation of toxic metabolite), or glutathione depletion (inability to detoxify toxic metabolite):

- Carbamazepine
- Phenobarbitone
- Phenytoin
- Primidone
- Rifampicin
- St John's Wort
- Regular alcohol excess
- Malnutrition (e.g. eating disorders, cystic fibrosis, Muscular dystrophies, AIDS)

N-ACETYLCYSTEINE (NAC)

- Treatment is most effective if started within 8 hours of ingestion.

**Adult dosing schedule for N-acetylcysteine:**

- 150mg/kg IV in 200mls 5% dextrose over 15 minutes, then:
- 50mk/kg IV in 500mls 5% dextrose over 4 hours, then:
- 100 mg/kg in 1000mls 5% dextrose over 16 hours

ANAPHYLACTOID REACTIONS TO N-ACETYLCYSTEINE

- A histamine-mediated reaction occurs in 10% of patients, usually within 30 min: features include flushing, vomiting, rash, and rarely bronchospasm and hypotension. True anaphylaxis does not tend to occur.
- Infusion should be stopped, and symptoms often subside within 20-30 minutes. In some cases, antihistamines may be needed (e.g. IV chlorphenamine 10-20 mg). Occasionally, bronchodilators are required (e.g. nebulised salbutamol 5 mg) and, rarely, IM adrenaline and IV hydrocortisone are required for severe hypotension. See chapter 2.
- When symptoms have resolved the NAC infusion should be recommenced at 50% of the normal administration rate.
- Reactions to NAC do not necessarily recur. Therefore, the normal treatment schedule should be used even if patients have a history of a reaction to a previous N-acetylcysteine infusion.
PRESENTATION 8-15 HOURS POST INGESTION

- If >150 mg/kg (or >12g) or >75 mg/kg in a high risk patient has been ingested start N-acetylcysteine immediately (see Adult Dosing Schedule above)
- Check FBC, U&Es, LFTs and prothrombin time (INR) and paracetamol concentration
- If paracetamol concentration is below treatment line, ALT and INR are normal and the patient is asymptomatic discontinue NAC.
- Otherwise continue with the normal infusion protocol.

PRESENTATION 15–24 HOURS

Patients presenting late are at greatest risk of developing liver damage.

- If >150 mg/kg (or >12g) or >75 mg/kg in a high risk patient has been ingested start N-acetylcysteine immediately
- Check FBC, U&Es, LFTs and prothrombin time (INR) and paracetamol concentration
- The paracetamol concentration is less reliable at this time, and the presence of an elevated prothrombin time and ALT are better markers of possible liver damage
- If blood tests are all normal and the patient is asymptomatic, N-acetylcysteine can be discontinued otherwise continue with the normal infusion protocol.

PRESENTATION >24 HOURS

Patients presenting late are at greatest risk of developing liver damage.

- Check FBC, U&Es, LFTs and prothrombin time (INR)
- If investigations are normal, and the patient is asymptomatic no further medical treatment is required
- If abnormal give N-acetylcysteine
- Patients require frequent monitoring of U&Es (including bicarbonate), LFTs, INR, lactate and glucose
- Progressively rising INR and ALT, metabolic acidosis, renal impairment, hypoglycaemia and hepatic encephalopathy are poor prognostic indicators and the patient should be discussed with the NPIS and on-call gastroenterology/ liver team (via switchboard RIE).
STAGGERED OVERDOSE

- If >150 mg/kg (or >12g) or >75 mg/kg in a high risk patient has been ingested within a 24-hour period, then N-acetylcysteine infusion should be given
- Check FBC, U&Es, LFTs and prothrombin time (INR)
- Plasma paracetamol concentration will confirm ingestion but cannot be used to determine the need for treatment
- If repeat blood tests are normal 24-hours after ingestion of the last tablets, and the patient is asymptomatic, then N-acetylcysteine can be discontinued

COMPLETION OF N-ACETYLCYSTEINE

- Check U&Es, ALT and prothrombin time (INR) at the end of the infusion.
- If ALT and creatinine are normal, and INR \( \leq 1.3 \), then the patient can be discharged after appropriate psychiatric review (*N.B. N-acetylcysteine directly causes a rise in INR that is not related to liver function*).
- If ALT or creatinine are abnormally high, or INR >1.3, or there is metabolic acidosis, then N-acetylcysteine should be continued at 150 mg/kg over 24 hours. Coagulation and LFTs should be checked every 8-12 hours. When there is a sustained improvement the N-acetylcysteine can be discontinued.
- In a small number of cases, INR and ALT continue to rise despite N-acetylcysteine therapy. Patients may develop liver failure, renal failure, hypoglycaemia and metabolic acidosis. These patients may need consideration for liver transplant, and should be discussed with senior staff urgently.

BENZODIAZEPINES

**Features**

Drowsiness, hypotension, coma and respiratory depression. Toxicity is worse when co-ingested with alcohol or other CNS depressants, e.g. opioids.

**Toxicity**

Serious toxicity from pure benzodiazepines is uncommon

**Management**

- Consider need for gut decontamination if present within 1 hour of ingestion
If significantly reduced conscious level or respiratory depression, then call 222 for urgent endotracheal intubation and ventilatory support in a critical care area

Flumazenil (Anexate®), a benzodiazepine antagonist, may be used if immediate access to critical care is not available. It has a short half-life (around 1 hour) and can provoke seizures, especially in patients with:
1. Pre-existing epilepsy
2. Benzodiazepine-dependence
3. Mixed overdose, particularly common after tricyclic antidepressants

FLUMAZENIL should not be used as a ‘diagnostic test’.

**SALICYLATES**

**Features**
Vomiting, tinnitus, deafness. In severe cases confusion, seizures, metabolic acidosis, pulmonary oedema and coma may occur.

**Toxicity**
Likely if >250 mg/kg ingested; >500 mg/kg can cause severe toxicity/death.

**Management**
- Give activated charcoal if >120 mg/kg ingested less than 1 hour ago.
- It can take several hours to reach peak plasma concentrations. Salicylate concentrations should be checked in patients who have ingested >120 mg/kg.
- In symptomatic patients: check at 2 hours post-ingestion, then repeat after further 2 hours in case of on-going drug absorption.
- In asymptomatic patients: check at 4 hours post-ingestion.
- In patients with features of toxicity, a repeat level should be checked in case of prolonged drug absorption, and repeated until levels are falling.
- Poisoning severity is indicated by plasma salicylate concentrations taken together with clinical and biochemical features. Concentrations >350 mg/l (2.5 mmol/l) are associated with toxicity, and concentrations >700 mg/l (5.1 mmol/l) are associated with severe toxicity and may be fatal. Confusion, impaired consciousness, metabolic acidosis and high salicylate concentrations all indicate severe poisoning.
• Check U&Es, prothrombin time (INR) and blood glucose. If serum potassium is low this must be corrected first. After correction of serum potassium, metabolic acidosis should be corrected with IV sodium bicarbonate.

• If salicylate >500 mg/l, then IV 1.26% sodium bicarbonate 1500 ml should be administered over 2 hours to enhance salicylate clearance. This should be repeated as necessary to obtain optimal urine pH 7.5-8.5. It is important to monitor electrolytes and acid-base status closely (particularly to avoid hypokalaemia).

• Patients with plasma salicylate level >700 mg/l, those with renal failure, severe metabolic acidosis, pulmonary oedema or CNS toxicity should be considered for haemodialysis (discuss with NPIS for further information).

ANTIDEPRESSANTS

Features
• **Tricyclic antidepressants (TCAs)** e.g amitriptyline, dosulepin
  Tachycardia, dilated pupils, urinary retention, hyperreflexia, divergent squint, hypotension, seizures, coma, arrhythmias, prolonged QRS duration, metabolic acidosis.

• **Selective serotonin reuptake inhibitors (SSRIs)** e.g. paroxetine, sertraline. Nausea, vomiting, tremor, prolonged QTc, serotonergic syndromes.

• **Selective norepinephrine reuptake inhibitors (SNRIs)** e.g. venlafaxine. Tachycardia, tremor, agitation, prolonged QRS and QTc duration, arrhythmia, seizures, coma.

• **Mirtazapine**. Drowsiness, nausea, vomiting.

Toxicity
• In general the most toxic in overdose are venlafaxine and tricyclics (particularly dosulepin) due to the risk of seizures and arrhythmia.

• Toxicity greatest when two or more antidepressants taken together.

Management
• Consider activated charcoal if within 1 hour of ingestion.

• Organise early intubation and intensive care admission if reduced conscious level.

• Correct electrolyte or acid-base disturbance, ensure adequate hydration.

• Perform ECG and monitor cardiac rhythm.

• If QRS >120 ms after TCA overdose, administer IV 8.4% sodium bicarbonate 50 ml (=50 mmol) via central or large peripheral vein,
even in the absence of acidosis, to reduce risk of arrhythmia and seizure. Repeat as necessary.

- Arrhythmias are best treated by correction of hypoxia and acidosis (metabolic and respiratory). Torsade de pointes should be treated with IV magnesium sulphate 8-10 mmol over 1-2 minutes. Consult TOXBASE® or contact NPIS for further advice.
- Treat seizures with IV lorazepam (2-4 mg) or diazepam 5-10mg; repeated doses may be required.
- Serotonin syndrome may occur after ingestion of 2 or more drugs with serotonergic effects e.g. TCAs, SSRIs, monoamine oxidase inhibitors, tramadol. Features include alteration of mental status, neuromuscular hyperactivity and autonomic instability. If suspected, monitor temperature and check serum creatinine kinase (CK). Discuss management with NPIS.

**OPIOIDS**

For example codeine, diamorphine, dihydrocodeine, fentanyl, methadone, morphine, pethidine, tramadol.

**Features**

Reduced conscious level, respiratory depression, pinpoint pupils and hypotension. (N.B. opioids and their active metabolites accumulate in patients with renal impairment: opioid toxicity should be suspected in any patient with unexplained type-2 respiratory failure)

**Management**

- ABCDE as Chapter 2.
- Monitor respiratory rate and ensure adequate airway and support ventilation.
- If reduced conscious level or respiratory depression, then administer IV naloxone 0.4-2.0 mg: repeat the dose if inadequate response after 2 minutes.
- Naloxone (Narcan®) is a competitive antagonist and large doses (>4 mg = 10 ampoules) may be required in severe cases.
- Naloxone can be administered by the IM route if IV access is not possible, or if the patient is threatening to self-discharge when its effects might be more prolonged.

> The plasma half-life of naloxone is shorter than that of most opioids, so repeated doses are often required. This is especially true of long-acting opiates (e.g. MST or methadone), where a naloxone infusion might be needed.
Naloxone infusion is usually started at around 60% of the initial dose per hour. A solution containing 5 mg (12.5 ampoules) reconstituted in 25 mls dextrose gives a 200 micrograms/ml solution for IV infusion via a syringe driver.

