MANAGEMENT GUIDELINES FOR COMMON MEDICAL CONDITIONS

FIFTH EDITION 1994

Internal Medicine Services

Canterbury HEALTH
Manaaki tahu • Caring for everyone

CONTENTS

INTRODUCTION - FIFTH EDITION 1994

A formal continuing medical education (CME) programme for physicians began in Christchurch in 1979. Medical audit, carried out as part of this CME programme, revealed a need for treatment guidelines to improve standards of medical care. Each sub-specialty of Internal Medicine Services produced recommendations which have been edited to achieve a reasonably uniform format. In the fifth edition there have been significant changes to a number of sections. The poisons and drug overdose section has been rewritten and a new section added on a problem orientated approach to emergency presentations.

These guidelines should not be followed in a rigid manner. The treatment given to the patient must always be considered in the light of that patient's individual problems and needs. Although these recommendations may often need modification in practice, they should provide a useful guide to the provision of good medical care. They do not apply to children.

Remember that the delivery of medical care is a group activity. Always listen to advice from the patient and relatives, from other members of the staff and from the General Practitioner. Try to get as much accurate information about the patient as possible. Get all available past medical notes, and if necessary telephone the General Practitioner. In some situations, for example a suspected fit, an interview with a witness may prove to be crucial. Above all remember that patients are people and that coming into hospital is probably the worst thing that has ever happened to them. Relatives may be fearful that they are about to lose a loved one. The correct treatment is devalued if it is given in an uncaring or inconsiderate manner and the reasons for giving it are not explained to the patient.

Finally remember the dollar costs of your actions. It should be possible to save money by avoiding expensive treatments and investigations when adequate, cheaper alternatives are available.

We are pleased to acknowledge the enthusiastic help we have received from many consultants, registrars and other hospital staff not only in Medicine but from other disciplines. We are grateful to Mrs Mary Mann for her skills in producing this book. We would like to acknowledge the financial support of Canterbury Health Ltd.

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FOR
Internal Medicine Services
CANTERBURY HEALTH LTD

(Copies can be obtained from the Department of General Medicine, Christchurch Hospital, Christchurch)

ABBREVIATIONS

GENERAL

CXR	Chest X-ray
CBC + Diff	Hb, PCV, MCV, WBC and differential, platelets
DKA	Diabetic Ketoacidosis
CPR	Cardiopulmonary Resuscitation
MSU	Mid-stream Urine
IVU	Intravenous Urogram
MCV	Mean Cell Volume
ESR	Sedimentation Rate
NSAID	Non-Steroidal Anti-inflammatory Drug
LP	Lumbar Puncture
DIC	Disseminated Intravascular Coagulation
Na	Sodium
K	Potassium
PO_4	Phosphate
Ca	Calcium
AST	Aspartate Aminotransferase
GGT	Gamma Glutamyltransferase
Alk. Phos.	Alkaline Phosphatase
Bili	Bilirubin
Mg	Magnesium
Alb	Albumin
Cl	Chloride
KCl	Potassium Chloride

FLUID PRESCRIPTIONS

0.9S	Normal Saline
0.45S	Half Normal Saline
D5W	5% Dextrose
D4S	4% Dextrose and Fifth Normal Saline
SPPS	Stable Plasma Protein Solution

DRUG DOSAGE

IV	Intravenous
IM	Intramuscular
SC	Sub-cutaneous
PO	Oral
PR	Rectal
BD/TID/QID	Twice, three times or four times during the normal day, i.e. implies not during the night
q24h or q6h	Every 24 hours or every 6 hours respectively. This means that the drug is given exactly at those times

SYMBOLS AND UNITS

ml	Millimetre
L	Litre
mcg	Microgram
mg	Milligram
g	Gram
kg	Kilogram
mmol	Millimole
mcmol	Micromole
IU	International Unit
U	Units

USE OF ANCILLARY SERVICES

Try to organise requests for such services early in the day. Try to minimise the number of tests done out of normal working hours or at weekends. Remember to be courteous when requesting emergency tests from ancillary staff.

CANTERBURY HEALTH LABORATORIES

Most departments provide a routine diagnostic service from 8 a.m. to 5 p.m. Monday to Friday. These same departments maintain a skeleton staff for urgent specimens between 5 p.m. and midnight (Monday to Friday) and from 8 a.m. to midnight on Saturday, Sunday and public holidays. Between midnight and 8 a.m., two staff members provide an emergency service for all departments.

Between 5 p.m. and midnight communication can be made by contacting the Department concerned. After midnight all communications should be through the Christchurch Hospital telephone office.

RADIOLOGY

A full specialized radiology service is available at Christchurch Hospital. Interventional procedures, arteriography, CT and MRI are not available at the other institutions. There are, however, specialized ultrasound services available for obstetric and gynaecological cases at Christchurch Women's Hospital, and full ultrasound facilities are also available at the Princess Margaret Hospital.

We endeavour to perform all x-ray examinations within 24 hours of them being ordered for hospital inpatients. Reports on those procedures are usually completed the same day and despatched from the department. In an endeavour to avoid losing x-rays, they are kept in the consultants' viewing room within the department. The movement of x-rays elsewhere is discouraged.

- **Consultation forms**
  It is essential that all consultation forms be filled in adequately. This includes the patient's full name, patient number, and accurate date of birth. It is useful to know whether there have been previous examinations, and please supply relevant clinical data so that the most appropriate radiology examination is performed.

- **Normal working hours**

Christchurch Hospital	0800-1700 with a 24 hour presence of medical radiation technologists (radiographers) in the department for acute work. Registrars are usually present in the early evening and there are registrars and consultant radiologists on call at all times.

Princess Margaret Hospital	0830-1600 Monday to Friday. Medical radiation technologists and radiologists are available on call.
Christchurch Women's Hospital	0800-1600 Monday to Friday with medical radiation technologists, registrars and radiologists available for acute work.
Burwood Hospital	0800-1700 Monday to Friday.

- **X-ray consultations for special examinations**

Where possible, it is advisable to discuss special examinations with a registrar or consultant so that the most appropriate examination is done. These examinations include ultrasound, CT, MRI, arteriography, and interventional studies. After hours, urgent special examinations must be approved by the clinical consultant and discussed with either the radiology registrar on call or the consultant radiologist.

- **Radiology after hours**

Remember that apart from simple examinations, special procedures are very expensive. If not critical to the patient's management such procedures can be delayed until normal working hours so that unnecessary expenditure is avoided.

NUCLEAR MEDICINE

Refer to "Nuclear Medicine Procedures" for explanations and information for Nursing and Medical staff, and indications and interpretations of scans. This booklet should be available in every ward area. For all routine scans the appropriate request form should be sent to the Department of Nuclear Medicine. Relevant x-rays should be available and all lung scan patients must have had a chest x-ray within 24 hours. If the scan is considered urgent the Department (Scanning Room) should be contacted by telephone and every effort will be made to carry out the study that same day. It should be noted that some procedures such as bone scans require several hours between radio-pharmaceutical injection and scanning and so the Department must be contacted at the earliest available opportunity. A limited service is usually available for urgent scans during the weekends and public holidays. Contact Dr J. Turner or Dr B. Brownlie before 10.00 am.

COMMON EMERGENCY PRESENTATIONS

INTRODUCTION

This section is intended to supplement the systems based sections which follow with a brief account of an approach to the unwell patient which is:

PRIORITIZED AND PROBLEM ORIENTATED

Attention is first directed to the **airway, breathing and circulation**, before consideration of the specific manifestations and management of the underlying disease process.

The airway, breathing and circulation will be discussed first and then some undifferentiated emergency presentations are described.

THE ABCs

A AIRWAY IMPAIRMENT

- **Recognition:**
  - Altered level of consciousness (common association).
  - Noisy breathing.
  - Laboured breathing (especially a "see-saw" pattern of opposite chest and abdominal movement).
  - Not breathing.
- **Management options** (in order of invasiveness):
  - Supplemental oxygen.
  - Positioning:
    - Recovery position.
    - Chin lift.
    - Jaw thrust.
  - Suction.
  - Oropharyngeal (Guedel) airway.
  - Orotracheal intubation.
  - Surgical airways:
    - Needle cricothyroidotomy.
    - Surgical cricothyroidotomy.
- **Causes:**
  - Altered level of consciousness (most common cause).
  - Mass (infective, neoplastic, inflammatory).
  - Foreign body.
  - Palsy (bulbar, pseudobulbar, vocal cord).

B BREATHING IMPAIRMENT

- Recognition:
  - Altered level of consciousness (cause and effect).
  - Hypoxia:
    - Pulse oximetry/arterial blood gases.
    - Cyanosis (important only if present).
  - Hypercapnia - arterial blood gases.
  - Tachypnoea or bradypnoea.
  - Laboured breathing.
- Management options:
  - Supplemental oxygen (high flow, reservoir bag, minimal air entrainment - will provide an FIO_2 approaching 80%).
  - Assisted ventilation:
    - Mouth to mouth.
    - Mouth to mask.
    - Bag to mask.
    - Bag to endotracheal tube.
- Causes:
  - Central respiratory depression.
  - Airways disease.
  - Lung disease.
  - Chest wall disease.

C CIRCULATORY IMPAIRMENT

- Recognition:
  - Impaired brain perfusion (anxiety, confusion, lowered level of consciousness).
  - Impaired skin perfusion (coolness, pallor).
  - Impaired renal perfusion (decreased urine output).
  - Tachycardia, low pulse volume.
  - Hypotension (late).
- Management options:
  - Supplemental oxygen.
  - Intravenous fluids.
  - Pressor agents.
  - Other specific treatment.
- Causes:
  - Hypovolaemia.
  - Cardiogenic (arrhythmias, myocardial damage).
  - Vasodilation (sepsis, drugs, anaphylaxis).
  - Obstruction (tension pneumothorax, massive pulmonary embolism, cardiac tamponade).

SOME EMERGENCY PRESENTATIONS

Each of these presentations is discussed as it would be handled in a prioritized manner, with a concurrent problem orientated diagnostic process leading eventually to a specific diagnosis and definitive treatment.

The approach is thus:

- Initial assessment and resuscitation i.e. the ABCs and specific resuscitation measures.
- Complete assessment.
- Definitive management.

As this approach is followed a differential list develops. As more information comes to hand, the list will get smaller (although occasionally, it will be added to) and eventually a final diagnosis will guide definitive management.

The initial assessment and resuscitation take priority and should not await a final diagnosis, although they may be guided by the differential problem list.

ALTERED LEVEL OF CONSCIOUSNESS

Initial assessment and resuscitation

- Airway, (commonly impaired by altered level of consciousness), breathing, circulation
- Consider the "three coma antidotes".
  - **Glucose** - check capillary blood glucose, if hypoglycaemia confirmed give 50 ml of 50% dextrose solution (25 g of glucose) IV.
  - **Thiamine** 100 mg IM, if possibility of Wernickes encephalopathy.
  - **Naloxone** 0.4 mg IV and repeat 0.4 mg in 5 minutes. If no response and narcotic overdose suspected give Naloxone up to a maximum of 2.0 mg. (Anexate [flumazenil] is available for benzodiazepine reversal but is rarely indicated in the emergency setting).

The complete assessment

- History - patient, relative, ambulance, GP etc.
- Examination:
  - The Glasgow Coma Scale (see below).
  - Temperature.
  - Neurological and other system examination.
- Investigations:
  - Arterial blood gases, glucose, Na, K, creatinine, Bili, alk. phos., GGT, AST, CBC + diff.
  - Other tests according to suspected cause e.g. CT scan of head, urine or gastric toxicology, carboxyhaemoglobin etc.

Glasgow Coma Scale

EYE OPENING	Spontaneously	4
	To Voice	3
	To Pain	2
	None	1
VERBAL RESPONSE	Orientated	5
	Confused	4
	Inappropriate Words	3
	Inappropriate Sounds	2
	None	1
MOTOR RESPONSE	Obeys Commands	6
	Localizes Pain/Purposeful movement	5
	Withdraws From Pain	4
	Abnormal Flexion	3
	Abnormal Extension	2
	None	1
SCORE:	TOTAL POSSIBLE	15

Definitive management

- According to the cause. Possible causes are:
  - Anatomic:
    - ~ Damage to, pressure on, the reticular activating system.
    - ~ Occasionally bilateral cortical insults (bleeds, infarcts, masses, hydrocephalus).
  - Toxic / metabolic:
    - ~ Drugs.
    - ~ Hypoxia, hypoperfusion.
    - ~ Abnormalities of sodium, glucose, acid base.
    - ~ Encephalopathies.
    - ~ Hypothermia / hyperthermia.-
    - ~ Infections (meningitis, encephalitis).
    - ~ Endocrine causes.
  - Psychogenic.
  - Post ictal.
- For further details **(see page 122 Altered Level of Consciousness under Neurology).**

SYNCOPE

Definition - a transient loss of consciousness in a now conscious patient.

- Initial assessment and resuscitation:
  - Airway]
  - Breathing] including monitoring of these in case of recurrence
  - Circulation]
- The complete assessment:
  - History]
  - Examination] as described in "Altered level of consciousness"
  - Investigations]
- Definitive management according to the cause. Possible causes:
  - Seizure disorder.
  - Cardiac causes (arrhythmias, ischaemia, obstructive lesions).
  - Cerebrovascular causes (TIAs, subclavian steal, carotid sinus syncope).
  - Peripheral vascular causes (vasovagal, orthostatic, hypovolaemic, others).
  - Miscellaneous causes (hyperventilation, hypoglycaemia, psychogenic, others).

SHOCK

Simple definition - inadequate delivery and utilization of oxygen by vital organs.

- Initial assessment and resuscitation:
  - Airway]
  - Breathing] oxygen delivery must be optimized
  - Circulation - management of circulation will vary according to the cause of the shock but will generally require intravenous access (one or two large bore IV cannulae) and fluid infusion.
  - Assessing the degree of shock and the rate of intravenous infusion.
    - The management of hypovolaemic shock is described in detail below. Initial resuscitation of 'distributive' shock (e.g. sepsis) will also include fluid resuscitation. Resuscitation of cardiogenic shock (arrhythmias or myocardial insufficiency) and obstructive shock (tension pneumothorax, massive pulmonary embolism, cardiac tamponade) will rely on specific therapy, often after a trial of a fluid bolus.
    - Assessment of the degree of shock can be difficult as signs and symptoms will vary with the cause, the speed of onset, the patients pre-morbid state and the treatment so far. Generally speaking, if the patient displays signs of shock, then the shock has reached a severity beyond the patient's ability to compensate and demands aggressive treatment.

- For hypovolaemic, and other forms of shock, invasive monitoring of the circulation (CVP or Swan Ganz catheter) provides useful objective information but requires expertise in application and interpretation.
- CVP measurement is appropriate in some non-ICU patients but requires skill in insertion and an educated interpretation. An absolute CVP is less useful than changes in CVP in response to fluid challenges. A low CVP (with a patent well placed catheter) means low volume but a high CVP may mean adequate volume, pulmonary hypertension, COAD, left ventricular failure or increased pulmonary vascular resistance (as can occur in trauma or other unwell patients).
- Pulmonary capillary wedge pressure measurements provide the ultimate measure of 'volume status' but are only practicable in ICU, CCU or the Operating Theatre.
- The urine output is a useful objective measure of renal perfusion assuming no diuretics have been given.

The following description is of acute haemorrhagic shock, and provides a framework only.

Note:

The average 70 kg man has approximately 5 litres of blood.

CLASS I SHOCK	Blood loss up to 15% blood volume (750 ml) CNS] Skin] no discernable abnormality Urine] Pulse] BP]
CLASS II SHOCK	Blood loss of 15 to 30% blood volume (750-1500 ml) CNS agitated Skin cool, pale Urine decreased Pulse tachycardia (>100 per minute) BP normal
CLASS III SHOCK	Blood loss of 30-40% blood volume (1500-2000 ml) CNS agitated to confused Skin cool, pale Urine decreased Pulse tachycardia (>120 per minute) BP falling
CLASS IV SHOCK	Blood loss of over 40% of blood volume (>2000 ml) CNS confused (unconscious by 50%) Skin white and cold Urine Nil Pulse >140 per minute, peripheral pulses lost (all pulses lost by 50%) BP very low (absent by 50%)

Therefore, haemorrhagic shock with hypotension suggests 1500-2000 ml of blood loss and demands rapid infusion of 2000 ml of crystalloid. Crystalloid will 'splint' the circulation temporarily before travelling out of the vascular space and therefore more will be required (another 2000 ml) unless the response to the first 2000 ml was dramatic. For Grade III or Grade IV shock transfusion of blood will invariably be required. If the early crystalloid does not restore satisfactory circulation then this transfusion is urgent and may not wait for a crossmatch. Type O negative blood is available in the transfusion fridge in the Operating Theatres, and type specific blood may be available from the Blood Bank prior to full crossmatch.

Note:

- The elderly and those on drugs such as ß-blockers are less able to compensate and therefore will become hypotensive earlier. There is a greater blood volume in advanced pregnancy and the ability to shunt blood from the placental circulation (at the fetus' expense) means that shock manifests later in the mother (but earlier in the fetus).
- Management of haemorrhagic shock includes controlling the haemorrhage - i.e. pressure on external bleeding, surgery for internal bleeding - involve the surgeons early.

♦ The complete assessment:
- History]
- Examination] according to the likely cause
- Investigations]

♦ Definitive management according to the cause. Possible causes:
- Haemorrhagic shock as above.
- Other hypovolaemic shock:
  - ~ Plasma loss - burns, exfoliative dermatitis.
  - ~ Fluid loss or redistribution - vomiting, diarrhoea, sweating, hyperosmolar states (diabetic ketoacidosis, non ketotic hyperosmolar coma), third space losses (pancreatitis, ascites, bowel obstruction).

    Note:

    Dehydration precedes shock in extracellular fluid loss, shock is very advanced dehydration. Electrolyte disturbance is commonly associated and must also be addressed.
- Cardiogenic shock:
  - ~ Arrhythmias, myocardial dysfunction, acute valvular dysfunction, ventricular or septal rupture.
  - ~ Fluid therapy may occasionally be useful to increase filling pressure but more often specific therapy is most appropriate.
- Obstructive shock:
  - ~ Tension pneumothorax (obstructs venous return), pericardial tamponade or constriction, obstructive valvular disease (aortic or mitral), pulmonary hypertension, massive pulmonary emboli, cardiac tumours.

    Note:

    JVP/ CVP may be raised but does not represent fluid overload.
  - ~ Fluid therapy commonly used but specific treatment is required.
- Distributive shock:
  - ~ Septic shock, anaphylactic shock, neurogenic shock, vasodilator drugs.
  - ~ Skin is warm and pink.
  - ~ Relative hypovolaemia due to expanded vascular space.
  - ~ Fluid resuscitation and specific treatment are required.

ANAPHYLAXIS

Definition - a severe hypersensitivity reaction resulting in cardiovascular and/or respiratory compromise. Anaphylactoid reactions manifest identically but do so independent of specific IgE antibodies. Severity varies but generally three systems are involved, with occasionally a fourth.

SKIN	Urticaria
RESPIRATORY	Airway spasm / oedema
CARDIOVASCULAR	Shock
GASTROINTESTINAL	Cramps and diarrhoea

- Initial assessment and resuscitation:
  - Airway.
  - Breathing.
  - Circulation - large volumes of fluid may be required.
  - Adrenaline:
    - **0.5 ml of 1:1000 IM (= 0.5 mg).**
    - Repeat every 5 minutes as required.
    - IV adrenaline is indicated if the situation is life threatening. Dose **0.3** to **0.5 mg i.e. 3 to 5 ml of 1:10000** (dilute 1 ml of 1:1000 to 10 ml with 0.9S). Repeat every few minutes as required.
    - Nebulized salbutamol 5 mg for bronchospasm.
    - Nebulized adrenaline (2 ml of 1:1000 (2 mg) diluted to 4 ml with 0.9S) for laryngeal oedema (stridor).
- The complete assessment:
  - History]
  - Examination] Try to identify any allergens
- Definitive management:
  - Moderate/severe reaction:
    - Hydrocortisone 200 mg IV.
    - Promethazine 25 mg IV.
    - Admit for observation.
    - Discharge with provision and instruction in the use of parenteral adrenaline.
    - Prednisone 40 mg daily for 2 days.
    - Promethazine 25 mg BD for 2 days, warn about sedative effects, caution with regard to driving and working.
  - Mild / moderate reaction:
    - Prednisone 40 mg PO stat and then daily for 2 days.
    - Promethazine 25 mg PO stat and then BD for 2 days.
    - Discharge after resolution of symptoms and signs to observation by family / friends.
- Arrange follow up with Clinical Immunology for moderate/severe reactions.

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HEAD PAIN

- Initial assessment and resuscitation:
  - Airways]
  - Breathing] brief appraisal
  - Circulation]
- Complete assessment:
  - History]
  - Examination] directed according to the differential problem list
  - Investigations]
- Definitive management according to the cause.
  Possible causes include:
  - Trauma - scalp, skull, intracranial haematoma.
  - Chronic, recurrent, unchanged, e.g. tension headaches, migraines, sinusitis, cluster headaches, temporomandibular joint disease, cervical spine disease, hypertension, oral contraceptive or other drug induced headaches.
  - New headache:
    - ~ Meningeal irritation - subarachnoid haemorrhage, meningitis / meningoencephalitis.
    - ~ Hypertensive encephalopathy.
    - ~ Pre-eclampsia / eclampsia.
  - Paracranial causes - temporal arteritis, eyes, ears, sinuses, teeth, cervical spine.

Note:

Focal signs - chronic or new headaches need investigation. CT scan should precede lumbar puncture if focal signs or if significantly altered level of consciousness.

CHEST PAIN

- Initial assessment and resuscitation:
  - Airway]
  - Breathing] including oxygen, cardiac monitoring and
  - Circulation] intravenous access in all but trivial cases
- The complete assessment:
  - History]
  - Examination] directed according to the differential problem list
  - Investigations]

- Definitive management according to the cause or possible causes:
  - Traumatic - chest wall, lung, heart, great vessels, diaphragm, oesophagus, spine.
  - Non traumatic:
    - Chest wall - (pleuritic pain, tenderness).
    - Lung - (pleuritic pain, focal signs), pneumothorax, infective inflammatory, pulmonary embolism.
    - Heart - ischaemia, pericarditis.
    - Great vessels - dilatation, dissection.
    - Oesophagus - inflammation, spasm, rupture.
    - Abdominal - peptic ulceration, pancreatitis, cholecystitis etc.
    - Psychogenic.

Note:

Investigations usually include CXR and ECG but often require pulse oximetry/arterial blood gases, cardiac enzymes, ventilation/perfusion scan, abdominal ultrasound, aortography, CT scan, echocardiography.

ABDOMINAL PAIN

- Initial assessment and resuscitation:
  - Airway] if sepsis or hypovolaemia evident, oxygen delivery
  - Breathing] should be optimized and intravenous fluids given
  - Circulation]
- The complete assessment:
  - History:
    - Site - Abdominal (upper, lower, mid, left, right).
      - Pelvic.
      - Retroperitoneal (flank / back - consider the anatomy).
    - Nature - Severity: mild, moderate, severe.
      - 'Visceral' - dull, ill-defined.
      - 'Somatic' - sharp, localized.
      - 'Peritoneal' - constant, patient lies still.
      - 'Colicky' - intermittent, patient writhes around
      - Radiation: - to back; retroperitoneal.
        - to groin; ureteric.
        - to thigh; genitourinary, major vessel.
        - to shoulder; diaphragmatic irritation
    - Associated symptoms - vomiting, diarrhoea, genitourinary, possible pregnancy.
  - Examination:
    - General - Perfusion, hydration.
      - Colour: pallor, jaundice.
      - Peripheral manifestations of liver disease.
      - Peripheral manifestations of vascular disease.

- ~ Abdomen - Appearance: scars, masses, distension.
  - Palpation - tenderness - inflammation.
    - guarding - peritoneal inflammation
    - rigidity - generalized inflammation
    - masses
  - Auscultation: bowel sounds, bruit.

- Investigations:
  - ~ Urinanalysis, urine microscopy and culture.
  - ~ Urine pregnancy test.
  - ~ CBC + diff.
  - ~ Urea, creatinine, Na, K, Ca, glucose, amylase, Bili+, AST, GGT, alk. phos.
  - ~ Erect CXR (? perforation). Abdominal x-rays, supine/erect (obstruction).
  - ~ Ultrasound - liver, biliary system, pancreas, kidneys, ureters, pelvis, aorta.
  - ~ Other radiology - contrast studies, CT scan.

♦ Definitive management according to the cause. Possible causes: gastrointestinal, hepatobiliary, pancreatic, urological, gynaecological - complications of pregnancy (e.g. ectopic), musculoskeletal, respiratory, vascular, metabolic.

SHORTNESS OF BREATH

♦ Initial assessment and resuscitation:
- Airway]
- Breathing] will require at least supplemental oxygen
- Circulation]
- Supplemental oxygen therapy:
  - ~ Titrate according to PaO_2 or pulse oximeter.
  - ~ To optimize oxygen delivery use high flow O_2, reservoir bag.
  - ~ Use regulated FIO_2 via ventimask if COAD with CO_2 retention.

♦ Complete assessment:
- History:
  - ~ The patient with chronic or recurrent shortness of breath can provide a very valuable assessment of their severity.
  - ~ The patient's past history of severity may provide a warning to observe the patient closely. Obtain previous records urgently.
  - ~ Symptoms of infection should be sought - fever, rigors, productive cough.
  - ~ Shortness of breath may be a symptom of disease in another system, e.g. ischaemic heart disease, metabolic acidosis (diabetic ketoacidosis), anxiety, pulmonary embolism, anaemia.
- Examination:
  - ~ Severity is best assessed from the end of the bed.
  - ~ Respiratory rate, heart rate, pulses paradoxus, peak expiratory flow rate and use of accessory muscles are useful objective signs.

- ~ Auscultation and percussion of the chest are done not to assess severity but more to confirm or exclude infection or pneumothorax.
- Investigations:
  - ~ Pulse oximetry is the best guide to oxygenation (real time, non invasive, accurate but needs an educated interpretation).
  - ~ Arterial blood gas essential to assess pH and $PaCO_2$.
  - ~ CXR - particularly for pneumothorax, as it is difficult to exclude clinically, pneumonia and cardiac failure.
  - ~ Other investigations as indicated.

♦ Definitive management according to the cause. Possible causes:
- Lung disease.
- Heart disease.
- Airway disease.
- Chest wall disease.
- Neurological disease (abnormal patterns of breathing).
- Other disease (as above).

HYPOTHERMIA

Definition:	-	Mild	Temperature 32-35°C (shivering)
	-	Moderate	Temperature 30-32°C (unable to shiver)
	-	Severe	Temperature 25-30°C (<28°C high risk for ventricular fibrillation)

A low reading core temperature probe is required (e.g. rectal). Standard thermometers do not go below 35°C.

♦ Initial assessment and resuscitation:
- Airway]
- Breathing] warmed humidified oxygen
- Circulation
  - ~ Warmed IV fluid may be required but be cautious as fluid overload can occur.
  - ~ Defibrillation and antiarrhythmic drugs are less effective at low temperatures.
  - ~ Vital organs are protected by hypothermia.
  - ~ CPR should not be abandoned until the patient has been warmed beyond 32°C (the patient is not dead until he or she is "warm and dead").
- Passive rewarming:
  - ~ This is the method of choice.
  - ~ Warmed dry blankets (foil blanket can be put over the warm blankets but be sure to replace warm blankets as they become cool).
  - ~ Warmed humidified oxygen.
  - ~ Warmed IV fluids - contribute little to rewarming but will help prevent further cooling by cold IV fluids. Do not attempt to warm the patient with IV fluid. Limit the IV fluid volume unless hypovolaemic.

- Other methods - more aggressive methods are generally not used.
  - ~ Active external rewarming - heating with electric blanket or warm bath is contraindicated as it shunts blood to the periphery exacerbating hypotension and further cooling the core.
  - ~ Active internal rewarming - invasive methods of warming e.g. warmed peritoneal dialysis, are only indicated in the patient with severe hypothermia and refractory cardiac arrest in whom an adequate circulation cannot be maintained. Use only at the discretion of the Consultant.

♦ The complete assessment:
- History - three general types:
  - ~ The healthy person with exposure to extreme cold e.g. immersion.
  - ~ The previously healthy person with exposure to cold after ingestion of drugs or alcohol.
  - ~ The patient with underlying disease who may have been exposed to only moderate cold e.g. the elderly, the inactive, cerebrovascular disease, trauma, cardiovascular disease, diabetic ketoacidosis, hypoglycaemia etc.
- Examination] for traumatic injuries, underlying disease and
- Investigation] complications of cold.

♦ Definitive management:
- Moderate/severe hypothermia is best managed in ICU.
- Treatment of the underlying disease or complication.
- Hypothermia is 100% reversible, i.e. the patient has the potential to return to exactly the condition they were in prior to becoming cold.

HYPERTHERMIA

Definition - 'heat stroke' - temperature greater than 41°C with altered mental status (confusion to coma), with underlying dysfunction of the heat regulatory mechanism. It may be a continuum of 'heat exhaustion' which is a systemic reaction to prolonged heat exposure and is characterized by salt and water depletion. Cardiovascular and respiratory stimulation and sweating eventually give way to depression and a hot dry skin. Many systems can be damaged with rhabdomyolysis, renal failure, and coagulopathy common.

♦ Initial assessment and resuscitation:
- Airway]
  Breathing] supplemental oxygen
  Circulation
  - ~ Large volumes of fluid may be required to resuscitate.
  - ~ Subsequent fluid management is aimed at maintaining a urine output of >50 ml/hour and may best be guided by invasive monitoring of the circulation in ICU.
- Cooling measures:
  - ~ Rapid cooling is essential.
  - ~ Remove all clothing.

- ~ Apply ice packs to groin, axillae, and neck (large superficial vessels).
- ~ Thoroughly douse in iced water if available (ice slurry in towels). An alternative is to spray with water and fan to cause evaporation. If done well this may be the most effective means of heat loss.
- ~ Cooled peritoneal lavage has been used but other invasive 'lavages' have not been well evaluated in humans.

Note:

Tentative cooling may simply cool the skin and further limit heat loss by the core - be aggressive. Chlorpromazine 10-25 mg slow IV may control shivering and promote cooling by vasodilation.

- ♦ Complete assessment:
  - History:
    - ~ Usually exposure to extreme heat or strenuous activity in the heat.
    - ~ There may be contributing factors e.g. elderly, infirm, cardiovascular disease, cystic fibrosis, diabetes, alcoholism, obesity, infection, anaesthetic agents (via a muscle hypermetabolic state and requiring treatment with Dantrolene - contact ICU and the duty anaesthetist if secondary to suxamethonium, inhalational or local anaesthetic agents), antipsychotic and other drugs (via a central dopamine blocking action).
  - Examination - directed to causes and complications.
  - Investigations - directed clinically, but including CBC + diff, coagulation profile, urea, creatinine, AST, GGT, alk. phos., Bili, Na, K, Ca, urine for myoglobin.
- ♦ Definitive care - usually cooling/monitoring continues in the Intensive Care Unit including management of the many potential complications.

DRUGS

HOSPITAL PREFERRED MEDICINES LIST (PML)

The PML is available to all hospital doctors, trainee interns, pharmacists and Unit Nurse Managers. The content reflects local prescribing practice and includes common daily dose ranges and costs.

- Drugs should be chosen from the list unless there is a compelling reason for an alternative choice.
- If an alternative drug is prescribed, a pharmacist will phone the prescriber to ask if the drugs in the formulary had been considered. If "no", and the prescriber is happy with a PML drug, then the prescription will be changed accordingly. If "yes", and the prescriber still desires the alternative drug, this will be dispensed (if available). A record of the discussion will be kept so that at intervals (e.g. six monthly) patterns of drug usage can be analysed and the PML adjusted if necessary. Alterations to prescriptions will not be imposed.
- Patients admitted to hospital taking drugs not included in the PML should continue on these drugs unless change is deemed appropriate on clinical grounds.
- Drugs which may be prescribed only with the approval of a consultant are noted.

PRESCRIBING OF DRUGS

Ask yourself the following questions each and every time you prescribe a drug:-

- What is the diagnosis?
- What drugs are appropriate for this condition? (Check PML).
- Which drug is the most cost effective?
- Is this drug appropriate for this patient? (Consider age, allergies, underlying diseases, pregnancy, lactation, other drugs etc).
- Is a loading dose necessary?
- What is the appropriate maintenance dose and dose interval? (Remember that compliance is best with medication taken once or twice daily).
- Should the drug be administered with or without food?
- When should the effect of the drug be reviewed?
- How long should the patient remain on the drug or at this dose?
- Are any drug interactions likely?
- What major side effects might occur and should patients be warned of these?
- What minor side effects might occur and how should the patient handle these?
- What should the patient do if a dose is missed?
- Have you discussed the treatment programme with the patient?
- Is the prescription CLEAR, CONCISE, CORRECT, COMPLETE and WRITTEN IN CAPITALS?

DOSE ALTERATION IN RENAL IMPAIRMENT

Drugs, (or active metabolites) with a high fraction excreted unchanged (fu) in the urine **and** low therapeutic index, require dose-adjustment in renal impairment. This list includes:-

	fu (fraction excreted unchanged in the urine)
ACE inhibitors	
- enalapril	0.9 (metabolite)
- captopril	0.5
Aminoglycosides	0.9
Digoxin	0.8
Allopurinol	0.8 (metabolite)
Lithium	1.0
Metformin	0.9
Methotrexate	0.85
Ethambutol	0.85
Vancomycin	0.9

(Other drugs - phone Clinical Pharmacology Ext: 80900)

HOW TO ADJUST THE DOSE

- Decide on an appropriate dose-rate (i.e. total dose in 24 hours) for a patient with normal renal function (DR_{normal}).
- Calculate patient's creatinine clearance (CrCl). Normal is >1.5 ml/sec.

$$\text{CrCl (ml/sec)} = \frac{(140\text{-age}) \times \text{lean body weight (kg)}}{50{,}000 \times \text{serum creatinine (mmol/L)}} \times 0.85 \text{ if female}$$

- lean body weight (males) = 50kg + 0.9kg for each cm over 150 cm in height.
- lean body weight (females) = 45kg + 0.9kg for each cm over 150cm in height.

- Calculate dose-rate (DR) for this patient

$$DR_{patient} = \left[(1\text{-fu}) + fu \times \left[\frac{\text{calculated CrCl}}{1.5} \right] \right] \times DR_{normal}$$

- Decide whether you should decrease the dose itself or prolong the dose-interval (usually prolong dose-interval)

DOSE ALTERATION IN LIVER IMPAIRMENT

- Serum albumin and the prothrombin ratio best reflect liver metabolic capacity.
- Decide on approximate dose-rate for a patient with normal liver function.
- Check with Drug Information Service (Ext: 80900) and the drug package insert.

DOSE ALTERATION IN THE ELDERLY

- Adjust dosing for renally eliminated drugs as above.
- For other drugs use doses at the lower end of those recommended for young adults.
- Remember older patients may react differently to drugs than younger patients e.g. memory dysfunction with anticholinergic drugs.

PREGNANCY AND LACTATION - Check with the Drug Information Service (Ext: 80900).

THERAPEUTIC DRUG MONITORING

Measurement of plasma concentrations of the following drugs can assist management.

ESSENTIAL	IMPORTANT	SOMETIMES USEFUL	USEFUL IN OVERDOSES
lithium aminoglycosides	phenytoin carbamazepine phenobarbitone quinidine theophylline digoxin cyclosporin methotrexate amiodarone vancomycin	lignocaine platinum compounds valproic acid ethosuximide tricyclic antidepressants tetracyclic antidepressants 5-fluorocytosine	paracetamol methanol ethanol ethylene glycol salicylate

SAMPLING

- Aminoglycosides and vancomycin require sampling at specific times.
- For other drugs, TROUGH concentrations (just prior to next dose) give the best guide to accumulation.
- Sampling should be done at STEADY STATE (i.e. 4 half-lives after the first dose, or after a change in dose), unless a loading dose was given.
- Times of dosing, duration of dosing, and times of sampling should be recorded accurately for adequate interpretation.
- The specimen - one full 7 ml heparinized tube (green top), except for antibiotics which require 2 ml in a red-stoppered plain tube.
- If unsure, consult:
  - Toxicology (Ext: 80322) re sampling.
  - Clinical Pharmacology (Ext: 80900) re interpretation.
  - Specialist in relevant area re interpretation.

AMINOGLYCOSIDES

The Department of Clinical Pharmacology has a special interest in aminoglycoside dosing. Gentamicin is the aminoglycoside of choice except in special circumstances (e.g. tobramycin for pseudomonas, netilmicin if multiple courses (>3), amikacin for particular resistance).

- Computerized dosing (via toxicology or clinical pharmacology departments) should be performed in nearly all circumstances. Exceptions include the use of low dose therapy in urinary tract infections (UTIs) or in addition to ß-lactams in bacterial endocarditis.
- There are two dosing strategies:

  - **24 HOUR DOSING** - this is being introduced into the Christchurch hospitals. Recent evidence suggests that it may be superior to conventional dosing with reduced toxicity and at least equal efficacy. It should **not** be used if calculated **CrCl is less than 1.2** ml/sec, in **paediatrics,** and in **neutropenia**. It is not well studied in these situations and conventional dosing should be used.

    Method

    ~ First dose should be 5-7 mg/kg, depending on severity of infection, given as a 30 minute infusion in 100 ml normal saline. (Record exact infusion start and stop times).

    ~ A 'peak' blood sample should be taken about 30 minutes after the end of infusion. (Record exact sample time).

    ~ A second sample should be taken between 6 and 12 hours post dose. (Record exact time).

    ~ Consult Clinical Pharmacology (Ext: 80900) for dosing prediction when concentrations are available.

    ~ Repeat 30 minutes and 6-12 hours post infusion concentrations should be checked 2-3 times per week.

- **CONVENTIONAL DOSING**
  Method
  - Loading dose 4.5 mg/kg.
  - Check gentamicin concentrations at one hour and four hours post loading dose (second sample should be delayed if impaired renal function).
  - Record exact times of dose and samples.
  - While awaiting gentamicin concentrations give 1.5 mg/kg q8h, or less frequently depending on calculated CrCl.
  - Modify regimen according to computer predictions from Toxicology (Ext: 80322).
  - Gentamicin concentrations should be peak 6-10 mg/L and trough <1.5 mg/L.
  - Follow up concentrations should be measured every 2 or 3 days, or more frequently if clinical situation dictates.

VANCOMYCIN

Vancomycin has more complex pharmacokinetics than the aminoglycosides. The Department of Clinical Pharmacology is available to give advice about the interpretation of blood concentrations (Ext: 80900).

Dosing Method

- Conventionally, loading doses are not used.
- Start dosing with 15 mg/kg (usually 1 g) as a slow IV infusion in either 0.9S or D5W - minimum volume 100-200 ml) over a minimum of one hour.
- Draw blood for concentrations at ½ hour post-infusion and at the trough (immediately pre-dose).
- In patients with normal renal function the dose-interval will be 12 hours. In the presence of renal impairment the dose-interval should be longer.
- Modify regimen according to computer prediction from Clinical Pharmacology.
- Concentrations should usually be in the range of peak 20-60 mg/L and trough 5-10 mgL.
- Follow-up concentrations should be measured every 3-4 days, or more frequently if the clinical situation dictates.

OUTPATIENT PRESCRIPTIONS

Medicines prescribed for patients being discharged, or patients attending outpatient clinics, are not dispensed by the hospital pharmacy, except when not available from a retail pharmacy as outlined below.

The writing of outpatient prescriptions is governed by law in the Medicine Regulations 1984 and subsequent amendments. All such prescriptions must clearly state the following -

- The patient's name and address.
- The patient's date of birth if aged under 13.
- The name, strength, dose and dose frequency of the medicine.
- The quantity to be dispensed.
- Be signed personally by the prescriber.

The prescriber's name, address and registration number must also be **CLEARLY** shown on the prescription. A maximum of 90 days supply may be prescribed.

A small number of medicines are only available from a hospital pharmacy. Prescriptions for such medicines should be written on a separate form and the patient directed to the hospital pharmacy. Failure to do so may result in considerable inconvenience to the patient. A list of hospital only medicines is provided to wards. If in doubt, contact the ward pharmacist or the pharmacy (Ext: 80840).

CONTROLLED DRUG PRESCRIPTIONS FOR OUTPATIENTS

Prescriptions of Class B Controlled Drugs **MUST** be written on the special triplicate controlled drug prescription form. A maximum of thirty days supply may be prescribed.

PRESCRIPTION CHARGES

Most prescriptions incur the Government prescription charge. The current charge ranges from $2-$15 per item. Patients possessing a Community Services Card or a High Health User Card are entitled to lower charges. The status of the patient should be indicated using the codings on the top left of the prescription form.

Y	Under 5 years
J	5 years to under 16 or 18 (varies, depending on financial independence)
A	Adult
O	All contraceptives
1	Patient has Community Services Card (patient applies for this)
2	
3	No Community Services Card
Z	High User Card (Doctor applies for this on behalf of patient)
X	Has had greater than 20 prescriptions that year

Additional charges may apply where medicines are only partially subsidized by the Government.

NON SOCIAL SECURITY MEDICINES

Medicines not included in the Drug Tariff are not supplied free to patients by either retail or hospital pharmacies. A supplementary benefit approval may be obtained in some circumstances from the Ministry of Health for individual patients, which will then allow a free supply. Applications should be made to:

Health Benefits Centre
Private Bag 3015
WANGANUI

The Centre's Fax No. is (06) 345-1121

Approval must be received before supplies to patients can be made. Medicines not yet registered in New Zealand cannot be supplied free to patients.

DRUG INFORMATION SERVICE (Ext: 80900)

The Drug Information Service is available to answer any drug-related questions which cannot be answered by readily available texts.

- A verbal answer will be provided immediately or as soon as possible.
- Written, referenced answers will be provided for more complex, but specific, questions.
- Reviews are not performed.
- You are encouraged to come to the Department to discuss any drug-related questions.

ALCOHOL RELATED PROBLEMS

SCREENING FOR ALCOHOL RELATED PROBLEMS

This section provides guidance for the screening of alcohol related problems and lists the supporting services available in the Canterbury area.

A Social Worker who specializes in alcohol-related problems is employed at Christchurch Hospital in the Social Work Department (Ext: 80420). This service is restricted to patients attending Christchurch Hospital.

RECOGNIZING ALCOHOL PROBLEMS

The essence of recognition lies in thinking "could alcohol be contributing to this patient's problems?"

Some pointers to harmful drinking - gastrointestinal problems, alcohol withdrawal, anxiety, epileptic fits, recurrent accidents, memory failing, blackouts. Examination findings include alcohol on the breath, tongue tremor, rapid pulse, hypertension, peripheral neuropathy cerebellar signs, spider naevi, evidence of portal hypertension, testicular atrophy and gynaecomastia.

If looked at only from a physical point of view, many problem drinkers will be found to have no evident pathology. However, further enquiry about their lives and clear questioning about their drinking may reveal an alcohol problem.

It's often a good idea to ask about alcohol use at the same time you ask about diet, exercise and smoking, so that it forms part of a general health screen.

USEFUL QUESTIONS

- Do you drink alcohol at all?
- On average how many days a week do you drink?
- How many standard drinks would you consume?

