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Management Guidelines for Common Medical Conditions

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Eighth Edition 2000

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Internal Medicine Services

Date of Issue: 1 March 2000
Date of Expiry: 1 November 2001

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Canterbury Health Limited

Management Guidelines for Common Medical Conditions

8th EDITION 2000

INTERNAL MEDICINE SERVICES

OBSOLETE

Date of Issue 1 March 2000
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Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in treatment and drug therapy are required. The editors and the publisher of this work have checked with sources believed to be reliable in their efforts to provide information that is complete and generally in accord with the standards accepted at the time of publication. However, in view of the possibility of human error or changes in medical sciences, neither the editors, nor the publisher, nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete and shall have no liability for any loss or damage arising out of any reliance on the information.

Readers are encouraged to read the product information sheet included in the package of each drug they plan to administer to be certain that the information contained in this book is accurate and that changes have not been made in the recommended dose or in the contraindications for administration. This recommendation is of particular importance in connection with new or infrequently used drugs. Readers should also consult their own laboratories for normal values.

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1. Introduction - Eighth Edition 2000

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. This booklet has proven popular and this is the Eighth Edition.

These guidelines are not designed to 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 Paediatrics.**

Remember that the delivery of medical care is a team 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 most stressful 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 clearly explained.

Finally, remember the financial costs of your actions. It is often 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 would like to acknowledge the financial support of Canterbury Health Ltd.

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2. 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
Ca	Calcium
PO_4	Phosphate
AST	Aspartate Aminotransferase
GGT	Gamma Glutamyltransferase
Alk. Phos.	Alkaline Phosphatase
Bili	Bilirubin
Mg	Magnesium
Alb	Albumin
Cl	Chloride
KCl	Potassium Chloride

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Fluid Prescriptions

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

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

The correct symbol for micro (grams, moles, etc.) is the Greek letter μ. However when prescribing, this is often written poorly and this can be dangerous. In Canterbury Health when prescribing in micrograms, the abbreviation mcg must be used not μ.

The units used to express normal ranges for white cells in CSF, pleural, peritoneal and joint fluids and urine are confusing.

The following are the approximate upper normal values for white cells in:

CSF	<5	All expressed as	10^6/L or
Pleural	<1000		per mL or
Peritoneal	<300		per mm^3 or
Synovial	<3000		per μL^{-1}
Urine	<10		All these are the same!

Check which units your laboratory is using and always obtain that laboratory's normal range.

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3. General Medicine Services

Main Office 1st Floor, Riverside ☎ 81020 Fax 81025

Inpatient Services

Primarily in Wards 23, 24, 29 and 30. There is an assessment area adjacent to the Emergency Department. There are ten General Medical teams rostered on a 1 in 5 basis with two teams per 24 hours (8.00 am - 8.00 am). One team does the cardiac arrest calls and the other surgical consults.

Consultation Guidelines

An acute consult service is provided mainly to the surgical areas. Contact the appropriate acute medical registrar/consultant via the Hospital Switchboard.

Staff

Currently 19 physicians work in General Medicine and Dr J O'Donnell is the Clinical Director.

Outpatient Consultations

At the present time two General Medical clinics are held each week.

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4. 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.
Further information is available in Diagnostic & Support Division Manual Volume 4 (Green colour code)

4.1 Computer Access to Results

- Most laboratory and radiology results are available online and in printout form, in wards and departments. The system used at Christchurch Hospital is referred to as 'Éclair' and access to this requires a simple training process. Phone Ext 80303 to arrange training.

4.2 New Zealand Blood Service

- Transfusion Medicine services are provided 24 hours daily by the New Zealand Blood Service.

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4.2.1 Contact Telephone Numbers

Cross matching	80310
Tissue Typing Laboratory	89874

4.2.2 Specimen Labelling

Hand labelled specimens are the only ones acceptable to Transfusion Medicine.