Measure U&Es and CK. **N.B. patients who have reduced conscious level are at high risk of rhabdomyolysis, pressure injuries and compartment syndromes.**

### RECREATIONAL DRUGS

**Features**
- Stimulants such as MDMA (ecstasy), amphetamines, cocaine, lysergic acid diethylamide (LSD) may cause severe agitation, tachycardia, sweating, pyrexia, dilated pupils, hypertension, arrhythmia and seizures. Severe cases result in coma, rhabdomyolysis, renal failure, subarchnoid haemorrhage, myocardial infarction, refractory seizures and death.

**Specific features**
- Cocaine also causes coronary artery spasm, myocardial ischaemia and infarction and aortic dissection.
- Ecstasy may cause severe hyponatraemia.
- Gamma hydroxybutyrate (GHB) may cause bradycardia, hypotension, reduced conscious level and coma.

**Management**
- Measure U&Es, LFTs and CK.
- Perform ECG and monitor cardiac rhythm.
- Control agitation and seizures with diazepam. Large doses and repeated administration may be required.
- Hypertension usually settles after administration of diazepam. If hypertension persists despite diazepam, then consider intravenous nitrates (e.g. glyceryl trinitrate 1-2 mg/hour) and gradually increase the dose until blood pressure is controlled.
- Treat cocaine induced chest pain and ECG changes with aspirin, diazepam and nitrates.
- Tachycardia usually responds well to adequate sedation and control of agitation, and specific therapy is not normally needed.
- Correct metabolic acidosis with sodium bicarbonate.
- Hyperthermia should be treated with passive cooling and sedation with intravenous diazepam (large doses may be required). However, when body temperatures exceed 40ºC, then more active cooling is preferable, and the patient should be transferred to a critical care area.
ACUTE RHEUMATOLOGY

Chapter 11

ACUTE MONO OR OLIGOARTHRITIS

Commonest causes are summarised by the abbreviation GRASP. Acute arthritis in > 1 joint should be considered to be sepsis until proven otherwise.

- Every effort should be made to aspirate involved joints, involving on-site orthopaedic teams/radiology for ultrasound guided aspiration if necessary.
- It is imperative to send blood cultures on admission.
- Once aspirates and blood cultures are sent, empirical IV antibiotics (see below) should be commenced. Err on side of diagnosis of sepsis until proven otherwise.

GOUT

- 1\textsuperscript{st} MTPJ > ankle > knee > upper limb: tophi.
- Middle age to elderly.
- Men > women.
- Polyarticular in 10%.
- Can mimic sepsis: see above.
- Atypical subacute onset in hands in elderly women with renal impairment on diuretics.
- History of previous attacks, alcohol or diuretic intake, obesity, renal disease.
- Family history.

REACTIVE ARTHRITIS

- Young male > female.
- Large joint, lower limb: usually more than one.
- Can mimic sepsis: see above.
- History must include GI, Genito-urinary and sexual information.
- Balanitis, keratoderma blenorrhagicum, nail changes.
- Conjunctivitis, iritis.
SEPSIS

- Any age, any joint, may be more than one joint.
- General symptoms: malaise, fever.
- Skin infection may be seen e.g. pustules, boils.
- *Staphylococcus aureus* is most common organism in adults.
- Is there reduced immunity? e.g. Rheumatoid arthritis, steroids, NSAID, liver or renal disease.
- Gonococcal arthritis should be considered in young adults. Patients are usually female with polyarticular disease. There may be no clinical evidence of concurrent STD.

PSEUDOGOUT

- Middle aged or elderly.
- Knee or wrist.
- Can mimic gout/sepsis.
- Previous attacks likely.
- May have chondrocalcinosis on x-ray.

OTHER CAUSES

Other causes include: haemarthrosis, monoarticular presentation of polyarticular disease, mechanical.

INVESTIGATIONS

For All

Every effort should be taken to aspirate involved joints: Involve on-site orthopaedic teams/radiology for ultrasound guided aspiration if necessary.

- Record colour, viscosity and turbidity.
- Microscopy for cell count, differential and Gram stain (Microbiology); polarising microscopy for crystals (Histopathology lab).
- Culture.
- Blood cultures x3.
- FBC and diff, ESR and CRP.
- X-ray joint on admission.

Selective Investigations

- Gout: serum urate (but a poor discriminator).
• Reactive: stool for Salmonella, Campylobacter, Shigella, Yersinia.
• STDs: endocervical swab or first pass urine for chlamydia. Endocervical, urethral, rectal and throat swabs (as applicable on history) for culture for gonococcus.
• Yersinia serology (if stool culture negative).
• Serology in polyarthritis: parovirus, ASOT, mycoplasma. Consider also, if possible exposure history, Lyme.

**MANAGEMENT**

Seek rheumatological advice early in suspected septic or reactive arthritis via WGH switchboard.

If gonorrhoea confirmed, contact tracing should be arranged via Genito Urinary Medicine.

GUM do not contact trace for chlamydia: arrange yourself or via patient’s GP.

**Analgesia**

- Paracetamol
- NSAIDs
- Others

In cases with infected joint prosthesis obtain specialist Orthopaedic or Rheumatological advice.

**Gout**

- Bed rest plus high dose indometacin (indomethacin) 50mg qds oral or alternative NSAID e.g. diclofenac 50mg oral bd to maximum dose. Colchicine (0.5 mg oral od-tds depending on tolerance) is useful in patients in whom NSAIDs are contraindicated (e.g. renal failure, allergy, GI complications). Should be used under expert supervision. Leave 3 clear days between courses, halve dose if creatinine clearance <10ml/min.
- Intra-articular steroid may be used in difficult cases: consult Rheumatologist.
- Do not use allopurinol until attack has settled for at least 2 weeks and only introduce with NSAID or colchicine (0.5mg bd) cover. Adjust dose of allopurinol if renal function impaired: normal renal function 300mg od oral, creatinine clearance 30-60ml/min 200mg od oral, creatinine clearance <30ml/min 100mg od oral.
Reactive Arthritis

- Bed rest plus NSAID in adequate dose +/- intra-articular steroid. Treat associated/triggering infection. If active arthritis persists consult Rheumatologist.

Septic Arthritis

- Rest joint in appropriate position.
- Antibiotic therapy
  a) First line therapy: flucloxacillin 2 g iv 6 hourly
  b) In circumstances where there is increased likelihood of Gram negative infection (chronic or acute urinary tract infection, chronic prostate symptoms, recent intra-abdominal surgery) use flucloxacillin 2 g iv 6 hourly plus ciprofloxacin 500mg oral bd (ciprofloxacin 400mg iv bd if unable to take oral).
  c) Seek Microbiological advice if suspected (risk factors present) MRSA or confirmed MRSA positive.
- Treatment will vary locally e.g. Orthopaedic patients may be different to others. Discuss with rheumatologist/orthopaedic surgeon and specialist microbiologist.
- Once cultures available treat according to sensitivities and on microbiology advice.
- Duration of antibiotic therapy: minimum of 2 weeks IV, then prolonged oral or IV therapy depending on whether prosthetic joint, whether patient immunosuppressed and pathogen. Seek specialist advice (from Rheumatology/ID/Microbiology).

Pseudo-gout

- Bed rest, joint aspiration, single injection of intra-articular steroid usually sufficient. NSAID may be used.
Chapter 12
PSYCHOLOGICAL MEDICINE

ALCOHOL

PRESENTATION

- Acute intoxication.
- Withdrawal “DT’s” see below.
- Seizures: withdrawal or intoxication, or hypoglycaemia.
- Associated problem e.g. pneumonia, rhabdomyolysis.
- Incidental e.g. admission for unrelated problem.

MANAGEMENT

- Check plasma alcohol level, FBC, U&E’s, glucose, LFT’s, clotting, and other tests indicated e.g. amylase if abdominal pain.
- Start thiamine 300mg od oral.
- **Pabrinex** may be required if NBM, actual or incipient Wernicke’s encephalopathy or Korsakoff’s psychosis (see below).
- Indications for Pabrinex:
  - Acute confusion
  - Reduced conscious level
  - Memory problems
  - Ataxia
  - Ophthalmoplegia
  - Hypoglycaemia
- **Pabrinex** IVHP (No1 and No2 mixed) by IV infusion in 100ml 5% dextrose over 30mins then 8 hourly for 48 hours.
  - **N.B.** Risk of anaphylaxis - facilities for treating this must be readily available.
- **Alcohol withdrawal management** guidelines are detailed below and updates are available on the Intranet.
- Never prescribe hypnotics as discharge drugs.

### Information

**High dependency or intensive care and nursing observation is required with IV sedatives.**

- Treat any associated problems. Screen for infection including CXR.
  - **N.B.** Remember spontaneous bacterial peritonitis and tap any ascites and send for culture (in blood culture bottles), and urgent Gram stain and cell count, WCC (>250 per microlitre suggestive of SBP). Check cytology on ascitic tap.
• Consider a referral to the liaison psychiatrist/alcohol dependence team/social worker.

NOTE

• Myelo-suppression, with a reduced platelet count is not uncommon, as is folate deficiency.
• In chronic pancreatitis the amylase may be normal. A raised CRP is the best guide.
• TB is more common in alcoholics. Request AFB’s on sputum sample x3 (preferably early morning).
• Don’t assume alcohol is responsible for a fit. Could the patient have meningitis, or intra-cerebral pathology following a fall?
• Check for hypoglycaemia.
• Encephalopathic patients may have flap, ↓ LOC, signs of chronic liver disease. Distinguish from DT’s (tremor, restlessness).
• Common precipitating causes of encephalopathy are infection, GI bleed, electrolyte disturbance, constipation.
• Withdrawal may occur two to three days after hospitalization.

MANAGEMENT OF ALCOHOL WITHDRAWAL

Alcohol dependence and withdrawal are associated with significant morbidity and mortality. People who admit to drinking more than 10 units a day are likely to have withdrawal symptoms. Delirium tremens is rare at a consumption of less than 15 units per day. Hypoglycaemia, hypokalemia, hypocalcaemia and fever may predispose patients to seizures or delirium tremens.

INITIAL ASSESSMENT

History
Ask the patient ‘Do you take a drink sometimes?’ or ‘What have you had to drink in the last week?’ Make a note of alcohol consumption in units wherever possible. If you suspect alcohol dependence ask ‘have you experienced tremor or shakiness in the morning - and taken a drink to relieve this?’ Ask when they last had a drink. Try to take a history from an informant if the patient is unable to co-operate.

Examination
Look for excessive capillarisation of the conjunctivae or facial skin, palmar erythema, and alcohol on the breath.