A standard drink is		**1 glass (100 ml) wine** **¾ can beer (335 ml)** **1 nip spirits (pub nip)** **1 small glass sherry (60 ml)**
1 jug beer (1000 ml)	=	**4 standard drinks**
1 bottle beer (750 ml)	=	**3 standard drinks**

SCORE: Positive for alcohol related problems if:-

Men - Weekly intake exceeds 35 standard drinks

Women - Weekly intake exceeds 21 standard drinks

- Most people have days when they drink more than usual, how many times in the last year have you drunk more than 10 standard drinks?
- Have you ever been admitted to hospital because of accidents?
- Have you any blood relatives who are heavy drinkers?
- Does anyone annoy you by telling you to cut down on your drinking?
- What have you noticed about your drinking that bothers you?

If the answers indicate that an alcohol problem is present tell the patient what was found in the way of blood tests or physical examination and then provide him or her with some straight forward advice. It can be effective if done in a caring and concerned manner, e.g. "firstly, I have to tell you that the amount you have been drinking, although it doesn't seem much to you, has caused some damage to your liver".

The treatment options range from outpatient to inpatient counselling. The Alcohol Social Worker offers assessment, outpatient counselling and provides access to all treatment facilities. All referrals to the Social Work Department (Ext: 80420). Family members may also be referred.

Facilities available to help with alcohol related problems

City Mission - (Ph: 365-0635)
275 Hereford Street
(Assessment / Overnight Stay)

Community & Alcohol Drug Service - (Ph: 365-0839)
262 Armagh Street
(Assessment, Outpatient)

John Dobson Clinic - (Ph: 365-0983)
258 Armagh Street
(Intravenous Drug Users / Methadone)

Kennedy Villa - (Ph: 338-5059)
Sunny-side Hospital
(Assessment / Detoxification)

Mahu Clinic - (Ph: 338-5059)
Sunnyside Hospital
(Assessment / Outpatient)

Odyssey House - (Ph: 358-7791)
98 Greers Road
(Residential - 40 beds)

Salvation Army - Bridge Programme - (Ph: 338-4436)
35 Collins Street
(Inpatient Programme)

The Queen Mary Centre - (Ph: 03 315-7016)
Hanmer Springs

IV drug users should be referred directly to the John Dobson Clinic (see above).

ALCOHOL WITHDRAWAL

Following alcohol withdrawal the following sequence of events may be seen: tremor, hallucinations, seizures, delirium tremens (autonomic hyperactivity, disorientation and hallucinations).

- Assess nutritional status and administer thiamine 100 mg IM **before** glucose given.
- Attention to fluids, electrolytes, hypoxia, sepsis.
- Early withdrawal - diazepam 10 mg PO per hour until some signs of light sedation. Then stop diazepam - the half-life of diazepam and its metabolites is long enough to completely cover the danger period.
- Intermediate phase of withdrawal - chlormethiazole 50-1500 mg QID. Avoid oversedation. Chlormethiazole can easily oversedate and cause death from aspiration. Review patient regularly with particular attention to airway.
- Alcoholic hallucinations - Haloperidol 5 mg IM then 1-3 mg BD maintenance. Oral therapy when appropriate. **Note** Haloperidol may provoke fits or hypotension. Therefore only use if indicated.
- Delirium tremens:
  - Diazepam 2.5-10 mg IV infusion over 5-10 minutes then maintenance dose 1-2 mg/hr infusion. Oral therapy when appropriate.
  - Or chlormethiazole 50-100 ml 0.8% solution over 3-5 minutes then 500-1000 ml over 6-12 hours to maintain light sleep.

  Note:

  Phenothiazines are to be avoided because they are epileptogenic.
- Seizure prevention is required for patients who have past history of seizures. Use carbamazepine 400 mg stat then 200 mg TID for 5 days. Some authorities also recommend phenytoin or sodium valproate. To treat seizures use diazepam and see section on epilepsy.

  Note:

  Maintenance anticonvulsant treatment is contraindicated in patients with alcohol withdrawal fits as non-compliance favours further seizures.
- Many alcoholics are thiamine deficient and glucose infusions may precipitate Wernicke's encephalopathy. The classic features of Wernicke's encephalopathy are not always present and mild confusion may be the only manifestation. Always consider giving thiamine 100 mg IM stat in alcoholics presenting to hospital and for all patients with undiagnosed seizures, confusion, stupor and coma.

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CARDIOLOGY

HEART FAILURE

DEFINITION

"Heart failure" is a pathophysiological complex not a diagnosis or a pathological process.

MANAGEMENT REQUIRES EACH OF THE FOLLOWING:

- Recognition of the pathophysiological disturbance(s).
- Identification of the pathological process.
- Identification of precipitating cause(s).

AETIOLOGY

Primary disease processes

- Ischaemic heart disease: myocardial infarction, ischaemic cardiomyopathy.
- Hypertension: systemic or pulmonary (end stage).
- Heart valve disease: especially mitral and aortic valve disease.
- Cardiomyopathy: alcohol, idiopathic.
- Pericardial disease: constrictive pericarditis, tamponade.
- Congenital heart disease.
- High output states: cardiac beri-beri (alcoholics), Paget's Disease.

Contributing factors

The following are not generally the primary cause of heart failure but may exacerbate the physiological disturbance and therefore need to be considered when managing heart failure;

- Arrhythmias.
- Drugs.
  - ß-blockers]
  - Calcium antagonists] Negative inotropes
  - Most antiarrhythmics]
  - Withdrawal of diuretics due to poor compliance
  - Fluid retention: steroids, NSAIDs, liquorice.
- Anaemia.
- Thyrotoxicosis - particularly in the elderly.
- Infections (especially endocarditis and pulmonary infections).
- Pulmonary embolism.
- Fluid overload - e.g. transfusion, renal failure.

INVESTIGATIONS

May be delayed while acute therapy instituted and initial symptoms controlled.

- CXR (pulmonary venous congestion/oedema, cardiac size, pulmonary infections).
- ECG.
- Arterial blood gases (hypoxia, metabolic acidosis suggests lactic acidosis due to compromised peripheral circulation).
- Na, K (urgently if ECG or rhythm abnormal), creatinine, Mg, Ca, PO_4.
- CBC + diff (usually non urgent).
- Echocardiography (urgent if tamponade or bacterial endocarditis suspected).
- Radionuclide scan is useful to document degree of LV dysfunction.
- Thyroid function tests.

THERAPY

- Correct any contributing factor such as arrhythmias, infection etc.
- **Acute pulmonary congestion, pulmonary oedema**
  - Sit patient upright
  - Oxygen at 4-6 L/min.
  - Glyceryl trinitrate - 0.6 mg tablet sublingual or nitrate spray under tongue.
  - Morphine 2.5-5.0 mg IV slowly over 3-5 minutes, watch for evidence of hypopnoea. Care needed in patients with diminished level of consciousness and/or CO_2 retention.
  - Frusemide 40 mg IV - repeat as necessary to initiate diuresis. The effective dose will vary and a larger dose may be needed if patient is on frusemide maintenance treatment or has renal impairment.
  - Less distressed patients may not need morphine and oral frusemide may be sufficient. Be alert to poor absorption from an oedematous GI tract.
  - **If patient does not respond** to initial procedures then nitrate infusion (**see page 39**), positive pressure ventilation and haemodynamic monitoring in ICU or CCU should be considered.
- **Compromised myocardial function** - this can be managed by increasing myocardial contractility (inotropic support) or reducing the cardiac work load (after load reduction).
  - **Inotropic support**
    - Digoxin - indicated for control of ventricular response in atrial fibrillation and atrial flutter (and has value as third line agent in sinus rhythm with refractory heart failure). Loading dose (if not already on maintenance treatment) of 0.5 mg (IV or oral) then 0.5 mg after 4 hours then 0.25 mg 4 hours later. Each dose adjusted by factor of Wt/70 kg if necessary. Maintenance dose 0.25 mg per day usually given at night. In renal failure and the elderly reduce the dose.

- ~ Intravenous adrenergic agonists are useful in patients with severe heart failure on the basis of diminished myocardial function. They generally require ECG monitoring for arrhythmias and this is best done within the CCU or ICU.
  Dobutamine is probably the best drug to use for its positive inotropic effect as it causes little tachycardia and minimizes the increase in myocardial oxygen consumption compared to other drugs. Place 500 mg (2 ampoules) in 500 ml D5W (1 mg/ml) and run at 10 ml/hour (approximately 2.5 mcg/kg/min). Increase dose as required to achieve clinical response. One can give up to 10-15 mcg/kg/min but at these dose levels ECG monitoring is required.
  If BP remains below 80 mmHg systolic on dobutamine, a vasoconstrictor drug should probably be added (dopamine or adrenaline) to keep the BP above 80 mmHg and thus maintain coronary perfusion. Where renal impairment is an early feature use a "renal dose" of dopamine (2.5-5.0 mcg/kg/min) by IV infusion Can increase dopamine up to 7.5-10 mcg/kg/min if necessary (2 hourly steps of 2.5 mcg/kg/min). **Adrenaline is rarely needed.** If considered unavoidable place 1 mg adrenaline in 1000 ml D5W or 0.9S and run at 1-4 mcg/min IV infusion.

- **After load reduction**
  - ~ If BP well maintained use vasodilator therapy. Angiotensin converting enzyme (ACE) inhibitors are the treatment of choice. They can cause hypotension especially when given after intensive diuretic therapy and if there is hyponatraemia, therefore use with care. Aortic stenosis is a relative contraindication to ACE inhibition.
  - ~ In the acute situation, where oral therapy may not be suitable use intravenous vasodilators (glyceryl trinitrate or sodium nitroprusside) with close monitoring including Swan Ganz and arterial pressure monitoring in CCU or ICU (**see page 39 for IV glyceryl trinitrate instructions**).
  - ~ ACE inhibitor dosing - initial dose of captopril or enalapril then maintenance dose according to renal function and clinical status.

CREATININE CLEARANCE (ml/sec)	CAPTOPRIL DOSE		ENALAPRIL DOSE	
	Starting	Maximum	Starting	Maximum
>1.5	6.25 mg	50 mg q12h	5-10 mg	30 mg q24h
0.8-1.5	6.25 mg	37.5 mg q12h	5 mg	20 mg q24h
0.4-0.8	6.25 mg	31.25 mg q12h	2.5-5 mg	10 mg q24h
0.2-0.4	6.25 mg	25 mg q24h	2.5 mg	5 mg q24h
<0.2	6.25 mg	12.5 mg q24h	2.5 mg	2.5 mg q24h

Cockcroft and Gault Creatinine Clearance formula (**see page 25**).

Side effects of ACE inhibitors include;

Renal impairment - Use minimum dose possible, reduce dose if creatinine rises and reassess diuretic dose.

Hyperkalaemia - Need for K supplements usually reduced or unnecessary; avoid giving spironolactone, amiloride, triamterene at the same time.

Further management

- Daily weigh. Fluid balance in first 24 hours is essential to check diuresis, thereafter a daily weight will provide the best indication of the effectiveness of diuretic therapy. Check previous weights from old notes.
- Repeat CXR prior to discharge or when dyspnoea and/or clinical features fail to respond.
- Heparin 5,000 units SC BD unless contraindicated. Start on admission. Consider full heparinization then warfarin in those with marked left ventricular impairment, increased atrial size or chronic atrial fibrillation. Consult with Cardiologist.
- Potassium supplements will be needed with most diuretics. Requirements reduced or unnecessary in renal failure, ACE inhibitor treatment or when using potassium sparing diuretic therapy.
- Re-evaluate the primary cause of the heart failure - attempt to confirm the primary disease process and exclude contributing factors. This may include cardiac catheterization in selected cases.

MYOCARDIAL INFARCTION

DEFINITIONS

The diagnostic criteria for an acute myocardial infarction are 2 out of 3 from:

- Central chest pain lasting >30 minutes.
- ST elevation (transmural or Q wave infarction).
- Cardiac enzyme release.

CAUSES

- Ischaemic heart disease.
- Emboli (rare).
- Spasm (Prinzmetal's angina).

CLINICAL FEATURES

A history of severe crushing retrosternal chest pain radiating to neck and arms is typical but not invariable. May present as collapse, LVF, hypotension, peripheral embolus, stroke, or "malaise". A difficult diagnosis to exclude even

with normal ECG. Generally if in doubt, admit to hospital. If the initial ECG is normal then the diagnosis may be suspected on the basis of history alone and ECG repeated within 2 hours.

INVESTIGATIONS

- ECG daily for 3 days. If spasm is being considered (ST elevation and atypical pain) repeat ECG when pain resolves to document ST change. An ECG should be done prior to discharge.
- Cardiac enzymes: Troponin T (TNT), CPK (MB fraction), AST should be done on admission, at 12 hours and 24 hours after onset of symptoms. For those patients admitted more than 24 hours after the onset of pain, serial enzymes and LDH should be performed on 3 consecutive days.
- CXR. Indications for urgent X-ray:
  - Suspicion of aortic dissection (widened mediastinum ± separation of calcified intima).
  - Moderate or severe cardiac failure.

  Otherwise CXR can wait until normal working hours or at discharge.
- CBC + diff.
- Na, K, Ca, PO_4, creatinine, glucose.
- Fasting cholesterol and triglycerides the morning after acute episode and repeat at 6 weeks.

Complications of myocardial infarction

- The following problems may complicate even small myocardial infarcts:
  - Left ventricular failure.
  - DVT/PE.
  - Dressler's syndrome (pericardial and/or pleural inflammation).
  - Arrhythmias.
  - Cardiogenic shock/low cardiac output states.
  - Valvular dysfunction.
  - Myocardial rupture (septal or free wall).
  - Mural thrombi (with systemic embolization).

MANAGEMENT

- **Placement** - any patient with acute myocardial infarction is at risk from an acute arrhythmia and should be closely monitored, preferably in a coronary care unit for 12-24 hours from onset of symptoms. For advice on admission to CCU or ICU contact the CCU or ICU registrar on call.
- **IV access** - IV insertion on admission. Flush 4-6 hourly with 0.9S.
- **Oxygen** is unnecessary in uncomplicated infarcts if the patient remains well and pain-free. Otherwise give oxygen by mask or nasal cannulae at 1-4 L/min for 24 hours or longer if necessary.

- **Pain relief -** continuing pain suggests ongoing ischaemia which should be treated with nitrates, ß-blockers, calcium antagonists and morphine as required. Give morphine IV according to severity and repeat up to 4 hourly if necessary. Draw morphine 10 mg (1 ml) up with 9 ml of water for injection (1 mg/ml). Give 2-3 mg (2-3 ml) increments until pain is controlled observing the patient's BP and respiration. Metoclopramide (Maxolon) 10 mg IV or prochlorperazine (Stemetil) 12.5 mg given IV at the same time may reduce nausea and vomiting.
- **Nitrates** may be helpful for continuing pain (patch, isosorbide dinitrate or mononitrate tablets). If pain is severe consider IV nitrates. Use glyceryl trinitrate (Tridil) 50 mg in 250 ml 0.9S. Start at 3 ml/hr in average-sized adult and titrate up by 3 ml/hr steps every 5 minutes until pain relieved or BP falls (can go as low as 90 mmHg systolic if otherwise well).
- **Aspirin** 300 mg sublingual stat then 300 mg orally daily if no contra-indications.
- **Thrombolytic therapy -** streptokinase (SK) is the best established thrombolytic. Tissue plasminogen activator (tPA) is also available but is much more expensive and should not be used as the initial thrombolytic agent. However, consider tPA if SK has been used during the past 2 years, if there has been a recent definite streptococcal infection, or if SK provokes unacceptable side effects requiring early cessation of the infusion. Consultation with a cardiologist is essential if there is any doubt as to which agent to use.

Current indications for SK therapy

- All patients presenting with acute myocardial ischaemic symptoms lasting more than 30 minutes with ST elevation on ECG.
- New ST elevation greater than 1 mm in at least 2 limb leads **or** greater than 2 mm in at least 2 pre-cordial leads **or** new left bundle branch block with typical symptoms.
- If pain has waxed and waned and ST elevation is present, you cannot be certain when ischaemia progressed to infarction. If in doubt, give thrombolytic therapy.
- Continuing pain with ST elevation is an indication for thrombolysis regardless of the time from the onset of symptoms.

  Note:

  SK should be given as soon as possible. Major benefits have been seen if given within 6 hours of the onset of symptoms. Some beneficial effects may be seen up to 24 hours and thus the administration of SK should also be considered between 6-24 hours from the onset of pain.

Flow diagram for the use of SK therapy

Check Exclusion Criteria →

EXCLUSION CRITERIA

- Active bleeding (including menstruation) / known bleeding disorder
- Major surgery or trauma in last 6 weeks
- Recent non compressible vascular puncture
- Extensive CPR (>5 mins)
- Recent CVA (<6 weeks)
- Active peptic ulcer disease
- Severe liver disease
- Pregnancy
- Severe diabetic retinopathy
- SK in last 12 months or sustained known allergy
- Recent streptococcal infection
- Uncontrolled hypertension (diastolic >115 mmHg) (systolic >180 mmHg)

↓

No contraindication for SK

↓

Sublingual nitrates
Aspirin 300 mg PO

↓

Insert IV Lines x 2 and take blood for coagulation profile, Group & Hold

↓

Streptokinase 1.5 million units over 30 minutes. Slow rate if BP falls.

↓

Repeat ECG

↓

Exclude

↓

Sublingual nitrates
Aspirin 300 mg

↓

ß-blocker
Discuss with Cardiology Reg/ Consultant

Administration of streptokinase

- Add 1,500,000U of streptokinase to 100 ml 0.9S and infuse over 30 minutes.
- After 6 hours start Heparin 5000U SC BD.

Monitoring requirements for thrombolytic therapy

- Continuous ECG monitoring with senior nurse and doctor in attendance.
- 15 min pulse, blood pressure and temperature for 1½ hours, then hourly for 4 hours, then as needed.
- Maintain availability of drugs and defibrillator.
- Patients who are not already in CCU should be transferred promptly to CCU after the infusion provided the patient is stable.

Record times of:
- ~ Onset of pain.
- ~ First ECG / start SK / finish SK.

Note:
- If ST segment depression is present, or ST-T wave changes are non specific but symptoms/risk factors are suggestive, then give ß-blocker (atenolol 50 mg PO daily, may need 100 mg) as well as aspirin and nitrates.
- Check right sided leads for ST increase when ST depression is confined to V1-3 in std 12 lead (i.e. look actively for RV infarction) - consider 'true' posterior' infarct - **if in doubt give thrombolysis.**
- Move to CCU **early**.
- Err on side of **excess consultation** with cardiology registrar/ cardiologist, acute medical registrar/general physician.

Complications

- Hypotension associated with SK infusions:
  - Slow or stop streptokinase temporarily.
  - Head down tilt.
  - Consider giving IV 0.9S 250 ml boluses x 2-3 (contraindicated in LVF, particularly useful in right ventricular infarcts).
  - If above unsuccessful, consider aramine IV 0.5-1 mg, adrenaline 1 ml 1:1000 very cautiously (wait and repeat at 5-10 minute intervals). Adrenaline IV in a patient with an acute MI should only be given in the event of a catastrophic anaphylactic reaction since it may precipitate ventricular fibrillation.
- Allergic or febrile reactions which vary in severity from rigors to typical anaphylaxis. Give hydrocortisone 100 mg IV and/or phenergan 12.5-25 mg IV stat. For severe anaphylaxis consider adrenaline SC/IV but the need for this is rare.
- Haemorrhage - apply local pressure. If significant bleeding administer 2-3 units fresh frozen plasma and seek advice from Haematology.
- If bradycardic atropine may be helpful. Give atropine 0.3 mg IV with further doses over 30 minutes to a total of 1.2 mg.

Other treatments

- **Heparin** 5,000 units SC BD starting at admission if thrombolytic treatment not given.
- **Hypnotics** if sleep disturbed.
- **ß-Blockers** - continue if patient is already on them and no contraindication to using them. There is now evidence that ß-blockers improve prognosis and unless contraindications are present ß-blockers such as atenolol, metoprolol or timolol, should be commenced on admission, IV if continuing pain/arrhythmias and continued for 1-2 years.

- **Lignocaine** - reserve for those patients who have had **recurrent** episodes of ventricular fibrillation or tachycardia. Give lignocaine 200 mg by IV infusion over 10-20 minutes; if necessary following with infusion of 1000 mg in 500 ml D5W (i.e. 2 mg/ml). Give 2 mg (1 ml) per minute initially and reduce to 1 mg per minute if possible. Can increase to 3 mg/min if required but if this is necessary for any length of time consider doubling the concentration to reduce fluid load. Watch for lignocaine toxicity such as sedation and convulsions if the patient has impaired hepatic function.
- **Continuing chest pain** in spite of appropriate morphine IV and sublingual nitrates. Consider nitrate infusion (**see page 39**) or full IV heparinization (**see page 191**).

CARDIOGENIC SHOCK

CLINICAL FEATURES

The presence of shock following myocardial infarction implies the loss of a large area of myocardium and carries an extremely high mortality (>80% in hospital).

- Dobutamine is probably the best drug to use for its positive inotropic effect; as it causes little tachycardia and less increase in myocardial oxygen consumption than other drugs (**see page 36 for dosing instructions**). If BP remains below 80 mmHg systolic on dobutamine, a vasoconstrictor drug should probably be added (dopamine or adrenaline) to keep the BP above 80 mmHg and thus maintain coronary perfusion. Consider early addition of 'renal dose' (2.5-5.0 mcg/kg/min) of dopamine in presence of baseline or evolving renal impairment.
- About 20% of patients with cardiogenic shock have low LV filling pressures (eg RV infarction or patients on diuretic therapy) and may benefit from fluid infusions (250 ml bolus 0.9S and maybe a second bolus). This should only be done with monitoring of wedge pressures using a Swan-Ganz catheter. CVP monitoring alone in cardiogenic shock is insufficient.
- All young patients (under 60 years) with cardiogenic shock should be managed in CCU or ICU. Call the CCU/ICU registrar for advice. If shock occurs in the hours following an MI coronary angioplasty is the treatment of choice.
- Treat any arrhythmias.

CARDIAC ARRHYTHMIAS

Note:

Inappropriate treatment of arrhythmias can be rapidly fatal. Whenever possible seek expert advice.

CLASSIFICATION

- Ectopic activity (atrial and ventricular).
- Heart block.
- Bradyarrhythmias.
- Supraventricular tachycardias.
- Ventricular tachycardias.

AETIOLOGY

- Common in the presence of structural cardiac disease, especially acute myocardial infarction.
- Electrolyte imbalances (especially hypokalaemia) and acid/base imbalance may initiate and/or perpetuate the arrhythmia and these should be corrected.
- Drugs including tricyclics, phenothiazines, theophylline, digoxin and anti-arrhythmics.
- Hyperthyroidism.

CLINICAL FEATURES

- Check pulse at apex and wrist, blood pressure, tissue perfusion.
- If there is evidence of hypotension or heart failure then urgent treatment is required.
- Assess venous pressure waves:
  - Regular cannon waves in junctional rhythm.
  - Irregular cannon waves in ventricular tachycardia or heart block.

INVESTIGATIONS

- ECG - 12 lead and rhythm strip with the best P wave. If bizarre/wide QRS complexes then check speed of paper.

  Note:

  An oesophageal lead can be easily placed and may greatly aid diagnosis of tachyarrhythmia by clearly identifying the P wave and its relationship to the QRS complex.
- Check for abnormalities of K, Mg, Ca, acidosis and hypoxia. Metabolic factors may contribute to the initiation/perpetuation of the arrhythmia.
- Consider the possibility of drug induced arrhythmias.
- Thyroid function tests.

MANAGEMENT

Ectopic activity

- Atrial ectopics - often normal, usually benign in MI. Look for atrial beat (may just deform preceding T wave) when diagnosing "extrasystole". Does not require treatment.
- Ventricular ectopics - common, usually benign. May be confused with aberrant atrial ectopics. Treatment usually not required.

Heart block

- Prolonged PR Interval:
  - 1st degree block does not require treatment. Monitor closely in anterior infarcts and consider pacing as this may precede complete heart block.
  - 2nd degree block
    - ~ Type I (progressive increase in PR interval until beat is dropped) may be observed in inferior infarcts but is more serious in anterior infarcts.
    - ~ Type II (PR interval normal or increased but beats lost in unpredictable fashion) may become complete heart block in anterior MI.
- Bifascicular block - stable asymptomatic bifascicular block does not necessarily require pacing. However, following anterior myocardial infarction it may progress to complete heart block.
- Complete heart block - if stable with regular ventricular escape rhythm and satisfactory blood pressure, may be observed overnight. Be prepared to use isoprenaline (**see dosing instructions under Bradyarrhythmias**) to maintain rate if atropine is not effective. Discuss with Cardiologist. Symptomatic A-V block not associated with infarction usually merits placement of a permanent rather than temporary pacemaker. If syncope has occurred a temporary pacemaker should be placed until permanent pacing can be arranged.

Bradyarrhythmias

- Sinus bradycardia - check for excessive ß-blockade. Common after myocardial infarction. Treat with atropine 0.6 mg IV if heart rate <40 and aim to keep it above 50. Smaller additional doses of 0.3 mg may be required. Total dose of 2-2.5 mg before atropine side effects occur. Isoprenaline may also be used. Place 2 mg in 500 ml D5W (= 4 mcg/ml) and start at 60 ml/hour but then run as slowly as possible (0.5-10 mcg/min or ml/hr) to keep heart rate >60.
- Sinus arrest - common in inferior infarction and usually benign, as nodal escape rhythm maintains adequate heart rate. It may require treatment with atropine or isoprenaline but rarely needs pacing. When sinus arrest is not

associated with infarction, it is due to the sick sinus syndrome and requires pacing if symptomatic.

Note:

Inferior infarcts are associated with a wide range of rhythms without having much adverse effect on myocardial performance. A-V block is commonly seen. These arrhythmias are generally not treated vigorously apart from ventricular tachycardia and fibrillation. If they are persistent and cardiac function is impaired, treatment is indicated.

Supraventricular tachycardia

- **Sinus** - slow onset, rate usually below 150/min, slows with carotid sinus massage. Does not require treatment itself but requires an explanation as to its cause (e.g. LVF, anxiety, pain, hyperthyroidism, infection, hypoxia).
- **Paroxysmal tachycardia** - sudden onset, rate usually >150/minute. Carotid sinus massage causes either no response or reversion to normal or increased AV block. Atrial flutter usually gives a ventricular rate of approximate 150/min (2:1 block) and may be misdiagnosed as paroxysmal tachycardia. If not distressed and not in failure and history of short-lived attacks either:
  - Do nothing, or
  - Valsalva manoeuvre (supine)
  - Dive reflex - face into iced water
  - Carotid sinus massage at the upper point of the thyroid cartilage for 2 cms up and down (one side at a time).

  Monitor the effect of these manoeuvres with ECG, as this may induce 2:1 block.
- Adenosine IV in increasing doses 5 mg, 10 mg, 15 mg, 20 mg in step wise fashion at 2 minute intervals (effective for AV nodal re-entrant tachycardia but will not revert atrial flutter).
- If unsuccessful and not on ß-blockers:
  - Verapamil 5 mg by slow IV bolus (5 minutes) followed by 1 mg/min to a total of 15 mg. Do this with the patient on a monitor, measuring BP and with resuscitation equipment nearby as asystole may result.

    Note:

    Verapamil should never be used for a broad complex tachycardia as this may be ventricular tachycardia. It has considerable negative inotropic effects and should not be used in the presence of ventricular dysfunction.
  - If on ß-blockers and no structural cardiac disease present use disopyramide 50-100 mg IV and/or flecainide 2 mg/kg (max 150 mg) over 10 minutes IV or consider further ß-blockade (make sure patient is not asthmatic) but DO NOT USE VERAPAMIL as it may cause complete heart block. If necessary proceed to cardioversion.
  - If unsuccessful, proceed to cardioversion. The patient should be in CCU or ICU. Use thiopentone 100-500 mg IV (until loss of eyelash reflex. Give slowly as thiopentone will lower the BP if given too quickly or in

too high a dose). An experienced assistant is essential to maintain ventilation using an oral airway and Ambu-bag ventilation. Be prepared for endotracheal intubation if required. Start with 50 Joules, then 200 Joules, then 400 Joules. Do not shock more than twice with 400 Joules - consult with Cardiologist.

- **Atrial flutter** - this rhythm is often mislabelled as paroxysmal atrial tachycardia because the doctor has not performed carotid sinus massage to increase AV block, decrease ventricular rate and demonstrate flutter waves. If compromised, cardiovert with low energy shock. If not compromised, digitalize using oral protocol on **page 35**. If spontaneous reversion to sinus rhythm does not occur within 24 hours, the patient should be referred for cardioversion.
- **Atrial fibrillation** - initial treatment is digitalization. Cardioversion is indicated if compromised. Cautious additional verapamil (e.g. 40 mg TDS orally) or ß-blockers may be useful if a high ventricular rate persists despite digoxin.

  Note:
  - Digoxin levels will rise on verapamil.
  - Failure to control rate suggests underlying pathology, (e.g. thyrotoxicosis).
  - Do not treat Wolff-Parkinson-White syndrome with digoxin, verapamil or lignocaine. Pre-excitation syndrome is likely if ventricular rate >200. Use cardioversion if urgent intervention is required.
  - Flecainide is useful in resistant atrial arrhythmias.
  - Amiodarone may be helpful - consult Cardiologist.

Ventricular arrhythmias

- **Nodal rhythms** (rate < 100/min, idioventricular) - these are common after myocardial infarction and do not require treatment. Remember digoxin toxicity as a cause. Slow rhythms respond to treatment with atropine and/ or isoprenaline. Ventricular bigeminy may be due to digoxin toxicity and does not require therapy.
- **Ventricular tachycardia** (VT) - may be confused with SVT when aberrant AV conduction causes broad QRS complexes. Cannon waves and a variable first sound are suggestive of ventricular tachycardia. ECG diagnosis depends on P waves, and these are best seen in V1 or V2 or an oesophageal lead if possible. P waves independent of ventricular rate or fusion beats are diagnostic. Remember VT may be prolonged and not associated with collapse. Treatment is lignocaine 100 mg IV bolus and repeat before proceeding to cardioversion. Unless an emergency this should be undertaken in CCU or ICU. In an emergency situation proceed to 200-400 Joule shock.

  If in doubt assume that all regular, broad complex tachycardias are VT. Treatment of choice is cardioversion.
- **Ventricular fibrillation** (VF) - D.C. shock (see below).

CARDIAC ARREST

A precordial thump should only be used in a witnessed cardiac arrest.

- Commence basic life support - using the ABC's of CPR. Call the Cardiac Arrest Team and trolley.
  REMEMBER:
  - External cardiac compression at 80-100/min.
  - Ventilate once every 5 beats for 2 person CPR and twice every 15 beats for 1 person CPR.
  - Use mouth piece and bag, or mouth to mouth rather than intubate unless you are confident of success. If you insert an endotracheal tube basic life support must not stop for more than 30 seconds.
  - When the defibrillator arrives identify the rhythm utilizing the paddles and/or by attaching ECG limb leads.
  - Paddle positions at right of upper sternum below the clavicle, and left of the left nipple in the anterior axillary line. Use either paste on the paddles or pre-jelled pads on the chest to decrease impedance.
  - Do not use dilated pupils as an indication to stop resuscitation.

IDENTIFY THE CARDIAC RHYTHM

- **Ventricular fibrillation (VF):**
  - Defibrillate immediately using 200 Joules.
  - Check rhythm: if still in VF, repeat defibrillation using 300 Joules.
  - If still in VF, repeat defibrillation using 360 Joules.
  - If unsuccessful give adrenaline 1.0 mg IV (1 ml 1:1000). This may be repeated every 3-5 minutes.
  - If still in VF defibrillate using 360 Joules.
  - If still in VF give lignocaine 50-100 mg IV bolus and repeat defibrillation using 360 Joules.
  - If still in VF give bretylium 5 mg/kg IV and repeat defibrillation.
  - If further resistant VF give 0.5 mg propranolol IV and defibrillate.
- **Ventricular asystole:**
  Note:
  Exclude the possibility of monitor failure resulting in apparent asystole.
  - Give adrenaline 1 mg IV (1 ml 1:1000). This may be repeated frequently.
  - If still asystole give atropine 1 mg IV.
  - Consider giving sodium bicarbonate 50-75 ml of 8.4% solution.
  - Consider an adrenaline infusion. Add 1 mg (1 ml of 1:1000) adrenaline to 100 ml 0.9S (10 mcg/ml) and infuse at an initial rate of 15 ml/hr (2.5 mcg/min) and thereafter at a rate, generally 15-60 ml/hr (2.5-10 mcg/min), sufficient to produce an acceptable heart rate.
  - Consider transcutaneous or transvenous pacing.
- **Bradycardia and heart block:**
  - Atropine 0.6 mg IV and repeat if necessary.
  - Isoprenaline infusion as described under bradyarrhythmias.

- Adrenaline infusion as described under asystole.
- Definitive management is by pacemaker, transcutaneous (temporary) or transvenous.
- Thump pacing may be effective in inducing ventricular depolarization and an adequate cardiac output.

♦ **Electromechanical dissociation**, i.e. organised electrical activity on ECG but failure of effective myocardial contraction:
- Consider and treat various possible causes including hypovolaemia, tension pneumothorax, cardiac tamponade, pulmonary embolism, overdose, anaphylaxis.
- In absence of other specific therapy give adrenaline 1 mg IV (1 ml 1:1000). This may be repeated every 3-5 minutes.
- Consider sodium bicarbonate 50-75 ml of 8.4% solution.

POST-ARREST MANAGEMENT

♦ Maintain basic life support unless the patient has an adequate spontaneous circulation and respiration.
♦ Provide high inspired oxygen.
♦ Monitor ECG and transfer when stable to CCU or ICU, depending upon level of consciousness and requirement for artificial ventilation.

HYPERTENSION

CLASSIFICATION

Primary	-	Idiopathic, "essential".
Secondary	-	Renal, endocrine or neurological disease.
	-	Drug induced.
Malignant	-	Severe hypertension with rapidly progressive end organ damage e.g. acute left ventricular dysfunction, encephalopathy, retinopathy (haemorrhages, exudates and papilloedema) and renal failure.

AETIOLOGY

Renal	-	Acute nephritis, renal impairment (acute or chronic), renovascular and volume overload (especially dialysis patients).
Endocrine	-	Cushings Syndrome, phaeochromocytoma, Conn's, hyperparathyroidism, hypothyroidism, acromegaly.
Neurological	-	Raised intracranial pressure, autonomic neuropathy.
Respiratory	-	Obstructive sleep apnoea.
Drugs	-	Presence or absence (clonidine withdrawal). NSAIDs, steroids, sympathomimetics (including non prescription drugs), alcohol, liquorice, cocaine, erythropoietin, cyclosporin.

INVESTIGATION

- Blood pressure measurement - lying and standing (should be confirmed by medical staff).
- ECG and CXR.
- Collect blood for catecholamines before treatment if phaeochromocytoma possible as therapy will alter the blood levels (5 ml of blood into a purple top EDTA tube. Requires rapid separation and storage at -20°C. Consult with Special Tests in Endocrinology any week day morning).
- Plain abdomen x-ray or ultrasound for renal size and calcification.
- Urinalysis (microscopy for cells and casts).
- Plasma, Na, K, Cl, creatinine, Ca.
- 24 hour urine collection for protein (plain container):
- 24 hour urine for creatinine clearance, Na, K, VMA, motanephrines and free catecholamines (acidified container).

MANAGEMENT OF ACUTE HYPERTENSIVE CRISIS

Medical staff should personally monitor blood pressure frequently;

- The excessive use of powerful IV agents may lead to severe cerebral and myocardial insufficiency. Gentle reduction over hours enables compensatory vasodilatation and cardiovascular changes to develop and decreases possibility of end organ damage.
- Hypertensive encephalopathy in adults is usually associated with systolic BP >200 mmHg and diastolic >130 mmHg but can occur at lower levels if there has been a rapid rise in pressure. Aim to reduce diastolic to around 100 mmHg only. Oral therapy is generally best but patients with evidence of hypertensive encephalopathy (confusion, restlessness, convulsions, hypoventilation, papilloedema) require IV treatment. Consider admission to ICU or CCU.
- **Oral therapy -** sublingual nifedipine is probably first choice. Get patient to bite 10 mg capsule and retain drug in mouth to allow absorption. This tastes bitter. Dose may be repeated ½ hourly to a maximum of 30 mg. Alternatively captopril 6.25 mg PO may be used but is best avoided in the presence of hyponatraemia. Labetalol gives combined α and ß-blockade and may be used if no contraindications to ß-blockade. (200 mg PO stat then repeat as required to 1200 mg daily).
- **IV therapy** - for true acute hypertensive encephalopathy, i.e. sudden severe rise in diastolic blood pressure, headache, convulsions, and neurologic signs including papilloedema; labetalol 50 mg IV over 1 minute followed by further slow IV push to total 300 mg. An effective safe alternative is nitroprusside (50 mcg in 500 ml, titrated against BP, in the range 0.3-1.0 mcg/kg/min), only in the CCU or ICU.

Note:

- Do not treat cerebrovascular accidents with IV therapy - oral therapy with a slower reduction in blood pressure is mandatory as cerebral autoregulation may be lost.
- If hypertension is associated with acute LVF or volume overload IV frusemide should be used.
- Phaeochromocytoma, if suspected, requires α-blockade (phenoxybenzamine) or the combination of α plus ß-blockade (e.g. labetalol). Avoid ß-blocker monotherapy. It may cause paradoxical hypertensive crisis via unopposed adrenergic activity.
- Plasma sodium gives some index of volume depletion and activity of the Renin-Angiotensin-Aldosterone system (RAAS) in hypertension. A low sodium usually indicates low circulating volume and high RAAS activity. The use of ACE inhibitors may produce profound hypotension.
- If hypertension is associated with withdrawal of clonidine or other centrally acting drugs used in hypertensive treatment avoid giving a ß-blocker alone. Stopping clonidine may induce a phaeo-like state which is exacerbated by giving a ß-blocker. Labetalol is recommended.

AORTIC DISSECTION

CLINICAL FEATURES

This diagnosis should always be specifically considered in all cases of acute chest pain. Pain in the back, hypotension and an abnormal mediastinum on CXR, suggest aortic dissection. Seek urgent expert advice from the Cardiologist.

AETIOLOGY

- Atheroma.
- Hypertension.
- Hereditary defects, e.g. Marfan's Syndrome.
- Cystic medial necrosis.

INVESTIGATIONS

- ECG - dissection involving the aortic root may occlude the coronary arteries and produce myocardial infarction. LVH may be present from long standing hypertension.
- CXR - calcified intimal line separated from aortic outline. High aortic arch.
- Proceed to urgent contrast CT scan or trans oesophageal echocardiogram (TOE).
- Crossmatch blood.

TREATMENT

- Aim to reduce systolic pressure to 100-120 mmHg and reduce contractility of left ventricle.
- Treat with propranolol (10 mg IV over 10 minutes) or labetalol. Avoid diazoxide and hydrallazine.
- Analgesia, morphine 10-15 mg. Give prochlorperazine (Stemetil) 12.5 mg IV to prevent vomiting.
- Consult cardiologist on call who will make decision on transfer of patient to Cardiothoracic Unit.
- Monitor BP and urine output.

BACTERIAL ENDOCARDITIS (Reference: Lancet i: 603, 1984)

Fever of unknown origin, especially if in association with heart failure or heart murmurs, must be considered suspicious. If in doubt treat after blood cultures have been taken. Urgent cardiology and infectious disease consultation is essential.

INVESTIGATIONS

- Blood cultures. Three venepunctures inoculating 2 bottles each time (even only 10 minutes apart) or 6 venepunctures (12 bottles) if antibiotics given in last 2 weeks. **(See page 39 for blood culture technique).**
- CXR.
- ECG.
- MSU x 2 before therapy.
- Na, K, Ca, glucose, creatinine, Bili, alk. phos., AST.
- CBC + diff.
- Echocardiography.

TREATMENT

- Initial therapy - penicillin 2 mega units q4h IV, plus gentamicin. Flucloxacillin should be added if staphylococcal sepsis suspected (e.g. IV Drug User).
- Gentamicin dosage (**see page 27**). Full dosage should be given for the first 48 hours . Depending on the organisms isolated, lower doses of gentamicin may subsequently be adequate. Seek advice.
- Revise therapy in the light of the organism(s) isolated and their potential clinical significance and sensitivities, e.g. urgent valve replacement may be needed if staphylococcal or fungal endocarditis suspected.
- Observe, closely monitoring cardiac function, renal function and antibiotic levels.

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SUB-ACUTE BACTERIAL ENDOCARDITIS, PROPHYLAXIS

(NZMJ:1992;105:192-194).

PATIENTS AT RISK

Antibiotic prophylaxis should be given to patients with the following disorders:

- Congenital heart disease, including all patients with lesions which are haemodynamically significant and patients with known or suspected minor lesions of the left side of the heart. Included in the latter category are mitral valve prolapse of sufficient haemodynamic significance to produce a systolic murmur and idiopathic hypertrophic subaortic stenosis. Patients with surgically created shunts or conduits are also included. It is preferable to err on the side of caution and give prophylaxis if in doubt.
- Most patients who have been treated surgically still require prophylaxis except for those who have undergone surgery for patent ductus arteriosus and uncomplicated ostium secundum atrial septal defect. Prophylaxis may be required after correction of ventricular septal defect or coarctation of the aorta because of minor associated anomalies such as aortic valve lesions.
- Rheumatic and other acquired valvular heart disease.
- Patients with a prosthetic heart valve including heterograft or homograft tissue valves.
- The risk is small in patients with permanent transvenous pacemakers but prophylaxis is indicated if major bacteraemia is likely.
- Prophylaxis is not required for patients who have had coronary artery bypass graft surgery or have trivial pulmonary ejection systolic murmurs.

HIGH RISK PATIENTS

- Patients with prosthetic, heterograft, or homograft valves or with severe lesions of their own aortic or mitral valves. The consequences of endocarditis are worse in these patients.
- Patients with a history of bacterial endocarditis. These patients have an increased risk of recurrence.

OTHER PATIENTS REQUIRING SPECIAL CONSIDERATION

- Those who have recently received penicillins (including those on continuous oral or intermittent intramuscular penicillin for rheumatic fever prophylaxis) or cephalosporins in whom the oral flora may include organisms relatively resistant to penicillins.
- Patients allergic to the penicillins.

DENTAL AND RESPIRATORY TRACT PROCEDURES

Dental procedures

- All procedures which cause bleeding require prophylaxis, particularly in the presence of gingival disease. These include extraction of teeth, oral surgery, scaling periodontal surgery, application of matrix bands (if bleeding is expected), endodontics, re-implantation of avulsed teeth, incision or drainage of infected tissue, and biopsy procedures.
- Endodontic treatment should be completed as quickly as possible. With the treatment schedules recommended, repeat dental procedures with the same antibiotic prophylaxis are safe after one week.
- Application of an antiseptic such as chlorhexidine gluconate to the gingival margins, or as a mouth wash before dental treatment, reduces the severity of the bacteraemia and may be used to supplement antibiotic prophylaxis.
- Prophylaxis is not required for spontaneous shedding of deciduous teeth, adjustment of orthodontic appliances or restorations above the gingival tissues.
- Edentulous patients with ill-fitting dentures which cause ulceration are at risk from bacteraemia.

Respiratory tract procedures

These regimens are suitable for those undergoing tonsillectomy and adenoidectomy, rigid bronchoscopy, or operations involving respiratory mucosa.

Flexible bronchoscopy with or without biopsy, endotracheal intubation and tympanostomy tube insertion are not now generally regarded as indications for prophylaxis.