4.3 Canterbury Health Laboratories

4.3.1 Service Provided

- Microbiology services are provided by specialist technical staff from 0800 hours until midnight daily, including weekends and public holidays.

- The Core Laboratory functions 24 hours daily providing routine chemistry, haematology and haemostasis testing. After midnight, urgent microbiology is also available through the Core Laboratory.

- Biochemistry, Immunology, Haematology Special Tests, Surface Marker Laboratory, Cytogenetics, Cytology and Histopathology all provide a routine diagnostic service between 0800 and 1700 hours, Monday to Friday.

- Most laboratories provide an on-call service for urgent specialised tests not performed in the Core Laboratory.

4.3.2 Contact Telephone Numbers

Reception	80300
Microbiology	80350
Core Biochemistry	80376
Anatomical Pathology	80580
Core Haematology	80373

4.3.3 Specimen Labelling

The standard label (100x30 mm) must be used on request forms and the small label (50x30 mm) only on specimen tubes, except blood transfusion specimens.

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4.4 Radiology

Services provided and working hours at each hospital

- **Christchurch Hospital** - a fully specialized radiology service 0800-1700 with a 24 hour presence of medical radiation technologists (radiographers) and a registrar in the department for acute work.
  Consultants and radiologists are on call at all times.
- **Burwood Hospital** - general radiology and very limited ultrasound only 0800-1700 Monday to Friday. Radiographer on call at all times. Patients requiring more specialized examinations will need to be sent to Christchurch Hospital. Radiologist visits Monday, Wednesday, and Friday afternoons.
- **Christchurch Women's Hospital** - General radiology and ultrasound 0800-1600 Monday to Friday. Radiographer and radiologist on call at other times. Patients requiring more specialized examinations will need to be sent to Christchurch Hospital. 24 hour cover to NICU by radiographer and radiologist.

Consultation Forms

It is essential that all consultation forms are filled in adequately. Patient details must include the patient's full name, date of birth, and the patient number. A patient identification sticker is sufficient for this. It is of considerable help to know whether the patient has had relevant previous examinations, and if possible, where and in what year. Adequate clinical information is mandatory to make sure that the most appropriate examination is performed. Consultation forms must be signed by a doctor and a bleep number provided wherever possible.

Radiology consultations for special examinations

Where possible, it is advisable to discuss special procedures with a Radiology registrar or consultant so that the most appropriate examination is performed. This applies particularly to CT, MRI, arteriography and interventional studies. Special procedures after hours must be approved by the clinical consultant and arranged with the radiology registrar on call or the consultant radiologist.

Radiology After Hours

Radiology is expensive after hours if a radiographer has to be called in. If a radiologic procedure is not critical to the patient's management it should be delayed until normal working hours so that unnecessary expenditure is avoided.

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4.5 Nuclear Medicine

Refer to "Nuclear Medicine Procedures" for explanations and information for Nursing and Medical staff, and indications and interpretations of scans. This booklet is widely available. 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 the 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 on Sunday morning and public holidays. Contact Dr J. Turner or Dr B. Brownlie before 1000.

5. Common Emergency Presentations

5.1 Emergency Department

Main Office Ground Floor, Parkside ☎ 80270 Fax 80286

Staff House staff, registrars in fellowship training, casual medical staff and a number of senior medical staff including:

- Dr A. Pitchford (Director),
- Dr M. Ardagh, (Director of Training)
- Doctors P. Helliwell, R Mitchell, R Ojala, P Gee, M Wallace, M Roberts, S Peddie, D Graber and S Pearson.