Key investigations: alcohol level or breathalyser, Gamma GT and liver function tests, MCV.
MILD SYMPTOMS
Tense, irritable, poor concentration.
If there is suspicion of withdrawal then review regularly

Alcohol intake?

<50 units/week

Encourage fluids

Continue to observe

No medication required on discharge

MODERATE SYMPTOMS
Tachycardia, nausea, tremor, sweats, anxious, headache, irritable, flu-like symptoms, seizures

>50 units/week or previous history of alcohol withdrawal

Oral diazepam 10-40mg up to four times daily and hourly (if necessary) are required (max 160mg/24 hours) titrated to response.
If no response after 160mg, stop diazepam and go to Step 2 of Severe Symptoms.
NB: syrup form available

SEVERE SYMPTOMS
Confusion, visual or auditory hallucinations, irrational thought/fears, bizarre, aggressive or unco-operative behaviour

1. GAIN CONTROL
IV diazemuls by slow injection. Up to 40mg in divided doses during the first 30 minutes whilst monitored
Adjunctive therapy - haloperidol 5-10mg IM or IV. If still symptomatic contact admitting consultant for advice (see notes).
Options
(i) IM paraldehyde (5-10ml) max 20ml
(ii) Midazolam infusion 0.03-0.2 mg/kg/hr
Consider transfer to higher dependency unit. Call ICU consultant or anaesthetist if appropriate.

2. TO MAINTAIN CONTROL
Haloperidol (oral) 5mg twice daily increasing to 5mg four times daily if required.

VITAMIN SUPPLEMENTATION

ORAL - Thiamine 300mg stat dose then 300mg once daily.
IV - Indications:
(i) Vomiting/malabsorption/general debility,
(ii) Risk of Wernicke/Korsakoff Dose of Pabrinex (2 pairs) up to 8 hourly until oral intake adequate. If in doubt - give Pabrinex without delay.
See CSM advice below.

CSM Advice - Pabrinex
I - 1. Restrict to patients in whom enteral treatment is not possible
II - 2. Administer over at least 10 minutes
III - 3. Facilities for treating anaphylaxis should be available

REVIEW PATIENT TWICE DAILY
I - • Increase in medication? Signs of major toxic side effects of treatment medication?
   (a) IV benzodiazepine - monitor pulse, oximetry and respiratory rate. Reversed with flumazenil.
   (b) Haloperidol extrapyramidal side effects. Reversed with procyclidine.
II - • Reduction in medication?

CONTINUATION THERAPY
Convert to oral diazepam as soon as possible or reduce dose of haloperidol

On discharge prescribe:
• Thiamine 300mg once daily if chronic alcohol problem (GP to review need)
• Diazepam 2-3 days reducing course if committed to abstinence and if under appropriate review.

SPECIAL NOTES
1. Dose with caution in the elderly.
2. Thiamine/Pabrinex should always be given before the administration of dextrose fluids to avoid precipitating Wernicke Syndrome.
3. Reality orientation and reassurance is encouraged.
4. Transfer patients to oral medication as soon as possible.
5. For complicated cases or cases that are difficult to control seek specialist advice:
   At RIE - consult Psychiatric Team, Medical Registrar/Consult on call at RIE or the duty psychiatrist (REH).
   At WGH - consult consultant on-call or in charge.
   At SJH - contact Psychiatry SHO on-call, radio page via switchboard.
6. For follow up contact the Alcohol Liaison Service, RIE ext 21396, WGH contact ext 31834.
7. These guidelines may not be appropriate in the peri-operative period.

Consider referral to Intensive Care if requiring more sedation.
The service operates at the **Royal Infirmary Monday to Friday 09:00-17:00**.

Direct referral should be made by telephone to extension 21396/21398 or by bleeping **Sr Leslie (#6426)**.

If patients are admitted and discharged over a weekend, referrals can be made via the service answerphone (as above) or complete a weekend referral form kept in doctors’ rooms in CAA.

The following details are required when making a referral:

- Referrer
- Patient name
- Address
- DOB
- Reason for admission

**PATIENT’S PERMISSION MUST BE SOUGHT PRIOR TO REFERRAL**

**N.B.** For patients to be seen promptly, referrals must be made as early in the day as possible. Where possible, a same day service is offered.

There is currently no alcohol liaison service at the WGH. Referrals from the WGH should go either to psychiatry or the Alcohol Problem Service at the Royal Edinburgh Hospital.

St John’s: radiopage via switchboard

**Alcohol Withdrawal Seizures**

Initial treatment with 10mgs diazepam (as Diazemuls) by intravenous injection over two minutes may be given. Status epilepticus should be treated according to the guidelines.

**Fluid and Electrolyte Balance**

Examine for features of fluid depletion and check U&Es. Oral fluid intake of 2-2.5 litres per day should be given. Intravenous replacement of fluid and electrolytes may be required; potassium and magnesium supplementation should be tailored according to blood chemistry. Hypoglycaemia should be excluded by blood sugar measurements and treated accordingly.

**Vitamin Supplementation:** see above.
ACUTE DISTURBANCE

Guide for medical practitioners on the granting of an emergency detention certificate under section 36 of the Mental Health (Care and Treatment) (Scotland) Act 2003.

Registered medical practitioner (see notes 1) carries out a medical examination and recommends hospital admission.

Patient Refuses Admission

Patient Agrees to Admission

The patients must meet these grounds for detention:

1. You consider it likely that conditions (a) and (b) are met:
   (a) the person has a mental disorder (see notes 2) : and
   (b) because of that mental disorder, the person’s ability to make decisions about the provision of medical treatment for that mental disorder is significantly impaired.

   AND

2. You are satisfied that conditions (a) to (c) are met:
   (a) it is necessary as a matter of urgency to detain the patient in hospital for the purpose of determining what medical treatment requires to be provided to the patient:
   (b) if the patient were not detained in hospital there would be a significant risk to the health, safety or welfare of the patient or to the safety of any other person if the patient were not detained in hospital.
   (c) making arrangements with a view to granting a short-term detention certificate would involve undesirable delay.

   AND

3. Immediately before the medical examination, the patient was not detained in hospital by way of certain provisions of the Act (see note 3).

   AND

4. There was no conflict of interests in relations to the medical examinations (see note 4).

Detention criteria are met

Detention criteria are not met: emergency detention certificate may not be granted.

You must, where practicable, consult a mental health officer (MHO) and obtain their consent to the granting of the certificate. See notes 5 and 6.

MHO consent obtained.

Impracticable to consult and obtain the consent of an MHO.

MHO consent refused.

1. Inform patient of decision to grant the certificate
2. Complete and sign the emergency detention on certificate within prescribed timescales (see notes 7,8 and 9)
3. Ensure that arrangements are in place for the patient’s transfer to hospital where this is required.
4. Ensure that the detention certificate is passed to the relevant hospital managers (see note 10).

Throughout the process of granting an emergency detention certificates, you are bound to have regard to the principles of the legislation as laid out in sections 1 to 3 of the Act.

*AMP: approved medical practitioner
This section is still under development. Please refer to the final version on the Intranet.

**Note 1:** Any registered medical practitioner may grant an emergency detention certificate. You do not have to be an approved medical practitioner.

**Note 2:** Section 328(1) of the Act defines “mental disorder” as “mental illness, personality disorder or learning disability, however caused or manifested”. Section 328(1) further states that a person is not mentally disordered by reason only of sexual orientation, sexual deviancy: transsexualism: transvestism: dependence on, or use of alcohol or drugs: behaviour that causes, or likely to cause harassment, alarm or distress to an other person: or acting as no prudent person would act.

**Note 3:** The relevant provisions are set out at section 36(2) of the Act and they are: an emergency detention certificate: a short-term detention certificate: an extension certificate issued under section 47 of the Act pending an application for a CTO: section 68 of the Act (i.e. the extension to the detention period authorised once a CTO application has been submitted to the Tribunal): a certificate granted under sections 114(2) or 115(2) of the Act (i.e. a certificate issued subsequent to a patients non-compliance with the terms of a community-based interim CTO or a CTO).

**Note 4:** [DN - conflict of interest material to be added once regs/Code of Practice material has been finalised]

**Note 5:** The medical practitioner must consult and seek the consent of an MHO to the granting of the certificate. All reasonable efforts should be made to contact a MHO. However, where the urgency of the situation is so great that it would not be practicable for this consultation to take place then it is permissible for the practitioner to grant the EDC without consent. [DN - Add best practice material about consulting and discussing the situation with other members of the multi-disciplinary team depending on what the final version of the Code says.]

**Note 6:** Best practice would be that if one MHO refuses to grant consent, then.... [DN - revise according to the final version of the Code]

**Note 7:** A valid emergency detention certificate can be issued on any document if form [x] is not available. However, it is strongly recommended that form [x] be used in all circumstances. If form [x] is not used, the emergency detention certificate must state the
practitioner’s reasons for believing the conditions mentioned at points 1 and 2 on the blue box overleaf to be met and must be signed by the medical practitioner.

**Note 8:** The emergency detention certificate must be completed either by the end of the day on which the medical examination takes place (if the examination takes place before 8pm) or within 4 hours of the medical examination being completed (if it takes place after 8pm).

**Note 9:** The emergency detention certificate authorises first the patient’s transfer to hospital within 72 hours of the certificate being granted: and secondly, the patient’s detention in hospital for 72 hours.

**Note 10:** Section 36(7) of the Act states that the patient’s detention in hospital is only authorised if the emergency detention certificate is given to the managers of the hospital before the patient is admitted to hospital under the authority of the certificate. If the patient is already in hospital when the certificate is granted, then the certificate must be given to the hospital managers as soon as practicable after it was granted [DN - clarification of “hospital managers’ required? depends on final content of Code on this point.]

The purpose of the above information is to act as a guide only. It does not provide full and comprehensive coverage of everything you ought to know about emergency detentions. For fuller information please consult the Act and its Code of Practice.

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**OBTAINING INFORMED CONSENT POLICY/PROCEDURE**

Care for patients in general as well as psychiatric hospital settings.

Commonest problem area that is covered by the Adults with Incapacity Act. However, other areas such as the inability to manage money or to agree to discharge arrangements may be important.

**Frequently asked questions and answers**

**Q1 Why use the Mental Health Act in general hospital?**

A1 If someone with a mental disorder is at risk of self-harm, self-neglect or of harming others they may be prevented from leaving hospital by use of the Act. Sometimes the Act is used to authorise restraint of a violent person. Authorisation of compulsory treatment for mental illness may occasionally be required.

**Q2 Who could be detained?**
A2 Someone who has a mental disorder and impaired judgement about the treatment of that disorder could be detained on a short-term detention certificate (STDC). If using a STDC would involve ‘undesirable delay’ an emergency detention certificate (EDC) may be used. Detention may be necessary to allow for full assessment of suicidal intent. Violent behaviour is not a mental disorder but may be a sign of underlying mental disorder such as mania or schizophrenia. Similarly, violence associated with drug or alcohol intoxication or dependence is not a mental disorder but delerium or a confusional state may result from drug or alcohol withdrawal and may justify detention. Psychotic illnesses may result from drug or alcohol use. Intoxication may increase the risk of harm to self or others and this should be taken into account when considering the detention of someone with an underlying mental disorder.