ANTIBIOTIC PROPHYLAXIS FOR DENTAL AND RESPIRATORY TRACT PROCEDURES

1. **Standard regimen** (Note 1)
   - Amoxycillin 2.0 g PO one hour before procedure
   - Amoxycillin 1.0 g PO six hours later
2. **Allergy to the penicillins**, recent penicillin treatment, long term penicillin prophylaxis (Note 2)
   - Erythromycin stearate 1.0 g PO 1-2 hours before procedure
   - Erythromycin stearate 0.5 g PO six hours later **or**
   - Clindamycin 300 mg PO one hour before procedure
   - Clindamycin 150 mg PO six hours later
3. **Intravenous regimen** for patients unable to take oral medications (e.g. under general anaesthesia)
   - Amoxycillin 1.0 g IV immediately before procedure
   - Amoxycillin 1.0 g PO or IV six hours later

   For those **allergic to the penicillins**, recent penicillin treatment, long term penicillin prophylaxis
   - Vancomycin 1.0 g IV infused over one hour before procedure **or**
   - Clindamycin 300 mg IV immediately before procedure
   - Clindamycin 150 mg PO or IV six hours later
4. **Preferred option for high risk patients** (Note 3)
   - Amoxycillin 1.0 g IV immediately before procedure
   - In the presence of infection, gentamicin 120 mg IV may be added
   - Amoxycillin 1.0 g IV or PO six hours later

   For those **allergic to the penicillins**, recent penicillin treatment, long term penicillin prophylaxis
   - Vancomycin or clindamycin as in (3) above.

Note 1: Oral penicillin VK is an alternative to oral amoxycillin. Regimens using parenteral benzylpenicillin are an alternative to all parenteral amoxycillin recommendations.

Note 2: Recent penicillin treatment means more than one dose of any penicillin or cephalosporin within the last month.

Note 3: High risk patients are those with prosthetic, heterograft or homograft valves; those with severe lesions of their own mitral or aortic valves; those with a history of bacterial endocarditis.
The standard oral regimen (1) may be used in such patients, but is not the preferred option.

GASTROINTESTINAL AND GENITOURINARY TRACT PROCEDURES

- Bacteraemia often accompanies surgery or instrumentation of the genito-urinary or gastrointestinal tracts and endocarditis may result. Prophylaxis is directed against enterococci.
- Prophylaxis is recommended for the following: cystoscopy, urethral dilatation, prostatic surgery, insertion or withdrawal of urethral catheters in the presence of suspected or proven infection, urinary tract surgery, vaginal

hysterectomy, gall bladder surgery, colonic surgery, oesophageal dilatation, sclerotherapy of oesophageal varices and incision or drainage of infected tissue.

- Prophylaxis is not now generally recommended for upper gastrointestinal tract endoscopy, colonoscopy or sigmoidoscopy, either with or without biopsy, or for barium enema, i.e., these are low risk procedures.
- Prophylaxis is not usually necessary for obstetric or gynaecological procedures such as uncomplicated childbirth, forceps delivery, manual removal of placenta, uterine dilatation and curettage, caesarian section, therapeutic abortion, sterilization procedures or intrauterine device insertion or removal i.e. these are low risk procedures.
- In high risk patients or where infection is suspected, prophylaxis is recommended for the procedures listed as in sections above.

SPECIFIC ANTIBIOTIC REGIMENS FOR GASTROINTESTINAL AND GENTIOURINARY TRACT PROCEDURES

- **Standard regimen (includes high risk patients)** - amoxycillin 1.0 g IV plus gentamicin 120 mg IV immediately before the procedure followed by amoxycillin 1.0 g IV (or oral) six hours later.

- **Alternative oral regimen for minor or repetitive procedures in low risk patients** - amoxycillin 3.0 g PO one hour before procedure followed by amoxycillin 1.5 g PO six hours later.

- **For patients with allergy to the penicillins,** recent penicillin treatment, long term penicillin prophylaxis - vancomycin 1.0 g IV infused over one hour before procedure plus gentamicin 120 mg IV before procedure. No repeat dose is necessary.

ENDOCRINOLOGY / METABOLIC DISORDERS

ADRENAL INSUFFICIENCY

CAUSES

- Primary adrenal failure
  - Autoimmune.
  - Tuberculosis.
  - Haemorrhage.
- Secondary
  - Pituitary failure.
  - Adrenal suppression - steroids stopped or not increased at time of stress. May occur with topical or aerosol administration.
  - Aminoglutethimide.

INVESTIGATIONS

- Biochemistry - low Na, high K, high urea, low/ish glucose (may look like inappropriate ADH).
- CBC + diff - may be eosinophilia and neutropenia.
- CXR - cardiac size (may be decreased).
- Draw blood for cortisol, ACTH, renin and aldosterone (25 ml into EDTA tubes). Contact Biochemistry for immediate 4°C centrifugation and freezing. Contact Steroid Laboratory re assays after discussion with Endocrine Department.
- ECG - to exclude a silent myocardial infarction since this may present as unexplained hypotension.
- Arterial blood gases - for unrecognised acidosis.
- 24 hour urine for sodium and potassium excretion and creatinine clearance.

TREATMENT

- Fluid replacement with 0.9S to restore arterial and venous pressure. In critical situations a CVP line may be needed. May require 1 litre or more over 2 hours. May require 5%-25% dextrose to raise glucose levels. Amount of potassium infused (if any) based on plasma levels.
- Hydrocortisone 100-200 mg IV then 50 mg 12 hourly. Continue at this level until the patient's condition has stabilised, then reduce daily dose of hydrocortisone gradually (e.g. daily dose rapidly reduced from 100-200 mg to 100, 50-75, 30-50 mg/day on successive days depending on metabolic status) then down to a long term maintenance level of 20-30 mg per day. 9α-fludrocortisone may also be required once hydrocortisone dose is approximately 50 mg/day.
- Diagnostic work-up should be completed in consultation with the Endocrine Department.

- Steroid induced adrenal suppression may be managed by prednisone 30-60 mg per day which is then rapidly reduced to 7.5 mg daily over 10-14 days. Then reduce by 1 mg each week and hold at 3 mg per day. Leave at this level until Synacthen test shows adequate increment. May need to decrease to 2 mg daily if no increment seen after 2 months. The tailing off process of steroid medication will need to be slowed if the patient is unwell.
- Antibiotics will be needed acutely if there is any suggestion of sepsis. Take appropriate baseline cultures. If no particular organism is suspected then broad spectrum therapy, e.g. IV cefuroxime and gentamicin.

MANAGEMENT OF STRESS SITUATIONS IN PATIENTS WITH STEROID DEFICIENCY ON MAINTENANCE STEROIDS

- All patients should have a steroid card and medic alert.
- Minor procedures, e.g. dental extraction under local anaesthetic - one should double the daily dose.
- Major surgery or stress.
  - 100 mg hydrocortisone IM or IV ½ hour preoperatively. Then 100 mg over the 4 hours of surgery, thereafter 50 mg q6h and reduce as above.

DIABETES - GENERAL COMMENTS

Unstable blood glucose - patients with diabetes, who are admitted to hospital for other reasons, often experience difficulty with their blood glucose control. Factors which tend to elevate blood sugars include stress (both physical and psychological) and immobility. Changes in their usual diet will also affect blood glucose and if this occurs they should be seen by a Dietitian. Any sustained increase in blood glucose will lead to a delay in wound healing and the slow resolution of infection.

Some patients who were previously well controlled on diet and tablets as an outpatient, may require insulin on a temporary basis during their hospital stay. Most patients on insulin will require a temporary adjustment to their insulin dose if they are in hospital more than 48 hours.

Patient autonomy - most patients on insulin are competent at diabetes self care, including self-adjustment of insulin. Maintenance of this autonomy should be encouraged during hospitalization. A common complaint from patients is that they were not given the opportunity to suggest changes to their insulin dose, whilst in hospital. This can be particularly frustrating, when they have unique personal experience regarding their own insulin requirements and are often better at predicting their insulin requirements, than the medical staff. **Changes in insulin dose should therefore be made in consultation with the patient**.

Ward capillary blood glucose testing - many patients require frequent testing when admitted acutely or during the peri-operative period. Once the patient's

condition has stabilized, four times a day testing is usually adequate. Patients on insulin should be tested before main meals and at bed-time. If you order 4 hourly blood testing, results will be difficult to interpret, as the relationship of test results to meal times will be highly variable.

The frequency of capillary glucose testing in inpatients with non-insulin dependent diabetes needs to be individualized, depending on the medical question you are trying to answer. For example, if you are wondering whether a patient on maximum oral hypoglycaemic agents needs to be on insulin, you will require regular tests, both before and two hours after meals. If the patient is well controlled on diet only, fasting blood glucose tests alone will be sufficient.

Pre-discharge planning - this is particularly important in insulin dependent diabetes and should be undertaken at least 24 hours before the patient leaves hospital. Questions you should ask include:

- Does the patient need to go back onto their usual insulin dose at discharge, particularly if they are resuming their usual eating and activity patterns?
- Have you discussed a plan of action, if blood glucose results do not stabilize, after discharge?
- Have your prescribed the right sort of insulin? (Remember, pen injector cartridges come in two sizes - 1.5 ml and 3 ml, and patients using syringes should be prescribed 10 ml vials of insulin).
- Have you prescribed insulin syringes, if required? (You can only prescribe insulin syringes together with 10 ml vials of insulin, and can prescribe a maximum of 26 syringes per three months insulin prescription).
- Have you prescribed the right sort of glucose test strips for the patient's blood glucose meter? (At the time of writing, there are nine different strips available - the wrong strip in the wrong meter may produce incorrect blood glucose results, with potentially dangerous consequences).

Contact the Diabetes Centre if you require further advice about diabetes inpatient management, from either the Diabetes Physician or Diabetes Nurse Educator.

DIABETIC KETOACIDOSIS (DKA)

DKA is associated with significant mortality, particularly in the older patient with an underlying acute medical condition precipitating ketoacidosis. Deaths from DKA in young, otherwise healthy patients, are often associated with inadequate electrolyte (particularly potassium) and fluid replacement. Cerebral oedema may complicate childhood DKA. A deterioration in the level of consciousness, despite improving biochemistry, suggests this complication.

Some young patients with insulin dependent diabetes present with hyperglycaemia, ketonuria but no acidosis (normal pH or bicarbonate) and can be safely managed as a Day Case. Make sure the patient does not have

hyperosmolar non ketotic diabetic coma (**see page 61**). **Discuss the urgent management of these patients with the Diabetes Physician on call.**

COMMON CAUSES FOR DKA

- Insulin withdrawal or reduction.
- Myocardial infarction, stroke, trauma or other medical stress.
- Infection such as pneumonia, gastroenteritis, influenza, UTI, meningitis.
- Drugs (e.g. steroids, thiazides).

BASELINE INVESTIGATIONS

- K, Na, urea and creatinine. (Creatinine may be falsely elevated if ketones are high due to interference with the assay).
- Blood and urine ketones.
- Arterial blood gases (venous bicarbonate may be sufficient if patient has mild DKA only).
- CBC and diff.
- Cultures of blood and urine and any other material as indicated.
- CXR.
- ECG.

TREATMENT

If the patient is severely ill (pH <7.1 or obtunded or has DKA complicated by other medical conditions) admit to the Intensive Care Unit.

Monitor the patient - all patients requiring intravenous insulin need a **flow chart** documenting potassium, fluid balance, insulin dose, blood glucose, pH and/or bicarbonate. If the patient is severely ill, Na, K and glucose should be checked hourly for the first 4 hours then at 4 hourly intervals, over the next 12 hours. Vital signs should also be monitored in severely ill patients (e.g. pulse, temperature, respiration, blood pressure, weight and mental status).

IV fluids - 0.9S is the usual first choice of rehydrating fluid. The amount and speed of fluid replacement will be dictated by the clinical findings (e.g. degree of weight loss at presentation, hypotension, JVP or CVP, concomitant heart failure). A common replacement regimen in patients without heart failure is one litre 0.9S over the first hour, then 500 ml over the second hour, then 500 ml 2-4 hourly thereafter, adjusted according to urine output and other clinical findings. Consider 0.45S if Na >150 mmol/L. When the blood glucose approaches 15 mmol/L change to D5W. See below for K replacement. Additional insulin and K may be given through 3-way connector.

Insulin - Give a stat bolus dose of 10 units IV Actrapid (soluble insulin). Add 500 units (IU) insulin to 500 ml of 0.9S. Flush 50 ml of this solution through the tubing to saturate insulin binding sites on the plastic tubing. Run insulin infusion

at 6-10 ml per hour ('piggy backed' together with IV fluids) via infusion pump. Increase or decrease according to rate of blood glucose fall. Aim to normalise glucose over 24 hours, no faster. When the glucose reaches 15 mmol/L start to slow the insulin infusion to 1-2 units per hour and change IV fluid replacement to D5W.

Potassium replacement - patients with DKA are depleted in total body potassium despite the fact that most have a normal, or even elevated, serum potassium at presentation. Unless the patient is anuric, K replacement will therefore be required within two hours of commencing insulin, or sooner if baseline K is low:

SERUM POTASSIUM (mmol/L)	POTASSIUM VIA INFUSION PUMP (mmol/kg/hour)
<3	0.5
3-4	0.4
4-5	0.3
5-6	0.1-0.2
6	withhold K replacement

For example, a 70 kg patient with a K between 3-4 mmol/L will require about 30 mmol K per hour. The K can be added to the IV fluid replacement bag. For safety reasons K replacement must be given via an infusion pump.

Changing from IV to subcutaneous insulin - when acidosis has been corrected and the patient is eating well, consider discontinuing IV fluids and IV insulin. The half-life of IV insulin is short and there should be a 2 hour overlap of IV and SC insulin. If the patient has newly diagnosed IDDM, estimate the likely SC insulin requirements from the previous 24 hours IV insulin requirement. All patients changing over to SC insulin should be commenced on an insulin regimen which includes a long-acting component, for example twice daily premixed insulin before breakfast and tea.

Use of sliding scale Actrapid on its own is inappropriate and is likely to delay stabilization of diabetes.

Additional notes

- Do not strive for rapid correction of hyperglycaemia - the underlying principle is to avoid hypoglycaemia and correct salt and water loss.
- DKA in young patients may be complicated by cerebral oedema.
- If gastric stasis is present and you are concerned about aspiration of gastric contents, consider inserting a nasogastric tube.
- Consider IV bicarbonate only if pH is very low (<7.0) and then give enough to raise the pH to 7.1 e.g. try giving 1 mmol $NaHCO_3$ per kg and review in one hour.

- Always refer the patient to the Diabetes Centre or Diabetes Physician to assess overall diabetes management.

NON-KETOTIC HYPEROSMOLAR DIABETIC COMA

Differentiated from patients with DKA by:

- Absence of significant ketosis.
- High blood glucose and plasma osmolality.
- Profound dehydration.

These patients are often drowsy, confused or comatose, due to cerebral intracellular dehydration. Hyperosmolar coma tends to occur in older patients with non insulin dependent diabetes. Precipitating causes include infection, diuretic therapy and myocardial infarction. Hyperosmolar coma is associated with mortality rates of up to 40%.

INVESTIGATIONS - as for DKA but include plasma osmolality.

MANAGEMENT

The key to adequate management is appropriate fluid replacement. The correct choice of fluid replacement and correct speed of administration are critical.

The following management plan will obviously need to be tailored to the individual patient, and will depend on factors such as degree of dehydration, urine output, serial serum Na readings and concomitant medical problems such as underlying cardiac disease. If management does not result in a steady improvement in the level of consciousness, serum sodium and osmolality urgent specialist review is indicated.

- Consider admission to ICU. Many patients will benefit from monitoring of CVP (this is likely to be of particular benefit in patients with congestive cardiac failure or renal insufficiency).
- Flow chart, plotting fluid replacement, urine output and serum glucose and electrolytes. Venous blood samples should be measured two hourly for the first four hours then at least every four hours thereafter.
- The flow chart should also document level of consciousness. With adequate fluid and electrolyte replacement, this should gradually improve.

Fluid and electrolyte replacement

- 1 L 0.9S over 30-60 minutes. Consider a second litre of 0.9S only if the patient remains hypotensive. **Thereafter all fluids should be hypotonic.** 0.45S is available on the acute admitting wards and in ICU.

- 2-3 L 0.45S at 500 ml/hr. The rate of subsequent 0.45S infusions will depend on the patient's clinical state.
- Add D5W to the 0.45S when the blood glucose is <15 mmol/L and Na <150 mmol/L. If glucose is <15 mmol/L and Na >150 mmol/L, change to D4S.
- K replacement will probably not be needed initially, but, after a few hours rehydration, K may be needed at a rate of 10-20 mmol/hr. (Total body K deficiency will be less marked than in diabetic ketoacidosis).

IV insulin replacement

- Infuse at a rate of 6-10 units/hr, initially.
- Once the glucose has reached 15 mmol/L, decrease the rate to 1-2 units/hr and once the patient is fully rehydrated (which may take >36 hours), consider instituting SC insulin, as for the management of diabetic ketoacidosis. Longer term, the patient may manage on diet or diet plus oral agents. Discuss this with the Diabetes Physician.

Prevention of venous thrombosis

These severely dehydrated, comatose patients are at particular risk of DVT. Consider heparin 5000U BD, SC.

PERIOPERATIVE MANAGEMENT OF DIABETES

This will usually be supervised by the anaesthetist. If not, here are some suggested regimens:

- If on oral agents omit drug. Restart when eating for at least 12 hours.
- If on insulin, omit morning subcutaneous insulin. Start infusion, using a pump, of 1L D5W with 10 mmol/L KCl, at 100 ml/hr, plus an insulin infusion at 1 U/hr. Measure blood glucose 4 hourly pre and post operatively and every hour during surgery. Do not change infusion rate if glucose remains between 6.5-10 mmol/L. Increase infusion rate to 1.5 units/hr if glucose >10.0 mmol/L. Decrease infusion rate to 0.5 units/hr if glucose <6.5 mmol/L.

The above regimen is suitable for most patients but those on high daily doses of insulin may require more IV insulin than above. Also remember that the half-life of IV insulin is measured in minutes and discontinuation of IV insulin, in the absence of SC insulin, will result in a rapid rise in blood glucose.

A high blood glucose for a brief period does not matter but hypoglycaemia does. Give the patient's usual insulin dose when they have been eating satisfactorily for 12 hours and overlap IV and SC insulin by 2 hours.

MANAGEMENT OF THE NEWLY DIAGNOSED PATIENT WITH DIABETES

A diagnosis may be made on random or fasting blood glucose. An oral glucose tolerance test is rarely needed. A random blood glucose above 11.1 mmol/L, and a fasting blood glucose above 7.8 mmol/L, are diagnostic of diabetes. Intermediate levels are likely to represent impaired glucose tolerance, which should be treated with lifestyle management (exercise and diet) because of the increased risk of cardiovascular complications.

The non-obese patient - if the patient has no ketones in the urine, they may safely be given a trial of diet (plus a sulphonylurea if presenting blood sugars are very high), rather than insulin. The thinner and younger the patient at presentation, and the higher the presenting blood sugars, the more likely the patient is to require insulin in the longer term. Sulphonylureas can cause hypoglycaemia and should be used with caution in the elderly and in patients with renal impairment.

The obese patient - weight reduction and exercise are the cornerstones of management. If the body mass index is >28 kg/m^2, patients often do better on metformin, rather than a sulphonylurea. BMI = patient's Wt (kg) divided by the square of the height (m). Ideal BMI = 20-25 kg/m^2. The risk of developing lactic acidosis on metformin is increased in the presence of renal, cardiac or liver disease and metformin should also be used with caution in the elderly.

Diabetes nurse educators are available to help with the practical management of patients with diabetes (contact via the Diabetes Centre). They will help with home blood glucose monitoring, selection of an appropriate blood glucose meter and also insulin injection technique, including the use of pen injector devices. They can facilitate discharge arrangements with the diabetes domiciliary nurse and also liaise with the Diabetes Centre.

HYPOGLYCAEMIA

This is common in patients on insulin or a sulphonylurea. If the patient is unconscious deal with any ABC difficulties (airways, breathing, circulation), before confirming the diagnosis with a bedside finger prick blood test and also a laboratory blood glucose. Get blood for these tests before giving 50 ml 50% IV dextrose. When the patient has regained consciousness, give the patient food (long-acting carbohydrate). If the patient is hypoglycaemic due to a long-acting sulphonylurea, the hypoglycaemia may recur and management with a 10% dextrose drip is required. If the patient is hypoglycaemic but conscious, and can be persuaded to drink, oral glucose is appropriate but this should also be followed up by food.

Once the patient has recovered, consider precipitating causes (alcohol, dose of insulin or sulphonylurea too high). If the precipitating cause is found to be

related to diabetes self-care, consider referral to the Diabetes Centre for further patient education.

THE SYNDROME OF INAPPROPRIATE ADH SECRETION (SIADH)

CAUSES

- Tumours - particularly bronchogenic carcinoma.
- Sepsis.
- Drugs:
  - Vincristine / vinblastine.
  - Clofibrate.
  - NSAID.
  - Chlorpropamide.
  - Carbamazepine.
  - Tricyclics.

May not be easy to distinguish from the sodium and potassium loss and water replacement often associated with diuretics. Potassium deficiency contributes and may need correction.

INVESTIGATIONS

Diagnosis established by the following tests associated with the appropriate clinical picture. (Includes normal renal function).

- Plasma osmolality < urine osmolality. (Normal plasma osmolality 285-299 mOsm/kg).
- Low plasma sodium and chloride.
- Urinary sodium usually > 30-40 mmol/L.
- ADH levels. Take 10 ml blood into EDTA. Put on ice, centrifuge at 4°C and freeze. If ADH level is deemed necessary then Endocrinology will need to be contacted.

MANAGEMENT (always necessary if plasma Na <125 mmol/L).

- Water restriction 500-1000 ml over 24 hours.
- Subsequently relax fluid restriction in response to improved plasma osmolality and serum Na levels.
- CXR (to exclude tumour).
- Withdraw inappropriate drugs (may include diuretics).
- Treat sepsis.
- If treatment is unsuccessful consider drugs acting on ADH release / action (e.g. demeclocycline).
- If severe and symptomatic may need hypertonic saline ± frusemide.

HYPONATRAEMIA NOT DUE TO SIADH

- Consider history (volume depletion, diuretics etc).

HYPONATRAEMIA WITH NORMAL ECF

- Syndrome of inappropriate secretion of SIADH **(see above).**
- Antidiuretic drugs.
- Cortisol deficiency.
- Hypothyroidism.
- Severe potassium depletion.
- Chronic renal disease.

MANAGEMENT - treat underlying cause and treat as for SIADH as appropriate.

HYPONATRAEMIA WITH LOW ECF

- Renal:
  - Cortisol deficiency.
  - Diuretics.
  - Chronic renal disease.
- Extrarenal:
  - Gastrointestinal losses.
  - Burns.
  - "Third space" losses.

MANAGEMENT

- Treat with 0.9S (sometimes up to 5-10% body weight large volume may be required). Bicarbonate and potassium may also be required. Aim to raise plasma Na to 125 mmol/L over 48-72 hours, depending upon severity of illness, symptoms etc.

HYPONATRAEMIA WITH RAISED ECF

- Acute renal failure.
- Cardiac failure.
- Liver disease.
- Nephrotic syndrome.
- Inappropriate IV fluids.

MANAGEMENT

- If clinically important restrict fluid to:
  1000 ml/day if Na <130 mmol/L
  500 ml/day if Na <120 mmol/L

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HYPERCALCAEMIA

Note:
If marked this is a medical emergency.

CAUSES

- Malignant disease - myeloma, carcinoma (e.g. breast, lung, kidney).
- Primary hyperparathyroidism.
- Milk/alkali syndrome.
- Thiazide diuretics.
- Sarcoidosis.
- Thyrotoxicosis.
- Vitamin D intoxication.
- Bed rest in patients with active Paget's disease/malignancy.
- Lithium treatment.
- Cortisol deficiency.

SYMPTOMS

May be none. Common symptoms - nausea, vomiting, constipation, abdominal pain, thirst, polyuria, confusion, coma.

INVESTIGATIONS

Note:
If hypercalcaemia occurs during the course of a documented malignancy e.g myeloma, then only a few investigations will be needed, e.g. CBC + diff, Na, K, urea, creatinine, Bili, Alb, AST, GGT and alk. phos. Otherwise, the tests listed below should be considered and the Endocrinologists consulted.

- Na, K, Ca, Mg, PO_4, alk. phos., total protein, Alb, creatinine, Cl (the ratio of chloride:phosphate may be helpful - >100:1 suggests primary hyperparathyroidism; <80:1 strongly favours malignancy).
- Ionised calcium or ultrafilterable calcium if hyperparathyroidism suspected (10 ml plain tube - Biochemistry).
- CXR (lung cancer, sarcoidosis).
- CBC + diff and ESR.
- Thyroid function tests.
- Serum protein electrophoresis, urinary Bence Jones protein screen (multiple myeloma) - may require bone marrow aspirate.
- X-ray painful bones (metastases, myeloma).
- X-ray hands (best site for hyperparathyroidism).
- Ultrasound examination of kidneys + plain abdominal X-ray (calculi).
- 24 hour urine collection - calcium excretion, creatinine clearance.
- Parathyroid hormone levels (contact Endocrine Laboratory). Draw samples before giving hypocalcaemic drugs.

- 25 OH and 1-25 diOH vitamin D assay (contact Steroid Laboratory) and PTH related peptide if malignancy suspected.

  Note:

  Observed calcium levels will need to be corrected if there are major alterations in plasma albumin levels.

Correction formula:

Corrected calcium = observed calcium + (44 - albumin g/L) x 0.025 mmol/L.

MANAGEMENT

- This will depend on the severity and clinical context. Minor elevations of serum calcium will usually not require additional therapy apart from ensuring adequate hydration, monitoring any rise associated with immobilization, and establishing its cause. A marked elevation is a medical emergency. If hypercalcaemia is causing significant symptoms and active treatment is deemed appropriate then the following is recommended.
- Rehydration - this is the cornerstone of management:
  - Correct dehydration with 4-5 litres in 24 hours orally and IV. Monitor closely to avoid fluid overload. Start with 2 litres 0.9S over 2 hours then 1 litres 0.9S 6-8 hourly and reassess at 18-24 hours. Potassium supplements 10-20 mmol KCl per 500 ml may be required.
- Glucocorticoids:
  - Prednisone 20-60 mg daily may provide effective initial treatment especially in malignancy or hypervitaminosis D, and in sarcoidosis. If hypercalcaemia is the sole abnormality requiring treatment in sarcoidosis, control may be maintained by low doses of prednisone and later possible switch to cellulose phosphate.
- APD (aminopropylidine diphosphonate) if prednisone fails or deemed inappropriate.
  - 30 mg in 0.5 litre 0.9S IV over 4 hours. Ensure no extravasation (irritant to tissues). Fever may occur. Plasma calcium falls progressively with nadir at 3-5 days. Repeat doses may be necessary.
- Stop thiazides.
- Calcitonin will lower plasma calcium temporarily.
- Oral phosphates may be helpful. Do not give phosphates IV.
- Hypercalcaemic patients who have or may have an underlying malignancy, such as myeloma, should be referred to a Haematologist or Oncologist as soon as possible.
- Parathyroid surgery may be indicated in primary hyperparathyroidism.

HYPOCALCAEMIA

Check albumin and if necessary adjust the calcium level **(see above)**. If <2.0 mmol/L needs investigation. Symptoms may not be prominent if problem is long standing.

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CAUSES

- Hypoparathyroidism or resistance to parathyroid hormone.
- Renal failure.
- Vitamin D deficiency (fasting morning phosphate level usually also low).
- Low magnesium states.
- Pancreatitis, rhabdomyolysis.

INVESTIGATIONS

(Check Chovstek and Trousseau signs, and history of fits, tetany etc).

- Fasting morning Ca, PO_4, creatinine, Mg, and parathyroid hormone levels.
- May also need check for 25 hydroxy - Vitamin D level and assessment for osteomalacia etc.

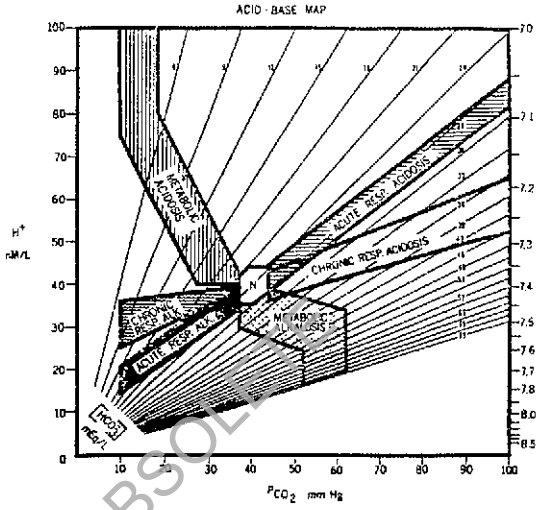
MANAGEMENT

- If severely symptomatic.:-
  - Give calcium gluconate IV e.g. 10 ml of 10% solution as bolus over 2 minutes.
  - Start oral calcium e.g. 1000-2000 mg elemental calcium daily.
  - In severe cases repeated IV calcium gluconate by continuous IV infusion - e.g. 2-3 ampoules in 500 ml dextrose over 4-6 hours (each ampoule of 10% solution calcium gluconate contains 90 mg elemental calcium). Dose and rate is monitored by repeated checks of serum calcium.
  - Consider magnesium replacement if hypomagnesaemic.
  - If the cause is hypoparathyroidism, also start 1α-25 dihydroxy Vitamin D (calcitriol) e.g. 0.25 mcg/day and monitor daily serum calcium. Consult Endocrinologists.

ACIDOSIS / ALKALOSIS

The diagram shown below allows easy interpretation of most clinical disorders of acid/base balance.

See also the similar diagram on **page 158**. See which diagram you find easiest to use.



A diagnosis of **respiratory acid/base disturbance** reflecting abnormal pulmonary function should be investigated and managed as discussed in the respiratory section. In general, respiratory acidosis is managed by increasing effective ventilation thereby reducing $PaCO_2$.

The diagnosis of metabolic acidosis may be more difficult. Acidosis may be associated with an increased anion gap which is of some value in diagnosis. The gap may be increased by ketones, lactate, some poisonings e.g. salicylates, and in late renal failure.

ANION GAP = (Plasma Na) - (Plasma Cl + HCO_3)

Normal - less than 15 mmol/L.

CAUSES OF METABOLIC ACIDOSIS

- Diabetic ketoacidosis.
- Renal acidosis:
  - Renal failure.
  - Renal tubular acidosis.
- Lactic acidosis (biguanide induced, severe shock, post alcoholic binge).

- Formic acid (in methyl alcohol poisoning may need treatment with ethanol urgently).
- Ethylene glycol poisoning.
- Salicylate poisoning.

INVESTIGATIONS

- Na, K, Ca, PO_4
- Arterial blood gases.
- Toxicology - as appropriate.
- Lactate level if lactic acidosis suspected.

TREATMENT

- TREAT UNDERLYING CAUSE.
- Recent evidence suggests that in most forms of metabolic acidosis, $NaHCO_3$ offers no benefit and may even be harmful, although some recommend using alkalinizing agents for pH less than 6.9.
- If $NaHCO_3$ is used give 1 mmol/kg and review pH in one hour. Do not overcorrect, aim for pH of 7.1.
- Specific indications for $NaHCO_3$ include methanol and tricyclic antidepressant poisoning - maintaining a normal pH probably reduces toxicity.

GASTROENTEROLOGY

HAEMATEMESIS

CAUSES

- Ulceration/oesophagitis.
- Cancer.
- Mallory Weiss tear (after retching).
- Varices (including gastric) (note: high mortality).
- Peptic ulceration (ask about NSAID + aspirin use).
- Acute stress erosions (shock, sepsis, NSAID).
- Abnormal haemostasis.
- Swallowed blood.

MANAGEMENT

- Assess degree of blood loss:
  - History often unreliable.
  - Resting tachycardia.
  - Hypotension or postural drop >15 mmHg.
- Stabilize patient and monitor:
  - Give 0.9S IV, then blood (use volume expanders e.g. SPPS if necessary).
  - CBC + diff. Crossmatch 6 units resuspended cells then whole blood as required. Give group O Rh-ve blood if required urgently.
  - Check Na, K, creatinine.
  - Check coagulation profile.

Resuscitation takes precedence over diagnostic investigations. Gastroscopy may be delayed 12-24 hours without significantly reducing diagnostic value unless varices suspected. May consider therapeutic intervention early (injection of bleeding ulcer/varix). The rapidly bleeding patient may require immediate surgery without other investigation, unless varices suspected.

THERAPY

- If VARICES are suspected:
  - Urgent sclerotherapy indicated.
    If unavailable:
  - Sengstaken-Blakemore tube and transfer to ICU. (Consider endotracheal intubation first if impaired level of consciousness).
  - Somatostatin analogues (Octreotide) 25-50 mcg/hr in 0.9S or D5W **or** IV vasopressin 20 units in 100 ml D5W over 20 minutes. This can be repeated 4 hourly, or given as 0.2 units/kg/hr infusion (± IV nitroglycerin to reduce side effects). Use with caution in the elderly and in patients with ischaemic heart disease.

- ACUTE STRESS ULCERATION:
  - Liquid antacids 30 ml PO (1-2 hourly).
  - Correct underlying condition.

  Note:

  H_2 receptor antagonists of uncertain value.
- PEPTIC ULCERATION:

  H_2 receptor antagonists are effective when given orally to heal ulcers. They have no effect on acute bleeding, even IV.

SURGICAL CONSULTATION

- Required urgently for:-
  - All bleeding where >3 units of blood required.
  - Continuing bleeding.
  - If perforation suspected.

  Note:

  High pulse rate should raise the possibility of continued bleeding.

Early referral is recommended for patients over 60 years as they withstand bleeding and surgery poorly.

VOMITING

CAUSES

- Visceral:
  - Organic disease of oesophagus/stomach/bowel.
  - Pseudo obstruction.
  - Mechanical - bowel obstruction/gastric stasis.
  - Acute abdomen.
  - Liver metastases.
- Toxic/metabolic:
  - Acute febrile illness/sepsis.
  - Ketoacidosis/uraemia/hepatic failure etc.
  - Drugs (e.g. digoxin, theophyllines, cytotoxics).
- Neurological:
  - Vestibular/middle ear.
  - Increased intracranial pressure.
  - Cerebrovascular accident (especially brain stem).
- Other:
  - Pregnancy.
  - Excess smoking, alcohol and other addictive drugs.

COMPLICATIONS

- Aspiration pneumonia.
- Haematemesis (Mallory Weiss tear).

- Oesophageal perforation (pain is a prominent feature).
- Malnutrition/dehydration.
- Electrolyte/volume depletion (hypokalaemic alkalosis).

TREATMENT

Determine and treat the **UNDERLYING CAUSE**. If antiemetics are indicated:

- Dopamine antagonists:
  - Metoclopramide PO, IM, IV, PR 10 mg q8h but higher doses may be required.
  - Domperidone PO 10-20 mg QID.
- Peripheral Cholinergics
  - Cisapride 10 mg PO TID.
- Phenothiazines:
  - Prochlorperazine PO, IV, IM, PR 12.5 mg q8h.
  - Chlorpromazine PO, IM, IV 25 mg q8h.
- Anticholinergics:
  - Scopolamine transdermal patch every 2-3 days.
  - Promethazine PO, IV 25 mg q8h.
- Sedatives and hypnotics may be used:

ACUTE DIARRHOEA (e.g. less than 2 weeks).

HISTORY

- Try to assess whether this has an infectious basis.
- Initial clinical assessment is important. Include severity of diarrhoea, passage of bloody stool, any upper GI symptoms, history of recent surgery, radiation, drugs (especially antibiotics) and overseas travel or infectious contacts. Also record the food eaten and occupation.

EXAMINATION

- Look for signs of dehydration, sepsis, abdominal tenderness and rigidity.
- Digital rectal examination and sigmoidoscopy (biopsy may be required).

INVESTIGATIONS

- An urgent plain abdominal x-ray may be required.
- CBC + diff, urea, creatinine, Na, K.
- Blood cultures if patient is febrile or has been abroad.
- Stool examination - a freshly collected stool specimen should be examined for:

Microscopy	Leucocytes and red cells. Parasites (especially cryptosporidia in immunosuppressed).

Bacteria	Salmonella, Shigella, Yersinia, Aeromonas, Campylobacter and Plesiomonas are routinely cultured at Christchurch. (Toxic forms of E.coli can be cultured on request).
Viruses	Rotavirus is looked for in paediatric samples and other viruses will be tested on request.
Cl. difficile toxin assay	Available on liquid stool if appropriate. Culture not routinely done
Parasites	3 faecal samples on separate days in PVA fixative for parasite examination.

MANAGEMENT - GENERAL

- Enteric isolation procedures required if infection suspected - (follow Hospital Protocol).
- IV fluids may be required. Remember faecal losses of electrolytes may be very high. 100-120 mmol/L Na and 5-15 mmmol/L K may be lost per litre of stool. An adult may lose more than 2-3 L of fluid per day.
- Avoid constipating drugs (especially in children) as these may prolong symptoms.
- Antimicrobials are not indicated for the majority of infectious diarrhoeas.
- **Specific infections:**
  - Salmonella/Shigella/Campylobacter are usually self limiting and antibiotics should only be used when illness is severe with systemic upset/septicaemia. These are notifiable diseases (**see Appendix 1**).
  - Pseudomembranous colitis; always suspect when antibiotics have been taken within last few weeks. Sigmoidoscopy may sometimes be diagnostic but often is not necessary. If suspected check for clostridium difficile toxin and treat. Treatment of choice metronidazole 400 mg q8h PO or IV. Alternatives - vancomycin 125 mg PO, QID or cholestyramine 9 g PO, TID before meals.
  - HIV - always suspect in at risk populations. Almost all have some gut manifestation either directly due to HIV or secondary to CMV, Cryptosporidia, Giardia, Mycobacterium avium intracellularae, Kaposi's sarcoma, lymphoma etc. (**see section on AIDS**).
  - Amoebic dysentery - metronidazole 800 mg PO, TID for 10 days.
- **Acute inflammatory bowel disease is suspected.**
  - Toxic megacolon (diameter >5.5 cm) should be considered in any person with inflammatory bowel disease, systemic toxicity and increasing diarrhoea. Requires daily plain abdominal x-ray and review with early gastroenterology and surgical referral.
  - Steroids are drugs of choice in acute situation. Give IV hydrocortisone 100 mg q6h then prednisone 30-60 mg/day PO.
  - Gastroenterology/Surgical consult if not responding in 48-72 hours.
  - Sulphasalazine 1 g QID, PO or Olsalazine or Mesalazine commencing at 250 mg PO/day may be of benefit pending diagnosis in less severe attacks.

- IV fluids, nutrition and antibiotics may be needed. Always consider other causes of diarrhoea and/or bleeding.

  Note:

  - Other causes of diarrhoea include carcinoma, ischaemic colitis, diverticulitis, and constipation with overflow.
  - Laxative abuse may cause dehydration, muscular weakness and hypokalaemia. Consider this in chronic diarrhoea.

TREATMENT OF CONSTIPATION

GENERAL MEASURES

- Look for treatable causes - pregnancy, cancer, diabetes, hypothyroidism, hypercalcaemia.
- Avoid constipating drugs (e.g. codeine, opiates, tricyclics, anticholinergics, calcium channel blockers, aluminium hydroxide).
- Dietary control e.g. increase fluid, fibre, fruit.

SPECIFIC MEASURES

- Bulking agents (e.g. metamucil, granocol, isogel). If no response then consider:
  - Faecal softeners (e.g. coloxyl).
  - Colonic stimulants (e.g. bisacodyl, senna) useful in acute constipation. Side effects include cramps, electrolyte imbalance, melanosis coli, and "cathartic colon" and should not be used long term.
  - Lactulose has an osmotic effect but may cause excess flatulence.
  - Bowel washout with Golytely may be needed.
  - Glycerine suppositories.

JAUNDICE

Is the urine positive for bilirubin ?

If **no** implies unconjugated hyperbilirubinaemia. Diagnostic possibilities are prehepatic disease such as haemolysis, Gilbert's Syndrome etc.

If **yes** conjugated bilirubin is present. Causes include hepatic and bilary obstructive lesions.

Obstructive jaundice (cholestasis)

- Ultrasound is investigation of choice to exclude duct dilatation.
- Check coagulation and if necessary correct with vitamin K.
- If intrahepatic cholestasis, check medications.
- Consider ERCP.

Hepatic jaundice

- Infectious hepatitis A, B, C, Delta, EBV, CMV, non A,B,C (HEV)
- Acute alcoholic hepatitis.
- Chronic liver disease:- alcohol, autoimmune hepatitis, primary biliary cirrhosis, sclerosing cholangitis, Wilson's Disease.
- Drugs, toxins.

LIVER FAILURE

Where this is suspected commence treatment early.

CLINICAL AND BIOCHEMICAL FEATURES

- Jaundice.
- Coagulation defects (check PT and Echis ratio).
- Hypoalbuminaemia.
- Encephalopathy.
- Ascites.

CAUSES/PRECIPITANTS

- **Acute severe hepatic necrosis**
  - Hepatitis B (rare) ± Delta superinfection.
  - Autoimmune - submassive necrosis.
  - Toxins:
    - Paracetamol (**see page 140**).
    - Carbon tetrachloride (consider dialysis).
  - Fatty liver of pregnancy.
- **Chronic liver disease with acute deterioration**
  - GI haemorrhage.
  - Sepsis (especially Gm-ve).
  - Spontaneous bacterial peritonitis.
  - Drugs (especially alcohol, benzodiazepines).
  - Electrolyte disturbance (diuretics, hypokalaemia).

INVESTIGATIONS

- Na, K, urea, creatinine (hepatorenal syndrome).
- Glucose (may require IV dextrose infusion).
- Albumin, Bili, alk. phos., AST, GGT.
- CBC + diff, coagulation profile.
- Drug screen (30 ml urine to Toxicology. Blood alcohol, and other drugs as indicated).
- Hepatitis B testing (assume infectious until result available).
- Blood cultures.
- Consider smooth muscle and antinuclear antibodies.

TREATMENT

- Treat any underlying correctable cause (e.g. bleeding varices, sepsis).
- Stop all offending drugs.
- Correct hypokalaemia, hypotension, hypoglycaemia.
- If ascites present tap for microscopy and culture, (consider protein, glucose, amylase, tuberculosis).
- Correct coagulation defects with vitamin K 10 mg IV slowly and fresh frozen plasma as indicated.

IF ENCEPHALOPATHY SUSPECTED

- Give high carbohydrate/low protein diet.
- Gut sterilisation with neomycin 1 g q4h PO **or**
- Purge with lactulose 30 ml TID adjusted to produce three loose stool per day.
- Watch for alcohol withdrawal **(see page 33)**.
- Consult gastroenterologist promptly.

ACUTE PANCREATITIS

CLINICAL FEATURES:

- Pain is the dominant symptom and may range from mild to excruciating and may radiate to back.
- Fever, tachycardia, hypotension, abdominal distention and rigidity may occur.
- Shock.
- Hypoxia.
- Hypocalcaemia.

  Note:

  Bacterial sepsis may also be present.

DIAGNOSIS

- Serum amylase is usually elevated at least x 5 above normal range in appropriate clinical setting. Other abdominal diseases may cause a lesser elevation of amylase.

AETIOLOGY

- Biliary tract disease (especially gallstones).
- Alcohol.
- Idiopathic.
- Other - drugs, types I and V hyperlipidaemia.