Secretary ☎ 89614
Triage Nurse ☎ 80274

Patient Handling

- Patients requiring resuscitation or stabilization in the Emergency Department will be managed in a 'shared', co-operative manner by staff of both the Emergency Department and the relevant inpatient team.
- Patients for whom admission is warranted, in the opinion of the Emergency Department staff, will be admitted, alternatively they can be assessed in the Emergency Department by the inpatient team and discharged at their discretion.
- Inpatient teams who are expecting a patient referred by a GP or Outpatient Clinic, should advise the Triage Nurse.
- The Emergency Department's main tasks are to ensure patient safety, patient comfort and appropriate patient disposition. Tasks beyond these will not routinely be undertaken in the Emergency Department

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5.2 Common Emergency Presentations

Introduction

This section is intended to supplement the systems based sections with a brief account of the initial approach to the unwell patient.

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:

1. Initial assessment and resuscitation i.e. the ABCs and specific resuscitation measures. This should take priority and should not await a final diagnosis, although it may be guided by the differential problem list.
2. Complete assessment.
3. Definitive management.

As this approach is followed, a differential diagnosis is developed. 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.

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.

5.3 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 and removal of foreign bodies.
  - 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).

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B Breathing Impairment

- Recognition:
  - Altered level of consciousness (cause and effect).
  - Hypoxia:
    - Pulse oximetry / arterial blood gases
    - Cyanosis.
  - Hypercapnia - arterial blood gases.
  - Tachypnoea or bradypnea.
  - Laboured breathing.

- Management options:
  - Supplemental oxygen (high flow with a mask and reservoir bag, 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 problem.

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, decreased pulse pressure.
  - Hypotension (a late sign).
- 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).

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5.4 Altered level of Consciousness

(See also Stupor and Coma in Neurology Section)

1 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 IV.
  - **Thiamine** 100 mg IV, if there is the possibility of Wernicke's encephalopathy.
  - **Naloxone** 0.2-0.4 mg IV and repeat in 5 minutes if necessary. If no response and narcotic overdose suspected give Naloxone up to a maximum of 4.0 mg. (2.0 mg may be required to reverse Methadone overdose.) Flumazenil is available for benzodiazepine reversal but is rarely indicated in the emergency setting.

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2 The complete assessment:

- History - patient, relative, ambulance, GP etc.
- Examination:
  - The Glasgow Coma Scale *(see page 19)*.
  - Temperature.
  - Neurological and other system examination.
- Investigations
  - Arterial blood gases, glucose, Na, K, creatinine, Bili, alk. phos., GGT, AST, CBC + diff, ECG, cardiac enzymes.
  - Other tests according to suspected cause e.g. CT scan of head, urine or gastric contents for toxicology, carboxyhaemoglobin, etc.
  - Glasgow Coma Scale *(see page 19)*

Table 1: Glasgow Coma Scale

Eye Opening	Spontaneously	4
	To voice	3
	To pain	2
	None	1
Verbal Response	Orientated	5
	Confused	4
	Inappropriate words	3
	Inappropiate sounds	2
	None	1
Motor response	Obeys commands	6
	Localises pain/purposeful movement	5
	Withdraws from pain	4
	Abnormal flexion	3
	Abnormal extension	2
	None	1
Score	**Total Possible**	**15**

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3 Definitive Management:
According to the cause *(see page 171 under Neurology)*

In the early management of the unconscious patient, consider the possible causes according to a "surgical sieve" and proceed accordingly.

- Trauma
  - Consider CT scan.
- Toxic
  - History, examine for signs of toxicity including the assessment of blood pH. Consider a toxic screen of urine or gastric contents (as above).
- Metabolic
  - Treatment for low blood glucose, hyponatraemia, etc. as defined by initial blood tests.
- Infective
  - Consider meningitis or encephalitis, a CT should precede a lumbar puncture in the unconscious patient, but if you suspect meningitis, give antibiotics early.

- Vascular
  - Intracranial bleed or brain stem infarct, are possible causes. If history and examination are suggestive, CT scan should be performed.
- Structural
  - Mass legion, e.g. bleed into tumour or a subdural haematoma. Do a CT scan if suspected.

Note: Supportive care with attention to A, B, and C are mandatory while the diagnostic possibilities are being considered.