Q3 How can someone be detained under the Mental Health Act?

A3 Only a senior psychiatrist, who is an Approved Medical Practitioner, can grant a STDC, which is effective for up to 28 days. The consent of a specialised social worker (Mental Health Officer/MHO) is required. An AMP may not be available out of hours in smaller hospitals. A fully registered medical practitioner can grant an EDC, normally with the consent of an MHO. If it is impossible to consult the MHO consent can be dispensed with. Under the previous Mental Health Act 1984 a relative could give consent, but this is no longer permitted. An EDC may be justified when detention in hospital is needed urgently in order to assess the need for treatment and where this is a risk to the person’s health, safety or welfare or the safety of others. A form called DET1 will need to be completed. Forms can be downloaded from the Scottish Executive website. The form must be passed to the Medical Records Department after completion for it to take effect. It should not be filed in case notes.

Q4 What measures are authorised by an EDC?

A4 If the person is not an inpatient, admission to hospital is authorised (within 72 hours) and detention in hospital is then authorised for up to 72 hours. A person in an Accident and Emergency department is not usually an inpatient and an EDC will not usually authorise detention of a person there. Under an EDC, treatment for mental disorder may be given if the person is able to consent and does so. Without consent, only urgent treatment may be given. There are some restrictions on the type of treatment that may be given e.g. treatment that entails significant physical hazard may not be given. The Mental Welfare Commission must be informed within 7 days, using form T4.
Q5 When does an EDC end?
A5 A person who is subject to an EDC should be assessed by an Approved Medical Practitioner as as possible. Normally the EDC will be rescinded and the person will either become informal or made subject to a STDC. An EDC should not usually remain in force for the full 72 hours. Occasionally a person may need to leave hospital temporarily during the period of detention. The EDC can be suspended by the doctor in charge of the patient’s care, the Responsible Medical Officer.

Q6 What measures are authorised by a STDC?
A6 Compulsory treatment for a mental disorder is authorised in addition to detention. Treatment can be given without the patient’s consent but with reference to the principles.

Q7 What is a Compulsory Treatment Order?
A7 This is a long-term order, with provisions similar to a STDC. Compulsory treatment under these orders can be given in hospital or in the community. A Compulsion Order is similar but is granted by a court. Occasionally patients subject to these orders are admitted to general hospital for treatment of a physical condition. A psychiatrist (AMP) must be responsible for mental health care. Liaison between the relevant psychiatric and general medical records departments is essential to ensure that the necessary legal arrangements are made to allow the patient to be admitted to the general hospital. If the psychiatric unit is in the same hospital, no special arrangements are necessary. There is no requirement under the Act that such patients should be cared for by mental health nurses but local arrangements may be made if this is appropriate.

Q8 Who is responsible for the patient’s treatment under a compulsory order?
A8 Hospital managers must appoint an AMP to be the patient’s Responsible Medical Officer. He or she is responsible for the patient’s mental health care on the general ward but responsibility for the treatment of the patient’s physical disorder remains with the appropriate physician or surgeon. If the patient’s detention has been suspended, the psychiatric hospital is still responsible for appointing an AMP. However, if the patient’s order has been transferred to the general hospital, its managers are responsible for appointing the AMP. There should be clear arrangements for liaison between the AMP and the medical/surgical team.
Q9 Can treatment for a physical condition be given without consent?

A9 The Mental Health (Care & Treatment) Scotland Act authorises treatment for mental disorder or for conditions that are a consequence of mental disorder. Treatment of conditions that are a direct cause of mental disorder, such as infection causing delirium, is authorised, as is treatment of conditions directly resulting from mental disorder, such as self-poisoning resulting from a depressive illness. Artificial feeding of a person with anorexia nervosa or severe depression may be authorised by the Act although a second opinion is required.

Where a person is unable to consent to treatment because of mental disorder, treatment can be authorised under the Adults with Incapacity Act by completing a Section 47 certificate, providing that person does not object or resist. In an emergency, if a person objects or resists, treatment can be given under common law, but the AWIA procedure should be used if time allows.

Another person may be authorised to give consent under AWIA. This may be through Power of Attorney, an intervention Order or Welfare Guardianship. The consent of the person delegated to make a decision should always be sought whenever practicable. Occasionally consent may be withheld. In these circumstances treatment may not be given. The AWIA includes arrangements to resolve such disputes, including a second opinion procedure.

Where treatment is not urgent and the patient objects or resists, there is no simple procedure to authorise treatment. Guardianship or an intervention order may be approved by a court, and an enforcement order applied for subsequently, but the Commission does not know of any examples of this leading to successful treatment.

Further information about the Mental Health and Incapacity Law in Scotland is available from the Commission’s website www.mwcscot.org.uk

The website provides links to the Acts and their Codes of Practice. Mental Health Act forms can be downloaded from the Scottish Executive website. A link to forms is also provided from the Commission site.
Other useful information sources:
www.gmc-uk.org
www.bma.org.uk
www.nmc-uk.org

Guidelines for Continuing Care

- Nurse in good lighting, a cool ambient temperature with good ventilation and supportive staff. Augment psychosocial and alcohol history.
- Perform a detailed physical examination looking for the stigmata of alcohol abuse.
- Feed back diagnosis to the patient, with the results of tests, in an open, but helpful manner.
- Abstinence should be advised if there is alcohol dependence with physical damage.
- Follow up should be arranged to aid this. Possibilities include: their GP, the Department of Psychological Medicine at the Western General, Social Work department, the Alcohol Problems Clinic at the Royal Edinburgh Hospital, Alcoholics Anonymous, Lothian Council on Alcoholism. In any event, always inform the GP by letter.
SHARING DIFFICULT INFORMATION WITH PATIENTS/RELATIVES

Be truthful but sensitive to the amount of information wanted. The communication process should be two way.

PREPARE FOR THE INTERVIEW

- Plan the meeting - a relative/close friend should be present, if possible:
  - allow enough time; not too early or late in the day.
  - ensure the patient is awake and comfortable.
  - in hospital, avoid giving bad news at the bedside if possible.
  - ensure you have all the relevant information.
- Place - quiet, private, equipped with tissues, notes/results, written information, booklets etc.
- Protect against interruption.
- Clinical staff who know the patients should give the results of tests; preferably in person not by phone.
- A nurse who knows the family should be involved.

GIVING INFORMATION

- Manage the whole interview by summarising, clarifying what has been understood and checking for outstanding issues/concerns. Use clear, simple, unambiguous language.
- Check how much the patient/relative knows already, e.g. “Can you tell me what you understand about the illness?”
- Check how much they want to know, e.g. “Are you the sort of person that likes to know exactly what is happening?”
- Clarify the current situation and give any new information, tailored to the person’s needs.
  BUT, if the person is unaware of the situation - give a warning shot, e.g. “I’m afraid things are not so good”
  - break the news slowly in small steps - pause after each.

Coping with patient’s/relative’s reactions & distress:

- A slow pace, with pauses, allows the person to take the information in.
- Avoid premature advice/reassurance - it may be misinterpreted or not heard.
• Acknowledge/empathise with distress and encourage the person to talk about their feelings, e.g. “It sounds as if you feel ....”
• Help the person identify specific concerns resulting from the information given, e.g. “Can I ask you what exactly worries you about...?”
• Summarise and prioritise the person’s concerns.
• Take the person’s concerns in order of priority and give appropriate information/advice.
• Give reassurance of ongoing support and agree a joint plan of action.

At the end of the interview
• Summarise the conversation and offer to write down key information.
• Offer relevant written information/booklets.
• Arrange a later opportunity to ask further questions or go over the information again.
• Check if there is anything else they need now.
• Offer the patient/relative time alone if they wish.

After the interview
• Record details of: the information given
  any resulting concerns/issues
  follow-up arrangements
• Ensure that other key staff including the patient’s GP/consultant are aware of what has been said.

GIVING DIFFICULT INFORMATION BY TELEPHONE

This should be avoided, if possible, but may be necessary e.g. to inform relatives of a death.

In advance:
• Find out if the family want to be informed of changes in the patient’s condition by phone.
• Do the family want to be contacted overnight or not?
• Is one family member to be contacted first?
  ➔ Record these details clearly in the patient’s record with contact numbers.
• If the death is “sudden and unexpected” it is always better if the GP, emergency social work service or the police go and break the news to relatives.
1. Write down or review what you are going to say before you phone.

2. **Speak slowly**

3. Check if you are speaking to the right person.

4. State who you are and where you are phoning from.

5. Warn them you have some bad news and check if they are alone. **PAUSE.**

6. Give the person the opportunity to phone you back later if they wish.

7. Give the information slowly, simply and clearly.
   - If the patient has died, it is better to tell the truth. **(Avoid euphemisms e.g. passed away).**

8. Express your regret. **PAUSE.**

9. **IF ALONE -** offer to phone a relative/friend to be with them. 
   **IF NOT ALONE -** offer to speak to the relative/friend who is there.

10. Check when and how relatives will be coming into the unit. They do not need to rush.

11. Assure them medical and nursing staff will be available to talk to them.

12. Phone and inform the GP of a patient’s death.
END OF LIFE CARE

WHEN DEATH OCCURS - CARE GOALS

If you have a bleep, ask someone to hold it while you speak with the family. Turn off mobile phone.

GOAL
Family feel supported in the decision to be alone with the patient

- Reassure them that they will be given all the time they need.
- Continue to make regular contact to provide support, but do not imply haste.
- Provide a separate private area to enable the family to be together.
- After a period of time ascertain if family still comfortable staying, sometimes they are at a loss as to what to do or what happens next.

GOAL
Religious/cultural issues are identified

- Ask family if they would welcome support of minister/priest/other. Religious/Cultural issues?
- Have they been identified? (see “What to do after a death in Scotland Booklet” - Chapter 10)

GOAL
Family supported and advice given if they are waiting for the Death Certificate to be issued

- Medical staff will pronounce life extinct (PLE).
- Provide tea/coffee in separate room, to allow medical staff to confirm death and issue death certificate to the family in private.
- Medical staff will assess whether the Procurator Fiscal should be informed. The death certificate cannot be issued by hospital staff in the event of the fiscal taking over the case.
- Using professional judgement as to the appropriateness, sensitively ascertain if any arrangements have been made/discussed re burial or cremation. If cremation is chosen, or if intentions are not clear, a Cremation Form part B should be completed and sent to the mortuary. It should not be handed to relatives. The mortuary will arrange for Form C to be completed if a post mortem is not undertaken and will give the cremation form to the undertaker.
- Ward staff return belongings as per policy* and give bereavement booklet and invitation for bereavement support.