INVESTIGATIONS

- Serum amylase.
- CBC + diff.
- Na, K, Ca, PO_4, creatinine, glucose, LDH, Bili, alk. phos., AST, GGT.
- Blood cultures.
- Abdominal ultrasound.
- Arterial blood gases.
- Lipid analysis if for types I and V hyperlipidaemia.

MANAGEMENT

- Treatment of shock (**see page 13**).
- Pain relief - pethidine is first choice.
- Bowel rest - nasogastric tube and aspirate gastric contents.
- Oxygen therapy - serial blood gases (ARDS, acidosis).
- Correct electrolytes and calcium disturbances.
- Antibiotics - if sepsis likely.
- Consider surgical consult.

Consider **urgent ERCP** if gallstone pancreatitis suspected.

Prognostic factors in acute pancreatitis

The following are associated with a poor prognosis.

On Admission	At 48 Hours
Age > 55 years	Haematocrit decreased > 10%
WBC > 16 x 10^9/L	Urea increased > 15 mmol/L
Glucose > 7.5 mmol/L	Calcium < 2.0 mmol/L
LDH > 350 U/L	PaO_2 < 60 mmHg
AST > 250 U/L	fluid retention > 6L

HAEMATOLOGY

MANAGEMENT OF HAEMORRHAGIC DISORDERS

Platelet disorders usually result in surface bleeding such as epistaxis and petechiae. Coagulation disorders on the other hand produce deep bleeding such as haemarthrosis or muscle haematomas. There may be a mixed pattern of bleeding in DIC. Fatal intracranial haemorrhage may occur in either severe thrombocytopenia or a severe coagulation deficiency.

INVESTIGATION OF A PATIENT PRESENTING WITH A POSSIBLE HAEMORRHAGIC DISORDER

- Family history, history of pattern of bleeding, recent drugs, dietary history, possibility of HIV.
- Full CBC + diff, ESR, blood film examination.
- Prothrombin time, partial thromboplastin time, thrombin time and fibrinogen level. Use citrate tubes. Take care to add the correct amount of blood to these tubes and avoid heparin contamination from heparin containing IV lines, blood tubes, etc.
  Take blood samples **before** any transfusions are given.
  Note:
  These are only screening tests and do not necessarily exclude mild defects which may nevertheless result in abnormal bleeding following trauma. Consultation with the Coagulation Laboratory is strongly recommended (Ext: 80373).

TREATMENT

- This is entirely dependent on the results of the initial tests obtained. If a severe **thrombocytopenia** (say less than 20 x 10^9/L) is present then this constitutes a medical emergency. These patients may need platelet transfusions. An accurate diagnosis is required and this will often require bone marrow examination.
- Patients with known **coagulation defects** (Haemophilia A, Christmas Disease, etc) present special problems and consultation (day or night) is essential when these patients are admitted outside the Haematology Service. Patients with an established coagulation defect may carry a card giving essential details of their condition. Those living around Christchurch will have records available in the Haematology Department and Haematology Ward giving the relevant Factor levels and some clinical details. Always take a suspected bleed seriously; always take careful note of any advice the patient gives you, in this context they are usually right.
- In **haemophilia A** life threatening bleeding requires immediate IV Factor VIII infusions, with concentrated freeze-dried preparations. A rough guide is given by the following formula.

Units of Factor VIII required = $\frac{\text{weight in kilograms x \% rise desired.}}{2}$

Currently each CSL ampoule contains 250U.

- You will need to know what level of Factor VIII it is desirable to achieve in any particular clinical situation (see above formula). Urgent Haematology consultation is essential.
- In **Von Willebrand's disease** and mild haemophilia DDAVP or Factor VIII concentrate is used. In mild haemophilia, DDAVP may be given in a dose of 0.4 mcg/kg in 100 ml 0.9S over 20 minutes.
- In **Christmas Disease** (Factor IX deficiency) Factor IX concentrate (Prothrombinex H.T.) is the treatment of choice. Consult Haematologist for this and less common coagulation disorders.

MANAGEMENT OF SEVERE ANAEMIA

The following management is suggested for severe anaemia (a haemoglobin of 50g/L or less) in the absence of acute blood loss or shock. Some causes include: iron deficiency, B_{12} and folate deficiencies, leukaemias, myelodysplastic syndromes, aplasia, haemolysis, renal failure.

INVESTIGATIONS

- CBC + diff. Note particularly haemoglobin, MCV, white cell count, differential, platelets and reticulocytes.
- MCV <80 fl - probable iron deficiency, request iron, transferrin and ferritin. Check ESR.
- MCV >100 fl - could merely reflect an increased reticulocyte count - (haemolysis/blood loss), but if retics normal, B_{12} and folate levels should be done.
- MCV 80-100 fl - consider renal failure, hypothyroidism and acute blood loss.

Decide whether a bone marrow is required. Consider direct antiglobulin test if haemolysis suspected. These generalizations do not necessarily apply to patients with mild or moderate anaemia.

TREATMENT

- Once blood samples have been taken, and a bone marrow has either been performed or been deemed unnecessary, treatment may be started with oral iron and/or oral folic acid and/or IM hydroxocobalamin if one of these haematinic deficiencies seem likely. Recommended preparations are ferrous gluconate 300 mg PO, BD, folic acid 5 mg PO daily and hydroxocobalamin 1 mg every other day for 6 doses, followed by maintenance treatment.
- Transfusion should be given with extreme caution, if at all, if a deficiency state is present. A partial exchange transfusion may be needed for someone

in heart failure and in elderly patients. Close observation and diuretics will be needed. Transfusion may make subsequent diagnosis difficult, particularly in cases of haemolytic anaemia and some deficiency states.

- If in doubt a phone or written consultation with the Haematologist may be helpful as the appearances of the blood film may give further information of practical value (e.g. in haemolytic anaemias).

MANAGEMENT OF SEVERE NEUTROPENIA

- If the neutrophil count is $<0.5 \times 10^9$/L there is a significantly increased risk of severe or fatal sepsis. Try to identify the cause of this abnormal blood count.
- Chemotherapy, radiation treatment, drug toxicity, severe sepsis, leukaemias, myelodysplastic syndromes, aplasia are a number of possible causes.
- Unless the cause is obvious and temporary, investigations should include examination of the bone marrow.

TREATMENT

- Initial management should consist of isolation of the patient. Place the patient in a single room and institute strict hand washing for the attending staff. Restrict the number of visitors. Consider prophylactic oral ciprofloxacin (adult dose 500 mg PO BD).
- If febrile take blood cultures from peripheral vein and central line if present. Other appropriate investigations include MSU, swab of any lesion or pustule, sputum for Gram stain and culture, faecal culture if diarrhoea is present, CXR. If fever is maintained above 38°C for more than 2 hours parenteral broad spectrum antibiotics such as **gentamicin and piperacillin** or **imipenem** alone should be given immediately together with intravenous hydration. In addition, the choice of antibiotics used will be influenced if the site of infection is obvious (**see Septicaemia p 88**). **Do not await the results of cultures in this situation.** Suitable dosages for the above antibiotics in this situation are as follows:
  - **Gentamicin** 4.5 mg/kg as a loading dose and take blood levels at 1 and 4 hours. If renal function normal give gentamicin 1.5 mg/kg q8h and monitor peak and trough levels around the 3rd dose or use computer prediction from Toxicology (Ext: 80322) (**see page 27**).
  - **Piperacillin** 4 g q8h IV. This drug may be given by IV bolus or 30 minute infusion.
  - **Imipenem** 500 mg in 100 ml 0.9S over 30 minutes q6h. In moderate-severe renal impairment extend dose intervals.

HEALTH CARE OF THE ELDERLY

Care of elderly patients forms a significant component of hospital practice and with the ageing of the population the size of this component will increase. It is therefore important that all doctors are confident and competent when dealing with the elderly.

ATTITUDES

Poor staff attitudes to elderly people can adversely influence the standard of treatment and care they receive. It is important that the elderly are not considered an imposition or inappropriate admissions. In particular, labels such as "social admissions" and "nursing care" should not be used. So called "social admissions" have a high morbidity and mortality much of which can be avoided by accurate and prompt treatment.

HISTORY

- Always end admission clerkings with a diagnosis or problem list.
- The case history should follow the usual format of the medical history **but a collateral history should always be sought if the patient is unable to give a history themselves.** GP, spouse, carer, neighbour etc should be contacted in order to obtain a history.
- As well as the standard systems examination the specific Health Care of the Elderly check list should be completed.

HEALTH CARE OF THE ELDERLY CHECK LIST

- Eyes:
  - Visual acuity.
  - Have the correct spectacles been brought in to hospital?
- Ears:
  - Is there impacted wax?
  - Has the patient a hearing aid? Does it work? Can the patient use it?
- Mouth:
  - Check state and health of tongue.
  - Check dentition and fitting of dentures.
  - Check angles of the mouth.
- MSQ:
  - Check the standard 10 point MSQ **(see page 110).**
- Bladder:
  - Rectal examination.
  - Is the bladder palpable?
  - Should a continence chart be used?
  - Is vaginal examination required?

- Bowels:
  - Rectal examination.
  - Is the descending colon palpable?
- Temperature:
  - Infection may be present with a normal temperature. Is the patient hypothermic? - check rectal or freshly voided urine temperature with a low reading thermometer.
- Feet:
  - Can the patient attend to their own foot care?
  - Are there any painful lesions, corns, calluses that need attention?
  - Is the vascular supply impaired?
  - Is there a peripheral neuropathy?
  - Has the patient adequate, safe footwear?
  - Does the patient have correct, safe walking aids.

ALTERED PRESENTATION OF ILLNESS

Altered or abnormal presentation is the rule rather than the exception in the elderly. Beware the painless myocardial infarction or the pneumonia / septicaemia with normal temperature.

Falls, confusion, failure to manage, "gone off legs", are common presentations. Elderly people with such presentations need meticulous examination and work up.

MANAGEMENT OF THE CONFUSED ELDERLY PATIENT

Accurate diagnosis is the key to management. It is essential to find out the length of the patient's confusion and distinguish between acute confusional state (delirium) and chronic confusional state (dementia).

These patients are seriously ill. Delirium affects 25% of elderly patients admitted to hospital and is associated with a 1 month mortality of 33%.

Predisposing factors for confusional states are advanced age, pre-existing dementia, sensory impairment and Parkinson's Disease.

- Use the MSQ routinely.
- Make an accurate diagnosis.
- Treat any underlying cause (infection, dehydration, faecal impaction etc).
- Stop offending drugs.
- Avoid sedation unless absolutely required.
- **Do not** use "cocktails" of several drugs.
- **Do not** use intramuscular prn injections of preparations such as chlorpromazine or haloperidol.

- Select small doses of oral medication (e.g. haloperidol) on a **regular** basis. Titrate up or down according to need and withdraw as soon as possible. Aim to withdraw completely when the confusional state is improved.
- Aid orientation by getting the patient up, attending to spectacles, and hearing aids, communicating helpfully and providing clues to the environment with signposting and helpful layout of surroundings.
- Prohibit the use of cot sides which increase the confused patient's agitation and distress and inevitably result in the patient trying to climb over them with a longer fall to the ground.
- Nurse the patient low to the floor on a Hilo bed or mattress on the floor.
- Use a soft night light.
- Where possible use the same staff to deal with the patient rather than constant changes of personnel. Using relatives to stay with the patient may be helpful.

INCONTINENCE PROBLEMS

Urinary incontinence

- Make a diagnosis.
- Attempt to ascertain whether the patient has stress or urge incontinence.
- Check the medication list to see whether drugs are contributing.
- Check MSU.
- Remember rectal, vaginal and abdominal examination (to exclude faecal impaction, prostatism, urinary retention etc).
- Use an incontinence chart to identify any problem times or patterns of the incontinence.
- A trial of an anticholinergic (e.g. oxybutnin or imipramine) is worthwhile if uninhibited neuropathic bladder is suspected.
- Referral for urodynamic studies may be required in a small number of cases.

Faecal incontinence

- Faecal impaction with overflow is the leading cause in the elderly.
- Check rectal and abdominal examination (± abdominal x-ray).
- Check the medication list.
- Examine for painful rectal or anal conditions.
- Exclude other causes of diarrhoea (e.g. infective colitis, etc).
- Consider the use of bulking agents.
- Do not use constipating agents until it is certain that high faecal impaction is excluded.
- Use commonsense measures such as sitting the patient on the toilet after a meal.

REFERRAL TO THE ASSESSMENT AND REHABILITATION UNIT

These are generally for:-

- A Specialist opinion about an elderly patient.
- Assessment of patients for transfer to the A.R.U.
- Assessment of patients for ongoing treatment in the Day Hospital.
- Assessment of patients when continuing hospital care is being sought.
- When there is doubt whether a patient requires rest home or hospital care.

Points to remember

- The best value is obtained by using the referral as a "consultative" service rather than a "takeaway" service and by referring early.
- Medical wards have an identified Consultant Physician for the Elderly responsible to them. Please use this Consultant and don't hesitate to telephone.
- **There is a duty physician in Health Care of the Elderly on call 24 hours a day, 7 days a week.**
- **Regular consultations occur in acute admitting, surgical and orthopaedic wards.**

INFECTIOUS DISEASES

MENINGITIS

CLINICAL FEATURES

- Fever, headache, photophobia, neck stiffness and impaired sensorium.

CAUSES

- N. Meningitidis; S. pneumoniae; H. influenzae (usually paediatric). Listeria monocytogenes (immunosuppressed or pregnant); Mycobacterium tuberculosis.
- Syphilis, leptospirosis, Gram negative bacilli (rare but seen in neonates, post trauma, immunosuppressed).
- Viral - especially mumps and enteroviruses.
- Other - amoebae, fungi.

PATHOGENESIS

- Cryptogenic.
- Septicaemic illness.
- Secondary to head or neck sepsis e.g. ear, dental.
- Following head injury, CSF leak or sinus fracture.
- Complement deficiency, especially C7 and 8.
- Travel - may be insect borne.
- Immunosuppression including malignancy and HIV.

INVESTIGATIONS

- Blood cultures - 2 sets.
- Lumbar puncture. Caution: in patients with suspected meningitis and papilloedema, focal neurological signs or chronic ear infections - arrange CT scan urgently before doing lumbar puncture.
  Collect 2 ml of CSF into each of three numbered vials. Send to Microbiology. Routine tests done are cell counts, glucose, protein, and culture. Gram stain, antigen detection tests and viral culture will be done if WBC count is $>5 \times 10^6$ cells/L.
- CBC + diff.
- Na, K, glucose, creatinine, AST, GGT, alk. phos., Bili.
- Throat swab - bacterial and viral.
- Acute serum for serology.
- Stool for viral culture.
- Chest and sinus x-rays.
- Coagulation profile.
- Special investigation needed for cryptococcus, TB, amoeba, viruses - consult Microbiologists.

Note:

Antibiotics may have been given before the patient reached hospital. **You** may need to give antibiotics at once if LP cannot be done immediately **(see below)**

MANAGEMENT

All patients with papilloedema, focal neurological signs, chronic ear infections or who are semi-comatose or unconscious need a CT scan URGENTLY before doing a lumbar puncture. **Antibiotics may be needed first.**

- **Acute onset:**
  - Patients with a fulminating course of <24 hours, **or** who are semi-comatose **or** unconscious **or** if a purpuric rash present.
  - Take blood cultures and throat swabs.
  - Consider immediate lumbar puncture. If **contraindicated** or there will be a **delay. GIVE ANTIBIOTICS IMMEDIATELY**. Do **NOT** wait for the results of the lumbar puncture.
  - Antibiotics should be started within 45 minutes of arrival at Emergency Department.
- **Subacute onset 1-7 Days**
  - Careful history and physical examination.
  - Decision to treat based on review of CSF results and clinical state especially **level of consciousness**.
  - This should take less than 2 hours.
  - If CSF shows bacteria, bacterial antigen or pyogenic picture (see below) begin antibiotics.

Usual CSF patterns in meningitis

	PYOGENIC	TUBERCULOUS	ASEPTIC
Predominant Cells	Neutrophils	Mononuclear	Mononuclear
Numbers	>1000x10^6/L	10-350x10^6/L	50-1500x10^6/L
Glucose	<2/3 blood	<2/3 blood	= blood
Protein	>1.0 g/L	>1.5 g/L	<1.5 g/L

ANTIBIOTIC THERAPY

- **Pyogenic meningitis in otherwise normal host.**
  - Age >10 years, not immunosuppressed or pregnant, no head injury, no ear or sinus disease. Most likely to be due to meningococcus or pneumococcus. Penicillin G, 2 mega units IV q2h. If mild penicillin allergy

ceftriaxone IV 2 g q24h or chloramphenicol IV, 4-6 g/day in 4-6 divided doses.

- Under 10 years seek Paediatric advice.
- Meningococcus should be cleared from nasopharynx at end of treatment period regardless of throat swab, by giving rifampicin 600 mg BD, PO for two days or ceftriaxone 250 mg IM or IV.
- Close household contacts of patients with meningococcal meningitis should be given rifampicin prophylaxis as above as soon as diagnosis made. Throat swab **not** necessary.
- Notify MOH if applicable - see Appendix I.

♦ **Pyogenic meningitis in abnormal host.**

- Consult promptly. Patients who have ear or sinus disease, Immunosuppression, age >65 years or are pregnant. Beware Gram negative sepsis and Listeria. Add a third generation cephalosporin (e.g. ceftriaxone). Listeria is treated with IV amoxycillin 12 g/day in adults) with or without gentamicin.

♦ **Steroids.**

- Clear benefit in prevention of complications has been demonstrated for children with meningitis due to Gram negative organisms. There are no comparable studies in adults but many prescribe them in this situation. If used hydrocortisone 200 mg IV then prednisone 40 mg q8h PO should be given **before** or as soon as possible after antibiotics have been started.

♦ **Tuberculous meningitis**

- If TB meningitis is suspected it is vital to have a large volume (e.g. 10 ml) of CSF for ZN stain and culture. This may require repeat lumbar puncture. Ensure CXR, sputum, (gastric washings), EMU x 3 and Mantoux are done. May need bone marrow or liver biopsy for TB culture.
- Usual oral drug therapy isoniazid 8-12 mg/kg/day with pyridoxine 100 mg/day, rifampicin 10 mg/kg/day max 600 mg/day, ethambutol 25 mg/kg/day, pyrazinamide 20-30 mg/kg/day initially. Other drugs may be needed depending on history and clinicel state. Check HIV status.

♦ **Aseptic meningitis**

- Most often due to viruses - mumps, and enteroviruses most common. The seroconversion illness of HIV can present with aseptic meningitis.
- Many treatable and serious problems cause a similar CSF picture e.g. partially treated bacterial meningitis, TB, fungi, amoeba, syphilis, herpes simplex, leptospirosis, neoplastic, drugs, cyst related, Mollaret's, SLE, Bechet's, sarcoidosis, and others.

SEPTICAEMIA

This is life threatening. 30-50% of patients will die despite appropriate therapy. Early diagnosis and treatment are vital. Patients are usually toxic and febrile but beware of atypical presentations.

The patient may be in shock or just look unwell. Those who are apparently well may deteriorate rapidly. Those with chronic renal failure or advanced age may have no fever or be hypothermic. Systemic steroids may mask the symptoms and signs.

CLINICAL SITUATIONS WHICH MAY PREDISPOSE TO SEPTICAEMIA

- IV lines if they have been in longer than 24 hours.
- Urinary catheters.
- Local sepsis.
- Steroid therapy.
- Advanced age and debilitation.
- Drug addiction and alcoholism.
- Diabetes mellitus.
- Chronic renal failure.
- Post surgery or obstetric procedures.
- Splenectomy.
- Malignancy - leukaemia, myeloma etc.
- Immunosuppressive therapy - neutropenia etc.

CLINICAL CLUES TO CAUSE

- Skin lesions:
  - Ecthyma gangrenosum - pseudomonas.
  - 2-20 papules on extremities - N. gonorrhoea.
  - Purpura (may be necrotic) N. meningitidis, S. aureus.
  - Rose spots - Salmonella typhi.
  - Pustules - S. aureus.
  - Macronodular lesions - C. albicans.
  - Osler's nodes, Janeway lesions, splinter haemorrhages - endocarditis.
- Acute haemolysis - Clostridium sp.
- Multiple pulmonary infiltrates with rapid cavitation - S. aureus.

INVESTIGATIONS

- **Blood cultures.**
- The diagnosis is based on culturing organisms from the blood so good technique is essential. Blood cultures should be drawn by medical staff and not as a routine by bleeding staff.
- If septicaemia is suspected, urgent antibiotic treatment is necessary. Collect 2-3 sets of blood cultures with an interval as short as 5-10 minutes between venepunctures. Separate venepunctures are important as one set might be contaminated with skin organisms. If antibiotics have been given prior to blood cultures then further cultures will need to be taken over a more prolonged period of time.
- Using Bactec bottles. Wash hands and select vein. Remove and discard plastic cap of Bactec bottle. Clean patient's skin and rubber seal of Bactec

bottle with an antiseptic solution (avoid iodine based solution). Draw 10 ml of blood into syringe. Do **not** change needle on syringe. Inoculate one Bactec set (one blue bottle for aerobes and one yellow for anaerobes) with 5 ml blood into each bottle (i.e. 10 ml per set). Invert bottles gently to prevent clotting. Send to lab as soon as possible. Do not refrigerate. If lab is closed, keep at room temperature.

- If endocarditis is suspected 3 venepunctures (6 bottles) should be taken, ideally, spaced over 24 hours. If patients are acutely ill they may be taken stat from several sites. If antibiotics have been given during the past 2 weeks do 6 venepunctures (12 bottles).
- **Other cultures**
  - Sputum if possible.
  - MSU, throat and nose swab.
  - Swab skin lesions and ears if local sepsis likely.
  - Consider LP if meningitis possible.
  - Aspirate fluid from joints or serous cavities and send aspirated material to laboratory.
  - If IV cannula sepsis is suspected then swab skin over entry site with alcohol. Remove and cut intravenous section into sterile container with sterile scissors. Consult Microbiology if these samples have to be stored for more than an hour.
  - CBC + diff, coagulation profile for DIC.
  - Na, K, creatinine, glucose, AST, GGT, alk. phos., Bili.
  - CXR.
  - Arterial blood gases.

MANAGEMENT

- **Fluids**
  - Resuscitate with 0.9S then alternate 0.9S with D5W. Blood or plasma may be needed.
  - If the patient is in shock then a CVP line may be needed and larger volumes of fluid required. **(See page 13).** If you are not competent to place a CVP line do not attempt it. Consult ICU.
  - If patient remains hypotensive (systolic <80 mmHg) despite adequate hydration then dopamine infusion may be needed. **(See page 35)**.
  - Fluid management in septic shock can be difficult and CVP readings amy be misleading. Swan Ganz catheterisation is frequently required.
- **Oxygen therapy (see page 164).**
- **Monitoring.**
  - Urine output - If patient hypotensive/shocked may need catheter but catherization is best avoided if possible.
  - Daily creatinine.
  - Arterial blood gases and pulse oximetry - ARDS is common and the patient who has progressive hypoxaemia may need assisted ventilation.
  - Severe acidosis secondary to inadequate tissue perfusion may require partial correction **(see page 70)**.

- Repeat platelet count and coagulation profile as indicated. If bleeding occurs, this is most likely due to DIC. If so consider platelet transfusion and coagulation factor replacement.

♦ **Source** - seek source carefully and treat it promptly. This may determine survival.Relieve obstructed ureter or biliary system, drain abscesses, remove infected IV cannulae or IV solutions, evacuate septic uterus etc.

♦ **Antibiotic therapy.**

- Initial therapy is based on likely source of sepsis and common organisms associated with sepsis from this site. The sections on endocarditis, pneumonia, urinary tract infections, meningitis, bone and joint infections etc. may provide drugs appropriate as an initial choice.
- In hospitalised patients organisms may have been previously isolated and the sensitivities available.
- If infection is cryptogenic (no primary site identifiable) then cefuroxime plus gentamicin is a reasonable choice but this combination will not cover enterococci, anaerobes, Listeria and several other species.
- Reasonable choices Include:
  - ~ Cryptogenic sepsis
    - Community acquired - cefuroxime + gentamicin.
    - Hospital acquired - ceftriaxone and metronidazole.
  - ~ Intra-abdominal sepsis
    - Amoxycillin + gentamicin + metronidazole.
    - Ceftriaxone + metronidazole.
  - ~ Simple cellulitis
    - Penicillin + flucloxacillin.
    - Cefuroxime.
  - ~ Complex cellulitis e.g. associated with ulcerated skin in diabetes, ischaemia
    - Cefuroxime + metronidazole
  - ~ Pseudomonas sepsis
    - Piperacillin + tobramycin.
    - Ceftazidime + tobramycin.
  - Urinary tract
    - Gentamicin.
    - Ceftriaxone.
  - Short term (a few days) mild neutropenia (neutrophils >0.1 x 10^9L).
    - Gentamicin + cefuroxime
  - Severe neutropenia.
    - Piperacillin + gentamicin.

HIV AND AIDS

Managing these patients is complicated and requires close cooperation with infectious disease and microbiology. The indications for treatment with antiviral drugs such as zidovudine (AZT) are changing rapidly and require expert advice. The infections that have been found in association with HIV constitute a huge and expanding list and are often very unusual.

INFECTIVITY AND ISOLATION

- HIV may be carried by any patient within the hospital. **Please protect yourself.** The hospital policy is that **all** patients should be treated as if they are infected.
- The virus is present in body fluids and can cause disease if it penetrates skin or is splashed onto mucous membranes. It is **not** transmitted by aerosol, casual contact or physical examination.
- **Put a barrier between you and body fluids** from patients. Gloves, gowns and plastic aprons are generally only needed if patient is incontinent, demented, or for performing procedures.
- Goggles can be obtained if splashes likely eg: putting in a nasogastric tube.
- Venesection - Take container for sharps into patient's room. **Do not recap needles**. Drop sharps directly into box. If you have a minor skin lesion wear gloves. If skin is intact gloves are optional. They will not protect against needle stick and may make you more clumsy.
- **If you get a needle stick or splash of blood** obtain a blood sample from the patient unless known to be HIV positive. Contact microbiology **immediately.** Prophylactic zidovudine may be indicated and should be administered urgently.
- Follow protocol for needle stick injury. This is available in all wards and departments.

ANTIBODY TESTING

- Counselling and explanation. Consult with Infectious Diseases if you are uncertain.
- Obtain consent to test for HIV antibody.
- Tell patient of limits of the test.
- Preserve patient confidentiality. Tests should not have the patient's name on the form unless the patient agrees. A commonly used code is:
  - First two letters of surname.
  - First two letter of first name.
  - M or F (Sex).
  - Date of birth.

OTHER INVESTIGATIONS

If HIV infection suspected or proven a yellow "Infectious" sticky must be placed on all request forms accompanying blood or body fluids or if patient is to undergo invasive investigation.

CLINICAL PRESENTATION

- Acute infection:
  - "Mononucleosis like" - fever, lymphadenopathy, sore throat, truncal rash (macular papular), diarrhoea.

- Aseptic meningitis - retro-orbital pain often prominent, atypical lymphocytes often present in peripheral blood.
- Encephalopathy.
- These patients are infectious although the HIV antibody test is usually negative during the acute illness and may take 2-6 months to become positive.
- If diagnosis suspected ensure a sexual, drug and blood transfusion history taken.

♦ Persistent generalised lymphadenopathy:
- Lymph node enlargement in axillae, neck and groin present for over 3 months and for which no other explanation is found.
- HIV serology is positive.

♦ Complicated disease:
- Most patients who have progressed to complicated disease have sentinel infections in mouth and skin. These are important clinical clues.
  - ~ Mouth - candidiasis, hairy leucoplakia, herpes simplex, gingivitis
  - ~ Skin - herpes zoster, fungal infections.

♦ Suggestive laboratory findings:
- Anaemia.
- Thrombocytopenia.
- Leucopenia/lymphopenia.
- Reduced CD4 T lymphocyte count.

SOME SPECIFIC PRESENTATIONS OF COMPLICATED DISEASE

Pneumonia - Pneumocystis carinii is very common but bacterial, (e.g. pneumococcal, legionella and mycobacterial) and viral pneumonias also occur. If presentation is suggestive of a bacterial pneumonia investigate as usual (eg blood and sputum cultures) and treat as community acquired pneumonia. Otherwise treat as pneumocystis carinii pneumonia. This is life threatening and must be treated promptly.

♦ **Pneumocystis carinii pneumonia (PCP)** - symptoms are usually of slow onset over several days and up to eight weeks. Shortness of breath (initially on exertion), non productive cough, fever, and chills.

Investigations:
- Arterial blood gases - hypoxemia common.
- CXR - diffuse interstitial infiltration.
- Induced sputum (nebulized hypertonic saline - ask Physiotherapy department for help). Send for bacterial, legionella, mycobacterial and viral culture and stain for PCP.
- Throat swab - viral immunofluoresence and culture.
- CBC + diff.
- AST, GGT, alk. phos., Bili, creatinine.
- Bronchoscopy may be indicated consult Infectious Diseases and Respiratory physician.

Treatment

Begin treatment for presumed PCP with co-trimoxazole when specimens taken. If diagnosis clear and patient is unwell (PaO_2 on air <65 mmHg) add prednisone 40 mg BD PO. If a definite diagnosis has not been made and the patient is not responding within 48 hours bronchoscopy is indicated.

Co-trimoxazole - Dose to include trimethoprim 15-20 mg/kg/day (four divided doses). Begin with IV infusion therapy. This may be given in a smaller volume than recommended e.g. 320 mg in 500 ml. Change to oral after 5 days if clearly under control. Nausea is very common. Often responds to prochlorperazine. Folinic acid 15 mg/day if platelet count <100 x 10^9/L or neutrophil count <1.5 x 10^9/L. Rash occurs in up to 50% of HIV patients and **may** necessitate a change to pentamidine.

Pentamidine Isethionate - 4 mg/kg as infusion once each day. Hypotension common. Hypoglycaemia unusual but unpredictable and can be intractable.

CNS Disease

Most patients with CNS disease who are HIV positive have encephalopathy for which there is no specific treatment. Some have an infective or neoplastic cause of CNS disease but others will have **both**.

- **HIV encephalopathy** - Main features, forgetfulness, poor concentration, lethargy, loss of balance, poor handwriting, withdrawal, ataxia, hyperreflexia, weakness, with progression to dementia and incontinence over weeks to months. Usually due to HIV, but rarely may be CMV, HSV, lymphoma or atypical mycobacteria.
- **Meningitis** - Usually Cryptococcus neoformans. Headache universal, lethargy, fatigue, fever and weight loss common. Neck stiffness and photophobia often absent.
- **Space occupying lesions** - Lethargy and confusion progressing to fits and focal signs. Causes are lymphoma, toxoplasmosis and other infections.

Investigations:

- CT scan brain. If this shows:-
  - Diffuse atrophy then HIV encephalopathy is likely. Perform lumbar puncture.
  - Ring enhancing lesion - assume due to toxoplasmosis - seek advice.
  - Solid lesion - consider brain biopsy.
  - Low density lesions without enhancement - most likely progressive multifocal leucoencephalopathy.
- Lumbar Puncture:
  - CSF culture for bacteria fungi and viruses.
  - CSF antigen for cryptococcal antigen.

- Blood and Urine - cryptococcal antigen.

Treatment: - seek advice.

Retinitis - Most often due to cytomegalovirus.

Investigations:

- Ophthalmology opinion.
- Serology for CMV titres.

Treatment :

- Begin ganciclovir 5 mg/kg IV q8h as soon as possible. This infection causes **blindness** very rapidly.

GI disease

- Oesophagitis is generally due to candida or herpes simplex. Endoscopy or barium swallow may be needed for diagnosis.
  Treatment:

  H. Simplex - Acyclovir oral if able to swallow 200 mg 5 times/day or 5 mg/kg IV q8h
  Candida - Ketoconazole 200 mg PO once daily

- Diarrhoea can be due to many causes.

  Initial investigations include stools for ova and parasites, routine bacterial culture, and modified ZN stain for cryptosporidiosis and ZN stain for mycobacterium avium-intracellula're (MAI).

  Treatment - Cryptosporidia - No satisfactory therapy.

Constitutional disease

Systemic symptoms - fever, weight loss >10%, sweats, fatigue.

If fever is documented but no localising symptoms begin systematic search as follows.

- A blood culture - bacteria, CMV and MAI.
- Stool culture for bacteria and examination for protozoa and helminths.
- CXR.
- Arterial blood gases.
- Saline induced sputum.
- Urine culture.

- Bone marrow culture and special stains.
- Node biopsy.
- CT scan abdomen.
- Lumbar puncture.

All patients will need thorough work up for other STD's, and decisions made about appropriate use of zidovudine and prophylactic antibiotic regimens.

MALARIA

EPIDEMIOLOGY

P. vivax predominates
- India, Bangladesh, Pakistan, Sri Lanka and Central America.

P. falciparum predominates
- Africa, Papua-New Guinea, Haiti.

P. falciparum and P. vivax both prevalent
- South East Asia, South America, and Oceania.

Chloroquine resistant P. falciparum common
- South East Asia, South America PNG and East Africa.

Chloroquine resistance in P. vivax has recently been reported from Papua New Guinea and the Solomon Islands.

PRESENTATION

- Prophylaxis should be taken for four weeks after return from endemic areas. If this is not done clinical illness may occur.
- Incubation period - P. falciparum, 7-14 days, P. vivax 12-17 days.
- Prodrome of 1-7 days may resemble a viral illness - malaise, headache, fatigue and myalgias. May also have chest pains, abdominal pain, arthralgias.
- Paroxysms lasting 8-12 hours.
  - Cold phase 1-2 hours, chills, rigors, headache, pallor and cyanosis.
  - Hot phase 1-4 hours, fever up to 41°C, warm dry skin, headache, nausea, vomiting, backache, abdominal pain, delirium, orthostatic hypotension.
  - Sweating, flushing and vomiting often followed by euphoria and fatigue.
- Findings that may be associated - jaundice, petechial rash, retinal haemorrhage, pulmonary oedema.
- Complications .
  - P. vivax - rupture of spleen, be careful on palpation.
  - P. falciparum - cerebral malaria, focal signs uncommon, mortality 20%.
  - Renal failure and haemoglobinuria.
  - Severe liver involvement.

- Pulmonary oedema.
- Hypoglycaemia especially pregnancy and quinine therapy.

Note

The fever pattern may be suggestive but usually is not and may be continuous. Lymphadenopathy, muscle tenderness, and joint effusions DO NOT occur in malaria. Look for another cause.

INVESTIGATIONS

- Thick and thin blood films. If negative repeat examination daily. May need to examine bone marrow in partially treated patients.
- CBC + diff and film.
- Creatinine, AST, GGT, alk. phos., Bili, glucose.
- CXR.
- Arterial blood gases.
- Blood culture.
- Urinalysis.

MANAGEMENT

- P. vivax, P. ovale and P. malariae. These can usually be managed as an outpatient but you must be sure it is not P. falciparum. **If the patient has acquired the infection in Papua New Guinea then admission and close observation is warranted.**
  - Chloroquine 600 mg (base) stat PO, 300 mg 6 hours later then 300 mg daily for 3 days. If unable to take oral medicines, Chloroquine 10 mg/kg (base) IV q8h then 15 mg/kg over 24 hours.
  - Check for G6PD deficiency. If normal then primaquine is safe. If G6PD deficient check with Infectious Diseases/Haematology.
  - P. vivax and P. ovale - give primaquine 15 mg BD for 12 days. Primaquine treatment is not needed for P. Malariae.
- P. falciparum. All patients should be admitted.
  - Oral therapy:
    - ~ Quinine 600 mg q8h PO for 7 days PLUS tetracycline 250 mg q6h PO for 7 days. IV therapy: Quinine base, 16.7 mg/kg over 4 hours then 8.4 mg/kg over 4 hours q8h until patient can swallow.
    - ~ Oral mefloquine may be appropriate in some patients.
      Mefloquine 750 mg (3 x 250 mg tab) THEN
      500 mg after 6 hours THEN
      250 mg after 12 hours if >60 kg.
  - Cerebral malaria give phenobarbitone prophylactically 3.5 mg/kg IM stat.
  - Parasitaemia > 10%, consider exchange transfusion.

Note

Dose of quinine should be reduced in severe liver and renal disease.

MONITORING

- Blood films - contact laboratory, parasitaemia may not change for 24-48 hours but should be clear by day 5. Gametocytes persist for longer and do not necessarily indicate treatment failure.
- Blood glucose BD.
- CBC + diff, Na, K, creatinine, and Bili daily.

TYPHOID AND PARATYPHOID FEVER

Suspect in any patient who has been abroad and has fever. Beware of typhoid presenting with CNS and respiratory symptoms.

Organisms - S. typhi, S. paratyphi, S. schottmeulleri and S. hirschfeldii

Incubation period 7-21 days (usually 10-14).

PRESENTATION

- The onset is usually insidious with fever, malaise, anorexia, headache and myalgias. Fever gradually increases so that after one week it is usually 39-40°C. Sweats and chills are only found in one third of patients. Constipation or diarrhoea may occur. Hepatomegaly and splenomegaly common.
- Respiratory - cough and sore throat often prominent. May have cervical adenopathy and basal crepitations. In other words respiratory manifestations may predominate.
- CNS - confusions, seizures, psychotic behaviour.
- Skin - rose spots, 2-4 mm maculopapular lesions blanche on pressure.

INVESTIGATIONS

- Blood cultures x 3.
- Urine culture.
- Stool culture.
- CBC + diff.
- Coagulation profile for DIC.
- Na, K, creatinine, AST, GGT, alk. phos., Bili.
- CXR.
- ECG.

COMPLICATIONS

Meningitis, endocarditis, osteomyelitis, bone marrow suppression, bowel perforation.

TREATMENT

Blood cultures will be reported as positive for Gram negative bacilli. If typhoid fever is a possible diagnosis begin ceftriaxone 2 g IV stat or ciprofloxacin 750 mg BD PO or IV.

PENICILLIN ALLERGY

- It is unwise to give penicillins to patients who have a history of definite allergy to penicillin. It may be unavoidable in some situations e.g. enterococcal endocerditis or Listeriosis. These patients need a desensitisation protocol. Please consult the infectious diseases physician or clinical immunologist.
- Many patients who are said to have penicillin allergy do not in fact have this. Vomiting, loose motions and other vague symptoms do not represent allergy. An erythematous skin rash represents a mild allergy and is not likely to cause problems. Cephelosporins are safe in these patients.
- If there is a history of severe allergy e.g. urticaria, hypotension, or collapse, penicillins should not be given if there is an alternative. The risk of a reaction to cephalosporins in these patients is small - probably less than 5%. Nevertheless other agents may be available and should be used in preference.

INTRAVENOUS CANNULA CARE

INTRAVENOUS LINE SEPSIS

Infusion therapy and intravascular devices carry a substantial and unappreciated risk for producing iatrogenic harm. In Christchurch we have had an ongoing problem with infection from intravenous cannulae. These have resulted in severe metastatic infections including endocarditis, prosthesis infections, septic arthritis, and endophthalmitis. These complications can be prevented by good insertion technique and subsequent care.

COMMON ERRORS:

- Failure to wash hands before inserting the cannula.
- Placing non sterile dressings and tape over puncture site.
- Inadequate disinfection of site.
- Leaving in situ too long (>72 hours).
- No record of insertion date.
- Failure to replace lines inserted under emergency conditions (e.g. by ambulance staff, or in emergency department).

100

INSERTION AND CARE OF THE INTRAVENOUS CANNULA

Intravenous infusion is a common form of medication. From time to time infection occurs at the site of the intravenous catheter used in such therapy. Obviously any procedure which 'breaks' the protective skin surface has a potential to produce infection. Because these procedures are commonplace, it is easy to forget this risk. Observation of the following procedure is essential.

- **Insertion.**
  - Wash hands. Soap and water is adequate. Gloves are recommended and essential if there is significant risk of infection from blood contamination (e.g. severe dermatitis, open wounds).
  - Choose an upper limb vein, unless in an emergency. In this case, change to upper limb vein as soon as a satisfactory site is identified.
  - Prior to insertion, prepare site with antiseptic solution, hirsute arms might need clipping. The antiseptic must remain in contact with the skin for at least 60 seconds.
  - Insert cannula into vein. Avoid touching puncture site. Obtain flashback, remove needle and connect previously primed administration set or luer lock. Ensure puncture site is dry (using sterile gauze swab) before covering site.

    Two methods of stabilizing the cannula are acceptable.

    A sterile prepackaged transparent dressing (e.g. venigard) which will stabilize the cannula and act as a dressing.

Note:
The insertion date should be written on the tape and in the notes.

- Tape (e.g. leucopore) around the hub ensuring that the tape is not over the wound. Place sterile gauze over the wound and secure with tape.

Note:
The insertion date should be written in the clinical notes and on tape away from the insertion site. Convenient GREEN LABELS are available for this and should be used if possible.

IV CANNULA
INSERTION DATE / /
TIME

- **Care of catheter.**
  - Examine daily. Replace routinely every 48-72 hours, if not required earlier. Never leave longar than 72 hours. Clinical examination detects only 50% of infected catheters. Septic thrombophlebitis causes continuing bacteraemia after removal of the catheter, and will probably need surgical drainage.

Nursing staff may offer reminders of the need to change IV devices, but responsibility ultimately rests with medical staff.

- **Suspected catheter infection.**
  - Remove catheter and giving set.
  - Swab site as above and leave for 60 seconds.
  - Cut off SC portion of catheter using sterile scissors. Place in a sterile container.
  - Send to Microbiology Laboratory.
  - Consider whether infusion solution may be infected. If this is suspected, send solution and giving set to Microbiology. If related to blood transfusion, send to Blood Bank.

NEPHROLOGY

ACUTE RENAL FAILURE

Defined by elevation of plasma urea and creatinine (in other words a biochemical diagnosis) as 30% of these patients are not oliguric and may even be polyuric. The following are important aspects of the management of acute renal failure.

- Controlling hyperkalaemia.
- Correction of dehydration.
- Relief of urinary tract obstruction.

CAUSES

There are many causes - some important ones are listed below:

- Pre-renal
  - Hypovolaemia or hypotension
- Renal
  - Nephrotoxins including drugs and chemicals.
  - Acute interstitial nephritis.
  - Acute glomerulonephritis.
  - Vasculitides.
  - Haemolytic - uraemic syndrome.
  - Acute-on-chronic renal failure, e.g. polycystic disease, analgesic nephropathy, diabetic nephropathy.
- Post-renal obstruction.
  - **Tubular** - urate or Bence Jones protein.
  - **Ureteric** - single kidney with calculus, bilateral uric acid sludging, retroperitoneal involvement by tumour or fibrosis. Pelvic involvement by carcinoma of bladder or cervix.
  - **Prostatic**.

INVESTIGATIONS

Evaluation of the state of hydration is crucial in the management of these patients. Initially one must assess hydration by means of weight change, blood pressure (lying and standing), and jugular venous pressure or possibly central venous pressure.

- Abdominal, rectal and vaginal examinations to detect a distended bladder, abdominal masses, prostatic enlargement or pelvic masses.
- Urine for microscopy (pus cells, casts), culture, Na, K and osmolality. Ward should do dipstix test for blood, protein and glucose.
- CBC + diff, Na, K, urea, creatinine and coagulation profile. The biochemical tests should be done at least daily. Plasma potassium may be required more often.