5.5 Syncope

Definition – a transient loss of consciousness in a patient who has regained consciousness

1 Initial assessment and resuscitation:
- Airway
- Breathing
- Circulation

} including monitoring of these in case of recurrence

2 The complete assessment:
- History
- Examination
- Investigations

} *as described on Page 172 in 'Neurology'*

3 Definitive management according to the cause:
- Seizure disorder.
- Cardiovascular causes (arrhythmias, ischaemia, obstructive lesions, hypersensitive carotid sinus syndrome).
- Cerebrovascular causes (subclavian steal, TIAs).
- Peripheral vascular causes (vasovagal, orthostatic, hypovolaemic, others).
- Miscellaneous causes (hyperventilation, hypoglycaemia, psychogenic, pulmonary embolus, others).

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5.6 Shock

Simple definition – inadequate delivery and utilization of oxygen by vital organs due to a problem with the circulation.

1 Initial assessment and resuscitation:

- Airway, Breathing, Circulation } oxygen delivery must be optimized
  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).
- **Distributive Shock**
  (e.g. sepsis) – initial resuscitation includes IV fluids and specific therapy.
- **Cardiogenic Shock**
  (arrhythmias or myocardial insufficiency) will rely on specific therapy, i.e. anti-arrhythmic agents, DC shock, inotropic agents etc.
- **Obstructive Shock**
  (tension pneumothorax, massive pulmonary thromboembolism, cardiac tamponade) will rely on specific therapy, but often after a trial of fluid bolus.
- **Hypovolaemic Shock**:
  - 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. The actual CVP level is less useful than changes in CVP in response to fluid challenges. A low CVP (with a patent well placed catheter) means low volume. A high CVP may mean volume overload, pulmonary hypertension, COPD, right ventricular failure or increased pulmonary vascular resistance (as can occur in pulmonary embolism or any very unwell patients). Other more unusual causes of high CVP include tricuspid stenosis, tricuspid regurgitation, constrictive pericarditis, pericardial effusion or SVC obstruction.

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- 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

Table 2: Shock

Class I Shock	Blood loss up to 15% blood volume (750 mL)	
	CNS	no discernible abnormality
	Skin	
	Urine	
	Pulse	
	BP	
Class II Shock	Blood loss up to 15 – 30% blood volume (750 – 1500mL)	
	CNS	anxious to agitated
	Skin	cool, pale
	Urine	decreased
	Pulse	tachycardia (>100 per minute)
	BP	maintained (reduced pulse pressure)
Class III Shock	Blood loss up to 30 – 40% blood volume (1500 – 2000mL)	
	CNS	agitated to confused
	Skin	cool, pale
	Urine	decreased
	Pulse	tachycardia (>120 per minute)
	BP	falling
Class IV Shock	Blood loss in excess of 40 % of blood volume (>2000mL)	
	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%)

OBSOLETE

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 moving out of the vascular space and therefore more will be required (another 2000 mL) unless the response to the first 2000 mL was dramatic. Use of colloids either alone or in conjunction with crystalloids, is a matter of choice. For Class III or Class IV shock transfusion of blood will invariably be required. If the early crystalloid (maximum of two boluses of 2000 mL) 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 β-blockers are less able to compensate and therefore will become hypotensive earlier.
- There is a greater blood volume in advanced pregnancy and an ability to shunt blood from the placental circulation (at the fetus' expense) and therefore 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 – urgent surgical consult.

OBSOLETE

2 The complete assessment:
   - History
   - Examination
   - Investigations

   } according to the likely cause

3 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 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 necessary.

- 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 this does not represent fluid overload.
  - Initial fluid therapy is 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.

OBSOLETE

5.7 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. Generally involves three systems with occasionally a fourth.