GOAL
Arrangements for organ donation or post mortem if appropriate

- If there is a possibility of organ donation or a post mortem examination is thought desirable, discuss with the family.
- A cremation Form C is not required if a post mortem examination has been undertaken but consultation with pathologist is necessary for completion of Q8a in Form B.

GOAL
Declaration of serious infection hazard to undertaker.

Complete a care of the deceased form (infection certificate) and send to mortuary.

GOAL
Family advised what to do next (if they do not wish to wait for the death certificate)

- Advise to return at a mutually convenient time the next day.
- Inform them that any member of the family / friend can do this as it is often too difficult for the immediate family.

GOAL
Advice given to the family about what to do next

Explain steps in booklet pertaining to registering the death and about the role of the funeral directors.

GOAL
Enquire whether the person has support at home

Discuss whether contact with family /friends or GP is required for support. If appropriate accompany to the end of the ward or to the car.

*If all the above have been addressed perform Last Offices identifying cultural beliefs and spiritual needs (refer to manual if required)

Ensure remaining patients concerns are addressed

<table>
<thead>
<tr>
<th>Initials</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above Goals met</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

If “No” record

a variance
(Code …)

Appendix 2

END OF LIFE CARE

WHEN DEATH OCCURS - CARE GOALS

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- A cremation Form C is not required if a post mortem examination has been undertaken but consultation with pathologist is necessary for completion of Q8a in Form B.

GOAL
Declaration of serious infection hazard to undertaker.

Complete a care of the deceased form (infection certificate) and send to mortuary.

GOAL
Family advised what to do next (if they do not wish to wait for the death certificate)

- Advise to return at a mutually convenient time the next day.
- Inform them that any member of the family / friend can do this as it is often too difficult for the immediate family.

GOAL
Advice given to the family about what to do next

Explain steps in booklet pertaining to registering the death and about the role of the funeral directors.

GOAL
Enquire whether the person has support at home

Discuss whether contact with family /friends or GP is required for support. If appropriate accompany to the end of the ward or to the car.

*If all the above have been addressed perform Last Offices identifying cultural beliefs and spiritual needs (refer to manual if required)

Ensure remaining patients concerns are addressed

<table>
<thead>
<tr>
<th>Initials</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Above Goals met</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

If “No” record

a variance
(Code …)
This checklist is used at ward level to ensure that all important steps are taken and to document timing and responsible individuals.

<table>
<thead>
<tr>
<th>Category</th>
<th>Initials</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient’s death confirmed by doctor?</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Senior nurse in charge informed of patient’s death?</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Next of kin notified of death? (See breaking bad news guidelines)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Procurator Fiscal Notification - <em>inform as appropriate</em></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death certificate prepared? (See instructions in Deaths Book)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death certificate given to family?</td>
<td></td>
<td></td>
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<tr>
<td>Family returning later for death certificate? (if yes please record at the bottom of the page)</td>
<td></td>
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</tr>
<tr>
<td>Bereavement booklet given to family?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valuables/belongings returned to family?</td>
<td></td>
<td></td>
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<tr>
<td>Valuables held in cashier office?</td>
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</tr>
<tr>
<td><strong>Post Mortem  <em>If required</em> - Patient’s family must sign</strong></td>
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<td></td>
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<tr>
<td>Copy of signed post mortem consent given to family?</td>
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<tr>
<td>Cremation Form B (<em>if appropriate</em>) completed?</td>
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<tr>
<td>Cremation Form B (<em>if appropriate</em>) sent to Mortuary?</td>
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<td></td>
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</tr>
<tr>
<td>Infection Certificate for undertaker sent to Mortuary?</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Consultant informed - within 24hrs</td>
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<td></td>
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<tr>
<td>GP contacted - within 24hrs</td>
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</tr>
<tr>
<td>Medical records informed - within 24hrs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cancel any follow up appointments if already booked prior to death</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Arrangements to collect death certificate:** Date: / / Time:

**Other comments:**

Determine families wishes regarding jewellery? To remain on the patient? Yes / No

Comments:

Initials:
Bereaved relatives

- Despite good quality intervention, it is inevitable that resuscitation will not always be successful. Appropriate handling of the situation can help relatives and friends to cope, to start the grieving process and to initiate the practical process relating to funeral arrangements.
- Insensitivity or poor communication at this time may cause long-lasting psychological distress.

Contacting the relatives

- Personal communication is best. Use the Police rather than giving the news by telephone, but if it has to be done by phone an experienced person should do it, and arrange for immediate support e.g. neighbour. Face-to-face communication is the best.

Who should tell them?

- The appropriate person may be a member of the medical team, the named nurse or another. There are no hard and fast rules. It is often appropriate for a doctor and nurse to see the family together.

Practical points

- If you have a bleep or mobile phone, ask someone to hold it while you are speaking with the family.
- Prepare yourself: make sure any blood or other fluid which has been around during resuscitation is cleaned up. Wash your hands, take a big breath in to steady yourself. Plan what you will say.
- Involve a nurse who may know the family.
- CONFIRM they are the correct relatives, who’s who, introduce yourself, find out what they know.
- Physical proximity is important. Sit down, don’t look rushed (even if you are), give them time.
- Make eye contact. Holding hands may be appropriate. Talk clearly in a simple straightforward way getting to the point quickly. Use the word died or death.
- Do not use euphemisms e.g. passed away.
- Emphasise and repeat. Give time for reactions and questions (i.e. be quiet).
- Be truthful, direct, compassionate and empathise. The only thing you may be able to offer is a hug, if this is accepted. If it feels right it usually is right.
Reactions vary: distress, anger, denial, guilt, numbness. Allow and encourage crying. Ask if there are questions: be sympathetic, honest but non-judgmental. Reassure them that pain and other distressing symptoms were dealt with.

- Physical comfort e.g. tea.
- Do not be afraid to show emotion.

Where to tell them?
- A quiet room.

Facilities which should be available
- Paper tissues. Comfortable chairs, a telephone to call out on (not an internal one which may ring at any time). A sink, drinks.

Seeing the patient or body
- Try to get the relatives in before death.
- Warn about equipment and any deformity.
- Encourage them to get close.
- Some relatives will want to help with cleaning the body.
- Remember Religious and Ethnic requirements. Involvement of the Chaplain, Minister, Priest or other religious officials may be welcomed.

Communication
- Contact the GP.
- Notify the Procurator Fiscal if required.
- Religious officers.
- Information on what to do next. Write the death certificate neatly and explain what it says.
- Leaving the hospital: ensure family or friends are available for support.
- Follow up: give the relatives contacts. They may wish to come back to discuss events at a later stage.
- Staff support and debriefing is to be encouraged.

Gaining experience of breaking bad news

Try to accompany experienced members of staff when they are speaking to relatives.

Thanks to the authors of the Lothian Palliative Care Guidelines for the sections on ‘sharing difficult information with patients & relatives’ and ‘giving difficult information by phone’.
GENERAL PRINCIPLES OF GOOD PRACTICE: INFUSION DEVICES

“Health Professionals are personally accountable for their use of medical devices and must therefore ensure that they have appropriate knowledge and training”

Medical Devices Agency 2001

What causes infusion device adverse incidents?

- Free-flow and siphonage.
- Incorrect setting of or failure to set infusion rate.
- Use of inappropriate accessories.
- Calculation errors.
- Lack of knowledge of infusion therapy.
- Patient/Visitor tampering.
- Using damaged devices.

Report mechanical or fluid spillage damage.
NEVER use damaged medical devices.

Free-flow and Siphonage

What is it?

- Uncontrolled fluid flow from container (syringe or fluid bag).

What causes it?

- Gravity: Fluid containers empty if raised above the infusion site and there is nothing to prevent flow (e.g. outflow tube is open).
- Volumetric set: A fluid bag empties if the roller clamp is not closed or the line is not clamped before removal from the pump.
- Syringe pump: Siphonage can occur if: (1) syringe is not properly inserted in pump, (2) the pump is located too high above patient or (3) the syringe is damaged.

Example of poor practice
Bandaged ‘temporary’ repair

Siphonage
A syringe can empty itself, pulling the plunger along, if the line is primed and the plunger is not restrained.

Note: If the syringe is cracked siphonage and emptying of the syringe can occur without movement of the plunger.

Anti-siphon valves are used to help prevent free-flow due to siphonage. They can be thought of as a valve held closed by a spring that requires about 100 to 150mmHg pressure to open and allow flow.
• Damaged syringes and siphonage: Air leaks can occur if the seal between barrel and plunger is broken or if the syringe is cracked. Siphonage then occurs without movement of syringe plunger.

What are the consequences?
• Free-flow and syphonage can cause over-infusion – in severe cases this can lead to death.

What steps should be taken to prevent free-flow?
Syringe pumps:
• Use anti-siphon valve where possible. Use Luer-lock syringes.
• Check that the syringe is not damaged.
• Clamp the syringe securely in the pump (plunger and barrel secured and syringe lip in pump groove) before attaching the line to the patient.
• Pump Height. Ideally, mount syringe pumps and drivers at or below the height of the infusion site.
  Never mount higher than 2 feet above the infusion site.
• Clamp the infusion line before removing the syringe from a syringe pump or driver.

Volumetric pumps
• Ensure that the correct infusion line is selected for use with volumetric pumps.
• Clamp the line before opening the door and removing the line from the pump.

What checks to make before starting an infusion?
• Check infusion rate. Does it match the prescription? **Ask a colleague to tell you the rate.**
• After an end-of-infusion alarm, don’t restart the pump before checking if the prescribed volume has been infused.
• Syringe pumps: Check that the pump correctly registers the syringe size being used (Graseby 3000 series and Alaris Asena series pumps).
Why should you carry out regular checks and what are you looking for?
Infusion devices are very reliable and rarely give problems. However, occasionally they do fail. It is also important to regularly check the infusion site for signs of extravasation or infiltration.

Regular checks

When:
• First check after 15-20 minutes
• Hourly thereafter

What: Patient
• Infusion site. Swelling, pain
• Patient comfort

Infusion System - pump, giving set and fluid container
• Infusion rate
• Volume left in bag or syringe - also check totaliser.
• The line. Has the line been left clamped.

When things go wrong:

Safety Action/Hazard Notices and Incident reporting

Safety Action Bulletins and Hazard notices
What are they? Issued by Scottish Healthcare Supplies to inform users of potential dangers involving medical devices, often following problems experienced in other hospitals.

Where do you find these safety warnings? These should be kept in a file in the clinical area. They are issued by e-mail and through the Messenger. If in doubt, contact the Clinical Skills Co-ordinators or Medical Physics.