- Urinary tract ultrasonography and plain abdominal film to exclude obstruction and calculi and to assess kidney size and echo texture. Renal area tomography may be indicated. Discuss with Radiologist.
- Urgent renal biopsy may be indicated.

MANAGEMENT

- Stop any potentially nephrotoxic drugs.
- Ensure optimal hydration with appropriate fluid - blood, 0.9S or albumin. Central venous pressure monitoring should be done in most patients. When the patient has been rehydrated give 600 ml plus urine output and other losses per 24 hours, either as oral fluid or D5W. Replace sodium losses as 0.9S within this volume.
- Do not give diuretics until the patient has been rehydrated. Then try 40-80 mg IV frusemide and give a double dose in 2 hours if no response. If still no response, give no further frusemide and seek advice.
- Ureteric obstruction - consult urology team immediately.
- Bladder outlet obstruction - suprapubic drainage probably best. Refer to urology urgently.
- Hyperkalaemia. This may be immediately life threatening and should be treated according to its severity.

5.5-6.5 mmol/L	Resonium-A 15-30g PO or PR q6h.
6.5-7.5 mmol/L	As above. Do ECG. Give 25-50 ml 25-50% dextrose IV plus 12 units actrapid insulin IV. Give $NaHCO_3$ 80-120 mmol IV over 30 minutes if not fluid overloaded.
>7.5 mmol/L	As above but also give 10-30 ml 10% calcium gluconate IV. Consult re immediate dialysis.

Salbutamol has been shown to be comparable to insulin and glucose for lowering the plasma potassium, but is not yet recommended as first line management. This can be given in a dose of 20 mg by nebuliser or 0.5 mg by IV infusion over 15 minutes.

- Indications for urgent dialysis:
  - Urea >35 mmol/L.
  - K >7.5 mmol/l.
  - Pericarditis.
  - Cardiac failure or fluid overload.

Note:

Care should be taken with IV line insertion - veins may be required for subsequent fistula formation. Try to use the dominant arm and avoid forearm veins. Avoid radial and brachial artery for blood gas sampling from the non-dominant arm. However, it would be inappropriate if the patients were inadequately resuscitated because good veins were not used due to a fear that they may be required for future vascular access.

Interpretation of urine biochemistry in oliguric patients, without cardiac or liver disease and who have not received diuretics, helps to distinguish pre-renal from renal causes of oliguria.

MEASUREMENT	REVERSIBLE OLIGURIA (PRE-RENAL ARF)	ESTABLISHED OLIGURIA (ARF)
Osmolality (mmol/L)	>500	<400
Na (mmol/L)	<20	>50
Urine/plasma urea	>10-20	<3
Urine/plasma creatinine	>30	<20
Fractional excretion Na		
U/P Na / U/P creatinine x 100%	<1*	>1

* Also in acute glomerulonephritis

DRUGS AND THE KIDNEY

- Most drugs (or their metabolites) used in hospital practice are excreted in whole or in part through the kidneys.
- The dose of most drugs should be modified in patients with renal insufficiency according to the fraction excreted unchanged (fu) and the creatinine clearance (**see page 25**). This is particularly important for drugs such as the aminoglycosides, cephalosporins, cimetidine, ranitidine, digoxin, procainamide, the ACE inhibitors and some of the ß-blockers. Drugs which are mainly metabolised do not usually require dose adjustment in renal insufficiency unless an active metabolite is excreted through the kidneys.
- Some drugs should be completely avoided or used with great care in the presence of renal insufficiency. These include - tetracyclines (except doxycycline), co-trimoxazole, nitrofurantoin, nalidixic acid, K-sparing diuretics (spironolactone, amiloride, triamterene) and the NSAIDs.

The **ACE inhibitors** are widely used for the management of hypertension and cardiac failure. However, many elderly patients may undergo a deterioration in renal function due to an excessive dosage in relation to their renal clearance of the drug. This is more likely to occur if the patients are also taking a diuretic or

NSAID, or get dehydrated from any cause. The recommendations for the doses of captopril and enalapril are based on the fact that for unmetabolised, renally eliminated drugs (or active metabolites), dosages should be reduced in proportion to the reduction in renal function. The recommended doses have been adjusted to suit tablet size, and the dose interval to suit conventional once or twice daily administration. These drugs should be stopped prior to surgery and should not be used in combination with potassium-sparing diuretics potassium supplements, or NSAIDs. **(See page 36 for dosage recommendations).**

ASSESSMENT OF RENAL FUNCTION

- The plasma creatinine alone is not a sufficiently accurate guide to the glomerular filtration rate, particularly for small or elderly patients.
- **Cockcroft and Gault** (Nephron 1976, 16:31-41) developed a simple bedside formula to predict the creatinine clearance without having to collect urine and using the variables of plasma creatinine, body weight, sex and age. The formula is as follows:

$$\text{CrCL (ml/sec)} = \frac{(140\text{-age}) \times \text{lean body weight (kg)}}{50{,}000 \times \text{serum creatinine (mmol/L)}} \times 0.85 \text{ if female}$$

- lean body weight (males) = 50 kg + 0.9 kg for each cm over 150 cm in height.
- lean body weight (females) = 45 kg + 0.9 kg for each cm over 150 cm in height.

This formula has not been tested in infants or young children, but is accurate from the age of 12 years onwards.

If in doubt concerning the prescribing of a drug in a patient with renal insufficiency seek advice.

LOWER URINARY TRACT INFECTIONS

- **Cystitis** is the syndrome of frequency and dysuria. Other lower urinary tract symptoms may, or may not, be present. As many as one-half of all women with this syndrome do not have a bacterial infection and are considered as having non-bacterial cystitis (urethral syndrome). The aetiology of the latter is multifactorial, but acute Chlamydia trachomatis urethritis should be excluded.
- **Bacterial cystitis and asymptomatic bacteriuria** - patients with bacterial cystitis will have typical lower urinary tract symptoms together with pyuria. All pregnant woman should be screened in each trimester for (asymptomatic) bacteriuria. Only about one-half of pregnant asymptomatic patients will have

pyuria (>10 x 10^6 WBC/L) indicating urinary tract inflammation. The prevalence is 5-6% in caucasian woman and 15-18% in Maori/Polynesian woman. These woman are at risk of developing acute pyelonephritis in the last trimester or puerperium. E. coli is the commonest pathogen followed by Staphylococcus saprophyticus (more prevalent in the spring and summer months) and Proteus mirabilis.

DIAGNOSIS

- The diagnosis is confirmed by culturing **either** an MSU specimen **or** urine obtained by suprapubic bladder aspiration. In asymptomatic patients 2 consecutive MSU samples should be obtained before concluding that a UTI is present.
- The current criterion for a significant bacterial count in symptomatic women is $\geq 1x10^6$ colony forming units/litre (cfU/L) of a uropathogen. A count of $>100x10^6$ cfU/L of a uropathogen has a specificity of 0.99, but a sensitivity of only 0.51 in women with an uncomplicated acute UTI. Approximately 30% of women who have lower urinary tract symptoms, pyuria and a good clinical response to antimicrobial treatment have true infection with $10-100x10^6$ cfU/L. The use of a criterion of $\geq 1x10^6$ cfU/L of a uropathogen results in a sensitivity of about 0.90 with little loss of specificity (approximately 0.80).

INVESTIGATIONS

- Always ask the question - **"Is this infection a pointer to some underlying abnormality in the urinary tract?**
- All adult men should have an ultrasound examination of the urinary tract together with a plain abdominal x-ray for kidneys, ureter and bladder with or without renal tomography. If any abnormality is noted an IVU should be considered. A urine flow rate may be appropriate in males in the 'prostatic age group'.
- Women with a UTI only require organ imaging of the urinary tract (as for adult men) if:
  - They had problems prior to the commencement of sexual activity.
  - Have had acute pyelonephritis.
  - The infections have become closely-spaced.
  - Are due to a Proteus species or an unusual organism.
  - Microscopic haematuria persists, or
  - Single dose therapy has failed.
- Cystoscopy should be considered for some males and elderly women.

MANAGEMENT

- For patients with bacterial cystitis or covert bacteriuria a single dose of an appropriate antimicrobial agent is as effective as a conventional 3 day course of the same drug. Because of the high incidence of bacterial resistance,

amoxycillin is no longer a first choice drug. Suggested regimens are:

SINGLE DOSE	3 DAY COURSE
Trimethoprim 600 mg*	Trimethoprim 300 mg q24h
Co-trimoxazole 1.93 g *	Co-trimoxazole 0.96 g q12h
	Nitrofurantoin 50 mg q8h (ineffective for Proteus spp)
Norfloxacin 800 mg	Norfloxacin 400 mg q12h
	Ciprofloxacin 250 mg q12h
Amoxycillin 3 g]	Amoxycillin 250 mg q8h
] results inferior	(Streptococcus faecalis may be
Cephalexin 3 g] to above drugs	only indication now)

* Co-trimoxazole should only be used if indicated on bacterial sensitivity testing. Trimethoprim is just as efficacious as co-trimoxazole in the urinary tract and has a lower incidence of side effects.

♦ **Follow-up** - ideally all patients should have a urine specimen taken for culture 7-14 days after completing treatment.

Prophylactic therapy for patients with recurrent urinary tractinfections

Patients with recurrent UTIs (e.g. >3 in 6 months) with normal renal function and a normal urinary tract merit consideration for low dose prophylactic antimicrobial therapy.

♦ Try simple measures - treatment of cervical erosion or vaginitis, increase in fluid intake, increase the frequency of micturition, post coital voiding, application of an antiseptic cream to the periurethral area prior to intercourse (e.g. 0.5% cetrimide + 0.1% chlorhexidine in the form of Savlon).

♦ Drugs which have been shown to be effective in prophylactic regimens include:
- Nitrofurantoin 50 mg]
- Trimethoprim 100 mg] preferred agents.
- Co-trimoxazole 240 mg (½ tablet).
- Norfloxacin 200 mg.
- Hexamine hippurate 1 g (less effective).

The above drugs should be taken after emptying bladder and before retiring. If patients have renal insufficiency cephalexin 125 or 250 mg or cephradine 250 mg can be used for prophylaxis.

♦ Prophylactic treatment should only be started after a UTI has been treated with a curative course of therapy and the post-treatment culture is sterile.

Prophylactic treatment should be continued for at least 3 and preferably 6 months, although the patient may wish to continue for longer.

- Recent trials have shown that a dose on alternate nights, 3 nights a week or even just after intercourse may be equally efficacious.

ACUTE PYELONEPHRITIS

- A syndrome of fever (>37.8°C) ± rigors, loin pain or tenderness together with infected urine.
- Lower urinary tract symptoms may be absent.
- Symptoms may be unilateral or bilateral.
- Patients with severe acute pyelonephritis require hospitalisation.
- One third will have a bacteraemia.

CAUSES

- Acute pyelonephritis may occur in a structurally normal urinary tract or as a complication of some underlying urinary tract disorder (i.e. it may be uncomplicated or complicated).

INVESTIGATIONS

- The clinical features are usually clear-cut, but the diagnosis must be confirmed bacteriologically. In a patient with acute pyelonephritis approximately 80% will have a colony count >100 x 10^6 colony forming units per litre (cfu/L), 10-15% will have 10-100 x 10^6 cfu/L and the remainder will have small numbers of uropathogens on culture of a midstream urine specimen. Significant pyuria (>10x10^6 white cells/L) will invariably be present. To eliminate the problem of contamination of voided urine samples it is preferable to obtain urine by suprapubic aspiration from the distended bladder. A specimen obtained in this manner can be examined microscopically and if any organisms are seen then treatment can be started at once. Moreover a suprapubic aspirate may be stored for several hours and still allow an accurate bacteriological diagnosis to be made.

- Rectal and vaginal examination should be done.
- CBC + diff.
- Na, K and creatinine.
- Blood cultures.
- Serum protein electrophoresis if over 50 years of age.
- A cystoscopy may very occasionally be indicated.

All patients with acute pyelonephritis should have an ultrasound examination of the urinary tract together with a plain abdominal x-ray (kidneys, ureter and bladder) with or without renal tomography. If any abnormality is noted an intravenous urogram should be considered. A DMSA scan may also be helpful.

MANAGEMENT

If the patient is dehydrated and/or vomiting, give IV 0.9S.

Parenteral antimicrobial therapy should be given initially, and continued until the patient can tolerate oral medication. Treatment should be continued for 5 days, although shorter courses may be successful. Parenteral ampicillin or amoxycillin should not be used, at least until the antibacterial sensitivity profile is known, as about 50% of E.coli locally are now resistant to these antibiotics.

The choices of **parenteral** agents are:

- Gentamicin **(see page 27 for dosage guidelines).**
- A 4-quinolone e.g. ciprofloxacin 100 mg q12h.
- A ß-lactam drug, e.g. ceftriaxone 2 g q24h, aztreonam 1 g q12h.

After the patient has improved and is taking food and fluids a switch to oral antimicrobial therapy should be considered. An advantage of the 4-quinolones is that some of these agents are available in both parenteral and oral formulations and a switch can often be made from IV to oral treatment within 24-48 hours, enabling the patient to go home earlier.

The urine should be recultured 7-14 days after completion of therapy.

NEUROLOGY

NEUROLOGICAL EXAMINATION

Some points to remember are:

- **Dilatation of the pupils** by mydriatic drops should be avoided in neurologic patients, particularly those who are ill.
- **Abbreviated Mental Test Score (MSQ)** - each question scores one mark.

  A quick routine test of mental function is recommended, particularly in the elderly:

  - Age.
  - Time (to nearest hour).
  - Address for recall at end of test - this should be repeated by the patient to ensure it has been heard correctly: e.g. 42 West Street.
  - Year.
  - Name of hospital.
  - Recognition of 2 persons (doctor, nurse, etc).
  - Date of birth.
  - Year First World War started.
  - Name of present Monarch.
  - Count backwards 20-1.

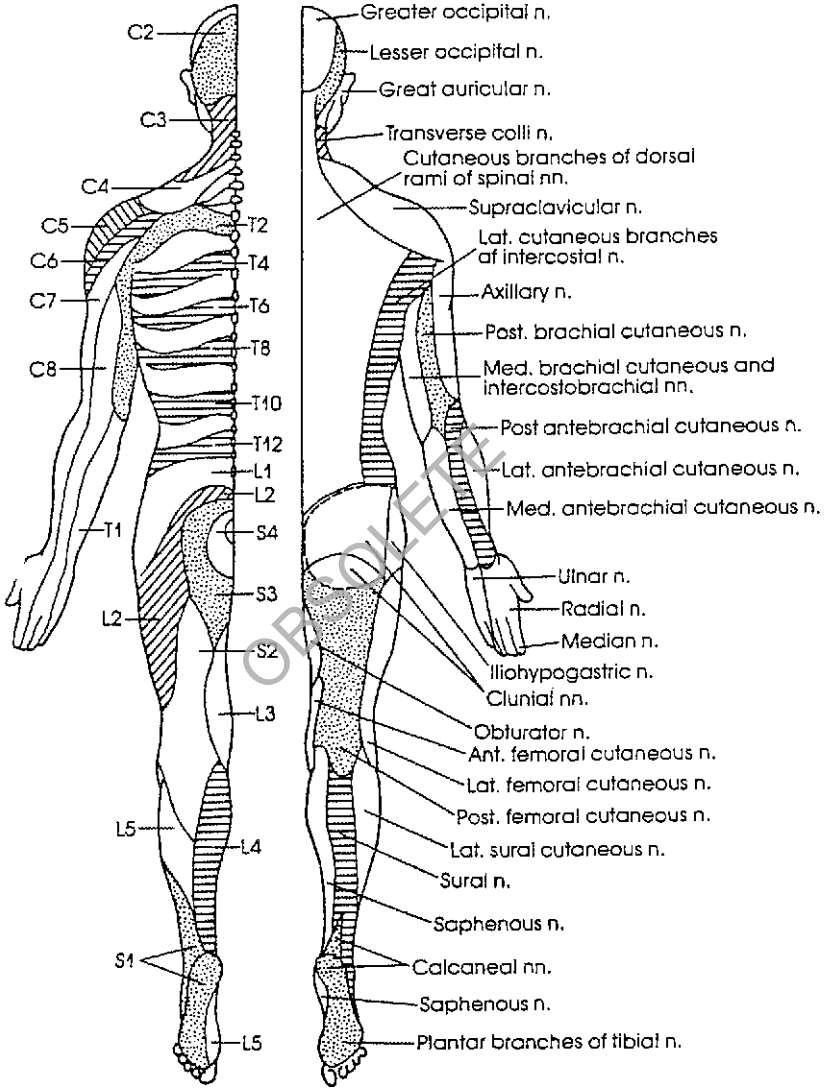
Note:
Although useful, this test is insensitive and mentation has to be severely affected before it is abnormal.

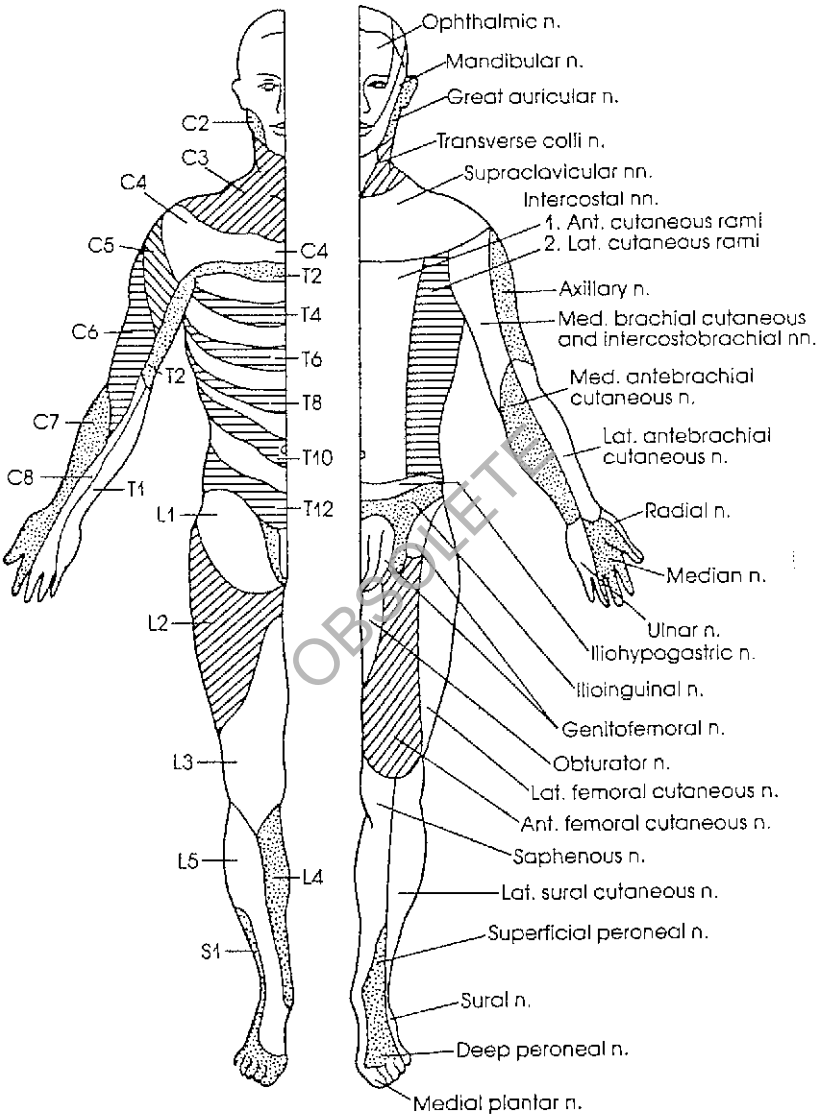
- **The principal spinal segments** responsible for the most commonly tested reflexes are:

Biceps jerk	:	C5, 6 (Musculocutaneous nerve)
Brachioradialis reflex	:	C5, 6 (Radial nerve)
Triceps jerk	:	C7, 8 (Radial nerve)
Knee jerk	:	L3, 4 (Femoral nerve)
Ankle jerk	:	S1, 2 (Tibial nerve)

- **The segmental innervation of the skin** is illustrated on the next two pages. This can be more readily recalled by remembering certain "key" dermatomal levels e.g.:

C8	:	middle finger	L1	:	groin
T4	:	nipples	L3	:	knee
T10	:	umbilicus	Sacral	:	buttocks


C2
C3
C4
C5
C6
C7
C8
T1
T2
T4
T6
T8
T10
T12
L1
L2
S4
S3
S2
L3
L4
L5
S1
L5
L2
Greater occipital n.
Lesser occipital n.
Great auricular n.
Transverse colli n.
Cutaneous branches of dorsal rami of spinal nn.
Supraclavicular n.
Lat. cutaneous branches of intercostal n.
Axillary n.
Post. brachial cutaneous n.
Med. brachial cutaneous and intercostobrachial nn.
Post antebrachial cutaneous n.
Lat. antebrachial cutaneous n.
Med. antebrachial cutaneous n.
Ulnar n.
Radial n.
Median n.
Iliohypogastric n.
Clunial nn.
Obturator n.
Ant. femoral cutaneous n.
Lat. femoral cutaneous n.
Post. femoral cutaneous n.
Lat. sural cutaneous n.
Sural n.
Saphenous n.
Calcaneal nn.
Saphenous n.
Plantar branches of tibial n.


C2
C3
C4
C5
C6
C7
C8
T1
T2
C4
T2
T4
T6
T8
T10
T12
L1
L2
L3
L4
L5
S1
Ophthalmic n.
Mandibular n.
Great auricular n.
Transverse colli n.
Supraclavicular nn.
Intercostal nn.
1. Ant. cutaneous rami
2. Lat. cutaneous rami
Axillary n.
Med. brachial cutaneous and intercostobrachial nn.
Med. antebrachial cutaneous n.
Lat. antebrachial cutaneous n.
Radial n.
Median n.
Ulnar n.
Iliohypogastric n.
Ilioinguinal n.
Genitofemoral n.
Obturator n.
Lat. femoral cutaneous n.
Ant. femoral cutaneous n.
Saphenous n.
Lat. sural cutaneous n.
Superficial peroneal n.
Sural n.
Deep peroneal n.
Medial plantar n.

STROKE

CAUSES

- Infarction:
  - Thrombotic.
  - Artery - artery embolism (from atherosclerotic plaque).
  - Cardio - embolic (atrial fibrillation is the most common).
  - Lacunar (common in chronic hypertension).
- Cerebral haemorrhage (hypertensive, coagulation disorders, haemorrhagic infarction, A-V malformation).

INVESTIGATIONSAND MANAGEMENT

This depends on the character of the initial illness and other factors such as age.

Completed strokes - investigations as listed below unless embolic or due to a non-atheromatous disease such as arteritis. This will require further tests. Rehabilitation will be needed.

Cardioembolic stroke - when there are strong clinical or laboratory grounds for believing that stroke has occurred from cardiogenic embolism anticoagulants may need to be considered. The risk of re-embolism must be weighed against that of converting bland infarction into cerebral haemorrhage. The larger the infarct the greater the risk of bleeding from anticoagulants. CT is essential prior to treatment. If the infarct is small or moderate in size, non-haemorrhagic and the patient normotensive, anticoagulants could be commenced early. In this case, repeat CT scan at 48 hours; if there is no evidence of any bleeding, it would be reasonable to anticoagulate. If infarct is large, immediate anticoagulation is likely to carry an unacceptably high risk and should be delayed for 7-10 days. Repeat the CT scan at this time; if there is no evidence of bleeding, it would be reasonable to anticoagulate. Large embolic infarcts carry a risk of spontaneous haemorrhagic transformation unrelated to anticoagulants. This may not be present on early CT but may occur later, despite no obvious change in the patient's clinical state. Neurology consultation may be appropriate.

Stroke in evolution - this clinical picture can be produced by causes other than progressive intra-arterial thrombosis, such as tumour and even subdural haematoma. CT is, therefore, essential to establish the cause and to exclude haemorrhage if anticoagulants are being considered. Neurology consultation may be necessary for some cases. In the progressing case, IV anticoagulants may be appropriate. If CT shows haemorrhage, however, this may warrant surgical evacuation. This is especially so if haematoma involves the cerebellum. Urgent neurosurgical consultation may be appropriate for these cases.

Transient ischaemic attacks - defined here as a neurological deficit which resolves completely within 24 hours.

- Treat hypertension if moderate or severe. Drugs not causing postural hypotension are preferred.

Vertebro-basilar territory	- Exclude subclavian steal: take BP in both arms. - Aspirin 300 mg daily. - Consider oral anticoagulants if attacks still occur, with extra care to control blood pressure in this situation.
Carotid territory	- Aspirin 300 mg daily and consider carotid ultrasonography if patient is otherwise a suitable candidate for endarterectomy.

- **Investigations** appropriate to all 4 problems may include:-
  - CBC (polycythaemia, thrombocytosis).
  - ESR (arteritis).
  - Syphilis serology.
  - Na, glucose, Ca, albumin, creatinine.
  - ECG.
  - CXR (heart size, malignancy).
  - Blood lipids.
  - Anti-nuclear antibodies.
  - Serum protein electrophoresis.
  - Echocardiogram.
  - Coagulation profile.
- Some patients with completed stroke or TIA's will need a CT scan for accurate diagnosis.

SUBARACHNOID HAEMORRHAGE

CAUSES

In descending order of frequency:-

- Intracranial aneurysm.
- Intracranial arterio-venous malformation.
- "No cause found" usually associated with systemic hypertension with subsequent negative intracranial angiography.
- Haemorrhage from an intracranial tumour.
- Coagulation disorder (usually iatrogenic).

Intracranial aneurysm as a cause of subarachnoid haemorrhage has a peak incidence of rupture between 40-60 years of age. Intracranial arterio-venous malformation has a peak incidence of haemorrhage between 20-40 years of

age. The mortality of the first bleed of an intracranial aneurysm is approximately 30% and the mortality of an early rebleed is at least 40%. The mortality of the first and probably subsequent bleeds of an arterio-venous malformation is approximately 10%.

- 5% of ruptured aneurysms rebleed within the first 24 hours and by 14 days a total of 20% have rebled.
- Approximately 30% of patients surviving a ruptured aneurysm, where the aneurysm is not treated surgically, will be alive at the end of 12 months, the deaths occurring from rebleeding.

SPECIFIC INVESTIGATIONS

- The patient should undergo a CT Scan as soon as possible after haemorrhage. CT scanning within the first 3 days has a high positive yield for subarachnoid blood and gives added information as to the possible site of the ruptured aneurysm and also ventricular size.
- Lumbar puncture is **contra-indicated** if the patient has an impaired conscious level, or has significant lateralizing neurological signs. This group of patients **must be assessed by CT scan** as the likelihood of an intracerebral haematoma is high. Such a haematoma may require evacuation as it may be life-threatening.
- In general terms however it is quite safe to perform a diagnostic lumbar puncture if the patient is conscious, alert and orientated with no lateralizing neurological signs.
- Once the diagnosis of subarachnoid haemorrhage has been made the patient will require intracranial angiography and early neurosurgical intervention in the form of a craniotomy to clip the ruptured aneurysm or to remove an arterio-venous malformation.

TREATMENT GUIDELINES

- Complete bedrest.
- Adequate analgesia (paracetamol, low dose parenteral narcotics e.g. morphine 5-7.5 mg IM).
- Intravenous fluids to ensure adequate hydration.
- Avoid straining. Anti-emetics if needed plus stool softeners.
- Raised blood pressure should be treated only if:
  - The patient was already on anti-hypertensive drugs before the haemorrhage.
  - The diastolic blood pressure is greater than 100 mmHg for several hours, in the absence of any evidence of high intracranial pressure. Avoid hypotension or large swings in blood pressure.
  - Sublingual nifedipine recommended.

CHRISTCHURCH ADMISSION ARRANGEMENTS

- All patients under the age of 60 in whom there is a strong clinical suspicion of subarachnoid haemorrhage should be admitted under the Neurosurgeon of the day.
- Patients over the age of 60 in whom there is a strong clinical suspicion of subarachnoid haemorrhage, and who are conscious, alert and orientated, should be admitted under the General Medical Team of the day who should then subsequently obtain a neurosurgical or neurological opinion as to further management.
- Patients between the ages of 60-75 in whom there is a strong clinical suspicion of subarachnoid haemorrhage **and who in addition** have a deteriorating conscious level, or who are unconscious, or who have significant lateralizing neurological signs should be admitted primarily neurosurgically or alternatively neurologically. This group of patients will need urgent CT scanning to exclude a life-threatening intracerebral haematoma which may require urgent neurosurgical evacuation.

STATUS EPILEPTICUS

DEFINITION

- Prolonged seizures or failure to regain consciousness between seizures.

CAUSES

- Commonest cause is anticonvulsant withdrawal or non-compliance in a known epileptic.
- If the patient is not known to be epileptic consider whether the cause is primarily intracranial or secondary to some toxic-metabolic disorder. Remember alcohol withdrawal, hypoglycaemia and infection.

INVESTIGATIONS

CBC and diff, Na, K, glucose, and anticonvulsant concentrations if aetiology unclear. Arterial blood gases. ECG.

MANAGEMENT

- Ensure adequate airway and oxygenation. Endotracheal intubation may be necessary. Respiration is impaired by continued seizures, and by many anticonvulsants. If status is not stopped promptly patient should be transferred to ICU.
- Insert IV line and give diazepam 5-10 mg IV over 2-3 minutes. Give further 5 mg increments if needed but stop if respiratory or cardiovascular depression.

- If no past history of epilepsy consider thiamine 100 mg IM followed by IV bolus of 50 ml 50% dextrose.
- If patient is not already receiving anticonvulsants start phenytoin. Place 15 mg/kg phenytoin in 100 ml 0.9S and give IV over 30 minutes but no faster than 50 mg/min. Continuous ECG monitoring and BP every 15 minutes. Thereafter maintenance phenytoin 100 mg q6-8h IV, orally or via NG tube. In the elderly, those who are hypotensive, or those with cardiac disease, it may be safer to administer all or part of the above loading dose via NG tube.

  Phenytoin precipitates in all IV solutions **except** 0.9S because of pH incompatibility. Phenytoin also precipitates in tissues if given IM so this route is contraindicated.
- If seizures persist while awaiting a therapeutic response from phenytoin give diazepam infusion, 50 mg in 500 ml 0.9S q4-6h or depending on response.
- If the patient is already known to be on anticonvulsants, obtain urgent blood levels. If the level is subtherapeutic continue to administer the same drug in modified dose. If the anticonvulsant level was "therapeutic" at presentation start on a second drug. Choices include phenytoin, phenobarbitone IM, carbamazepine or sodium valproate.
- Sedation and respiratory depression may result. Early ICU consultation and transfer may be necessary.

FURTHER POINTS

- Once status is controlled, it is mandatory to establish its cause. CT, LP, drug screen, EEG, CXR may be required if underlying cause is not obvious.
- Do not use IM diazepam or IM phenytoin in the treatment of epilepsy - absorption is erratic and unpredictable.
- The simplest way of managing status is with one anticonvulsant (in addition to diazepam) rather than using multiple drugs. Ensure full dosage with adequate blood levels, before discounting a drug as ineffective.

EPILEPSY : PATIENTS PRESENTING WITH THEIR FIRST SEIZURE

DIAGNOSIS

- Is clinical. Diagnosis is established on the basis of the patient's account and on the eye-witness description. A detailed neurological examination is required.

INVESTIGATIONS

- CBC + diff, ESR.
- Glucose, Na, Ca, creatinine, AST, GGT, alk. phos., Bili.
- Syphilis serology.
- CXR.

- EEG useful if diagnosis is in doubt. It may give a clue to the type of epilepsy (e.g. distinguishes the "absence" of temporal lobe epilepsy (TLE) from that of Petit Mal) and may show a lateralised abnormality.
- CT indicated if:
  - Focal features to the seizure (including TLE).
  - Focal features on clinical examination.
  - Focal features on EEG.
  - The epilepsy presentation is as status epilepticus.
  - The epilepsy is of adult onset (over 20 years) and not of long standing or of known underlying cause (e.g. previous severe head injury).

TREATMENT

- Most patients following a single seizure, who have recovered and are well, do not require hospital admission.
- Generally, do not commence anticonvulsant therapy following a single seizure if no obvious underlying cause is demonstrated. If a second seizure occurs within 1 year, commence treatment.
- If the seizure occurs as the result of a focal structural lesion in the cerebral hemisphere, commence anticonvulsants after the first fit.
- Phenytoin, carbamazepine or sodium valproate are the drugs of first choice for grand mal epilepsy.Carbamazepine is probably the optimal drug for TLE (complex partial seizures) with either carbamazepine or phenytoin being the drugs of first choice for focal (partial) seizures.
- Do not discount one anticonvulsant drug as being ineffective without having shown that therapeutic blood levels have been achieved.
- All patients should be advised against driving a motor vehicle for 12 months even after a single seizure. Patients should also be advised of potential risk of swimming alone, working at heights and other high risk activities.
- Phenytoin has dose dependent kinetics with a rapid rise in level for small increase in dosage. Therefore use 30-50 mg daily dosage increments when blood level is near the therapeutic range. Adult dose around 300 mg/day may be given in a single dose, provided adequate blood levels are present just before next dose is due.

RAISED INTRACRANIAL PRESSURE

CAUSES

- Include intracranial mass effect, obstruction to the flow of CSF, and brain swelling.

INVESTIGATIONS

- CT is mandatory to establish cause. Note that papilloedema may be a late sign of raised ICP or may never appear. Do not dilate pupils.

MANAGEMENT

- Consult Neurosurgeon/Neurologist.
- Close observation with 15-30 minutes neurological recordings may be needed in drowsy or deteriorating patients. This will require a special nurse. Do not however, substitute observation for action since this may be needed urgently.
- Depending upon clinical circumstances, consider dexamethasone 4 mg PO q6h, especially if a tumour is present. Give dexamethasone 12 mg IV stat if the patient is drowsy. If the mental state declines further the patient may need Mannitol 1-2 g/kg IV over 20-30 minutes (500 ml of 15% Mannitol contains 75 g). In an acute situation consider hyperventilation while awaiting neurosurgical intervention.

Note:

Lumbar puncture - never perform a lumbar puncture if a patient may have raised intracranial pressure. Always obtain a CT scan first. Clues to the presence of raised ICP include the following - lateralising (focal) features, focal seizures, drowsiness and papilloedema. When bacterial meningitis is strongly suspected, but features consistent with raised ICP are present other than drowsiness alone, it may be appropriate to administer intravenous antibiotics immediately. Then arrange for urgent CT scan, and then lumbar puncture for CSF examination if CT does not reveal a mass lesion.

ENCEPHALITIS

CLINICAL FEATURES

These usually include fever, meningism and signs of involvement of the brain. The latter signs most commonly include confusion, altered conscious level, seizures, myoclonus, papilloedema, or focal signs reflecting involvement of one or both hemispheres.

CAUSES

- **Herpes Simplex virus** - this is the most urgent to identify as it requires immediate therapy. It often produces a rapid onset illness with little prodrome. Cutaneous herpetic lesions are uncommon.
- **Endemic viruses** - mumps, measles, rubella, chickenpox, adenovirus, enteroviruses, EBV, CMV, HIV.
- **Travel related viruses** - many severe viral encephalitides are transmitted by biting insects and are found in USA, Asia, Africa and Europe.

INVESTIGATIONS

A number of disorders may mimic encephalitis and many will require exclusion by appropriate tests. Most common are meningitis, severe sepsis, cerebral

tumour, or one of the many causes of toxic metabolic encephalopathy. (**See page 122 - Altered Level of Consciousness**). The differential diagnosis includes brain abscess, listeria, nocardia, toxoplasmosis, mycoplasma, tuberculosis, malaria and syphilis.

- CT/MRI - to exclude other diseases mimicking encephalitis and to aid in the diagnosis of HSV encephalitis.
- CSF - routine culture (viruses, TB, bacteria and fungi), biochemistry and microscopy. Cell counts almost always show lymphocytic pleocytosis. A normal result casts some doubt on the diagnosis. Take enough CSF for PCR testing (consult microbiology) if HSV suspected.
- CBC + diff, Na, K, Ca, glucose, urea, creatinine, AST, GGT, alk. phos. Bili.
- Blood cultures, throat swabs (bacteria and viruses), stool culture for viruses, serum for storage, monospot.
- CXR.
- EEG - this is not specific but is almost always abnormal in encephalitis. The finding of periodic complexes may be of more specific help when HSV is suspected.
- Brain biopsy - may be necessary.

TREATMENT

- HSV - the diagnosis is only clearly established by brain biopsy. The need for this is a matter for clinical judgement. (Suggest Neurology and/or Infectious Diseases consult).
- For HSV give acyclovir 10 mg/kg IV, q8h for 10 days. Treatment is urgent. Outcome correlates with level of consciousness at commencement of therapy.
- Dexamethasone may be appropriate for selected cases of both herpetic and non-herpetic encephalitis.
- Anticonvulsant therapy will be necessary in some patients.
- Close neurological observation to detect signs of increasing intracranial pressure.

SPINAL CORD COMPRESSION

CAUSES

- Trauma.
- Tumour - extrinsic / intrinsic.
- Haemorrhage.
- Extra-dural abscess.
- Disc prolapse.

INVESTIGATIONS AND MANAGEMENT

- **Remember that quick action may avoid irreversible paraplegia.**
- The urgency is dictated by the duration, the rate of progression, and the degree of the neurological deficit. Try to establish the level of cord involvement.
- X-rays of spine at the appropriate level and a CXR should be done.
- If recent onset, rapid progression, and/or significant neurological deficit, for immediate **(i.e. at once)** neurological/neurosurgical consultations re MRI or myelography ± CT scanning. **DO NOT** do a lumbar puncture before MRI / myelogram is performed.
- CBC + diff, ESR, Na, glucose, K, Ca, creatinine, AST, GGT, alk. phos., Bili, albumin, and may need serum protein electrophoresis and prostate specific antigen. Blood cultures. Urine for Bence Jones Protein. In selected patients a search for a specific malignancy, e.g. multiple myeloma or lymphoma, should be carried out and some of the above tests may be helpful in this regard.
- Remember that in some tumours, e.g. myeloma, a non-surgical approach with radiotherapy and/or chemotherapy may be the treatment of choice. Urgent consultation with a Haematologist or Oncologist is recommended.
- Catheterize if urinary retention present.
- Regular turning to avoid pressure sores.
- If patients with a known malignancy develop spinal cord compression it is desirable that the doctors who have been supervising their care be contacted immediately.

SUBDURAL HAEMATOMA

- A high index of suspicion is the key to diagnosis, especially in the elderly, in chronic alcoholics, and patients on anticoagulants.
- A preceding history of trauma is not necessary for the diagnosis to be considered.
- Consider a subdural haematoma if there is a:
  - History of headache plus progressive clouding of consciousness and localizing signs.
  - Clinical picture of headache, intellectual change, alteration in alertness, and signs of bilateral hemisphere dysfunction.
- It is uncommon for gross unilateral focal signs to be present e.g. a dense hemiplegia in an alert patient is unlikely to be due to a subdural.
- Fluctuation of signs from day to day or hour to hour is not uncommon.
- Diagnostic errors are common. Most frequent misdiagnosis is stroke.

INVESTIGATION AND MANAGEMENT

- CT scan.
- Commence neurologic recordings and consult Neurosurgeon for further advice on management.

♦ CBC + diff and coagulation profile.

ALTERED LEVEL OF CONSCIOUSNESS

Distinguish **anatomic** and **metabolic** causes. "Metabolic" implies any disorder which has a diffuse effect on cerebral metabolic pathways.

CAUSES

A ANATOMIC

Supratentorial

- Extradural or subdural haematoma.
- Cerebral - haemorrhage, infarction, abscess, cyst or tumour.
- Subdural empyema.
- Hydrocephalus.

Subtentorial

- Brainstem/cerebellar - infarction, haemorrhage, tumour, abscess or cyst.
- Rarer causes - brainstem demyelination, extradural and subdural haematomas.

B METABOLIC

- Drugs - e.g. alcohol, hypnotics, psychotropics, aspirin.
- Hypoglycaemia.
- Hypoxia/ischaemia - e.g. shock, cardiac arrest, syncope, carbon monoxide.
- Electrolyte or acid/base disturbance - e.g. acidosis, alkalosis, hyponatraemia, hypercalcaemia, hypercapnia, hyperosmolar coma.
- Encephalopathies - hypertensive, toxic (e.g. Reye's syndrome), hepatic, renal failure.
- Endocrine e.g. hypopituitarism, pituitary apoplexy, hypothyroidism, hypoadrenalism.
- Thiamine deficiency.
- Hypothermia/hyperthermia.
- Acute delirium (e.g. alcohol withdrawal, post-op).

C OTHER

- Head injury.
- Epilepsy/post-ictal.
- Hysteria.
- Sub-arachnoid haemorrhage.
- Infection - encephalitis/meningitis, septicaemia, typhoid, malaria.
- Miscellaneous - cerebral vasculitis, thrombotic thrombocytopenic purpura, basilar migraine, venous thrombosis.

Note:

Obtaining an accurate history is vital - this may have to wait until general supportive care has been commenced - contact relatives, GP, friends.

EXAMINATION

General

- Look for evidence of head injury, IV drug abuse, signs of chronic illness, gum hypertrophy (phenytoin).
- Temperature:
  - Remember hypothermia / hyperthermia.
  - Use high (up to 42°C) or low (down to 25°C) reading thermometers if necessary.

Neurological

- Level of consciousness - use 15 point Glasgow Coma scale which scores motor activity, verbal performance, eye-opening. (See Neurosurgery observation sheet, MR99 and **see page 12**).
- Respiratory pattern:
  - Cheyne-Stokes respiration suggests bilateral cerebral hemisphere dysfunction.
  - Hyperventilation - central neurogenic type due to pontine lesion is rare; most hyperventilation is due to pulmonary congestion (aspiration, infection, neurogenic pulmonary oedema) or acidosis.
  - Apneustic breathing (end-inspiratory pauses) generally accompanies pontine lesions.
- Look for neck stiffness - meningitis, subarachnoid haemorrhage, coning of cerebellar tonsils.
- Neuro-ophthalmic exam:
  Pupil size/equality/light reactivity:
  If entirely normal, implies midbrain intact, and cause of coma likely to be metabolic.
  - Enlarged (>5 mm) and unreactive pupil(s) suggest either a midbrain lesion (intrinsic or secondary to compression) or ipsilateral III nerve lesion. Exclude other causes such as mydriatic eye drops, anticholinergic drugs, or orbital trauma.
  - Bilateral pinpoint (<1 mm) pupils suggests bilateral pontine lesions, opiate overdose, or meiotic eye drops for glaucoma.
  - Horner's syndrome.

    Note:

    Pupillary pathways are relatively resistant to metabolic insults with the exception of drugs and anoxia.

Eye Movements:

- Spontaneous, conjugate, roving movements suggests midbrain and pons intact and favours bilateral hemisphere dysfunction. Lateral deviation of eyes suggests either an ipsilateral frontal lobe, or contralateral pontine lesion. Except for mild ocular divergence, dysconjugate ocular deviation

suggests structural brainstem lesion if pre-existing strabismus excluded. Eyes that are directed straight ahead have no localising value.

- Oculocephalic (Doll's eye) response (avoid if neck injury suspected). If normal, implies integrity of the brainstem. Faulty adduction of an eye suggests internuclear ophalmoplegia or third nerve lesion (look for pupil abnormalities). Faulty abduction of an eye suggests VI nerve lesion (pontine lesions or effect of raised ICP). Oculocephalic responses are generally normal in hemisphere lesions. Interpret subtle abnormalities of the Doll's eye manoeuvre with caution.