SKIN	Urticaria or generalised acute pruritis
RESPIRATORY	Airway spasm/oedema/feeling of pharyngeal constriction.
CARDIOVASCULAR	Shock
GASTROINTESTINAL	Cramps and diarrhoea

♦ Initial assessment and resuscitation:

• Airway • Breathing	} give high flow oxygen
Circulation	large volumes of fluid may be required. Prompt IV access. Fluids should be started early but don't delay adrenaline.

- **ADRENALINE: The pharmacological mainstay of treatment.**
  - **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 and cardiovascular monitoring is available. Begin with **0.5-1.0 ml of 1:10,000** and increase dose incrementally as required. Very rarely up to **1 mg (10 ml of 1:10,000)** may be required every five minutes.
  - 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 } try to identify any allergens
- Examination }

♦ Definitive management:
- Moderate/severe reaction:
  - Hydrocortisone 200 mg IV
  - Promethazine 25-50 mg, diluted in 10 mls of 0.9S and given as slow IV injection.
  - Admit for observation.
  - Upon discharge give instruction in the use of parenteral adrenaline. e.g. IM adrenaline – Ana-Kit, Epipen.
  - Prednisone 40 mg PO daily for 2 days.
  - Promethazine 25 mg BD PO 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.

Note:
- H_2-antagonist (e.g. ranitidine) may be useful in addition to H_1-antagonist, in resistant cases.
- Patients on beta-blockers respond poorly to adrenaline and may require additional adrenaline, glucagon in large amounts or atropine.
- Patients on tricyclic antidepressants are sensitive to adrenaline, therefore use much smaller initial doses (15% of above doses).

OBSOLETE

5.8 Head Pain

1 Initial assessment and resuscitation:

- Airways
- Breathing
- Circulation

} brief appraisal

2 Complete assessment:

- History
- Examination
- Investigations

} directed according to the differential problem list

3 Definitive management according to the cause.
Possible causes include:

- Trauma - scalp, skull, intracranial haematoma.
- Chronic - 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:

Chronic or new headaches with focal neurological signs or papilloedema need investigations. CT scan should precede lumbar puncture if focal signs, papilloedema, or an impaired level of consciousness is present.

4 Important to exclude - subarachnoid haemorrhage, space occupying lesions, meningitis.

OBSOLETE

5.9 Chest Pain

1 Initial assessment and resuscitation:

- Airways } including oxygen, cardiac monitoring and intravenous access in all but trivial cases.
- Breathing }
- Circulation }

2 Complete assessment:

- History } directed according to the differential problem list
- Examination }
- Investigations }

Investigations usually include CXR and ECG but may require pulse oximetry/arterial blood gases and cardiac enzymes/markers of myocardial damage. Myoglobin or rapid TNT are the most useful screening tests for an acute myocardial infarction. Remember, cardiac markers are slow to rise after myocardial damage (Myoglobin is quickest, but least specific) and therefore normal cardiac markers soon after the onset of pain will not exclude myocardial damage. If in doubt, keep the patient under observation and do Troponin T or I and Creatinine Kinase (CK) at 12 and 24 hours from the onset of symptoms. Other investigations that may be required include a ventilation/perfusion scan, abdominal ultrasound, aortography, CT scan, echocardiography.

OBSOLETE

3 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.

4 Important to exclude - ischaemic heart disease, pulmonary embolism, pneumothorax, pneumonia.

5.10 Abdominal Pain

1 Initial assessment and resuscitation:

- Airways } if sepsis or hypovolaemia evident, oxygen
- Breathing } delivery should be optimised and intravenous
- Circulation } fluids given.

2 Complete assessment:

- History
  - Site
    - Abdominal
    - Pelvic
    - Retroperitoneal (Flank/back)
  - 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 groins and thighs - genitourinary or major vessels.
      - to shoulder - diaphragmatic irritation.
  - Associated Symptoms
    - Vomiting, diarrhoea, genitourinary, possible pregnancy.
    - Always consider extra abdominal causes of the pain, e.g. MI, DKA, pneumonia etc
- 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, including aortic aneurysm
    - Examination of hernial orifices, genitalia.
    - Auscultation: bowel sounds, bruit.
    - Rectal examination.