Incident reporting
If an adverse event or a near miss occurs, fill in the incident report according to Trust protocol. You should also directly inform Medical Physics (x 22352 - RHSC and RIE; x 32167 - WGH; x 52148 - SJH). The giving set should not be removed from the pump (unless clinical care requires otherwise). The pump, together with the giving set and a copy of the infusion chart should be sent to Medical Physics.
Start-up time (Syringe pumps)

What is it? The time delay between starting an infusion and the patient receiving infusion at the prescribed rate. (Analogous to the time for a car to reach say 60mph from standstill.)

What causes it? Mechanical slack between the syringe and the pump and within the mechanism of the pump.

How can it be prevented or at least minimised?

- Prime line before installing syringe in the pump.
- Install the syringe correctly and firmly in the pump.
- Before connecting line to patient, use the pump’s PURGE facility. This takes up the pump’s mechanical slack, minimising start-up time delay.

Occlusion Alarm Pressure

What is it? Infusion pumps generate sufficient pressure to deliver the infusion at the set rate. If the line becomes fully or partially blocked, the pressure in the line will rise. For example, phlebitis in the vein can increase the resistance to flow causing the pump to increase the delivered pressure to overcome the increased resistance. When the pressure rises above the occlusion alarm pressure the pump alarms OCCLUSION.

What is the limit? Recommended at less than 500mmHg for adult and 300mmHg for paediatric infusions. Some pumps enable the user to adjust the limit - with in-line pressure-monitoring users can adjust the alarm pressure to about 30mmHg above the pressure needed to deliver the infusion.

What should you do if the alarm sounds?

1. Determine the cause of the occlusion, checking the venflon site.
2. Check that the line is not clamped or kinked.
3. Release the syringe plunger clamp in order to avoid a post-occlusion bolus.

Does the occlusion alarm prevent infiltration/extravasation?

No, it is not sufficiently sensitive. Always check clinical signs (redness, swelling, and pain).
Appendix 4

RESUSCITATION: SPECIALISED INFORMATION

PREGNANCY

Introduction
Resuscitation in pregnancy is complicated by a number of important factors.

- There are at least two patients.
- The physiological changes of pregnancy alter the response to acute illness, and to treatment.
- There are a number of diseases unique to pregnancy which may result in collapse.
- In view of these factors although the standard approach to resuscitation is applied, there are specific modifications.

Most of the causes of cardiac arrest in pregnancy are identifiable at a time when cardiac arrest should be preventable: e.g. hypoxaemia, hypovolaemia, and the aim is to avoid cardiac arrest.

Physiology
Changes in anatomy and physiology affect the approach to management of the pregnant patient.

Airways
- Oedema.
- Anatomical changes.
- Nasal congestion.

Breathing
- At 10 weeks 40% increase in tidal volume and normal respiratory rate with minute ventilation rising from 7.5 to 10.5 litres.
- \( \text{PaCO}_2 \) falls to 4kPa, and at term \( \text{PaO}_2 \) rises from 11.3 to 12.3kPa.
- FRC falls by about 30%.

Circulation
- Total body water rises by about 7 litres.
- Blood volume increases from 65 ml/kg to 80-85 ml/kg.
- Hb falls from 140g/l - 120g/l.
- Cardiac output increases by 1.5 l/min at 12 weeks, and by 44% in the third trimester.
• HR increases by 17% and stroke volume by 27%.
• Blood pressure and peripheral resistance fall.
• The combination of ventilatory and circulatory changes means that the ability to mount a compensatory response to acute illness (sepsis, hypovolaemia, haemorrhage) is diminished as the system is already working way beyond normal capacity.

GI
• Lower oesophageal sphincter tone falls.
• Intra-abdominal pressure rises.
• Gastric emptying may be delayed.
• Risk of regurgitation of stomach contents greater than normal, and increases the risks of pulmonary aspiration in situations where conscious level is depressed (including general anaesthesia).

Resuscitation peri-arrest and during arrest
• Call for help early: anaesthetist, obstetrician and paediatrician as appropriate.
• ABCDE
• High concentration oxygen: is the airway secure? Get anaesthetic help early.
• Large bore IV access and fluids: 20ml/kg colloid. Activate Major Haemorrhage protocol as appropriate.
• Left lateral tilt: as required.
• Refinements to BLS and ALS: see ALS manual in A&E/ARAU.

Specific problems: Haemorrhage; embolism; anaesthetics, eclampsia.
If alone after 1 minute call resuscitation team then continue CPR. If rescuers activate 222 call immediately stating paediatric cardiac arrest.

Note: Compress the chest by approx. one-third of its depth. Use 2 fingers for an infant under 1 year; use 1 or 2 hands for a child over 1 year as needed to achieve an adequate depth of compression.
PAEDIATRIC FBAO TREATMENT

Assess severity

Ineffective cough

- Unconscious
  - Open airway
  - 5 breaths
  - Start CPR

- Conscious
  - 5 back blows
  - 5 thrusts
  - (chest for infant)
  - (abdominal for child >1 year)

Effective cough

Encourage cough

Continue to check for deterioration to ineffective cough or relief of obstruction
FOREIGN BODY OBSTRUCTION SEQUENCE

If not breathing - Attempt 5 Breaths

Open Airway
Reassess for Breathing

5 Back Blows

Check Mouth
5 Abdominal Thrusts

Infant (<1 year)

If not breathing - Attempt 5 Breaths

5 Back Blows

Child (1-8 years)

Open Airway
Reassess for Breathing

5 Back Blows

5 Chest Thrusts

5 Back Blows

Check Mouth

Resuscitation Guidelines 2000-Resuscitation Council (UK), Marie Elen January 2001
CONSENT TO MEDICAL TREATMENT FOR CHILDREN IN SCOTLAND

This information is concerned with young people under the age of 16. Once a child reaches 16, he or she has full adult legal rights to decide whether to consent to treatment or not. Child Health in Scotland operates within the framework of Scots law, which differs from the law in England and Wales.

MEDICAL CONSENT IN GENERAL

Medical treatment is lawful, either:

- With consent
- Or in cases of urgent necessity (when consent cannot be immediately obtained).

It is important to remember that consent to medical treatment, whatever the age and capacity of the patient, is a matter that qualified medical practitioners must always make a decision about. In some cases where emergency treatment is required, the practitioner may decide that the situation is so urgent that the treatment cannot wait for consent. It is the practitioner who is making a judgement.

CONSENT FOR YOUNG PEOPLE OF 16 AND OVER

Scots Law treats the 16 year old as a full adult. He or she has the right to consent or refuse to consent to all medical, dental or surgical treatments or procedures.

CONSENT FOR YOUNG PEOPLE UNDER 16

(4) A person under the age of 16 years shall have legal capacity to consent on his own to any surgical, medical or dental procedure or treatment where, in the opinion of a qualified medical practitioner attending him, he is capable of understanding the nature and possible consequences of the procedure or treatment.

This means that for any child under 16 there is a right to consent to any form of treatment if the medical or dental practitioner considers that the child has capacity to understand:

- what the treatment or procedure is
- and its possible consequences.
For example it may be considered appropriate for a child to consent to a straightforward procedure such as setting a broken limb but not when considering treatments for more complex illnesses. If a doctor, dentist or other medical practitioner takes the view that the child has the capacity to consent, then only the child can consent or refuse consent. Although it would always be considered good practice for parents and if appropriate other family members to be included in the decision process.

It is important that all professionals who work with children are aware of the rights of the child rather than thinking that adults are the only people who have rights and whose views matter.

**Who consents if not the child or young person?**

If the medical practitioner does not think that the child is legally capable of consenting, the adults who could give consent would be as follows:

- Birth parents who have not lost their rights and responsibilities through adoption. A court could overrule the parents rights to consent or not to medical care, but this is very rare.
- Unmarried fathers do not automatically have parental responsibilities but carers rights.
- People who normally “care” for the child may also consent to medical treatment in certain circumstances. This is if the carer is over 16 years of age and the child is not capable of consenting and the carer has no knowledge that a parent of the child would refuse consent.
- School teachers and those under the age of 16 cannot give consent. It is also usual that in the event of the child being in care the local authority will have asked parents to sign a consent form, consenting to any required treatment.
- If the child is awaiting adoption then the local authority has all parental responsibilities for the child and the birth parents have no rights.

**CONFIDENTIALITY**

This is always a difficult area. The general opinion is that if a child is considered able to consent or refuse treatment, the child must also be entitled to patient confidentiality. It would always be considered good practice that the child and parents were included in any discussion. This would have to be done only if the child gave consent to it.
CONTRACEPTION AND ABORTION

It would be a breach of confidentiality if a doctor told a parent that their child had sought advice on contraception without the prior consent of the child.

The only exception is where the child is at risk and information may be disclosed in order to protect the child. When the girl is under 16, it could be argued that in seeking contraception she is “at risk” of being a victim of a sexual offence, but it is not always appropriate to inform the police or social services.

Similarly with abortion, consent to abortion or refusal of such consent is a matter for the young person, unless the girl has severe learning difficulties. Considerable support and counselling would be considered good practice and reasonable attempts to involve the family only if the child agrees. It is ultimately up to the girl to decide for herself and not up to her parents.

Bibliography

MALIGNANT HYPERTERMIA ACTION SHEETS - LUHD
RIE/WGH 2005-2007

TO BE KEPT IN THEATRE PROTOCOL FOLDER & MH BOX

DIAGNOSIS

Clinical
- Tachycardia + Tachypnoea / Raised EtCO₂
- Masseter Muscle Spasm after Suxamethonium.
- Rigidity / Fasciculations
- Arrhythmias
- Cyanosis / Low SpO₂
- Skin Mottling
- Temperature rise (Approx 1° / 5mins)
- Soda Lime hot & Rapidly Consumed
- Sweating +++
- Blood pressure unstable

Monitoring/laboratory
- SpO₂ decrease/Central Venous Hypoxia
- Hypercarbia
- Metabolic Acidosis
- Hyperkalaemia
- Myoglobinaemia
- Creatine Phosphokinase increase
- Clotting Screen Abnormality

ACTION

1. **DISCONTINUE ANAESTHETIC IMMEDIATELY WHEN POSSIBLE.**
   - Withdraw trigger agents immediately ie. All volatile agents.
   - Use new breathing system

2. **INTUBATE PATIENT & HYPERVENTILATE WITH 100% O₂ AT 3 x Vmin**
   (Aim for EtCO₂ of 3.5-4KPₐ)

3. **DISTRIBUTE “ACTION SHEETS”:**
   a) Treatment/Monitoring - Yourself + ODP - Page 1-3
   b) Malignant Hyperthermia Box - Anaesthetic Room Nurse - Page 4
4. **ASK THE SURGEON TO:**
   A. Abandon the operation rapidly.
   B. Insert urinary catheter.