  Note:

  Caloric testing (at least 20 ml iced water irrigation of ears) is useful as a substitute for dolling if neck injury is suspected or in diagnosing psychogenic unresponsiveness. The expected response in an (intact) unconscious person is tonic conjugate deviation of the eyes towards the syringed ear.

- Motor function
  - Observe responses to noxious stimuli applied to nailbeds, sternum or supraorbital ridges.
  - Normal responses include withdrawal of limb +/- grimace/groan, and implies intact sensory and motor pathways to and from cortex. Note that adduction/flexion of a limb can occur at spinal reflex level.
  - Abnormal responses include several stereotyped postures of limbs, **decorticate posturing/rigidity** (flexion of elbows and wrists, leg extension) and **decerebrate posturing/rigidity** (extension of arms and legs). Decorticate posturing generally carries a less serious prognosis and is associated with more rostral supratentorial lesions. Decerebrate posturing is often associated with brainstem or diencephalic injury. Note that these patterns are often incomplete, variable, and can interchange. Both may accompany metabolic coma.
  - Look for any asymmetry of limb movement or reflexes which would favour an anatomic lesion. (Hypoglycaemia is however a well described metabolic cause of focal neurologic signs).
  - The presence of asterixis or myoclonus favours a metabolic disorder.

INVESTIGATIONS

- CBC + diff.
- Glucose, Na, K, osmolality, Ca, urea, creatinine, AST, GGT, alk. phos., Bili.
- Arterial blood gases.
- Blood cultures x 2.
- Drug levels (consider gastric lavage).
- CT scan if structural lesions suspected.
- If meningitis a possibility, give antibiotics, do CT, then lumbar puncture if safe to do so.
- EEG may be considered to identify psychogenic unresponsiveness or partial complex epilepticus.

EMERGENCY MANAGEMENT

- Establish airway (intubate if necessary).
- Establish IV line.
- Monitor vital signs.
- Give thiamine 100 mg IM if malnourished or alcoholic.
- Give dextrose 50 ml of a 50% solution IV (if diagnosis not immediately apparent and not hyperglycaemic on BM strip).
- If severe acidosis (pH <6.9) give bicarbonate 50-100 mmol over 1 hour and review.
- Control seizures **(see page 116).**
- Lower intracranial pressure **(see page 118).**
- Naloxone 0.4 IV and repeat 0.4 mg IV in 5 minutes. If opioid overdosage likely up to 2.0 mg Naloxone may be given.

  Note:

  Subsequent management depends on making a specific diagnosis.

ONCOLOGY

Oncology specialists are willing to give advice at all times concerning the management of patients with malignant disease. During normal working hours phone Extension 80020, Christchurch Hospital. After hours or at weekends an Oncologist is on call and can be contacted through the telephone operator at Christchurch Hospital.

It is Departmental policy to re-admit under our care patients who are undergoing active treatment. Remember that any patient who is receiving chemotherapy and/or radiation treatment and who is febrile and neutropenic constitutes a medical emergency.

POTENTIALLY CURABLE MALIGNANCIES

Early discussion or referral to an Oncologist (or Haematologist if appropriate) is recommended for any patient with a potentially curable malignancy. This facilitates investigations, staging, and initiation of treatment. Please do not wait until all investigations or histology reports are complete. All require specialist consultation for staging and treatment.

- Testicular cancer.
- Germ cell tumours - ovary, extragonadal, retroperitoneal and mediastinal.
- Gestational trophoblastic malignancy including choriocarcinoma.
- Undifferentiated cancers, especially in young patients.
- Any malignancy in children (refer Paediatrician) or teenagers.
- Osteosarcoma, Ewing's sarcoma and other sarcomas.
- Leukaemias.
- Lymphomas - Hodgkins and non-Hodgkin.
- Early stage head and neck cancer, cervical cancer and prostate cancer.

SUPERIOR VENA CAVAL OBSTRUCTION

This condition may evade medical diagnosis for several weeks and may be advanced by the time the patient presents. Speed in consultation with an Oncologist, or Haematologist is recommended as in more than 95% of cases, superior vena caval obstruction is due to an underlying malignancy, usually carcinoma of the bronchus or malignant lymphoma. Response to radiation and/or chemotherapy is suboptimal if thrombosis has occurred, consequently we recommend urgent referral, **before** biopsy material is obtained.

HYPERCALCAEMIA

A high proportion of hypercalcaemic patients will have an associated underlying malignancy, the commonest being breast cancer, lung cancer and myeloma. Some 10-20% of patients with cancer will become hypercalcaemic at some

time during their course. Notably, some malignancies which may be cured or have a prolonged remission may present with hypercalcaemia. Therefore, any patient with hypercalcaemia who either has or is suspected of having an underlying malignancy should be referred promptly to an Oncologist or Haematologist.

The measures described in the Endocrinology section **(see page 66)** will ameliorate the hypercalcaemia, but the underlying cause must also be treated. The hypercalcaemia of most malignancies will not be controlled satisfactorily unless the cancer is treated specifically. In particular, the hypercalcaemia associated with myeloma, breast cancer, or lymphoma often resolves within 24-48 hours of specific chemotherapy. It is unwise to try to control hypercalcaemia in isolation and early consultation is recommended.

INAPPROPRIATE SECRETION OF ADH

There are a number of causes of this condition **(see page 64)** for a list of the causes and the recommended treatment). In malignant disease the most common cause of this and the other paraneoplastic syndromes is bronchogenic carcinoma. Small cell bronchogenic carcinoma is the most common associated tumour and this is, in the short term, eminently treatable and consultation is advised.

SPINAL CORD COMPRESSION

Early recognition and treatment are necessary for successful management of spinal cord compression. A high index of suspicion is required and pain at the site of the compression usually occurs before weakness and sensory loss. Tumours causing compression are extradural in the vast majority of cases and are most frequently metastatic from lung, breast, prostate or associated with deposits of lymphoma or myeloma **(see page 120)**.

NEUTROPENIC SEPSIS

This may occur in patients receiving chemotherapy or irradiation treatment. It may also occur in patients with bone marrow infiltration by malignancy. It is most commonly seen during intensive combination therapy used for curable malignancies and can be rapidly fatal. If the patient is under the Oncology or Haematology Units then the appropriate on call Consultant must be contacted. For management see section on Neutropenic Sepsis **(page 81)**, and the section on Septicaemia **(page 88)**.

URETERIC OBSTRUCTION

A number of malignancies may obstruct the ureters, and this is life threatening if it is bilateral or if it involves the only functioning kidney. Obstruction can be relieved by appropriate treatment of a known malignancy by hormones,

chemotherapy or irradiation. Sometimes temporary surgical decompression by nephrostomy or stent is necessary in responsive malignancies. An urgent ultrasound may be necessary for diagnosis.

LYMPH NODE OR OTHER TISSUE BIOPSIES

Consider, **before** such biopsies are organized, whether any additional tests will be needed on the material **other than** routine diagnostic histology. For example, if lymphoma is a possibility, fresh tissue may be needed for surface marker, cytogenetic or DNA analyses. Other samples may need to be cultured. It is recommended that an Oncologist or Haematologist be contacted and both Departments run an on call service. Alternatively, contact the Haematology Department at Christchurch Hospital so that the relevant extra tests may be done on any biopsy material obtained. If biopsy is done out of hours, please place the node in normal saline, refrigerate, and deliver to Surface Marker Laboratory next morning.

Biopsies of sarcomas, particularly of bone, should be deferred until after full staging/investigations, including plain x-rays, CT scans, bone scan, and MRI.

MANAGEMENT OF PAIN IN CANCER PATIENTS (see page 135 in the Pain section).

MANAGEMENT OF NAUSEA AND VOMITING

Hypercalcaemia, electrolyte imbalance, opiate use and increased intracranial pressure can all be associated with nausea and vomiting and it is important to screen for these problems before commencing treatment. If one of these causes is identified, specific treatment such as intravenous hydration for hypercalcaemia or dexamethasone for cerebral metastases should be instituted.

The standard antiemetic regimen for patients receiving intravenous chemotherapy is metoclopramide 10-20 mg IV up to 4 hourly ± dexamethasone 8-16 mg IV q8h. Patients receiving cisplatin and some other highly emetogenic regimens are given ondansetron 8 mg PO or IV q12h or granisetron 3 mg IV q12h in place of metoclopramide. These 5-HT_3 receptor antagonists are useful only during the 24 hours immediately following chemotherapy and are NOT of value in the management of chronic or delayed emesis.

Check that the patient is adequately hydrated and that there has been recent measurement of plasma Ca, creatinine, Na and K. It may be necessary to give intravenous fluids.

Prochlorperazine suppositories 25 mg q8h and haloperidol 1-3 mg IV q8h may be added if the response to metoclopramide has not been satisfactory.

Dexamethasone 8 mg PO, BD for 2 days, tapering to 4 mg BD for 2 further days is the currently recommended regimen for persistent emesis following chemotherapy. Check that other potentially emetogenic medicines, such as non steroidal anti-inflammatory drugs, have been stopped. NON STEROIDAL ANTI-INFLAMMATORY DRUGS SHOULD NOT BE GIVEN TO PATIENTS RECEIVING DEXAMETHASONE AS THIS COMBINATION CAN INCREASE THE RISK OF UPPER GI TRACT ULCERATION AND BLEEDING.

Dexamethasone is also often helpful in the management of nausea and abdominal discomfort associated with liver metastases.

Emesis attributable to bowel obstruction is best managed with cyclizine 50 mg IV q8h or prochlorperazine. Metoclopramide is best avoided in this setting.

Sublingual Lorazepam 1 mg q8h can be utilized in addition to the above measures if anxiety is a prominent feature.

Adequate antiemesis should have already been charted for patients receiving chemotherapy and, if this is not satisfactory, the situation should be discussed with the Consultant Oncologist on call.

PAIN MANAGEMENT

PRINCIPLES

Pain is a symptom which requires thorough and complete evaluation. The aim is to control pain adequately while treatment of the primary disease continues. Therapy depends on:-

- Type of pain.
- Its cause.
- The severity.

Individualise therapy - the optimum dose of analgesic can vary quite widely between similar patients and in the same patient from time to time. Titrate agent and aim for minimum side effects. Do not change a drug until it has been fully evaluated.

Liver and renal disease may reduce drug metabolism and excretion. Dose frequency may therefore need to be reduced. All opiates should be given cautiously to patients with respiratory disease. Morphine may cause bronchospasm in addition to respiratory depression.

Drug metabolism and excretion is also reduced in elderly patients.

SEVERE ACUTE PAIN

Opiates (morphine, pethidine) are usually the drugs of choice. Dosage should be sufficient to be effective in relieving pain without producing problems such as excessive respiratory depression. Acutely, they are given IV (especially if the patient is hypovolaemic or shocked). They are titrated in increments against the patients pain, level of consciousness and respiratory rate. Regional local anaesthetic blocks may be indicated e.g. femoral nerve block for fractured shaft of femur or digital ring block for finger injuries etc.

A wide range of modalities are used for acute post-operative pain. These include IV infusions (e.g. see paediatric morphine infusion sheet), regional techniques, epidural techniques including infusions, a combination of local anaesthesia and opiate, and patient controlled IV analgesia (PCA). Some of these may be organised or managed by anaesthetic staff. PCA is prescribed by anaesthetists and requires trained nursing staff.

DOCTORS RESPONSIBILITIES

- Initial assessment of patient:
  - Magnitude and cause of pain.
  - Existence of factors that might affect patients' handling of opiates e.g. weight, children, elderly, liver or renal disease, drug dependence.

- Contraindications e.g. airway obstruction, respiratory failure, hypovolaemia, raised intracranial pressure.

- Decide on drug, method of administration, safe dose range and dose interval (see Tables for general guidelines) and chart according to hospital protocols.
- Reassess at regular intervals and adjust prescription accordingly.
- Chart drugs for the management of expected side effects e.g. metoclopramide or prochlorperazine for nausea and vomiting, naloxone for severe respiratory depression.

NURSES RESPONSIBILITIES

- Administer opiate according to existing hospital protocols and patient's prescription.
- Decide on appropriate dose within the dose range on the patient's prescription form using patient's response to previous doses as a guideline.
- Monitor and record pain levels, degree of sedation, respiratory rate before and at appropriate intervals after the administration of the opiate (see Tables 2 and 3).
- Manage appropriately any untoward effects (see Table 4)

TABLE 1

GUIDELINES FOR DOSES OF OPIATES AND ANALGESICS IN ACUTE PAIN

DRUG	ROUTE	DOSE for adults	NOTES & DOSE INTERVAL
MORPHINE	Intramuscular	150 mcg/kg/dose i.e. 10 mg/dose/70kg	3-6 hourly
	Intravenous bolus	30 mcg/kg/dose i.e. 2mg/dose/70kg	Can be repeated at 5 min intervals until effect achieved and desired repeated as required
	Intravenous infusion	20-40 mcg/kg/hr i.e. 2-4 mg/hr/70kg	Use 1 mg/ml solution WITH infusion or syringe pump
PETHIDINE	Intramuscular	1.5 mg/kg/dose i.e. 100 mg/dose/70 kg	2-3 hourly
	Intravenous bolus	300 mcg/kg/dose i.e. 20 mg/70kg	Can be repeated at 4 min intervals until desired effect achieved & repeated as required
	Intravenous infusion	300 mcg/kg/hr i.e. 20 mg/hr/70kg	Use 10 mg/ml solution WITH infusion or syringe pump

Note:

- The oral and rectal routes are not usually recommended for acute pain.
- Doses for neonates and children vary. Refer to paediatrics guidelines.

TABLE 2

GUIDELINES FOR FREQUENCY OF OBSERVATION

ROUTE	AT LEAST
Intramuscular	1 hour after each dose
Intravenous	Every 5 minutes for 15 minutes, then hourly for 2 hours
Intravenous Infusion	1 hourly

Note:

Pulse, respirations, sedation score and pain score are the recommended minimum. More frequent observation may be required in some patients.

TABLE 3

PAIN SCORES	SEDATION SCORES
0 = 'No Pain'	0 = None, Alert
1 = 'Mild Discomfort'	1 = Mild. Occasionally drawsy, easily roused
2 = 'Moderate Discomfort'	2 = Moderate. Frequently drowsy, easily roused
3 = 'Painful'	3 = Somnolent. Difficult to rouse
4 = 'Severe Pain'	S = Normally asleep, easy to rouse (an attempt must be made to rouse patient)

TABLE 4

MANAGEMENT OF SEVERE COMPLICATIONS

COMPLICATION	MANAGEMENT
RESPIRATORY DEPRESSION	
Life Threatening	• Stimulate patient • Support ventilation and airway - bag and mask • Oxygen by mask • Give naloxone * • Stop opiate administration
Non Life Threatening	• Stop opiate administration • Give oxygen by mask
EXCESSIVE SEDATION (not rousable by verbal stimuli)	• Oxygen by mask • Stop opiate administration • Nurse in recovery position

* Naloxone 0.2-0.4 mg IV injection and repeat every 5 minutes until desired effect (maximum 2.0 mg).

Adjuncts to opiates for severe acute pain - other agents can be useful along with opiates e.g. a non-steroidal anti-inflammatory agent (NSAID) such as IM diclofenac (Voltaren) 75 mg IM or PO for an adult. Paracetamol or an NSAID which comes in suppository form may be combined with opiates where appropriate. NSAIDs are particularly useful for renal colic and musculoskeletal pain but take heed of relative contraindications (e.g. peptic ulcer disease, oral anticoagulant use, anti-hypertensive treatment, renal impairment, asthma, and possibly fractures and sepsis).

SEVERE CHRONIC PAIN

This is broadly classified into two categories - cancer pain and non-cancer chronic pain. For the latter, it is rare to use oral opioids on a long term basis, but common for cancer pain. Where severe pain from an acute situation is ongoing, attempts should be made to move to a non-opioid type analgesic. Nefopam (Acupan) can be combined with mild oral analgesics. Tricyclic anti-depressants or anti-convulsants may be useful for neuralgic type pain. Any problems in this area may be referred to the Pain Clinic, Department of Anaesthesia.

For chronic cancer pain , oral opiates are commonly used e.g. slow release morphine (MST). Because of the "first pass" liver metabolism effect, the equivalent oral dose is usually three times that of the IM or IV dose.

SIDE EFFECTS OF OPIATES

- Nausea - oral prochlorperazine 5 mg q8h or metoclopramide 5-10 mg q6-8h may be used. Both can be given IV as above.
- Constipation - bowel softeners such as coloxyl, lactulose, with stumulants such as dulcolax.

  Note:
  - Adjunctive agents are commonly used with oral opiates for cancer pain.
  - Other treatments such as nerve blocks, neurosurgical procedures, transcutaneous nerve stimulation etc. may all have useful effects. Again, consultation with the Pain Clinic, Department of Anaesthetics, may be helpful.

LESS SEVERE ONGOING PAIN

Mild analgesics and NSAIDs. Some equianalgesic dose recommendations are:

- Soluble aspirin 600 mg q4h is equivalent to paracetamol 1 g q4h or
- Codeine phosphate 30 mg q4h.
- Codeine has a synergistic effect with aspirin.
- NSAIDs.

Some patients respond to one NSAID and not to others. Sometimes these are available in suppository form which may reduce complications. In addition to their effect on the gastric mucosa NSAIDs may impair renal function, antagonise anti-hypertensive agents, and alter the sensitivity to warfarin. Aspirin should be avoided for patients on anticoagulants. Dextropropoxyphene a component of Di-gesic is not favoured because of the long duration of its effect, abuse potential and overdose danger.

MANAGEMENT OF CANCER ASSOCIATED PAIN

It is important to establish the causes of pain in cancer patients, e.g. muscle spasm is treated differently from a bone metastasis or a pressure area.

Cancer-induced pain is best controlled by specific anti-cancer treatment. e.g. irradiation of bone metastases.

Severe pain - morphine elixir 5-10 mg q3-4h, increase each dose by 50% until controlled. When morphine requirements stable, give same total 24 hour morphine dose as MST, in two divided daily doses (12 hourly), with morphine

elixir for breakthrough. MST is also effective when given rectally (same dose as orally) morphine and may be given subcutaneously as an infusion.

Pain is a physiological antagonist to morphine respiratory depression. Morphine doses can be increased until pain is controlled.

All narcotics cause constipation - use laxatives - e.g. coloxyl or lactulose with dulcolax. Narcotics may cause nausea - use anti-emetics e.g. metoclopramide 10 mg PO 4 hourly, haloperidol 0.5-1.5 mg PO TID, prochlorperazine 25 mg PR 8 hourly.

Co-analgesics may add to analgesic effects, e.g. NSAIDs to decrease inflammation and bone pain, corticosteroids to decrease swelling, muscle relaxants to decrease spasm, nerve blocks, transcutaneous nerve stimulation, antidepressants, anticonvulsants (e.g. carbamazepine) for neuralgic pain.

Avoid using NSAIDs and corticosteroids concurrently because of the risk of peptic ulceration. If such a combination must be used remember that misoprostol protects gastric mucosa and H_2 receptor antagonists protect the duodenum. Since NSAIDs and steroids are more likely to damage the gastric mucosa misoprostol is the preferred drug.

POISONING / DRUG OVERDOSE

INTRODUCTION AND PRINCIPLES OR MANAGEMENT

THE AIRWAY BREATHING AND CIRCULATION

Attention to the ABCs is the first priority in all unwell patients and each of A,B, & C should at least be mentally checked before proceeding further. See also under Emergency Presentations - Airway, Breathing, Circulation (**all on page 9**).

- Airway:
  - Altered level of consciousness is the most common cause of loss of airway patency.
  - Death due to airway obstruction in these patients is relatively common and absolutely avoidable.
- Breathing:
  - Central respiratory depression, primary lung damage from inhalation or ingestion or secondary lung damage from aspiration may warrant intervention.
  - Supplemental oxygen is warranted in most of these patients, and in patients with injury to other organ systems enhanced oxygen delivery may be beneficial.
  - Control of convulsions may be required to enable adequate ventilation.
- Circulation:
  - Many poisonings impair the circulation by effects on the pump (arrhythmias or myocardial depression), the pipes (vasodilation), or the fluid (vomiting, diarrhoea etc).
  - Intravenous fluids are required.
  - Antiarrhythmics are occasionally required.

GASTROINTESTINAL TRACT DECONTAMINATION

Attention to the ABCs must always take priority and then give due consideration to the risks and benefits of GI tract decontamination.

The options available are:

- Activated charcoal.
- Gastric lavage.
- Induction of emesis with syrup of ipecac.

IMPORTANT CONSIDERATIONS

- GI tract decontamination will at best, prevent systemic absorption of about 30% of the ingested dose.

- A number of studies have shown that activated charcoal is superior to the other two methods alone or in combination with activated charcoal.
- If a patient has vomited spontaneously then any further attempts at gastric emptying are unnecessary.
- Gastric lavage has no function as a punitive measure. It is unpleasant for all concerned and at times necessitates physical restraint of the patient which might be difficult to justify.
- Gastric lavage and induction of emesis carry the risk of iatrogenic injury.
- Corrosive and volatile substances only rarely warrant removal and then only after protection of the airway with a cuffed endotracheal tube.

ACTIVATED CHARCOAL

- First choice for GI tract decontamination. The large surface area binds drug preventing systemic absorption.
- Provided as 50 g in 300 ml - usually given as a single dose, but try to provide a dose of ten times the weight of the ingested poison - repeat dose may be required for some specific poisonings.
- Given via orogastric tube after lavage, or via nasogastric tube or sipped through a straw or from a cup in the co-operative patient.
- Will not bind alcohols (e.g. ethanol, methanol, eythlene glycol) other hydrocarbons (petroleum distillates, essential oils) nor some other substances (lithium, iron, potassium, lead).

GASTRIC LAVAGE

- Indicated if the possible removal of less than 30% of the ingested drug will significantly benefit the patient.
- The lavage should be followed by the instillation of activated charcoal before removing the orogastric tube.
- It should be performed by someone skilled in the procedure and able to manage an impaired airway or under the direct supervision of such a person.
- It should not be performed in the patient with an impaired gag reflex unless a cuffed endotracheal tube has been placed first.
- Best results are obtained if done within 2 hours of the ingestion, although lavage may be indicated up to 4 hours. Some would suggest up to 6 hours in overdoses that delay gastric emptying e.g. tricyclic antidepressants, and other anticholinergics, or in overdoses that form concretions e.g. aspirin, or with slow release preparations e.g. theophylline.
- Not indicated if the patient has been vomiting.

SYRUP OF IPECAC

- Commonly used in children, but has fallen out of favour in adult poisonings.
- Variable effect in adults.
- Effect delayed - potentially dangerous if the poisoning is likely to depress the level of consciousness or cause convulsions.

- Nausea and vomiting are at times a useful sign of significant poisoning, but not after syrup of ipecac.

SPECIFIC MEASURES

These may include antidotes (direct antidotes e.g. naloxone for narcotics and flumazenil for benzodiazepines, or indirect antidotes e.g. N-acetylcysteine for paracetamol or sodium bicarbonate for tricyclic antidepressants), or ways of enhancing the elimination of the drug (e.g. haemodialysis, hemoperfusion, alkaline diuresis etc.).

Specific measures demand a knowledge of the toxicity of the implicated substance. This implies access to appropriate reference material and advice:

- Clinical Pharmacology Drug Information Service (Ext: 80900).
- Poisindex - a computerized reference available in the Emergency Department and Clinical Pharmacology Drug Information Service.
- The Emergency Medicine Physician, Pharmacologist or General Physician.
- National Poisons and Hazardous Chemical Information Centre - (Telephone URGENT (03) 474-7000, NON URGENT (Monday to Friday 9 am - 5 pm) (03) 479-1200.
- Various texts:
  - The New Ethicals Compendium.
  - Handbook of Poisoning -Dreishbach, Lange Publications.
  - Clinical Management of Poisonings and Overdose - Haddard and Wincheste.

  All three are available in the Emergency Department.

INVESTIGATION

- Determining the poison ingested is mainly from the history. Occasionally a toxin screen is requested e.g. if the toxin is unknown, if other poisons may also have been ingested, or occasionally when investigating a patient with altered level of consciousness. The investigation should only be performed if the result is likely to influence the patient's management.

  Note:

  In the vast majority of cases management is 'supportive' - or more accurately 'reactive', i.e. problems are treated as they occur.

- In Christchurch, toxic screens are done only on urine and gastric contents.
- Specific drug levels may be required e.g. significant ingestion of paracetamol. Occasionally, they are also useful for salicylates. ethanol, methanol, lithium, iron, lead, carboxyhaemoglobin, cholinesterase levels for organophosphates, theophylline, digoxin. Antidepressant and anticonvulsant levels are usually not useful in the overdose situation.
- Other investigations may be indicated e.g. arterial blood gases, ECG.

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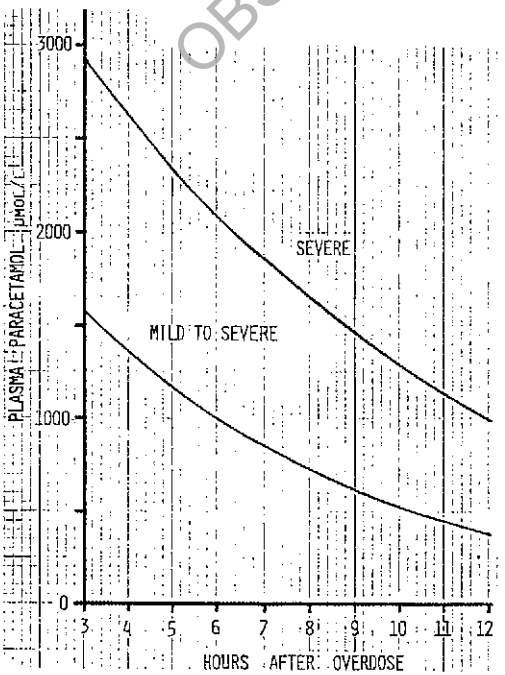
SPECIFIC MANAGEMENT OF SOME POISONINGS / OVERDOSES

PARACETAMOL

- 140 mg/kg approximates a toxic dose, although as little as 7g has caused death.

  Note:

  Chronic alcohol use, phenytoin, phenobarbitone, antihistamines, and other drugs that induce the cytochrome P450 enzyme system can increase toxicity for the same dose or blood level by shunting more paracetamol through the pathway that produces the toxic metabolite.
- Blood levels direct treatment, but should not be taken within 4 hours of ingestion.
- Do not await a blood level to treat if the patient has taken a significant amount on a mg/kg basis or on the basis of symptoms.
- Clinical toxicity follow four approximate stages:
  - ½ - 24 hours - Nausea, vomiting, malaise.
  - 24-48 hours - Right upper quadrant pain and raised transaminases. May be oliguric if dehydrated or with associated renal toxicity.
  - 72-96 hours - Peak of transaminase elevation, bilirubin and prothrombin time. Nausea and vomiting returns. Elevated creatinine (fetal death).
  - 4 days to 2 weeks - Resolution or hepatic failure.



- Treatment is directed by the nomogram above, (earlier if toxicity expected).

N-acetylcysteine	150 mg/kg in 100 ml D5W over 15 minutes THEN 50 mg/kg in 500 ml D5W over 4 hours THEN 100 mg/kg in 1000 ml D5W over 16 hours

- N-acetylcysteine is of most use when given within 15 hours of ingestion but there is increasing evidence that even beyond 24 hours it may be beneficial.
- The literature suggests that N-acetylcysteine given after 10-14 hours post ingestion assists in the repair of hepatic damage rather than providing an alternative source of sulphydryl groups as a protective substance. This will be independent of plasma paracetamol concentration making such measurements valueless in this regard. Therefore late paracetamol levels (>15 hours) will not be a useful guide to treatment and levels beyond 24 hours are pointless. Instead be guided by tests of liver function as described above.
- For N-acetylcysteine allergy use methionine instead. Simple rash or urticaria during infusion may respond to phenergan 25 mg IV and hydrocortisone 200 mg IV or by slowing the infusion

Note:

Combination tablets e.g. Di-gesic, may be toxic due to the dextropropoxyphene component e.g. 10 Di-gesic tablets gives a potentially toxic dose of dextropropoxyphene (500 mg) but contains only 3.25 g of paracetamol.

TRICYCLIC ANTIDEPRESSANTS

- Incidence declining since introduction of the newer, less toxic antidepressants (e.g. fluoxetine)
- Numerous receptor effects (sodium channel, calcium channel, muscarinic and α receptor blockades, some sympathetic agonist effects).
- High volume of distribution, liver metabolism with enterohepatic circulation.
- Main toxicity:
  - CNS depression, irritation, convulsions.
  - Cardiovascular depression / arrhythmias.

  Note:

  After some initial sympathetic stimulation, the main cardiac effects are due to slowing of impulse conduction, and hence widening of all phases of the ECG, cardiovascular depression, bradyarrhythmia and escape rhythms.
- Treatment depends on the dose taken, the time taken and the symptoms displayed.
- Sodium bicarbonate has both prophylactic and therapeutic effects on cardiac toxicity for two reasons. Firstly, alkalinization of the blood to a pH of greater than 7.5 seems to be cardio-protective. Secondly, and probably of more

relevance, it provides a bolus of sodium (the sodium channel blocking effect seems to be the most relevant in causing cardiac toxicity). Give 50-100 mmol $NaHCO_3$ over 30 minutes.

- The use of physostigmine is discouraged. Due to the complex receptor effects the response is unpredictable.
- When treating arrhythmias do as little as is necessary. Sodium bicarbonate is very useful. All drugs that prolong repolarization are contraindicated. Phenytoin and lignocaine are considered safe but their efficacy is debated, Lignocaine may aggravate seizure tendency. Pacing, cardioversion and defibrillation are relatively safe.
- Avoid suxamethonium if possible (it raises parasympathetic tone which can increase heart block).
- If pressor agents are required they must have ß-agonist effects, i.e. avoid isoprenaline and low dose dopamine.
- Treat convulsions with diazepam or midazolam and phenytoin if required.

BENZODIAZEPINES

- Rarely serious in isolation.
- Management is supportive (especially of airway and breathing).
- Flumazenil is available but is rarely required.

Note:

T½ flumazenil is one hour, i.e. sedation can recur. It may be helpful if trying to establish the cause of altered level of consciousness (although sedation due to alcohol and hepatic encephalopathy may also respond).

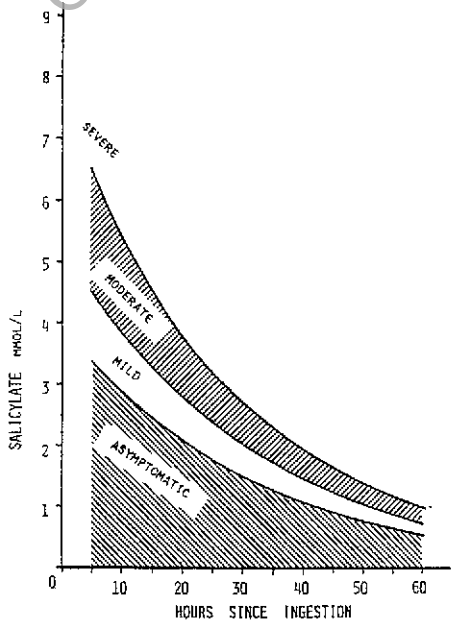
It is useful in reversing paradoxical agitation caused by benzodiazepine, seen occasionally in children and the elderly. Other indications are rare.

ANTIPSYCHOTICS

- Dopaminergic, adrenergic, muscarinic, H_1, H_2 blockade (and probably sodium channel blockade also).
- Treatment is supportive.
- The dopamine blockade produces the therapeutic effects but also many adverse effects - dystonia, akathesia, Parkinsonism, tardive dyskinesia, neuroleptic malignant syndrome.
- Dystonic reactions - treatment benztropine (Cogentin) 2 mg IM or IV.
- Neuroleptic malignant syndromes require identification and aggressive treatment prior to and after transfer to the Intensive Care Unit.
- In overdose, seizures and arrhythmias can rarely occur, and do so via mechanisms similar to tricyclic antidepressant poisoning. Therefore, treatment in such cases is similar.

ASPIRIN (AND OTHER SALICYLATES - e.g. oil of wintergreen)

- Acute poisoning:
  - Gastrointestinal effects - vomiting, haematemesis, respiratory centre stimulation; respiratory alkalosis. Inhibition of oxidative phosphorylation; metabolic acidosis. Raised blood glucose (mobilized glycogen stores) or low blood glucose (inhibition of gluconeogenesis, especially in children).
  - Manifestations - sweating, dehydration, tachypnoea, lethargy, confusion, convulsions, CNS depression, CVS depression and arrhythmias, coagulopathy, hyperthermia, ARDS.
- Chronic poisoning (over 12 hours) - no GI effects, often profound dehydration, acidosis, CNS depression, ARDS, coagulopathy. (The progressive acidosis increases the volume of distribution of aspirin and high tissue level of salicylates can occur despite relatively low plasma levels).
- Dosage:
  - Approx 150 mg/kg causes vomiting.
  - 150-300 mg/kg mild/moderate toxicity.
  - More than 300 mg/kg moderate/severe toxicity.
- Carbonic anhydrase inhibition increases the toxicity.
- Blood concentration - nomogram helpful in acute poisoning. Take a serum salicylate level at 6 hours or more post ingestion. Nomogram may dangerously under-estimate toxicity in chronic poisoning if previous salicylate taken within 24 hours, if poison taken over a prolonged period, or if enteric coated aspirin taken.



- Treatment:
  - If 6 hours post ingestion, concentration low and asymptomatic then patient may be discharged after appropriate psychiatric intervention.
  - If greater than 150 mg/kg of enteric coated aspirin, then admit for observation. Monitor salicylate levels and pH between 8-12 hours to detect delayed toxicity.
  - IV fluids - all significant salicylate poisonings are dehydrated.
  - Carefully follow blood glucose, electrolyte (especially potassium), prothrombin ratio and APTT.
  - If salicylate concentration is above 3.0 mmol/L or if significant symptoms, then alkalinize urine - $NaHCO_3$ 1 mmol/kg boluses until pH greater than 7.5, then 1000 ml of D5W, plus 100 mmol of $NaHCO_3$ + 40 mmol KCl started at 3 times the maintenance rate and adjusted according to regular measurements of pH, K, Na and hydration.
  - If Salicylate level greater than 6.0 mmol/L or if very unwell, then consider haemodialysis.

NARCOTICS

- Altered level of consciousness, respiratory depression, (typically a very slow respiratory rate, with a maintained tidal volume), meiosis.
- Treatment:
  - Naloxone 0.2-0.4 mg IV and repeat every 5 minutes. Some narcotic overdoses (e.g. dextropropoxyphene in Di-gesic) may need 4 mg or more.
  - Duration of action of Naloxone varies with the dose and route of administration but frequently repeat doses are required. The patient should be observed for evidence of returning narcosis. An infusion of 0.4 mg per hour may be required. Naloxone will reverse all the actions of the narcotic including analgesia, and may bring about an agitated 'withdrawal' state in an addict.

THEOPHYLLINE

- Arrhythmias - atrial/ventricular ectopics and tachyarrhythmias.
- CNS irritation/convulsions.
- Nausea and vomiting.
- Greater than 10 mg/kg may be toxic. Chronic overdosage is more dangerous than acute for any given concentration.
- Potassium falls with increasing acute toxicity.
- Slow release preparation will have delayed effects.
- Treatment:
  - Repeat doses of charcoal (50 g) orally or via NG tube every 4 hours.
  - Seizures:
    - ~ Often difficult to control.
    - ~ Diazepam is first choice but may fail.

- ~ Phenobarbitone 15 mg/kg IV over 20-30 minutes (avoid phenytoin).
- ~ Thiopentone, with ventilation, may be required.

- Arrhythmias - propranolol 1 mg slow IV every 5 minutes until control (or esmolol 25-50 mcg/kg/min).

  Note:

  Bronchospasm may ensue - consider risk benefit in asthmatics.
- Hypotension:
  - ~ IV fluids.
  - ~ Propranolol as above (hypotension is due to excessive ß adrenergic stimulation).
- Charcoal haemoperfusion:
  - ~ Effective and may be life saving - consult Nephrologist.
  - ~ Indications:
    - intractable seizures or persistent unstable haemodynamic parameters.
    - Acute intoxication with theophylline level >550 mcmol/L.
    - Chronic intoxication age 6 months to 60 years with theophylline level >330 mcmol/L.
    - Chronic intoxication age less than 6 months or over 60 years with theophylline level >220 mcmol/L.

BETA BLOCKERS

Consider using glucagon 3-10 mg IV boluses to reverse cardiovascular depression. Can also be given by infusion at 2-5 mg/hr.

CALCIUM CHANNEL BLOCKERS

- ♦ IV fluids for hypotension.
- ♦ Atropine for hypotensive bradyarrhythmias.
- ♦ Calcium chloride 10% 5-10 ml IV and repeat.
- ♦ Glucagon, dopamine may be useful.

DIGOXIN

- ♦ Acute poisoning:
  - Levels useful.
  - Plasma, K rises in proportion to toxicity.
- ♦ Chronic poisoning:
  - Levels may not be high (tissue distribution).
  - Plasma K may be normal or low.
- ♦ Treatment:
  - Bradyarrhythmias:
    - ~ Atropine to a maximum of 2 mg. May need pacing.
  - Tachyarrhythmias:
    - ~ Lignocaine 1.5 mg/kg IV, phenytoin 15 mg/kg IV infusion at no more than 50 mg/min, or magnesium sulphate 2-4 g IV.

- Cardioversion:
  - ~ May precipitate ventricular tachycardia.
  - ~ Use low energy (10-25 joules).
- Hyperkalaemia:
  - ~ Standard treatment (**see page 103**), but avoid calcium chloride / calcium gluconate as calcium may potentiate digoxin cardiotoxicity.
- Fab fragments:
  - ~ Indication - ventricular arrhythmias, unresponsive bradyarrhythmias, K >5.5 mmol/L. If ingested dose unknown use 10 vials. If ingested dose known or serum level available, calculate dose of Fab fragments required using the formulae available in 'Poisindex' (**see page 139**). Give over 30 minutes through a filter (bolus if life threatening). Beware - potassium level and cardiac output can fall rapidly. Ventricular rate in atrial fibrillation can rise rapidly.

LITHIUM

- Well absorbed orally. Low protein / tissue binding. Eliminated solely by the kidneys.
- Pathophysiology, Na, K, cAMP effects. CNS depression or stimulation, CVS stimulation progressing to conduction defects and block.
- Toxic dose approximately 40 mg/kg.
- Serum levels useful. Greater than 1.5 mmol/L considered toxic.
- Activated charcoal doesn't bind.
- Normal saline infusion aids renal elimination.
- Osmotic diuresis using Mannitol may enhance renal elimination.
- Haemodialysis useful if significant symptoms, deteriorating, not improving or if urine output inadequate - significant toxicity will usually require ICU monitoring.

CARBON MONOXIDE

- A high carboxyhaemoglobin level (>9%) is very significant. However lower carboxyhaemoglobin levels do not exclude significant toxicity.
- Carbon monoxide separates from haemoglobin spontaneously and therefore levels at the time of the blood test may not represent peak level. Also carbon monoxide toxicity occurs via other mechanisms (CVS depression and binding to cytochrome A3 causing paralysis of intracellular respiration).
- **Treatment:**
  - High flow oxygen via a system with a reservoir bag.
  - Hyperbaric oxygen - indications:
    - ~ COHb >25% (>20% if pregnant) **or**
    - ~ any loss of consciousness at any stage **or**
    - ~ any neurological sign or symptom (including cognitive, behavioural or psychological.

Note:
Hyperbaric treatment may be of value in more mildly affected patients. To arrange hyperbaric treatment, or if in doubt and you need advice call Princess Margaret Hospital Operator and ask for the "Hyperbaric Team". The appropriate member of the medical staff will contact you. **See Appendix 2.**

ETHANOL

- Intoxication:
  - Supportive Care.
  - Measure blood glucose if obtunded.

The chronic alcoholic with altered mental status may benefit from thiamine 100 mg IM.

Glucose infusion in thiamine deficiency can precipitate Wernicke's encephalopathy therefore always give thiamine to cover glucose infusion.

METHANOL

- 30 ml of 100% is potentially fatal.
- Causes a raised anion gap, metabolic acidosis and diffuse cellular hypoxia: visual effects ('snowstorm'), optic atrophy; CNS depression / irritation / seizures; gastritis and pancreatitis.
- Treatment:
  - Rapid absorption, don't gastric lavage beyond 2 hours.
  - Not bound by activated charcoal.
  - Sodium bicarbonate - often large amounts required to keep pH >7.2 (also sodium bicarbonate helps formate excretion).
  - Ethanol infusion:
    - If methanol level >6 mmol/L or visual symptoms or severe acidosis.
    - 0.6 g/kg bolus then 100 mg/kg/hr IV/orally/NG tube to maintain concentration of 100 mg/100 ml (22 mmol/L).
  - Haemodialysis if >20 mmol/L, visual impairment or severe acidosis.
  - Folate replacement may be helpful (acts as a co-factor for formate metabolism). Give 5-10 mg Folic acid orally or IV.

ETHYLENE GLYCOL

- Poisoning requires hospitalization.
- Toxicity via glycoxolate metabolite which causes a raised anion gap metabolic acidosis and also binds calcium to cause hypocalcaemia and calcium oxalate crystal deposition.

- Phases of toxicity:
  - 1-2 hours — CNS depression, focal signs or seizures due to calcium oxalate deposition, raised anion gap, metabolic acidosis, hypocalcaemia.
  - 12-72 hours — Cardiopulmonary toxicity - acute pulmonary oedema and congestive heart failure.
  - 24-72 hours — Acute renal failure - calcium oxalate crystals found in urine.
- Treatment:
  - Rapid absorption (don't gastric lavage beyond 2 hours).
  - Activated charcoal doesn't bind.
  - Sodium bicarbonate as for methanol.
  - Calcium chloride if hypocalcaemic. $CaCl_2$ 5-10 ml IV and reassess.
  - Thiamine and pyridoxine (act as co-factors for glycoxolate metabolism).
  - Intravenous fluid to maintain good urine output.
  - Ethanol as for methanol (see above).
  - Haemodialysis if significant intoxication.

LEAD

- CNS toxicity, PNS toxicity, anaemia, nephrotoxicity.
- Blood tests - total lead level and red cell protoporphyrins.
- Calcium sodium edetate IV chelation test may be a more accurate measure of intoxication.
- Treatment:
  - IV fluids to maintain adequate urine output. Excess IV fluid may precipitate cerebral oedema.
  - Calcium disodium edetate 1 g/m²/day usually for 5 days. Dimercaprol (BAL) can be used in children - see Paediatric Handbook.

IRON

- 4 stages of toxicity described:
  - GI tract stage - nausea, vomiting, haematemesis which may progress to shock (first few hours).
  - False reassurance stage - symptoms may abate (within 12 hours).
  - Shock, hepatic, renal and cardiac dysfunction (beyond 12 hours).
  - Days/weeks later - small bowel obstruction or gastric outlet obstruction due to stricture.
- Levels - useful at 4 hours but levels are not entirely reliable guide to the amount of iron taken:
  - Potentially toxic if level >60 mcmol/L.
  - If level greater than TIBC.

  But levels can be low due to tissue distribution and TIBC is artificially raised after overdosage.

- Begin treatment if:
  - Vomiting.
  - Diarrhoea.
  - Toxic level.
  - Level greater than TIBC.
  - Dose greater than 20 mg/kg of elemental iron (not the same as mg of drug).
- Treatment:
  - Avoid ipecac.
  - Desferrioxamine 15 mg/kg/hour IV (leave 5 g in the stomach if performing gastric lavage).
  - Increase infusion to as much as 45 mg/kg/hour if severe toxicity. Continue infusion until level falls, or clinically improved. Desferrioxamine may itself be toxic with prolonged infusion (>24 hours).
  - IV fluids to maintain urine output. Dialysis is required if renal failure develops, to remove the metabolite ferrioxamine.

OTHER HEAVY METALS

- e.g. arsenic, mercury.
- Treatment - Dimercaprol (BAL).

ORGANOPHOSPHATES

- Bind cholinesterase causing cholinergic toxicity.
- Check red cell and plasma cholinesterase activity (red cell level best reflect synaptic levels, but plasma levels are easier to do).
- Cholinesterase levels:

>50%	=	mild toxicity
20-50%	=	moderate
<20%	=	severe

- Treatment:
  - Atropine 1 mg test dose - if atropinized then the diagnosis is wrong. If not atropinized, then repeat 2 mg every 10-15 minutes until desired effect (massive dosages may be required).
  - Pralidoxime:
    - ~ To regenerate cholinesterases among other actions.
    - ~ Use only for moderate / severe toxicity.
    - ~ 1 g IV over 5 minutes (2 g if severe), (20-50 mg/kg for children).
    - ~ Repeat at 1-2 hours if required and then 12 hourly as required.

PARAQUAT

- Small amounts can be fatal. GI tract absorption is both slow and prolonged.
- GI tract, liver, kidney and pulmonary toxicity. Oxygen therapy makes the pulmonary toxicity worse.

- Treatment:
  - No oxygen.
  - Gastric lavage - following lavage instil into stomach -
    - ~ Mannitol 20% 3 ml/kg and **either** Fullers Earth 15% (150 mg per litre of water), 15 ml/kg **or** Bentonite 7% (70 g in 100 ml glycerol BP in 1 litre of water) 15 ml/kg.
    - ~ Save gastric sample, plasma and urine for Toxicology.
  - Transfer to ICU after consultation:
    - ~ Repeat dose of Mannitol / Fullers Earth.
    - ~ IV fluids to maintain urine output.
    - ~ Charcoal haemoperfusion and other therapy.

PSYCHIATRY

This section describes the management of acute psychiatric disturbance occurring in a general hospital setting.

Psychiatric disorders may be communicated directly by the patient who complains of a disturbed state of mind. But it is most often unusual behaviour that leads observers to infer a psychiatric condition. Control of this unusual behaviour is commonly requested when:

- It occurs at night.
- It disturbs other patients.
- It threatens the patient's own safety or that of others.

Disordered behaviour occurring in a non-psychiatric hospital will usually have arisen:

- As a severe disagreement or misunderstanding in staff / patient relations, sometimes augmented by alcohol or other drug abuse.
- As a symptom of a delirium.
- As an intercurrent exacerbation of a major 'functional' illness (schizophrenia, manic-depression).
- In the context of a drug withdrawal syndrome (alcohol, benzodiazepine).
- Occasionally as a factitious disorder, feigning craziness for some obscure advantage.

DELIRIUM

COMMON CAUSES

- **Systemic disease**
  - Toxic: - Drugs - iatrogenic
    - alcohol - intoxication
    - withdrawal
    - 'street' drugs of abuse
    - Heavy metals
  - Infections.
  - Metabolic: uraemia; liver failure; electrolyte disturbances; acid-base disorders; hypoxia; carcinomatosis; porphyria.
  - Endocrine: thyroid disorders; parathyroid disorders; hypoglycaemia, diabetic pre-coma.
  - Anoxic: cardiovascular disease; respiratory disease; anaemia; post-anaesthesia.
  - Vitamin deficiency: thiamine (Wernicke's); nicotinic acid; B12 and folate.

- **CNS disease**
  - Head injury.
  - Space-occupying lesion.
    Infection: encephalitis, meningitis.
  - Cerebrovascular disease: thrombosis, embolism; episode in atherosclerotic dementia; TIA; subarachnoid haemorrhage; hypertensive encephalopathy; SLE.
  - Epilepsy: post-ictal; psychomotor; petit mal status.

CHARACTERISTIC FEATURES

- Rapid onset.
- Fluctuating course.
- Night-time worsening.
- Impairment of consciousness - may be barely rousable but most often mild; a 'clouding' of consciousness, diminished attention, reversed sleep / wake cycle.
- Disorientation in:
  - Time - often the first noticed.
  - Place - especially in unfamiliar surroundings.
  - Person - seldom grossly disturbed.
- Memory disturbance:
  - Short term more than long term.
- Perceptual distortions:
  - Visual hallucinations.
  - Illusions (misinterpretations).
  - Other sensory modalities may be affected.
- Disturbances of thinking and mental grasp:
  - Impaired problem-solving, making sense of things.
  - Paranoid ideas common.
- Mood and behaviour:
  - Often unusually volatile.
  - Bewildered, afraid.
  - Can lead to sudden, unprovoked, uncharacteristic aggression.
- Disturbance of motor activity - hyperactivity, rarely hypoactivity (torpor).

Note:

In the elderly, minimal impairment of function may summate to produce a significant delirium. The very young and brain-damaged likewise have low delirium thresholds. Suicide in general hospitals occurs more frequently in the acutely delirious than in the acutely depressed patient. Delirium may be a fatal condition (suicide, exhaustion).

Do not confuse delirium and dementia

FEATURE	DELIRIUM	DEMENTIA
Impaired memory	+++	+++
Impaired thinking	+++	+++
Impaired judgment	+++	+++
Clouding of consciousness	+++	-
Major attention deficits	+++	+
Fluctuation over course of day	+++	+
Disorientation	+++	++
Vivid perceptual disturbances	++	+
Incoherent speech	++	+
Disrupted sleep-wake cycle	++	+
Nocturnal exacerbation	++	+
Insight	++	+
Acute or subacute onset	++	-

+++ Always present
++ Usually present
+ Present sometimes
- Usually absent

MANAGEMENT

- Seek, identify and treat underlying cause.
- Symptomatic treatment involves:
  - Optimal nursing care:
    - Close, sympathetic surveillance.
    - Humane restraint PRN.
    - Familiar faces (e.g. relatives).
    - Frequent short interactions.
    - Low-stimulus environment (single room).
    - Night light.
- If disturbed behaviour needs control before treatment of cause is effective, tranquillizers may be required:
  - **Haloperidol** (the tranquillizer of choice).
    - If the symptoms are minor give PO not IM.
    - In severe cases IV or SC administration is advisable. It should be injected over about a minute. Initial dose 1 mg (mild), 5 mg (severe), 10 mg (very severe). Double and repeat in 20-30 minutes until control. Await reappearance of symptoms and repeat the same dosage. Introduce oral dosing. Gradually phase out as delirium resolves.
    - Most patients require less than 20 mg/24 hours. Maximum single dosage 20 mg.
    - Maximum daily dosage 80-100 mg. Extrapyramidal side effects rare with IV. With oral haloperidol, benztropine 2 mg IV/PO may be required. Sedation usually achieved within 45 minutes.

- **Benzodiazepines** (short acting e.g. midazolam) useful adjuncts to enhance sedation, especially if alcohol or drug withdrawal states.
- **Opioids** in addition if pain, severe agitation.

♦ Additional principles governing management:
- PO before IM or IV administration if possible.
- Resist calls for heavy sedation ('pharmacologic straitjacket').
- The very elderly, frail and thin will need cautious dosage.
- Do not stop habitual benzodiazepines abruptly and censoriously (especially the short-acting).
- Benzodiazepines are rarely the mainstay of management, except perhaps in liver failure.
- Monitor treatment closely, adding and subtracting PRN. Try to have patient free of pscyhotropic drugs by discharge.

Note:

A time lag of a few days commonly occurs between apparent resolution of an organic cause and the resolution of a delirium.

ACUTE 'FUNCTIONAL' PSYCHOSIS

Acute 'functional' psychoses tend not to be highly differentiated despite the variety of psychiatric syndromes in which they may erupt. Context is vital and history essential to take the diagnosis past 'psychosis' to the perspective of, for example,

- ♦ Mania (in bipolar affective disorder).
- ♦ Puerperal psychosis.
- ♦ Acute schizophrenic episode.
- ♦ Paranoid psychosis .
- ♦ Major depressive disorder with delusions.
- ♦ Borderline personality disorder.

Sometimes, encountering disturbed behaviour in a general hospital, you will not have the benefit of either history or context and will be called upon to help de-escalate a situation characterized more by mutual fear than by feckless violence.

The symptoms of 'psychosis' come from a common pool representative of personal disintegration: impaired reality-testing, delusional thinking, hallucinations (commonly auditory), fear, suspicion, agitation and aggressivity, leading often to bizarre, reckless, assaultive or even suicidal behaviour.

Clouding of consciousness is not a feature so that cognitive disorganization, as in delirium, is not prominent, however peculiar the thinking may be.

Antipsychotic medications (butyrophenones and phenothiazines) are the mainstay of drug management, whose aim is the restoration of self-control without, if possible, the use of force or physical restraint.

Effective drug treatment should bring early resolution of the most alienating symptoms: hallucinations and delusions, the agitation, the unco-operativeness and raw hostility, the anti-social behaviour, the driven quality of the sleeplessness. Other socially interactive treatment influences then have a chance to repair the less responsive impairments.

SOME ORAL AND PARENTERAL ANTIPSYCHOTIC DRUGS

- **Range of dosage suggested:**

	ACUTE (mg)		
	IM	ORAL	DAILY (mg)
Haloperidol	5-10	5-15	5-80
Chlorpromazine	50-100	75-150	50-1200
Thioridazine	N/A	75-150	50-800

Common side effects

- **Low potency** (chlorpromazine, thioridazine).
  - Sedation.
  - Hypotension.
  - Marked anticholinergic effects; dry mouth; constipation; urinary retention.
- **High potency** (haloperidol).
  - Extra-pyramidal effects (especially oral dosage in range 5-20 mg/day).
  - Dystonia (torticollis, opisthotonus, oculogyric crisis).
  - Dyskinesia.
  - Akathisia.
- **Typical dosages of anti-parkinsonian agents.**
  Pre-emptive use is not recommended as long as you can respond at short notice (e.g. oculogyric crisis). Nursing staff should be forewarned of the possibility of adverse effects.

Benztropine	1-2 mg (IV, IM or PO)
Benzhexol	2 mg (PO)
Procyclidine	5-10 mg (PO)

Some practical management guidelines

- Review 12 hours after administration.
- Once symptoms show some modification, reduced dosage frequency.
- Chlorpromazine is the most sedating but IM administration is very painful.
- Write clear instructions to nursing staff about indications for 'repeat' dosage.

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MAJOR DEPRESSIVE DISORDER

A common condition in the general hospital (prevalence 30-40%).

- Chance association.
- Reactively precipitated:
  - Complicated grief.
  - Chronic debilitating illness.
- Organic.
  - Post viral (influenza, hepatitis, infectious mononucleosis).
  - Neurological (Parkinson's, CVA, multiple sclerosis, head injury).
  - Malignancy (pancreas, lung, cerebral, colon).
  - Immunological (SLE).
  - Endocrine (hypothyroidism, Cushing's, Addison's).
  - Medication (steroids, methyldopa, major tranquillizers, NSAIDs).

Symptoms may not be classical. Physiological (vegetative) symptoms can be hidden by co-existing physical illness. Psychological and cognitive symptoms (pessimism, suicidal ideation, hopelessness, depressive delusions) are more discriminative.

MANAGEMENT

- Beware of sensitivity to the side effects of antidepressant medications, particularly the tricyclics.
- Selective serotonin re-uptake inhibitors (fluoxetine, sertraline) and reversible inhibitors of monoamines (moclobemide) are better tolerated.
- If rapid response is critical, stimulants (e.g. methylphenidate 5-20 mg mane) should be considered.
- ECT should not be forgotten as an option.
- The physically ill should not be allowed to suffer an untreated depressive disorder.

DRUG WITHDRAWAL MANAGEMENT

- Benzodiazepine withdrawal syndrome.
  - 7-10 days after cessation.
  - Enhanced anxiety, anorexia, tremor, seizures, delirium.
  - Diazepam 10-20 mg PO hourly until control then reduce by 5-10% daily.
- Opioid intoxication.
  - Naloxone 0.4 mg IV and repeat in 5 minutes. May need up to 4 mg IV in this situation.
- Opioid withdrawal syndrome.
  - Not life threatening but very unpleasant.
  - 2-3 days post last dosage.
  - Sweating, dilated pupils, insomnia, nausea, goose flesh, rhinorrhoea, abdominal cramps, diarrhoea.

- Suppressed by 20-50 mg methadone, then reduce over 10-14 days, clonidine a useful adjunct.

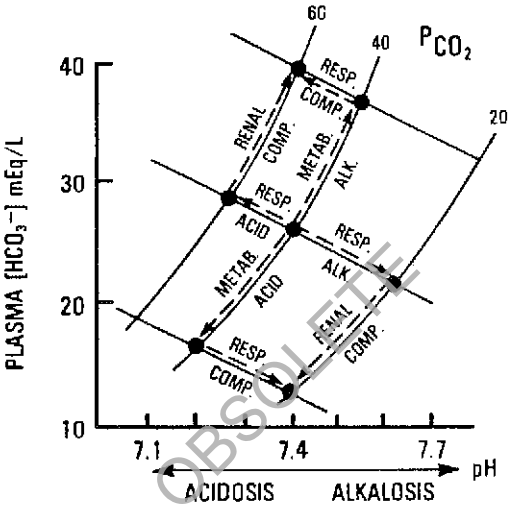
  Note:

  A general hospital admission is an opportunity to diagnose alcohol and drug abuse and initiate management.

RESPIRATORY MEDICINE

RESPIRATORY FAILURE

Classically defined as PaO_2 <60 mmHg or $PaCO_2$ >50 mmHg. Most commonly results from acute on chronic failure. Gases of this order in a non-respiratory patient mean the patient should be managed in a High Dependency Unit or ICU. See below for diagram to assist in interpretation of blood gas results.



Note:

This diagram differs from that given in the Endocrinology/Metabolic Section (**page 69**). We suggest you use the diagram that you prefer.

CLASSIFICATION

- **Type I** (Gas exchange/hypoxaemic) Respiratory Failure - Primarily due to pulmonary parenchymal disease with lowered oxygen transfer factor.
- **Type II** (Ventilatory/hypercapnic) Respiratory Failure - Primarily due to alveolar hypoventilation.

Note:

Many cases do not fit neatly into one or other type but identification of the primary process will be an important determinant of management strategy.

AETIOLOGY

Type I (hypoxaemic)

- Adult Respiratory Distress Syndrome.
- Pneumonia.
- Pulmonary oedema.
- Pulmonary embolism.
- Diffuse interstitial lung disease.
- Intracardiac and intrapulmonary shunts.

Type II (hypercapnic)

- Chronic Airflow Obstruction.
- Asthma.
- Drug induced (sedation, overdose, poisoning, muscle relaxants).
- Airway obstruction (laryngeal oedema, foreign body, mediastinal mass).
- Obstructive sleep apnoea.
- Thoracic restriction (obesity, kyphoscoliosis).
- Neurological disorders (Guillain Barré syndrome, myasthenia gravis, motor neurone disease).
- Muscle disorders (muscular dystrophies, myopathies).

INVESTIGATIONS

- Arterial blood gases.
- CXR.
- Ventilatory capacity; peak expiratory flow rate (PEFR), spirometry. Also static lung volumes, diffusing capacity, and maximum inspiratory pressure (MIP).
- Na, K, thyroid function tests, Ca, PO_4.
- CBC + diff.

MANAGEMENT

Consider each of the following:-

Airway protection

This is an important consideration in all cases where the upper airway defence mechanisms are compromised in some way, e.g. coma, profound sedation, bulbar palsy.

- Endotracheal tubes: secure but invasive. Allows suction but patient may require sedation.
- Oropharyngeal tubes: useful with patient in the "recovery position".

Reversal of precipitating cause

- Always consider the possible contribution of infection, cardiac failure and bronchospasm. They may not be the primary cause of the respiratory failure but are readily treatable.
- Drug depression - opiates may be reversed with naloxone 0.2-0.4 mg IV. As naloxone has a short half life respiration should be monitored frequently and naloxone repeated as necessary. May need to be hourly.
- Benzodiazepine induced respiratory failure may be reversed by giving flumazenil (dose 0.3-2 mg IV) precautions as for naloxone.
- Obstructive sleep apnoea - treatment with nasal CPAP can reverse respiratory failure. Discuss with Respiratory Physician.

Clearance of endobronchial secretions

Improves ventilation and helps prevent atelectasis and infection.

Patients may require regular chest physiotherapy to:

- Encourage effective coughing.
- Maximize inspiratory effort.
- Facilitate postural drainage.

Oxygen therapy (see page 164)

Mechanical ventilation

Indicated on the basis of the **overall clinical condition** rather than blood gases alone. Evidence of deterioration or lack of clinical improvement are strong indications for intervention.

Inform the ICU Team and Respiratory Physician on call that you have a patient with ventilatory impairment and ask for an early assessment so they will be able to detect any subsequent change in the patients clinical status.

INDICATIONS

- Severe hypoxia (<50 mmHg) despite high (>50%) inspired oxygen concentration.
- Significant hypoxia (<60 mmHg) and or hypercapnia (>45 mmHg) along with
  - Diminished/ing level of consciousness.
  - Diminished/ing chest expansion.
  - Evidence of respiratory muscular fatigue.
  - Retention of excessive endobronchial secretions.
  - Thoracic cage trauma/lung contusion.

OBSTRUCTIVE SLEEP APNOEA (OSA)

- Usually presents with day time hypersomnolence, snoring and apnoea[s]. It is a common and frequently missed diagnosis.

  Consider OSA in patients who have:

  - Unexplained respiratory or right cardiac failure.
  - Motor vehicle or industrial accidents related to sleepiness.
  - Hypertension.
  - Impotence.
  - Morning headaches.
  - Lethargy and depression.
  - Acromegaly, hypothyroidism, Marfan's Syndrome, retrognathia.

  If OSA is suspected refer to Respiratory Physician for full evaluation including a sleep study.

CHRONIC AIRFLOW OBSTRUCTION (COAD, CORD, COPD)

DEFINITION:

This is not a disease process, it is a clinical complex and consists of various admixtures of:

- Chronic bronchitis.
- Bronchial hypersensitivity/asthma.
- Emphysema.
- Small airways disease.

Identification of the relevant contribution of each of these components allows rational management and minimizes the possibility of misdiagnosis.

AETIOLOGY

- Smoking induced lung disease.
- Asthma.
- Bronchiectasis.
- Occupational exposures.
- Cystic fibrosis.
- Alpha-1 antitrypsin deficiency

Causes for acute deterioration of chronic airflow obstruction

- Lower respiratory tract infection (viral or bacterial bronchitis).
- Increased bronchial hyper-responsiveness.

Always consider the possibility of other contributing conditions:

- Pneumonia.
- Pneumothorax.
- Pulmonary embolism.
- Cardiac failure.
- Sepsis.
- Drugs.
- Acute abdomen.

INVESTIGATIONS

- Spirometry/PEFR.
- CBC + diff.
- Na, K, Ca, PO_4.
- Theophylline level.
- Sputum culture.
- Blood cultures.
- CXR.
- Arterial blood gases.
- ECG.

MANAGEMENT

Reconsider and reconfirm the diagnosis, identify the precipitating factor(s) and estimate the degree of functional impairment. Mistakes occur when previous diagnostic inaccuracies are perpetuated.

Oxygen therapy - aim to maintain a PaO_2 >55 mmHg which represents approximately 90% Hb oxygen saturation (see section below). Monitor for signs of hypercapnia, in particular drowsiness and/or confusion. Measure arterial blood gases regularly.

Infection

- Antibiotic therapy should be given to patients with purulent sputum, an increase in sputum volume or fever.
- Empiric therapy may be required. Possible choices include - amoxycillin, amoxycillin-clavulanate (Augmentin), doxycycline or a macrolide antibiotic.
- Oral antibiotics are usually adequate.

Bronchospasm/airways inflammation

- Bronchodilator therapy:
  - Nebulised ipratropium (Atrovent Respule 0.5 mg) q4-6h.
  - Nebulised $ß_2$-agonist; salbutamol (Ventolin nebule 2.5-5.0 mg) q2-4h may be added. Terbutaline 5 mg (Bricanyl) is equally efficacious.

- Change to inhaler when condition improves.
- IV salbutamol if inadequate response to nebulizers, initial dose 5 mcg/min. **(See Asthma Section).**
- IV aminophylline may also be considered (see below).

♦ Steroid therapy:
- Prednisone 40 mg PO /24 hours in a single morning dose.
- Hydrocortisone 200 mg IV q6h may be used if cannot take oral medication or if in extremis.
- Reduce prednisone over two weeks e.g. 40 mg for 1 week, 20 mg for 1 week.
- Inhaled steroids (400-1,000 mcg) may be useful in some cases as maintenance therapy.

♦ **Theophylline** preparations (aminophylline = 80% theophylline).
Indications for the use of parenteral theophylline include respiratory failure, drowsiness, or β_2 agonist intolerance.
Loading dose if not on oral theophylline. Give 5 mg/kg based on ideal body weight mixed with 100 ml of 0.9S over 20 minutes.
Maintenance dose. Add 500 mg aminophylline to 500 mg 0.9S (=1 mg/ml). Infuse at between 0.3 and 0.9 mg/kg/hr. Monitor levels daily. Target levels = 55-110 mcmol/L.

Doses recommendations for intravenous aminophylline:

	Calculated (mg/kg/hr)	
Non-smoker	0.5-0.7]	
Smoker	0.9]	mg/kg/hr
Cimetidine, erythromycin or ciprofloxacin use	0.3-0.4]	
Cor-pulmonale and hepatic insufficiency	0.25]	

Note:
If bronchospasm severe consider parenteral sympathomimetics as per Asthma Protocol.

Cardiac failure - emphysema may disguise the usual radiological and clinical signs of pulmonary oedema and/or heart failure. Recognition and treatment of even minor levels of pulmonary venous hypertension can significantly alter pulmonary compliance and result in symptomatic improvement. Objective assessment, such as a gated cardiac scan and even a trial of vasodilator/diuretic therapy should be considered in unresponsive cases.

An elevated JVP and peripheral oedema does not necessarily indicate "cardiac failure". In cor pulmonale the over use of diuretics can lead to a deterioration in cardiac output.

OXYGEN THERAPY

AIM - to prevent important tissue hypoxia.

Tissue oxygenation depends on two factors:

- Arterial oxygen content - the only factor affected by oxygen administration. A level of 65 mmHg (Hb saturation 90%) is all that needs to be achieved.
- Tissue perfusion - affected by cardiac output and peripheral vascular resistance.

INDICATIONS

- PaO_2 less than 60 mmHg.
- Hb saturation less than 90%.
- Conditions such as myocardial infarction, CO poisoning, acute anaemia.
- At risk of hypoxia such as post-op, LVF etc.

Oxygen therapy is indicated primarily to relieve hypoxia **not** dyspnoea.

METHOD OF ADMINISTRATION

- **Multi-vent mask:** use 24% initially in situations where there is a possibility of CO_2 retention, ($PaCO_2$ >40 mmHg). Monitor the arterial blood gases and if there is no CO_2 retention then consider nasal cannulae.
- **Nasal cannulae:** 1-4 L/min, provides an inspired oxygen concentration of 24% to 40% depending on the flow. Over 90% of patients can be treated with oxygen using nasal cannulae. This mode is most comfortable for the patient and in the absence of profound gas exchange problems, will provide more than adequate oxygen saturation levels. Adjust using pulse oximetry.
- **Hudson mask:** 6-10 L/min, provides about 50% oxygen. The initial method of choice in acutely hypoxic patients i.e. acute asthma, pneumonia, LVF and pulmonary embolism.
- **High flow humidified.** Used for long term therapy where drying the bronchial secretions needs to be avoided. It is only indicated in special circumstances. Contact ICU or Respiratory Technician.

The predicted oxygen percentage supplied by masks and nasal cannulae are unreliable. Whatever method you use monitor the patient with arterial blood gas estimations initially.

MONITORING

- **Pulse oximetry provides an estimate of capillary haemoglobin saturation. It doesn't assess the adequacy of ventilation.**
- Arterial blood gases analysis must ba sampled on admission and in many cases at regular intervals to assess response to treatment and oxygen dose.

- Hyperoxia can induce hypercapnia by a combination of worsening ventilation perfusion mismatch and to a lesser extent depression of respiratory drive. It is unpredictable and emphasizes the importance of arterial blood gas monitoring. If the patient is at risk, monitor blood gases every 30 minutes until stable. Sometimes, following the initiation of oxygen therapy the PCO_2 may risa by 10-15% then stabilize. This may be the cost of adequate oxygenation and is acceptable as long as there are no adverse clinical events.

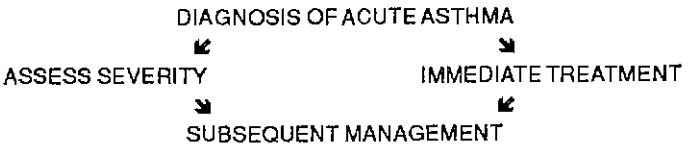
ASTHMA

Asthma is a clinical syndrome characterised by variable airflow obstruction secondary to inflammation of the airways. An acute asthmatic episode is usually the result of exposure to a trigger agent which may be either specific eg; pollen, animal dander, viral infection or non specific. The resulting symptoms usually consist of dyspnoea, wheeze, chest tightness and cough. They vary from being almost undetectable to severe, unremitting and sometimes life threatening.

The aims of management are:

- To prevent death.
- To restore the patient's clinical condition and lung function.
- To maintain optimum lung function and prevent early relapse.

The assessment of the severity of an acute attack of asthma and the immediate treatment occur in parallel.



The severity of asthmatic attacks is frequently underestimated by both the patient and doctor. It is therefore essential to objectively measure severity so that rational decisions regarding investigation and immediate treatment can be made.

All patients must have the following measured:

- Peak flow rate / spirometry.
- Respiratory rate.
- Pulse, blood pressure, temperature.
- Pulse oximetry/ arterial blood gases **(see page 168)**.

GUIDELINES FOR ASSESSING THE SEVERITY OF ACUTE ASTHMA

Individual features should not be interpreted in isolation. An overall assessment of severity should be made using your clinical judgement and the following guidelines:

	SEVERITY		
	MILD	MODERATE	SEVERE
Speech	Sentences	Phrases	Words
Peak expiratory flow (% of predicted or previous best)	>60%	40-60	Less than 40% or less than 150 L/min if best peak flow unknown
FEV1 (% predicted)	>60%	40-60%	<40% or less than 1 L
Respiratory Rate	Normal	18-25	>25
Pulse rate	< 100	100-120	>120
Oximetry	>94%	90-94%	<90%
Arterial PaO_2	Test not necessary	<80 mmHg	<60 mmHg
Arterial $PaCO_2$	Test not necessary	<40 mmHg	>40 mmHg
DANGER SIGNS: Exhaustion, confusion, cyanosis, bradycardia, unconsciousness, silent chest on auscultation, signs of respiratory muscle fatigue (indrawing of lower costal margin, abdominal paradox.			

IMMEDIATE MANAGEMENT

Specific treatment is dependent on severity and a number of factors which may vary between cases. The following table gives guidelines for the management of patients with asthma of different severities.

SEVERITY	MANAGEMENT	MONITORING
MILD	Nebulized salbutamol 5 mg q4h + PRN. Prednisone 40 mg orally stat then daily	Peak flow after initial treatment then qid. Pulse, respiratory rate QID
MODERATE	Nebulized salbutamol 5 mg q4h + PRN. Prednisone 40 mg orally stat then daily. Add: Oxygen to maintain O_2 sat > 95% (usually 2 L/min by nasal cannulae) Contact: Medical Registrar if not improving Perform: CXR if condition deteriorates or evidence of a complication (Note c)	Peak flow 2-4 hourly. Pulse oximetry. Pulse, respiratory rate, BP QID. Monitor serum potassium
SEVERE	Increase: Nebulized salbutamol 5 mg up to ½ hourly. Nebulized Atrovent 0.5 mg q4h. Oxygen 8L/min by Hudson Mask. Adjust to maintain O_2 sat >95% Add: Intravenous access. IV hydrocortisone 200 mg stat then q6h (for 24 hours). Fluids 0.9S 1 L 6 hourly. IV bronchodilator if not responding to nebulized bronchodilator (Note a) Contact: Respiratory Physician Perform: CXR in all cases	ICU or high dependency unit. Oximetry/arterial blood gases. Continuous ECG. Pulse, respiratory rate, BP ½ hourly. Special nurse. Serum potassium 12 hourly
DANGER SIGNS PRESENT	Increase: Oxygen to 100% via highflow system Contact: ICU team + Respiratory Physician Add: IV salbutamol 250 mcg loading dose then salbutamol infusion 3-20 mcg/min according to response (see next page)	Resuscitation or Intensive Care Unit. Nurse and doctor to stay with patient at all times

Notes:

a) Intravenous bronchodilators

SALBUTAMOL — Loading dose 250 mcg IV or IM. Make up intravenous infusion by adding salbutamol 1 mg to 100 ml of 0.9S.

concentration = 10 mcg/ml
rate = 18-120 ml/hr (3-20 mcg/min)

OR AMINOPHYLLINE (**see page 163**).

b) It is essential that all nebulised bronchodilators are given with oxygen 6-8L/min.

c) Patients with life threatening asthma, or severe asthma not responding to initial treatment, and patients in whom there is evidence of a complication require a CXR. Complications to look out for include pneumothorax, surgical emphysema, atelectasis and consolidation. All CXRs should be done at the bedside unless the patient is accompanied to X-ray by a nurse or doctor.

d) Pulse oximetry is very useful in assessing the adequacy of tissue oxygenation in patients with severe and life-threatening asthma. **It does not reflect the adequacy of ventilation.** An initial arterial blood gases measurement should be made in all patients admitted to hospital unless severity assessed as mild.

SUBSEQUENT MANAGEMENT

Depends on the severity of the attack and the patient's response to treatment.

A GENERAL MEASURES

- OBSERVATION - Close observation should continue in patients with severe asthma until there is objective evidence of sustained improvement.
- POSITIONING - recommend sitting upright and/or leaning forward.
- CONTINUE TREATMENT
  - Oxygen - according to arterial blood gases/oximetry.
  - β_2 agonist - if condition improving continue to give 4 hourly.
- MONITORING: repeat peak flow (or FEV1) 15-30 minutes after starting treatment then as required depending on severity. Arterial blood gases should be repeated within two hours of starting treatment in the following circumstances;
  - The initial $PaO_2 < 60$ mmHg.
  - The initial $PaCO_2$ was high normal or raised.
  - The patients condition deteriorates.

Measure and record heart rate and respiratory rate according to severity. Serum theophylline levels should be measured daily if IV aminophylline is continued more than 24 hours. Serum K and glucose daily.

B INVESTIGATIONS IN HOSPITAL

All patients admitted to hospital should have:

- CBC + diff.
- Na, K, glucose, creatinine.
- ECG - in patients over 40 years of age.

Indications for CXR:

- Severe or life threatening asthma attack - during resuscitation.
- Severe/moderately severe attack not responding to initial treatment.
- Patient suspected of having developed a complication or in whom another condition/diagnosis is suspected (see below).

All CXRs should be performed at the bedside unless the patient is accompanied to X-ray by a nurse or doctor.

C FAILURE TO IMPROVE

- WORSENING ASTHMA - check the adequacy of treatment e.g.: check drugs given, dosage and adequacy of drug delivery.

  Therapeutic options:
  - Increase the dose/frequency of β_2 agonist.
  - Add ipratropium bromide (Atrovent) 0.5 mg q4h .
  - Consider using an intravenous bronchodilator.

- The development of a complication or an alternative diagnosis:
  - Pneumothorax.
  - Cardiac arrhythmia.
  - Left ventricular failure.
  - Laryngeal or tracheal obstruction.
  - ARDS.
  - Pulmonary embolism.

  All patients who fail to improve or deteriorate despite initial treatment, must be monitored closely and discussed with the appropriate consultant or the Respiratory Physician on call.

D UNHELPFUL TREATMENTS

- Sedatives are absolutely contra-indicated unless in ICU.
- Antibiotics are not indicated unless there is evidence of bacterial infection (fever, purulent sputum, CXR opacity).
- Percussive physiotherapy.

E INDICATIONS FOR INTENSIVE CARE

Patients with the following features always require observation and management in intensive care.

- Hypoxia : PaO_2 <60 mmHg despite receiving high flow oxygen.
- Hypercapnia; $PaCO_2$ >50 mmHg.
- Exhaustion.
- Confusion, drowsiness, impaired level of consciousness.
- Respiratory arrest.

F MANAGEMENT DURING RECOVERY AND FOLLOWING DISCHARGE

Once the acute attack has been brought under control, attention must be redirected towards:

- Asthma control.
- Severity assessment - What is the risk of severe asthma recurring?
- Self-management skills.

ASTHMA CONTROL

Assess interval asthma control by asking the following questions;

- How many times in the past month have you been wheezy or coughing on first arising from bed? eg; 20/30.
- How many times in the past month have you been awoken from sleep wheezing or coughing? eg; 15/30.
- Does wheezing, breathlessness or cough limit your physical activity? eg; walking, cycling, sport or household activities?
- In the past month on average how many doses of your bronchodilator (reliever) would you take over twenty-four hours?
- In the past month what is the greatest number of doses of your bronchodilator (reliever) that you have taken in twenty-four hours?
- Do you measure your peak flow regularly and during an attack?
- What is your best and worst peak flow level over the past year?
- What is your usual range of morning and night peak flows?
- How much time in the past year have you had off work or school with any chest problems?

- How many courses of oral steroids have you had in the last year?
- How many times have you required treatment with a nebulised reliever in the past year?
- Your preventer is prescribed to be used two or three times daily. That is fourteen to twenty-one times per week. Many people find it difficult to remember to take their preventer inhalers. On average how many times would you take your preventer per week? eg; 10/21.

Patients with poor compliance, unstable features, or high β_2-agonist use should be referred to a Respiratory Physician.

SEVERITY ASSESSMENT:

The risk of a severe or fatal asthma attack is associated with:

- Previous severe asthma requiring ventilation or ICU admission.
- Frequent attendances to the emergency department.
- Nocturnal symptoms.
- Precipitous asthma attacks in the past - (severe attacks coming on over less than 3 hours).
- Frequent requirement for courses of oral steroids.
- Poor self-management skills.
- Poor social or financial circumstances.

SELF-MANAGEMENT SKILLS

The circumstances surrounding admission to hospital should be reviewed carefully.

- Was there an avoidable precipitant?
- How did the patient react to worsening asthma?
- Did the patient follow an asthma action plan?
- Were there problems with compliance?
- Was there an inappropriate delay in seeking help?

The key to asthma control is education and good self-management skills, Admission to hospital does not necessarily mean a failure of self-management but may provide an important learning opportunity.

All patients should have the following while recovering from an acute attack:

- Assessment of education needs - refer if appropriate to respiratory educator, nurse or physiotherapist.
- Check of inhaler technique and instruction on the use and interpretation of readings from a peak flow meter.
- Introduction to the asthma action plan and basic self-management skills.
- An arrangement for ongoing follow-up and education as an outpatient:

Options:

- Respiratory Outpatients Nurse.
- Respiratory Outreach for those with severe disability.
- General Practitioner / Practice Nurse.
- Community asthma educator - Canterbury Asthma Society.

Note:

Patients with recurrent admissions or life-threatening asthma should be referred to a Respiratory Physician.

Treatment on discharge will obviously vary from case to case. Usually the patient will receive:

- Inhaled corticosteroid - beclomethasone or budesonide 800-1200 mcg daily.
- Prednisone 40 mg mane for 1 week then 20 mg mane for 1 week (longer courses may ba required for chronic severe asthma).
- ß2- agonist inhaler to use as required (**NOT** regularly).
- Advice regarding common side effects of these medications :

$ß_2$ agonists	-	palpitations, anxiety, cramps.
Inhaled steroids	-	dysphonia, thrush - use mouth rinsing or a spacer.
Prednisone (short courses)	-	euphoria or dysphoria, hypertension, hyperglycaemia, mild indigestion.
Theophylline	-	indigestion, insomnia

Note:

For patients prescribed theophylline - arrangements will need to be made for a theophylline level to be taken following discharge.

PNEUMOTHORAX

CAUSES

- Traumatic (including pleural aspiration, insertion of CVP lines).
- Spontaneous.
- Chronic obstructive pulmonary disease.
- Asthma.

CLINICAL SIGNS

- Symptoms vary from mild breathlessness and/or chest pain to extreme respiratory compromise.
- Signs may include:
  - Decreased chest wall movement on affected side.
  - Trachea displaced towards or away from (tension) the affected side depending on air pressure within the pleural cavity.

- Diminished breath sounds.
- Surgical emphysema in the neck or over chest wall.

INVESTIGATIONS

- CXR - at the bedside if patient unwell.
- Arterial blood gases.

TREATMENT

- This varies from immediate life saving chest tube insertion to observation only. Sometimes no active treatment is needed.
- Indications for intercostal tube drainage:-
  - Tension pneumothorax. (If life threatening use needle decompression).
  - A deteriorating patient with pneumothorax complicating pre-existing lung disease.
  - Substantial collapse >50%. **Note** that percentage collapse is a volume assessment and may be underestimated on CXR.
  - Significant dyspnoea.
  - Traumatic pneumothorax or haemothorax.

Tube insertion - unless urgent, get experienced help if you have not done this before. Suggest contacting the Senior Respiratory Registrar, Respiratory Physician or Thoracic Surgical team. Follow hospital guidelines for the insertion and care of underwater seal drainage system.

CARE OF INTERCOSTAL TUBES

Intercostal tubes and underwater sealed drainage systems require specialized medical and nursing skills. Patients requiring these will normally be cared for by specialist Respiratory or Cardiothoracic Surgical teams.

Problems with intercostal tubes should be discussed with the appropriate specialist nursing and medical staff.

EMERGENCIES

- Acute deterioration in patient's condition:
  - Check all tube connections and underwater seals.
  - Administer high-flow oxygen.
  - Obtain an urgent CXR at the bedside.
  - Notify the Respiratory Physician on-call.
- Development of subcutaneous emphysema:
  - Check tubes for kinking / blockage.
  - Flush the tube with sterile saline.
  - Administer oxygen.

- Obtain an urgent CXR at the bedside.
- Notify the Respiratory Physician on-call.

COMMUNITY ACQUIRED PNEUMONIA (CAP)

GENERAL POINTS

- There is no unique clinical pattern or radiological appearance for a particular respiratory pathogen causing CAP.
- Management is initially based on an informed guess as to the likely causative agent **and** an assessment of the severity of the pneumonia.

DIAGNOSIS

- Differential diagnosis includes - lung carcinoma, lung infarction, cardiac failure and ARDS.
- The diagnosis is easily missed in the very young, the very old and the very ill.

ASSESSING THE MICROBIOLOGICAL CAUSES

- Streptococcus pneumoniae causes 50-70% of cases.
- With advancing age (>65 years) Gram negative and staphylococcal pneumonia become relatively more common.
- Seasonal variation occurs - influenza epidemics occur regularly in autumn and early winter.
- Patients with chronic lung disease often contract Haemophilus influenzae and Moraxella catarrhalis pneumonia.
- Consider HIV related infections (Pneumocystis carinii) in those patients in recognized risk groups (homosexuals, prostitutes, intravenous drug users and haemophiliacs).
- Mycoplasma often occurs in "epidemics" among young people every 2-3 years.

  Note:
  - Abnormal liver function tests and gastrointestinal symptoms may occur with any type of pneumonia.
  - Legionella pneumonia may present with headache and confusion.
  - Patients with mycoplasma pneumonia have often had symptoms for 2-3 weeks and have failed to respond to a broad-spectrum penicillin prior to admission.

ASSESSING SEVERITY

- This is essential as it directly influences initial management and patients with severe pneumonia can deteriorate rapidly.

Indicators of severe disease

HISTORY AND EXAMINATION	INVESTIGATIONS
Age >60 years	PaO_2 <65 mmHg on room air
Pre-existing medical illness	**Blood Urea ≥7 mmol/l**
RR ≥30/min	WBC <4 x 10^9/L or > 30 x 10^9/L
Diastolic BP ≤60 mmHg	CXR - multiple or spreading
Confusion	infiltrates

Those indicated in bold are signs of severe pneumonia and patients who have two or more of these signs of severe pneumonia have an increased mortality.

Note:

In patients with pre-existing lung disease, these variables must be interpreted in the context of previous lung function and arterial blood gases.

INVESTIGATIONS

The number of investigations depends on clinical circumstances.

- CXR - PA and lateral.
- CBC + diff.
- Na, K, urea, creatinine, glucose.
- Sputum sample for Gram stain:
  - Rinse mouth out with sterile water prior to collection.
  - Prior antibiotic usage must be recorded.
  - If storing overnight, refrigerate at 4°C.
  - Consider whether specific tests are indicated:
    - ~ Pneumococcal antigen.
    - ~ Stains for Pneumocystis carinii.
    - ~ Legionella immunoflorescence.
    - ~ ZN stain and culture for TB.
- Blood cultures - 2 sets prior to antibiotics.
- Arterial blood gases.
- Serology - acute specimen for the following:
  - Respiratory viruses.
  - Legionella species.
  - Mycoplasma pneumoniae (IgM and IgG).

Additional investigations

- Pleurocentesis:
  - Should be performed when a significant parapneumonic effusion is present on CXR. This should be supervised by a registrar.
  - Send for Gram stain, culture, cell count, pH, total protein, glucose and pneumococcal antigen.

- Bronchoscopy:
  Indications include:
  - Immunosuppressed patient.
  - Life threatening pneumonia.
  - Multiple CXR changes.
  - ICU patients.
  - Deterioration despite appropriate initial treatment.
  - Contact the Respiratory Physician on-call.

MANAGEMENT

- Assess severity.
- Resuscitate.
- Choose antibiotics.
  - Frequently empiric.
  - Depends on.
    - Clues as to likely pathogen.
    - Severity.
    - Initial Gram stain.

Note:

- **Antibiotics must be administered without delay in patients with pneumonia.**
- Initial antibiotic MUST cover Streptococcus pneumoniae which is the commonest isolated pathogen.
- Ideally one should obtain sputum and blood cultures (x 2) prior to giving antibiotics.

RECOMMENDATIONS FOR INITIAL ANTIBIOTIC TREATMENT

Mild-moderate community acquired pneumonia

♦ Those patients without any of the major risk factors for increased mortality.

	TARGETS	FIRST CHOICE	ALTERNATIVES
Majority - young, non-smoking, no underlying lung disease	Must cover strepto-coccus pneumonia	Amoxycillin 1g q8h IV	• Benzyl-Penicillin • Erythromycin
Older patient COPD Smokers	Must cover H. influenzae and M. catarrhalis	Augmentin 1.2g q8h	• Cefuroxime
Current mycoplasma epidemic	Should cover Mycoplasma pneumoniae	Erythromycin 1g q6h, IV	• Ciprofloxacin • Tetracycline • Roxithromycin • Clarithromycin
Little improvement with amoxycillin	Chlamydia species Legionella species	Erythromycin 1g q6h IV	• Clarithromycin

Change to oral therapy once patient afebrile for 48 hours.

Severe community acquired pneumonia

♦ Antibiotic choice is generally empiric and should cover serious bacterial causes.