5. **INJECT DANTROLENE.**

6. **COMMENCE BODY SURFACE COOLING WITH COOL WATER.**

7. **INFORM CONSULTANT IN CHARGE.**

---

**TREATMENT - OVERVIEW**

**THE ORDER WILL DEPEND ON AVAILABILITY OF DRUGS AND EQUIPMENT SPEED IS MORE IMPORTANT THAN ORDER.**

**HYPERVENTILATION**
- 100% Oxygen (Use new breathing system, NO VOLATILE AGENTS)
- Intubate
- \(3 \times V_{\text{min}}\) approx.
- Aim for \(ET_{CO_2}\) of 3.5 - 4 kPa.

**IV CANNULA**
- Large bore

**DANTROLENE**
- 1-2mg kg\(^{-1}\) IV rapidly.
  (i.e. 4 - 6 x 20mg vials for average adult)
- Vial Preparation:
  - Add 60 mls sterile water for injection to each vial
  - Further titrated Dantrolene up to 10 mg kg\(^{-1}\) may be required.

**COOLING**
- 2. Surface cooling with cool water sponging

*(Ice cooling is no longer recommended as it can cause intense vasoconstriction which retains body heat and can raise the core body temperature even more.)*
3. IV cooled fluids : (4 x 1000ml N/saline minimum stored in fridge)
RIE : Cardiothoracic theatre (Th 4-8),
Or Orthopaedic Theatre Clean Utility Area fridge.
WGH : Blood fridges in main theatre
(outside Theatre 3), DCN theatre &
Theatre 14.

Consider using a cardiac bypass pump heat exchanger in cooling mode.

**SUPPORTIVE TREATMENT TO COMBAT INCREASED METABOLIC RATE DUE TO SUSTAINED MUSCLE CONTRACTION:**

**Na BICARBONATE** : 1-2 mmols/Kg (100-200ml 8.4%) 
Reduces acidosis & serum K⁺
Repeat cautiously according to blood gas results.

**DIURESIS** : MANNITOL 20% (at room temp) 2ml/Kg/hr up to 500mls.
FRUSEMIDE 40 mg
Volume replacement as necessary (cold fluids)

**K⁺ REDUCTION** : Dextrose & Insulin infusion - 50ml 50% dextrose + 10 units Actrapid 
(Monitor Blood Glucose levels)

**ARRHYTHMIAS** : Usually secondary to acidosis & hyperkalaemia. Treat as appropriate.
β-Blockers frequently required. (Avoid the use of calcium channel blockers with dantrolene as hyperkalaemia can occur)

(DIC is common),
Creatinine Kinase (repeat at 24 hrs)
INSTRUCTIONS FOR ANAESTHETIST + ODP

1. STOP ADMINISTRATION OF VOLATILE AGENTS.

2. USE NEW BREATHING SYSTEM
If a circle system must be used the CO₂ absorbent must be replaced with new fresh granules to avoid absorbed volatile agents being released back into the system.

3. ARRANGE FOR MALIGNANT HYPERTHERMIA BOX TO BE COLLECTED FROM :

   **RIE:** GENERAL / ORTHOPAEDIC THEATRE RECOVERY
   Clean Utility Area – lower shelf.
   or ICU (Wd 118)

   **WGH:** ICU – Drug cupboard No 5
   WGH ICU also keeps another 12 vials of Dantrolene and 8x100ml vials of sterile water for reconstitution as a back up.
   Take them with you to save a second journey.

   **SJH:** In the Theatre Suite – Recovery Room on dedicated trolley.

4. ARRANGE FOR ON-CALL PHARMACST TO SEND A FURTHER 12 VIALS OF DANTROLENE & 8X 100ML VIALS STERILE WATER URGENTLY.

   Dosage: 1-2mg kg⁻¹ IV rapidly.
   (i.e. 4 - 6 x 20mg vials for average adult)
   Vial Preparation :
   Add 60 mls sterile water for injection to each vial
   Further titrated Dantrolene up to 10 mg kg⁻¹ may be required.

5. INTUBATE patient & hyperventilate with 100% O₂ at 3 x Vmin
   (Aim for ETco₂ of 3.5 - 4kPa)

6. SET UP:-
   - ECG
   - SpO₂
   - Et CO₂
   - Large bore IV cannula
   - Thermometer (Naso-pharyngeal/oesophageal/rectal)
   - Arterial line
   - Urine output - Surgeon will insert catheter (with urimeter)
   - Central Venous Pressure line
7. BLOOD SAMPLES:-

- Blood gases
- Potassium
- Clotting screen
- 10ml lithium heparin - for later analysis of creatine phosphokinase and myoglobin (At start / height / end of crisis)
- 20ml urine - for myoglobin (At start / height / end of crisis)

**ANAESTHETIC ROOM NURSE**
*(OR OTHER FAST RUNNER)*

**GET MALIGNANT HYPERTHERMIA BOX**

**LOCATIONS –**

**RIE** : GENERAL/Orthopaedic Theatre Recovery - Clean Utility Area – lower shelf or ICU (Ward 118) Clean Utility Area – Work surface corner

**WGH** : ICU - Drug cupboard No 5. WGH ICU also keeps another 12 vials of Dantrolene and 8x100ml vials of sterile water for reconstitution as a back up. Take them with you to save a second journey.

**PAEP** : Central Drug Store opposite Theatre 1 Anaesthetic room.

**RHSC** : Main Theatre – The middle of the recovery room by the emergency trolley.

**ROODLANDS HOSPITAL** : Main Theatre – in the safe in the ‘back corridor’

**St. JOHN’S HOSPITAL LIVINGSTON** : In the Theatre Suite – Recovery Room on dedicated trolley.

**Contents**

- Dantrolene Sodium : 12 Vials x 20mg
- Water for injection : 8 bottles x 100ml
- 1 bottle opener : For rapid filling of syringes using a quill
- 4 drawing up quills
- 12 luer lock 60 ml syringes
- 12 white needles
- 2 sponges for surface cooling
- 1 set Malignant Hyperthermia Protocol sheets
DRUGS - CIRCULATING NURSE

1. **MAKE SURE THAT THE FOLLOWING ARE HANDY :-**
   - Emergency drug box
   - Syringes needles and giving sets
   - 0.9% Normal Saline (NOT RINGER LACTATE), 5% Dextrose, 8.4% Bicarbonate
   - Foley catheter and equipment for urinary catheterisation
   - Basin / bucket to hold cool water for use with the sponges in the MH kit

2. Remind the anaesthetist to ask the surgeon to insert a urinary catheter when he has finished sewing up.

3. Take charge of the MALIGNANT HYPERTHERMIA BOX when it arrives:
   - Make up Dantrolene 1-2 mg kg\(^{-1}\) (SPEED IS IMPORTANT) :-
   - Add 60ml sterile water for injection to each vial.
   - The box contains quills and a bottle opener to speed up filling the syringes.
   - Shake vials well to dissolve the dantrolene.
   - (Average initial adult dose is 4 - 6 ampoules rapidly IV.)

4. Check that you have in theatre:
   - Mannitol 20%
   - Dextrose 20% or 50%
   - Insulin
   - Furosemide

5. Frequently ensure that stocks of IV fluids are not running out.

6. Arrange for more DANTROLENE to be brought to theatre from pharmacy.
   - *Note: You will need another 800ml (8 x100ml bottles) of sterile water for injection per box of 12 Dantrolene vials.*

Dispensary open hours:

<table>
<thead>
<tr>
<th>Dispensary</th>
<th>Days</th>
<th>Hours</th>
<th>Ext No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>RIE</td>
<td>Monday to Friday</td>
<td>0830-1830</td>
<td>22911</td>
</tr>
<tr>
<td></td>
<td>Saturday</td>
<td>0830-1500</td>
<td>22911</td>
</tr>
<tr>
<td></td>
<td>Sunday</td>
<td>1000-1400</td>
<td>22911</td>
</tr>
<tr>
<td>WGH</td>
<td>Monday to Friday</td>
<td>0845-1700</td>
<td>31461</td>
</tr>
<tr>
<td></td>
<td>Saturday</td>
<td>0900-1230</td>
<td>31210</td>
</tr>
</tbody>
</table>

Outwith these times contact the on-call pharmacist via the switchboard.

If necessary more Dantrolene can be mobilised from other hospitals.
(See page 8.) - Ask Pharmacist to co-ordinate
**COOLING - ASSISTANT SURGEON / RESIDENT / ODP**

**SPEED IS VITAL**

Cool patient by sponging with cool water over as much of the body as possible. This cools the patient by an evaporative heat loss process. Two sponges are in the MH box.

(The anaesthetist may arrange cardiac bypass pump cooling if required.)

**Important Further Information:**

*The following are no longer recommended for the treatment malignant Hyperthermia:*

1. Ice cooling is no longer recommended as it can cause intense vasoconstriction which retains body heat and can raise the core body temperature even more. Frostbite with loss of extremities could also occur.

2. Gastric or peritoneal lavage.
HELP - NURSE / RESIDENT

The following people should be contacted:

### RIE:
1. Supervising Consultant / On Call Consultant
2. Contact ward 118 ICU team in RIE (ext 2118) to arrange emergency transfer and admission.
3. The Assistant Operations Manager to report that the Malignant Hyperthermia policy plan has been actioned (RIE Bleep 2118)

### WGH:
1. WGH Consultant on call. Emergency Anaesthetist (Bleep 8155)
2. ICU Consultant on call. ICU team to arrange emergency transfer and admission (ext 31664/31665)
3. SHO on call (Bleep No 8112)
4. Theatre Assistant Operations Manager to report that the Malignant Hyperthermia policy plan has been actioned (Bleep # 6122)

### PAEP:
1. Contact ward 118 ICU team in RIE (ext 2118) to arrange emergency transfer and admission.
2. Inform Clinical Lead.
3. Theatre Assistant Operations Manager to report that the Malignant Hyperthermia policy plan has been actioned (Bleep # 1600)

### ROODLANDS HOSPITAL:
1. Contact ward 118 ICU team in RIE (ext 2118) to arrange emergency transfer and admission.
2. Inform Clinical Lead.
3. Theatre Assistant Operations Manager to report that the Malignant Hyperthermia policy plan has been actioned.
LOCATIONS OF SUPPLIES OF DANTROLENE IN THE LOTHIAN AREA

Large back-up supplies are available from the pharmacies in RIE, WGH & St Johns - Pharmacy will mobilise these sources if necessary

ROYAL HOSPITAL FOR SICK CHILDREN
12 vials of 20mg In Main Theatre - Middle of recovery room by emergency trolley.

WESTERN GENERAL HOSPITAL
12 vials of 20mg In ICU - in MH Box in ‘Drug Cupboard No 5’.
12 vials of 20mg In ICU - in ‘Drug Cupboard No 5’ as back-up.
12 vials of 20mg WGH Pharmacy

ROODLANDS HOSPITAL
12 vials of 20mg In Main Theatre - in safe in ‘back corridor’.