Erythromycin 1g IV q6h

AND

Cefuroxime 1.5g IV q8h

Mix IV erythromycin in 500 ml of 0.9S and give via a large vein or CVP as phlebitis is a common complication.

Note:

- If staphylococcal pneumonia suspected - add flucloxacillin 2 g q4h IV.
- If Klebsiella pneumonia suspected - Ceftriaxone 2 g q12h IV and gentamicin.(**See page 27**).
- If strongly suspect Legionnella infection and patient's condition is deteriorating, add rifampicin 600 mg PO q12h.

MANAGEMENT

- Patients must be reviewed regularly to ensure that they are not deteriorating.
- All cases of severe CAP should be discussed with:
  - Respiratory Physician.
  - Microbiologist.
  - ICU if respiratory failure evident.
- **Patients should be monitored in the ICU or similar high dependency area if:**
  - Deteriorating despite fluid resuscitation, oxygen and antibiotics.
  - In respiratory failure - with deteriorating arterial blood gases.
  - Looking tired or exhausted.

INITIAL GENERAL MANAGEMENT

- Controlled oxygen therapy - prescribed on basis of arterial blood gases/ oximetry.
- Insert intravenous line (large vein if using IV erythromycin).
- Fluids:
  - Use 0.9S.
  - Treat septic shock aggressively (in ICU with CVP or Swann Ganz catheter).
  - Monitor response to fluid challenge by measuring pulse rate, BP, peripheral perfusion and urine output.
- Antibiotics as detailed.
- Recordings:
  - The first 24 to 48 hours is the time for particular vigilance (monitor temp, pulse rate, BP, respiratory rate, urinary output, initially 1-4 hourly).
- Physiotherapy - indications:
  - May be useful to assist in obtaining sputum sample.
  - May help with sputum clearance, especially in patients with underlying COPD.
- Use IV antibiotics until afebrile for 48 hours.

At discharge

- Appropriate oral antibiotic
  - Total duration of course 7-10 days in uncomplicated pneumonia.
  - Longer course required for complicated disease (e.g. COPD, bronchitis, severe or Legionella pneumonia).
- Stop smoking - refer for smoking cessation programme.
- Check spirometry in all smokers and refer to Respiratory Physician if abnormal.
- Instruct patient to contact own GP if development of fever, chest pain or SOB.

- Follow up appointment either with GP or hospital team at 6 weeks to include:
  - CXR.
  - Convalescent serology if considered relevant.

  Note:
  - CXR may take up to 3 months to clear in patients with COPD.
  - Physiotherapy may be needed.

COMMON COMPLICATIONS

- Parapneumonic effusion - up to 40%. Should always be aspirated to exclude empyema.
- Empyema - 1-5% of pneumonia. Consult with Respiratory Physician and/or Thoracic Surgeon.

OTHER CONSIDERATIONS

- Any pneumonia that doesn't resolve at usual rate - consider endobronchial obstruction, tuberculosis.
- Recurrent pneumonia in same segment - consider endobronchial obstruction, bronchiectasis, foreign body.
- Recurrent chest infections - consider immune status:
  - IgG / IgA deficiency.
  - Acquired immunodeficiency syndrome.
  - HIV.
  - Cystic fibrosis.
- Consider referral to a Respiratory Physician.

ASPIRATION PNEUMONITIS

Chronic occult microaspiration of gastric fluid is an important cause of respiratory disease and should always be considered in patients with unexplained cough, worsening bronchospasm, nocturnal attacks of coughing/choking, "morning dip" pattern of asthma, diffuse pulmonary shadowing and chronic/recurrent pneumonia.

Macroaspiration of gastric contents usually occurs following a clearly identifiable episode such as trauma, anaesthetic induction, epilepsy, unconsciousness, drug overdose etc. It may lead to a mechanical airway obstruction (medium-large particles), a chemical endobronchitis and pneumonitis, and can cause severe ventilatory impairment and disturbance of gas exchange.

CLINICAL DIAGNOSIS

The right upper lobe and the upper segments of both lower lobes are the pulmonary segments most commonly affected although no area is immune. Patients may present with indolent, multi-segmental pneumonia and a low grade fever. Others will present in respiratory failure.

MANAGEMENT

- **Macroaspiration pneumonia**
  - Assisted ventilation - the early use of ventilatory support may substantially reduce mortality. Seek immediate advice from ICU team.
  - Fluid replacement - this requires careful management and assessment, and if large volumes are required this is best done in ICU with appropriate monitoring.
  - Antibiotics - routine administration of antibiotics has not been demonstrated to reduce mortality or the incidence of bacterial pneumonia. Some patients deteriorate after 1-3 days associated with development of bacterial pneumonia, and antibiotic therapy will then be required. Mixed infections +/- anaerobic organisms are common. Anti biotic therapy must be guided by culture results. There is no recognized standard regime and pulmonary isolates that are resistant are common.
  - Steroids are not helpful.
- **Microaspiration pneumonia** - antibiotics to consider include: penicillin ± metronidazole or clindamycin.

PLEURAL EFFUSIONS

CLASSIFICATION

The differentiation between **exudates and transudates** is the essential first step in the diagnostic evaluation.

INVESTIGATIONS

- Diagnostic pleurocentesis - may be undertaken by medical staff with appropriate experience. A lateral decubitus CXR will allow identification of free fluid. There should be at least 1 cm of free fluid before attempting pleurocentesis. If in doubt, arrange an ultrasound examination. Use a 20 ml syringe with a 22G needle under sterile conditions.
- Exudates meet one or more of the following criteria:
  - Pleural fluid protein >30 g/L.
  - Pleural fluid protein/serum protein ratio >0.5.
  - Pleural fluid LDH/serum LDH >0.6.
- Other tests:
  - Cytology.
  - Microscopy and culture.
  - CBC + diff.
  - Glucose.
- Send the laboratory 10 ml in a sterile pottle and 5 ml in an EDTA (purple top) tube. Other investigations may be indicated depending on the particular clinical problem.

CAUSES

Exudates:

- Associated with pneumonia - if effusion is culture positive or Gram stain positive then this is an empyema. This requires consultation regarding drainage. Para pneumonic effusions with an elevated WBC, low glucose and/or a low pH (<7.2) may indicate an early empyema. The upper range of normal for WBC in pleural fluid is 1000 x 10^6/L and in this context most of these cells should be neutrophils. Such patients may require intercostal tube drainage. Refer to a Respiratory Physician.
- Carcinomatous - primary and metastatic.
- Lymphoma - surface marker analysis of cells may be diagnostic. In these cases contact the Haematology Department for both cytology and surface marker analysis.
- Intra abdominal disease - pancreatitis, subphrenic/hepatic abscess, oesophageal perforation, uraemia, Meig's syndrome.
- Pulmonary embolism.
- Collagen vascular disorders.
- Drug induced e.g. nitrofurantoin, bromocriptine.
- Rare causes - Sarcoidosis, tuberculosis, asbestos exposure, yellow nail syndrome, Dressler's syndrome, trapped lung syndrome (rounded atelectasis).

Transudates:

- Congestive heart failure.
- Cirrhosis.
- Nephrotic syndrome.
- Glomerulonephritis.
- Myxoedema.
- Pulmonary embolism.
- Sarcoidosis.

The diagnosis of pleural disease may require a pleural biopsy or thoracoscopy. Referral to a Respiratory Physician is recommended.

RHEUMATOLOGY

ACUTE SWELLING OF A SINGLE JOINT

The cause of the acute swelling must be established before any rational form of treatment can be given.

POSSIBLE CAUSES

- Trauma ± haemorrhage.
- Infection (septic arthritis signs may be modified if on steroids).
- Crystal deposition (gout and pseudogout).
- Reactive to infections elsewhere, urethritis, colitis, rheumatic fever.
- Rheumatoid disease.
- Other conditions e.g. palindromic rheumatism, psoriasis, osteoarthritis, inflammatory bowel disease.

INVESTIGATIONS

Essential information which should be obtained at once **before consulting Rheumatologist:**

- CBC + diff, platelets and ESR.
- Aspirate joint fluid and send to Microbiology for:
  - Gram stain and culture. (Send aspirate in sterile tube, plugged syringe or inoculate into blood culture bottle).
  - Cell counts and differential (put fluid into EDTA tube and mix).
  - Compensated polarised light examination for crystals (heparin tube).
- Blood culture - 2 sets. Consider possibility of gonococcal infection. Inform laboratory as special culture techniques will be needed.
- Serum urate level.
- Coagulation profile if bleeding disorder suspected.
- X-ray joint. X-ray hands, wrists and feet for evidence of rheumatoid arthritis if previous attacks of arthritis.

When indicated from history:

- Tissue type - HLA B 27.
- Swab throat, cervix, urethra, anus (should be cultured at bedside to grow N.gonorrhoea and to do antigen test for Chlamydia).
- Culture faeces (Yersinia, Salmonella, Campylobacter).
- Ferritin if haemochromatosis suspected.

TREATMENT

- **Septic arthritis:**
  - Splint joint and give analgesia.

- Use appropriate antibiotic.
  If Gram positive cocci seen or Staphylococci suspected give penicillin 2 mega units and Flucloxacillin 2 g both IV q6h.
  If allergic to penicillin give cefuroxime 750 mg IV q8h if allergy mild or vancomycin if allergy severe. **See section on penicillin allergy (Page 99).**
- Repeat aspiration of synovial fluid daily.
- If not settling in 48 hours arthroscopic washout often needed. Consult orthopaedic service promptly.

♦ **Acute gout or pseudogout**

- Initial therapy - Indomethacin 75 mg PO stat then 50 mg q6h or naproxen 750 mg stat then 250 mg q8h. In the presence of previous peptic ulceration or renal disease alternative therapy may be indicated. Either a short course of corticosteroids parenterally or orally (e.g. prednisone 40 mg daily) **or** colchicine 1.0 mg stat, then 0.5 mg every 2-3 hours until pain disappears or GI symptoms develop. Maximum dose 10 mg.
- After an acute attack of gout has subsided consideration must be given as to the cause of the hyperuricaemia. If uric acid lowering drugs are used it is desirable to cover the period of initiation with NSAIDs or colchicine for up to 6 weeks.

♦ **Haemarthrosis**

- Immobilise joint.
- If bleeding disorder suspected **do not** aspirate joint before seeking advice. If however blood is found unexpectedly on a diagnostic tap, aspirate as much as possible. Remember to ask about family history of bleeding disorders.
- Unless trauma clearly the cause refer to haematologist as a bleeding disorder likely. Following consultation appropriate coagulation factor replacement may be indicated. A normal coagulation profile does not necessarily rule out a coagulopathy. Significant trauma requires referral to an Orthopaedic surgeon.
- X-ray if history of trauma.

THROMBOSIS, EMBOLISM AND ANTICOAGULATION

PERIPHERAL VENOUS THROMBOSIS

CLINICAL

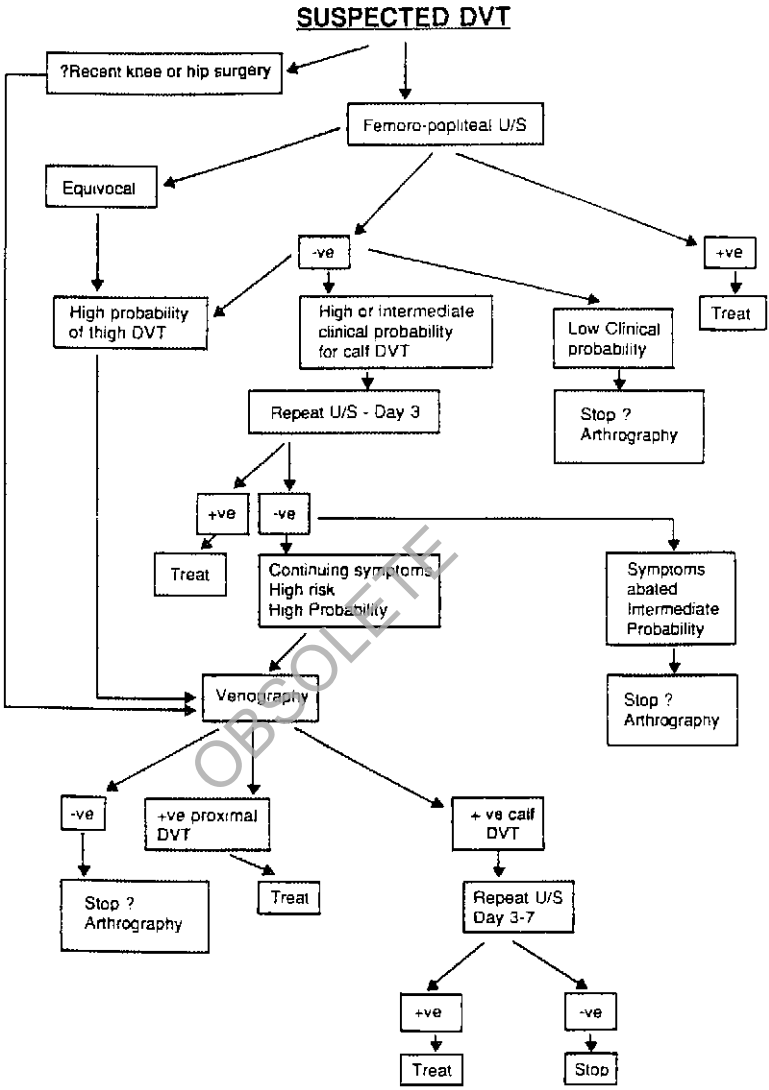
- Difficult to diagnose clinically especially if thrombus is restricted to calf veins. There is usually swelling, bluish discolouration, pain, tenderness, and dilated superficial veins.

PRECIPITATING CAUSES

- Surgery. Immobilization including travel. High oestrogen oral contraceptives. Stilboestrol. Polycythaemia. Thrombocytosis. Malignancy. Pregnancy and puerperium. **Suspect** anti-thrombin III, protein C or protein S deficiencies or the lupus anticoagulant if the patient is under 45, and has recurrent thrombosis or there is a family history of thrombosis.

INVESTIGATIONS **(see decision tree)**

- Ultrasound of the femoral and popliteal veins is usually the investigation of choice. A thrombus can be seen and if present the vein will not be compressible. This investigation will not detect calf vein thrombosis. Calf vein thrombi do not embolise and may not require therapy. They may however extend proximally. If a calf vein thrombosis is strongly suspected and the ultrasound is negative, the test must be repeated after three days to detect any extension. Occasionally calf symptoms may require definitive diagnosis and in this case a venogram is required. Check:-
  - CBC + diff, (polycythaemia, raised platelets).
  - PT, APTT before starting anticoagulant treatment.
  - If protein C, protein S or anti-thrombin III deficiency or lupus anticoagulant suspected take an extra 10 ml blood into citrate and request these assays. **These samples must be taken before starting treatment.**
  - If ultrasound negative and a ruptured Baker's cyst is a possibility consider arthrography.


SUSPECTED DVT
?Recent knee or hip surgery
Femoro-popliteal U/S
Equivocal
-ve
+ve
Treat
High probability of thigh DVT
High or intermediate clinical probability for calf DVT
Low Clinical probability
Repeat U/S - Day 3
Stop ? Arthrography
+ve
-ve
Treat
Continuing symptoms High risk High Probability
Symptoms abated Intermediate Probability
Stop ? Arthrography
Venography
-ve
+ve proximal DVT
+ ve calf DVT
Stop ? Arthrography
Treat
Repeat U/S Day 3-7
+ve
-ve
Treat
Stop

MANAGEMENT

For dosage details of anticoagulant drugs - **see page 191**

- Heparin infusion - the duration of treatment can be restricted to four days if the thrombosis is limited to calf or lower femoral veins. For ilio-femoral thrombosis or for pulmonary embolism a 7-10 day heparin infusion is still recommended. If a four day heparin infusion is planned warfarin will need to be started at the same time. For extended heparin therapy start warfarin 4 days before intended heparin termination.
- If massive thrombosis and no contraindication consider fibrinolytic therapy.
- If ruptured Baker's cyst - bed rest, 40 mg Depo-Medrol into the joint. Rheumatology consult.

PULMONARY EMBOLISM

- Pulmonary embolism should be considered in the presence of any of the following features:
  - Dyspnoea.
  - Chest pain (including typical angina).
  - Heart failure.
  - Unexplained raised venous pressure.
  - Syncope/collapse/shock.
  - Haemoptysis.
  - Respiratory failure.
  - Pulmonary hypertension/cor pulmonale.
  - Post operative atelectasis.
  - Acute arrhythmia.
  - Pleural effusion.
  - Lower respiratory tract infection.
  - Unexplained hypoxia.
  - PUO.
  - Apparent hyperventilation.
- The classical triad of chest pain, dyspnoea and haemoptysis is rarely seen.

CLASSIFICATION

- **Major pulmonary embolism:** compromised cardiac and/or pulmonary function. Cardio-pulmonary support will be required.
- **Minor pulmonary embolism:** cardio pulmonary function is not compromised.
- **Chronic pulmonary thromboembolism:** recurrent emboli with compromised cardio pulmonary function.

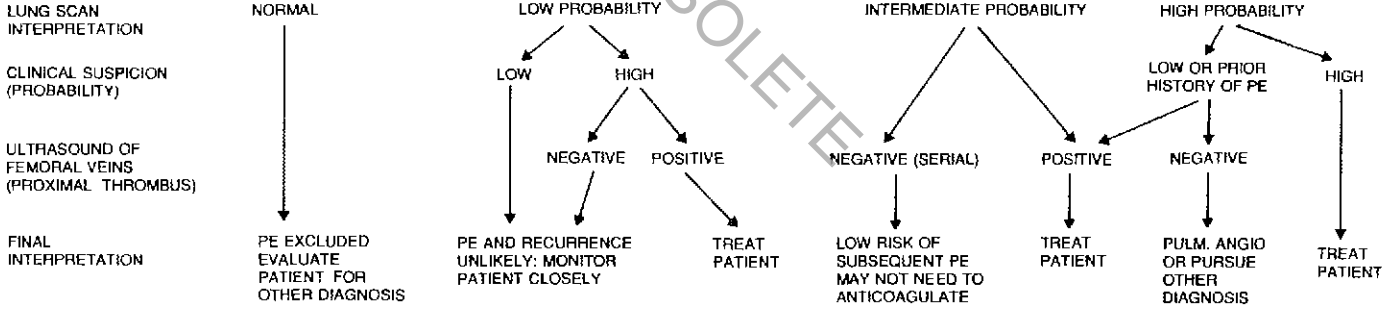
INVESTIGATIONS **(see decision tree)**

PULMONARY EMBOLISM (PE) DIAGNOSIS

PE SUSPECTED (SYMPTOMS AND SIGNS NON-SPECIFIC)

ALTERNATIVE DIAGNOSES CONSIDERED BUT UNLIKELY

PE STILL SUSPECTED IF NO CONTRA-INDICATION
BEGIN HEPARIN, ORDER URGENT VQ SCAN.



- INVESTIGATIONS SHOULD BE COMPLETED RAPIDLY AS THEIR SENSITIVITY DECLINES QUICKLY. LUNG SCANS SHOULD BE DONE WITHIN 48-72 HOURS OF ACUTE EVENT.
- COMBINING LUNG SCAN AND VENOUS ULTRASOUND/VENOGRAPHY DECREASES THE NEED FOR PULMONARY ANGIOGRAPHY

Pulmonary embolism may have to be initially treated on a presumptive basis. In all cases attempts must be made to establish the diagnosis as treatment can be hazardous.

There are only two definitive tests:

- **Lung scan** - patients with a clear CXR and a normal ventilation/perfusion scan have a probability of <0.01 for a pulmonary embolus. Large unmatched segmental perfusion defects have a sensitivity of >90% and some authorities consider this to be a "definitive test". Any other pattern cannot be "definitive".
- **Pulmonary angiogram** - the "gold standard". Abrupt cut off of a vessel and a filling defect are diagnostic but partial lysis and dissemination of the clot make interpretation difficult. The characteristic appearances are more likely to be seen if the pulmonary angiogram is performed early. The mortality and morbidity associated with the procedure are extremely low (<1%) if it is performed in a suitable unit with experienced personnel. Contact the radiology registrar on call or radiology consultant for advice.

All other tests are at best suggestive

- **Arterial blood gas** - some degree of hypoxia and/or increased A-a gradient is usually seen but normal gas exchange does not exclude the diagnosis. Alveolar hyper-ventilation with low $PaCO_2$ is often present.
  The level of hypoxia (or A-a gradient) does not necessarily reflect the level of cardiopulmonary impairment.
- **Chest radiograph** - excludes other pathology such as pneumothorax, but is never diagnostic. The most specific sign is segmental loss of vascular markings. Radiological shadowing takes at least 24 hours to develop and may be due to pulmonary infarction or segmental atelectasis/oedema.
  The following are compatible with pulmonary embolism:
  - Single or multisegmental shadowing (the classical pleurally based triangular shadow is rare).
  - Subsegmental atelectasis.
  - Pleural effusions.
- **ECG** - excludes obvious myocardial infarction but there are no diagnostic features in PE. Signs of acute pulmonary hypertension (right ventricular hypertrophy/strain) suggest significant cardiopulmonary impairment.
  Signs include:
  - Sinus tachycardia (most common ECG feature). Atrial fibrillation may occur.
  - Right axis deviation (S1/Q3/T3) is seen infrequently.
  - T inversion in right sided precordial leads.
- **Assessment of lower limb venous system** - if lung investigations are inconclusive an ultrasound examination of the femoral veins is recommended. A negative result lowers the probability of PE. A positive result increases the probability and in any case justifies anticoagulation in its own right.

- **Assessing the functional impairment** - the haemodynamic effects are mainly due to acute pulmonary hypertension Sudden increases in the mean pulmonary pressure of 30 mmHg can result in cardiovascular collapse and death. If there is evidence of right ventricular compromise which persists 30 minutes after adequate heparinization and oxygenation then thrombolytic therapy should be considered. Seek advice.
- **Evidence of right ventricular compromise include:**
  - Hypotension.
  - Parasternal cardiac impulse and loud P2.
  - Peripheral vasoconstriction.
  - Severe hypoxia (PaO_2 <50 mmHg).
  - ECG evidence of RV strain.

  Note:

  Avoid diuretics in this situation as they may aggravate hypotension.

MANAGEMENT **(see Table)**

TREATMENT OF DEEP VEIN THROMBOSIS OR PULMONARY EMBOLISM

INDICATION	DRUG THERAPY	DURATION	MONITORING
DVT	**Fragmin** 120u/kg sc bd or	4 days If PT ratio (INR) > 2 at day 4	Not usually required.
	Standard heparin 5,000 iu stat then with iv continuous infusion at 30,000 iu / 24 hrs, adjust according to APTT or	as above	APTT range* currently 50 - 70 secs. Check 6-12 hourly until stable then daily.
	Standard heparin 15,000 iu sc bd adjust according to APTT	as above	APTT range* currently 50 - 70s. Check mid dose.
		AND COMMENCE	
	Warfarin 15 mg po day 1 5 mg day 2 then dose according to PT ratio	6 weeks	PT ratio (INR) range 2.0 - 3.0. Check daily for 5 days. If controlled then twice weekly for 2 weeks then once a week
PE or Massive DVT	**Standard heparin**** 5,000 u iv stat then as above	7 days	As above
		THEN	
	Warfarin as above starting on day 3	3-6 months	As above although PT ratio may be done every 2 weeks if stable.
Recurrent DVT or PE	**Standard heparin**** as above and	4 days (DVT) 7 days (PE)	As above
	Warfarin as above	lifelong	PT ratio (INR) range 2.0 - 3.0 as above. If recurrence while on warfarin increase range to 3.0 - 4.5 Check for underlying cause

* Canterbury Health Laboratories

** Fragmin may well be effective in this situation but this remains unproven

♦ If **massive pulmonary embolism** is suspected and life saving treatment appropriate:

- Immediate heparinization.
- Cardiovascular support with inotropic therapy, oxygen, analgesia.
- Urgent pulmonary angiography.
- Give fibrinolytic therapy provided no absolute contra-indication exist and no improvement in cardiovascular function is seen over 20-30 minutes. Fibrinolytic therapy carries a higher risk of bleeding than heparin and the advice of a Haematologist should be sought. For contra-indications to fibrinolytic therapy **see page 193**.

  Note:

  If pulmonary angiography cannot be obtained, attempt a perfusion / ventilation scan although the patient may well not tolerate the latter. If neither is available, and other conditions excluded as far as possible, then heparinize. The decision to use fibrinolytic therapy under these circumstances must be left to the clinician in charge of the patient.

♦ **Minor pulmonary embolism:**

- Heparin infusion for 7-10 days followed by warfarin for 3-6 months **(see page 189).**
- Oxygen if PaO_2 less than 60 mmHg.
- Analgesia as required.

PERIPHERAL ARTERIAL EMBOLISM

CAUSES

♦ Consider when acute loss of function occurs with or without pain in limbs, gut, kidney or brain. Particularly following myocardial infarction, or in atrial fibrillation, valve disease.

INVESTIGATIONS

♦ Arteriography is indicated for the pale cold limb with absent pulses. This is urgent and may help distinguish between embolism and thrombosis.
♦ MSU/IVU or arteriography for renal embolus.
♦ CT scan head to exclude haemorrhage if cerebral embolus suspected.

THERAPY

♦ Surgical consult for an embolus causing ischaemic gut, limb or kidney is mandatory and urgent.
♦ Leave an ischaemic limb alone - do not heat or cool.
♦ Anticoagulant or fibrinolytic therapy may be appropriate if surgery is not indicated.

THE USE OF ANTICOAGULANTS AND RELATED DRUGS

Standard heparin (see page 189)

- **Check PT and APTT before starting treatment**, and take 3-4 citrate tubes so that tests to detect a thrombotic tendency (e.g. lupus anticoagulant) may be done later if indicated.
- The anticoagulant effect is immediate. Heparin is usually given by continuous infusion in syringe pump or rate controlled drip with an initial 5,000 IU IV bolus. Control with APTT and aim for the therapeutic time (currently 50-70 seconds). Check APTT 2-3 times daily till controlled then daily. Dose required usually lies between 20,000-80,000 IU/24 hours, and it is reasonable to start at a rate of 30,000-40,000 IU/24 hours but larger doses may be needed. Use the special heparin prescription chart (MR5B).

Low molecular weight (LMW) heparins (see table for dosages)

The therapeutic response to LMW Heparins is more predictable than that to standard Heparin. They are usually given by subcutaneous injection without monitoring. Although the direct drug costs are higher, other factors (? earlier discharge) favour their use. LMW heparin is of proven value in DVT prophylaxis and in the treatment of proximal and distal DVT.

Warfarin

- **Check PT and APTT before starting treatment.**
- Anticoagulant action begins in hours to days related to the half lives of the factors affected (II, VII, IX, X). Antithrombotic action takes some days to achieve. Recommended initial dosage is 15 mg on day one and 5 mg day two, if pre-treatment PT ratio <1.4. Aim to start warfarin 4-5 days before it is planned to stop heparin. During this time check PT and APTT at least daily. Use lower doses if there is any reason to suppose that the patient will be sensitive to warfarin e.g. old age, low body weight, initial PT raised, abnormal liver function tests or drugs known to cause increased sensitivity to oral anticoagulants **(see below)**.

The degree and duration of anticoagulation will vary according to condition being treated. The following is recommended:-

	Prothrombin Ratio (INR)*	Duration
Pre and perioperative anticoagulation	2.0-2.5	Days
Treatment of DVT	2.0-3.0	6 weeks
Treatment of PE or massive DVT	2.0-3.0	12-24 weeks
Treatment of recurrent DVT or PE**	3.0-4.5	? life long
Prosthetic heart valves	3.0-4.5	life long
Arterial disease	3.0-4.5	life long

* The ratio obtained will be influenced by the type of thromboplastin used and should be adjusted and reported by the Laboratory as the International Normalized Ratio (INR).

** Recurrence despite prothrombin ratio between 2 and 3.

Drug interactions with Coumarin type oral anticoagulants

Drugs expected to potentiate oral anticoagulants

Alcohol-----dose dependent
Allopurinol
Amiodarone
Anabolic steroids
Aspirin & its analogues----in large doses
Chloramphenicol
Clofibrate
Chlorpromazine
Cimetidine / Ranitidine
Co-trimoxazole
Danazol
Dextrothyroxine
Disulfiram
Ethacrynic acid
Glucagon
Ketoprofen
Ketaconazole
Mefenamic acid
Metronidazole
Moxalactam
Naproxen
Neomycin
Quinidine
Salicylates (see aspirin)
Thiouracils
Thyroxine

Drugs which antagonise or may antagonise anticoagulant therapy

Antacids
Antihistamines
Barbiturates
Carbamazepine
Cholestyramine
Corticosteroids
Glutethimide
Griseofulvin
Mercaptopurine
Oral contraceptives
Phenytoin
Primidone
Rifampicin
Vitamin K-only K_1 & K_2

Drugs potentiated by oral anticoagulants

Chlorpropamide
Phenytoin
Tolbutamide

Streptokinase, urokinase, tPA (fibrinolytic therapy)

♦ May be recommended for recent large vein thrombosis (e.g. ilio-femoral, axillary) or life threatening pulmonary embolus if no contraindications are present. Take citrate specimen (5 ml) to measure antistreptokinase antibodies. Give 250,000 IU over 30 minutes followed by 100,000 IU hourly either by pump or rate controlled drip (**see Cardiology Section page 39 for protocol details**).

We recommend controlling streptokinase treatment with thrombin times aiming for 2-4 times the control value. The length of the streptokinase infusion will depend on the clinical situation. IV standard heparin should be given by IV infusion in the dosage given above but without an initial bolus **before** thrombin time drops below twice normal. Hydrocortisone may be used to prevent allergic reactions. The Haematologist should be consulted as this treatment is more hazardous than heparin and any abnormal bleeding tendency is not easy to reverse.

Note:

Contra-indications to fibrinolytic therapy

- Active bleeding (including menstruation) / known bleeding disorder.
- Major surgery or trauma in last 6 weeks.
- Recent non compressible vascular puncture.
- Extensive CPR (>5 minutes).
- Recent CVA (<6 weeks).
- Active peptic ulcer disease.
- Severe liver disease.
- Pregnancy.
- Severe diabetic retinopathy.
- SK in last 12 months or sustained known allergy.
- Recent streptococcal infection.
- Uncontrolled hypertension (diastolic >115 mmHg) (systolic >180 mmHg).

Streptokinase and other activators of fibrinolysis such as tissue plasminogen activator (tPA)are used as an emergency treatment for acute coronary artery thrombosis (**see Cardiology Section page 39**). The dosages recommended and the monitoring used are different from those given above.

TREATMENT OF ANTICOAGULANT OVERDOSAGE

Heparin

- Note short half life (about 30 minutes). Reversal only necessary if serious bleeding. Protamine sulphate **must be given slowly** and may cause serious allergic adverse reactions. If necessary give protamine sulphate IV 1 mg per 100 IU of heparin estimated to be remaining in circulation. This is essentially an educated guess based on the amount of heparin given since its half life in the circulation is quite variable. Neutralization tests can be performed but are of limited value. Caution: Excess protamine sulphate may act as an anticoagulant itself.

Warfarin

- Rapidity and extent of reversal will depend on clinical situation. For example if ratio is high and there is no bleeding, withdrawal of warfarin may be sufficient. In desperate situations immediate but transient reversal may be

obtained with IV 1-2 units of fresh frozen plasma or prothrombinex. For delayed but complete reversal give IV 5 mg Vitamin K slowly. For delayed but partial reversal with return of ratio to therapeutic range but not to normal, IV Vitamin K 1 mg. In general Vitamin K takes about 24 hours to produce its full effect.

Streptokinase

- Abnormal haemorrhage may be very difficult to correct at least for some hours. If fibrinogen level is low, cryoprecipitate may help.

APPENDIX 1

DISEASES NOTIFIABLE IN NEW ZEALAND (includes suspect cases)

NOTIFIABLE INFECTIOUS DISEASES UNDER THE HEALTH ACT 1956

Section A - Infectious Diseases Notifiable to Medical Officer of Health and Local Authority

Amoebiasis
Anthrax
Brucellosis
Cholera
Diphtheria
Dysentery (see amoebiasis & shigellosis)
Enteric fever - typhoid, paratyphoid
Hepatitis A
Hepatitis B
Hepatitis non A or B
Leptospirosis
Meningococcal Infection
Meningoencephalitis-primary amoebic
Plague - bubonic, pneumonic, or septicaemic
Poliomyelitis
Psittacosis
Rabies
Relapsing fever
Salmonellosis
Shigellosis
Smallpox
Trachoma
Typhus
Yellow fever

Section B - Infectious Diseases Notifiable to Medical Officer of Health

Acquired Immune Deficiency Syndrome (AIDS)
Campylobacter infection
Congenital rubella
Encephalitis - acute arthropod-borne
  -Post infection
  -Post vaccinal
Lassa fever
Legionellosis
Leprosy
Listeriosis
Marburg virus-like disease
Neonatal infection - any infant who within 14 days of birth or whilst in a maternity hospital exhibits one of the following:
  - Congenital rubella
  - Congenital syphillis
Neonatal infection (continued)
- Eye infection due gonococcus
- Gastroenteritis
- Listeriosis
- Meningoencephalitis
- Septicaemia
- Staphylococcal skin infection
- Streptococcal Infection Groups A & B
- Toxoplasmosis

Puerperal infection - any woman who within 14 days of childbirth or abortion or whilst in a maternity hospital, has a temperature of 38°C or over or who has any infection either generalised or local arising from the genital tract or breasts.

Rheumatic Fever

NOTIFIABLE DISEASES OTHER THAN NOTIFIABLE INFECTIOUS DISEASES UNDER THE HEALTH ACT 1956

Notifiable to the Medical Officer of Health

Actinomycosis
Ancylostomiasis (hookworm disease)
Carcinoma of the nasal cavity or associated air sinuses
Cysticercosis
Damage to eyesight to include ulceration of the cornea or heat cataract
Decompression sickness arising from occupation
Decompression sickness arising from recreation
Denge Fever
Eclampsia
Food poisoning - all forms (chemical, bacterial, toxic)
Hydatid disease
Lead Absorption (not occupational) causing whole blood levels in excess of:
- for 0 - 10 years of age 1.45 umols/litre
- over 10 years of age 2.9 umols/litre

Malaria
Noise induced hearing impairment
Poisoning by: Arsenic, Cadmium, Chromium, Lead, Manganese, Mercury } and their compounds
Poisoning by: any chlorinated solvent
any organophosphate pesticide
any other pesticide
phosphorous
Poisoning arising from chemical contamination of environment
Primary malignant neoplasm of the mesothelium (mesothelioma) of the pleura or of the peritoneum.
Primary neoplasm of bladder
Pulmonary diseases due to the inhalation of dust fibres or chemicals at work
Ross River fever
Schistosomiasis (Bilharziasis)
Skin diseases arising from occupation
Taeniasis
Tetanus
Trichinosis

NOTIFIABLE DISEASES UNDER TUBERCULOSIS ACT 1948

Notifiable to the Medical Officer of Health

Tuberculosis (all forms)

INFORMATION FOR THE MEDICAL PRACTITIONER

COMMUNICABLE DISEASES AND SEPTIC CONDITIONS IN RELATION TO MATERNITY PATIENTS AND INFANTS

Obstetric Regulations 1975

Regulation 18. Duty of medical practitioner to notify - If any patient in a maternity hospital develops puerperal infection, or any mother or infant in any such hospital develops a septic condition, or develops symptoms that could lead to the diagnosis of a communicable disease or create the suspicion that a communicable disease exists, or an infant develops a neonatal infection the medical practitioner in charge of the patient, mother, or infant shall inform the medical superintendent or manager of the maternity hospital of that fact and of the precautions being taken. As soon as practicable after the medical superintendent or manager has been informed of a case under subclause (1) of this regulation, the medical superintendent or manager shall take all necessary steps to prevent the spread of the infection.

VENEREAL DISEASES

Duties of Medical Practitioners with Respect to Patients Suffering from Venereal Disease

Health Act 1956

Section 89 - Every medical practitioner who attends or advises any patient for or in respect of any venereal disease from which the patient is suffering shall, by written notice in the prescribed form delivered to the patient -

(a) Direct the attention of the patient to the infectious character of the disease, and to the penalties prescribed by this Act for infecting any other person with that disease; and
(b) Warn the patient against contracting any marriage until he has been cured of that disease or is free from that disease in a communicable form; and
(c) Give to the patient such printed information relating to the treatment of venereal disease, and to the duties of persons suffering from such disease, as may be issued by the directions of the Minister.

Veneral Diseases Regulations 1982

Regulation 7

(1) Where any medical practitioner (including any medical officer of any hospital) has been treating any person who is suffering from syphilis, gonorrheal infection affecting any site, chancroid or venereal granuloma in a communicable form, and the the patient either -

(a) Fails for 1 week after the date fixed for the purpose by the medical practitioner to attend for further treatment; or

(b) Fails on 2 or more successive occasions to attend for treatment as directed by the medical practitioner, -

the medical practitioner shall, unless he knows that the patient has in the meantime placed himself under treatment by another medical practitioner, forthwith send to the Medical Officer of Health a notice relating to the patient in form 1 in the Schedule to these regulations. (Form H 787).

(2) Where any medical practitioner has reason to believe that a patient whom he is or has been treating for syphilis, gonorrheal infection affecting any site, chancroid or venereal granuloma in a communicable form has had, within the period during which he was probably infected, intimate sexual contact with a person whose name, address or description is supplied to the medical practitioner, or of which he otherwise becomes aware, the medical practitioner may send to the Medical Officer of Health a notice in form 2 in the Schedule to these regulations. (Form H 793).

IMMUNISATION INFORMATION FOR INTERNATIONAL TRAVEL

Information on immunisation for overseas travel is available from the Medical Officer of Health of the District.

APPENDIX 2

TREATMENT AND REFERRAL OF PATIENTS TO HYPERBARIC UNIT

A double compartment hyperbaric chamber for the treatment with hyperbaric oxygen (HBO) is available at the Princess Margaret Hospital (PMH). (Hyperbaric Oxygen Therapy, Undersea, Hyperbaric Medical Society Publication No. 30, 1986):

Current indications for HBO are:

- Decompression sickness.
- Gas embolism (from any cause including iatrogenic).
- Clostridial myonecrosis or cellulitis (combined in a planned approach with surgical debridement and antibiotics).
- Mixed aerobic/anaerobic soft tissue infections with tissue necrosis, eg necrotising fasciitis.
- Carbon monoxide poisoning (possibly cyanide and H_2S also).
- Crush injury with acute traumatic ischaemia.

Referral for these conditions is **VERY URGENT.** Currently, any patient in one of these catergories will be accepted for assessment and therapy on an acute emergency basis.

HBO is also indicated in some other conditions:

- Osteoradionecrosis (especially of mandible).
- Refractory osteomyelitis.
- Diabetic ulcer.

These conditions require extended treatment courses. Since the chamber is staff voluntarily, this may only be considered on an individual basis with the approval of the General Manager for Hospital Services.

PATIENT REFERRAL (INPATIENT AND EXTERNAL REFERRALS)

Since these are nomally emergency referrals the following procedure will ensure a prompt response.

- Ring PMH Operator Extn 6000 or (03) 3377-899, and ask him / her to 'ALERT HYPERBARIC TEAM'.
- Give the Operator your name and contact phone number.
- Once a member of the volunteer medical staff has been contacted by the Operator they will return the enquirer's call.
- Trying to contact individual physicians may result in delay. However, in normal working hours, also ring the Department of Anaesthesia, Christchurch Hospital (03) 3640 640 (Extn 89345). The unit is administered by the Department of Anaesthesia with Dr Alastair Gibson as Medical Officer in Charge. Less urgent consultation requests should be sent to Dr Gibson, Department of Anaesthesia.

APPENDIX 3

TRANSFUSION REACTIONS

The following is derived from the Standing Orders on Blood Transfusion.

You are strongly urged to read the booklet - **Blood Transfusion Procedures In New Zealand** Part II: **Clinical use of blood and blood products, 4th Edition 1988.** This booklet is available on request from Blood Bank.

- The nursing staff have instructions to stop transfusions as soon as reactions occur and before notifying the House Surgeon. Reactions occurring during blood transfusion are extremely variable. Mild febrile reactions, temperatures less than 38.5°C, and transient skin rashes are common. Since a serious haemolytic reaction may initially present with such mild symptoms, any reaction occurring during blood transfusion must be immediately reported to the doctor responsible.
- The transfusion should not be stopped if the patient has a low grade fever and is symptom free. Mild skin reactions (pruritus, urticaria) merely require the administration of an antihistamine and slowing of the transfusion.
- If more severe reactions occur with fever over 38.5°C, chills, restlessness, nausea, rigors, shock, tachycardia, hypotension, the transfusion must be stopped immediately. The blood transfusion laboratory should be alerted, an adverse reaction form completed and the investigations below carried out.
  - The identity of the recipient and the name on the compatibility label should be checked.
  - The unit of blood should be inspected for signs of bacterial contamination and sent for Gram stain and culture.
  - The remains of the unit together with the giving set should be sent to the laboratory.
  - 10 ml clotted blood should also be sent to the laboratory, together with a post-transfusion sample of urine.

The blood transfusion laboratory will recheck the donor and the recipient blood groups, will screen for red cell and white cell antibodies, repeat the crossmatch, and will assess whether the donor unit was contaminated by bacteria. Inspection of the post transfusion plasma and urine for haemoglobin will provide an immediate guide to a serious haemolytic transfusion reaction.

Bacterial contamination of blood and platelet concentrates is a rare cause of an adverse transfusion reaction. The bacteria involved in contaminated blood are frequently Gram negative organisms. Yersinia enterocolitica has recently been implicated more frequently than in the past. Contaminated blood may appear either purple or darker than normal and may contain clots. When bacterial contamination is suspected empiric antibiotic therapy and general supportive treatment must be immediately commenced. A negative Gram stain result on

the blood bag contents does not exclude bacterial contamination. The recommended combination antibiotic regime is gentamicin IV 4.5 mg/kg loading dose plus **elther** ciprofloxacin 500 mg q12h **or** piperacillin 4 g IV q8h.

- If, after the above tests, no cause can be found for a moderately severe reaction, it may be presumed to have an allergic basis. The trensfusion can then be restarted following the administration of either paracetamol or hydrocortisone. These reactions are often due to leucocytes in the blood and can usually be avoided by using leucocyte poor blood. Blood filters will reduce the amount of white cell antigen given to the patient and reduce the likelihood of febrile reactions in repeatedly transfused patients. Filters are available from Pharmacy.

APPENDIX 4

CHRISTCHURCH HOSPITAL DISASTER PLAN

Each hospital in the city has a plan for managing a mass casualty incident and it is the responsibility of each staff member to acquaint themselves with the plan at their current workplace.

The response to a mass casualty event is centred on Christchurch Hospital and its Emergency Department. Activation is at the discretion of the senior staff on duty in the Emergency Department and key personnel will be contacted by the switchboard. A cascade notification structure is in place, relying on these key personnel to notify designated others and so on. It is your responsibility to know your role in the plan and who you must notify. Copies of the Disaster Plan and of departmental plans are available in each department.

Those staff whose role calls for their attendance in the Emergency Department should do so promptly and should report to the Emergency Department Controller who will allocate duties. Staff not required by the plan to attend the Emergency Department should continue with their normal duties, but, when possible, should avoid utilizing the pathology and radiology services. If additional assistance is needed in the Emergency Department, staff will be contacted by the switchboard.

Stand down after the emergency is the decision of the hospital controller and will be notified to staff by way of the cascade notification system initiated by the switchboard.

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