ROYAL INFIRMARY OF EDINBURGH
96 vials of 20mg In Pharmacy Dept - Drug store, injection store.
12 vials of 20mg ICU Ward - At Nurses Station, top shelf.
12 vials of 20mg General/Orthopaedic Theatre Recovery Room - Clean Utility Area - lower shelf.

St. JOHN’S HOSPITAL LIVINGSTON
12 vials of 20mg In Theatre Suite - Recovery Room on dedicated trolley.
RECOVERY & FURTHER TREATMENT

With rapid diagnosis and treatment most cases of Malignant Hyperthermia will recover. After the initial crisis has been stabilised the patient should be admitted to an intensive care unit noting the following:

RETRIGGERING - May occur.

*Oral Dantrolene (if possible) should be given for 48 hours:*

4mg/kg/day in divided doses.

Unnecessary stress should be avoided as this can trigger Malignant Hyperthermia.

HYPOTHERMIA - Can be induced by overvigorous cooling during recovery.

DIURESIS - Should be maintained to reduce the possibility of myoglobin induced renal failure.

BLEEDING DISORDERS - A DIC type coagulopathy is common.

PULMONARY OEDEMA - Is common.

MUSCLE OEDEMA - Compartment Syndrome may require fasciotomy.

(Spinal anaesthesia is often the method of choice for this)

Consider other diagnoses - Myopathy / Ecstasy ingestion / Neuroleptic Malignant Syndrome

Patients suspected of having Malignant Hyperthermia and their blood relatives should be referred later for formal investigation to:-

**Malignant Hyperthermia Services**
**University Dept. of Anaesthesia**
**St. James’s University Hospital**
**Leeds LS9 7TF**

**LEEDS MALIGNANT HYPERTHERMIA HOTLINE:** 07947 609 601
MAJOR HAEMORRHAGE PROTOCOL

- **WGH/RIE**: Attending clinicians should telephone switchboard on the emergency number (222), informing them that there is major haemorrhage, the name and location of the patient and a contact telephone number (and individual where possible).

  **SJH**: Telephone/bleep blood bank (as below) and porters (ext 2120) not 222.

  **Switchboard will inform:**
  - Blood Bank /Haematology Laboratory (by bleep).
  - Haematology/BTS duty doctor (by bleep).
  - Porter to go to the clinical area (porter will remain until stood down by clinical team).

- **Blood Issue (WGH Ext. 31912, SJH Ext. 53354/bleep 729, NRIE Ext. 27501/27502) should be rung directly to clarify the following:**
  - How urgent the need for blood is.
  - Patient’s minimum data set (full name, date of birth, hospital number if available, A&E number or Major Incident number if necessary).
  - The number and nature of blood components requested (a standard pack for an adult will consist of 10 units of red cell concentrate, 1 pool of platelets and 3 units of FFP. For children the normal dose is 10-15 ml/kg for these components).
  - The exact location of the patient.

- If required, emergency O negative stock is held in the RIE and WGH blood banks, and also in the fridges in the Acute Receiving Unit at WGH, A & E at RIE, Simpsons Centre for Reproductive Health Labour Ward, RHSC, and Roodlands. If required please use the nearest available stock.

- In order to speed up the coagulation screen, the fibrinogen will be done first and phoned to the clinical team and Haematology/BTS duty doctor. If this is less than 0.8g/l the PTR and APTT will be prolonged and fresh frozen plasma and cryoprecipitate are likely to be required.

- When the full blood count and coagulation screen are available they will be phoned to both the clinical team and to the Haematology/ BTS duty doctor. The Haematology/BTS duty doctor will liaise with the attending clinicians with regard to the haematological results and further blood component requirements.

For further FBC/coagulation or blood components, the clinical team should liaise direct with the appropriate laboratories on the emergency numbers. There is no requirement to go through the Haematology/BTS duty doctor, though he/she will be available as required.
MAJOR HAEMORRHAGE PROTOCOL

• Make two phone calls

TELL SWITCHBOARD: [Ext 222]
• There is a Major Haemorrhage
• Name and location of patient
• Contact name and telephone number

Switchboard to inform:
• Blood Issue
  (RIE 6535, WGH 31912, NRIE 27501 / 27502)
• Haematology Laboratory
• Haematology/BTS Duty Doctor:
  Porter/courier to clinical area

TELL THE BLOOD BANK:
(RIE Extension: 65353)
(WGH Extension: 31912)
(NRIE Extension: 27501/27502)
1. How urgent is the need for blood?
2. Patient’s Minimum Data Set:
   Name:
   Hospital no./CHI no./A&E no./Major
   Incident No.
   Sex and Date of Birth.
   ABO & Rh Group (if known).
3. The number and nature of the blood components requested.
4. Where the blood is to be sent.
5. Name and contact telephone number for Clinician.

Send FBC and Coagulation Screen to Haematology and Pretransfusion testing sample to Hospital Blood Bank
Remember to bleep Haematology technician (RIE#6550 WGH 5477) for follow-up FBC and coagulation screens


NOTE: O negative stock is often in critical supply. A sample should be sent to the Blood Bank ASAP to allow conversion to Group-specific blood.


NOTE: An antibody screen and crossmatch will be carried out on the released units within 30 minutes.

Send patient sample and request form urgently to Blood Bank.

ABO and Rhesus group specific red cells available for collection at Blood Bank. Blood available 15 minutes after sample received.

Send patient sample and request form urgently to Blood Bank.

ABO and Rhesus group, antibody screen and crossmatch will be carried out. Blood available 45 minutes after sample received.

NOTE: If the patient has a historic record and a Group and Screen on a current clinical sample, blood can be made available immediately by electronic release.

Are platelets, or FFP needed?

Allow time for preparation:
Platelets - Immediate Release if blood group available
FFP - 20 minutes

NOTE: Clinicians need to allow for the time it takes to deliver blood components from the blood bank to the clinical area.

Haematology/BTS Duty Doctor will liaise with attending clinicians re: haematology and coagulation results, further blood components and blood stock management.

NOTE: Cryoprecipitate can be ordered direct from blood bank if fibrinogen is less than 0.8 g/l.
CONTRIBUTORS TO PREVIOUS EDITIONS

LIST OF CONTRIBUTORS TO 1st WGH EDITION 1998
Editors: Dr G Nimmo, Dr D Northridge, Prof D Webb, Dr I Wilkinson.
Dr N Bateman, Dr R Benedikttsson, Dr GK Crompton, Dr M Dennis,
Dr M Denvir, Dr R Ellis, Dr M Ford, Dr S Ghosh, Sister F Good,
Dr IS Grant, Dr A Greening, Dr G Howard, Dr N Hurst, Dr JA Innes,
Mr M Johnstone, Dr C Leen, Dr R Lindley, Dr M Mackie,
Dr SJ Maxwell, Dr J McKnight, Dr C Mumford, Ms L Murray,
Dr GR Nimmo, Dr D Northridge, Prof G Nuki, Mr M O’Sullivan,
Ms R Oughton, Dr P Padfield, Dr K Palmer, Dr I Penman,
Prof L Prescott, Dr PWH Rae, Ms Jenny Scott, Dr K Slatford,
Dr A Webster, Prof DJ Webb, Dr I Wilkinson, Dr D Wilks.

LIST OF CONTRIBUTORS TO 1st LUHNT EDITION
Dr T Beattie, Dr D Bell, Prof J Bell, Dr B Chapman, Prof M Dennis,
Dr M Denvir, Dr M Ford, Dr P Gibson, Dr I Grant, Dr A Greening,
Dr G Howard, Mr M Johnstone, Dr C Kelly, Dr S MacKenzie,
Dr M Mackie, Dr C Maguire, Dr SJ Maxwell, Dr R Mitchell, Dr G Nimmo,
Dr S Nimmo, Dr A Patrick, Dr P Padfield, Dr K Palmer, Dr D Wilks,
Dr D Wright and all contributors to CCU Therapeutic Schedule.

SPECIALIST PHARMACISTS WHO PROOF READ CHAPTERS
Morag Naysmith, Anne Balfour, Julie Blythe, Alistair Brand, Carole Callaghan,
Jenny Carson, Heather Dalrymple, Katherine Harrington, Anne Kinnear,
Lesley Pacitti, Heather Paterson, Carol Philip, Karen Reid, Maureen Reid,
Jenny Scott, Sheila Selkirk, Laura Shaw, Lorna Thomson, Helen Veitch,
Susan Wilson, Sherry Wright.

LIST OF CONTRIBUTORS TO 2004/2006 EDITION
EDITORIAL COMMITTEE
B Chapman, C Kelly, S Maxwell, R Mitchell, G Nimmo, R Paterson,
J Pearson, M Watson.

CONTRIBUTORS TO THIS EDITION OF THE HANDBOOK
Dr J Amoore, Dr S Balata, Dr T Beattie, Dr D Bell, Prof J Bell,
Dr B Chapman, Dr N Colledge, Dr R Davenport, Prof M Dennis,
Dr M Denvir, K Farrer, Dr M Ford, Dr P Gibson, Dr I Grant, Dr A Gray,
Prof A Greening, Prof P Hayes, Dr G Howard, Mr M Johnstone,
Dr S Keir, Dr C Kelly, Mike Logan, Dr V Macaulay, Dr S MacKenzie,
Dr M Mackie, Dr C Maguire, Dr L Manson, Dr S Maxwell,
Dr S Midgley, Dr R Mitchell, Dr G Nimmo, Dr S Nimmo, Dr A Patrick,
Dr P Padfield, Dr K Palmer, Mr Z Raza, Dr M Strachan, Mrs L Waite,
Dr D Wilks, Dr R Winney, Dr D Wright

PHARMACISTS WHO PROOF READ SPECIALITY SECTIONS
J Blythe, H Dalrymple, L Goundry, C Hannah, A Kinnear, M Naysmith,
H Paterson, J Pearson, S Petrie, K Reid, L Shaw, C Stein
Useful links

The Anaphylaxis campaign:  
www.allergyinfo@anaphylaxis.org.uk

Meningitis Research Foundation:  
www.meningitis.org

British Thoracic Society :  
www.brit.thoracic.org.uk

Resuscitation Council (UK):  
www.resus.org.uk

The British Toxicology Society:  
www.thebts.org

National Poison Information Service:  
www.npis.org/npis/uk%20npis.htm

British Society Gastroenterology  
www.bsg.org.uk

Scottish Intensive Care Society (SICS):  
www.scottishintensivecare.org.uk

In the education section of SICS website you will find this handbook as a pdf and also sections on critical decision making, EDM in Intensive Care and teaching materials for Identifying Sepsis Early.