OBSOLETE

- Investigations
  - Dipstick 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.

3 Definitive management according to the cause. Possible causes:

Gastrointestinal, hepatobiliary, pancreatic, urological, gynaecological - complications of pregnancy (e.g. ectopic), musculoskeletal, respiratory, vascular, metabolic.

4 Important to exclude - abdominal aortic aneurysm [may present like renal colic], ischaemic bowel [non tender abdomen initially], ectopic pregnancy, torsion of testicle.

OBSOLETE

5.11 Shortness of Breath

1 Initial assessment and resuscitation:

- Airways, Breathing, Circulation — will require at least supplemental oxygen
- Supplemental oxygen therapy:
  - Titrate according to PaO_2 or pulse oximeter.
  - To maximize oxygen delivery use high flow O_2, with a reservoir bag
  - Use regulated FIO_2 (24-28%) via ventimask if COPD with CO_2 retention. Aim for O_2 saturation of 90% in long standing COPD
  - Need to check ABG in COPD patients to assess ventilation.
  - *See Respiratory Section (Page 227) on use of pulse oximetry.*

2 Complete assessment:

- History
  - The patient with chronic or recurrent shortness of breath can often 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 by observation
  - Respiratory rate, pulse rate, peak expiratory flow rate or preferably FEV_1, ability to speak, and use of accessory muscles are useful objective signs.
  - Auscultation and percussion of the chest may be helpful in identifying pneumonia, LVF or pneumothorax.
- Investigations
  - Pulse oximetry is a useful 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, (is difficult to exclude clinically), pneumonia and cardiac failure.
  - Other investigations as indicated.

OBSOLETE

3 Definitive management according to the cause. Possible causes:

- Lung disease.
- Heart disease.
- Airway disease.
- Chest wall problem.
- Neurological disease (abnormal patterns of breathing).
- Other disease (as above).

5.12 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.

1 Initial assessment and resuscitation:

- Airways, Breathing, Circulation: warmed, humidified oxygen
  - Warmed IV fluid may be required but be cautious as fluid overload can occur.
  - Defibrillation and antiarrhythmic drugs are less effective at low body 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").
- Simple rewarming 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).
    The "Bear Hugger" warm air blanket is available in ICU and in ED.
  - Warmed humidified oxygen.
  - Warmed IV fluids - contribute little to rewarming but will help prevent further cooling by cold IV fluids. Limit the IV fluid volume unless hypovolaemic.
- Other methods - more aggressive methods are generally not used
  - Avoid 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 (cardiopulmonary bypass is the ideal in this circumstance. Warmed gastric lavage is the most practical), 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.

OBSOLETE

2 Complete assessment:

- History - three general types
  - The healthy person with exposure to extreme cold e.g. immersion.
  - The 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
- Investigations } complications of cold.

3 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.

OBSOLETE

5.13 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, and complications like rhabdomyolysis, renal failure, and coagulopathy are common.

1 Initial assessment and resuscitation:

- Airways }
  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).
  - Alternatively, thoroughly douse in iced water if available (ice slurry in towels). Spray with water and fan to cause evaporation. If done well this may be the most effective means of cooling.
  - 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.

2 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.

3 Definitive management - usually cooling/monitoring continues in ICU including management of the many potential complications.

OBSOLETE

6. Medical Procedures

This section describes the current policy and practice in Internal Medicine Services at Canterbury Health Limited.

6.1 Intravenous Cannula Insertion and Care

Any procedure that 'breaks' the protective skin surface has the potential to introduce infection. Observation of the following procedure is essential.

- Insertion
  - Explain the procedure and why it is being done to the patient. Verbal consent should be obtained.
  - Wash hands. Soap and water is adequate. Gloves are recommended in all cases 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. We recommend 1% Chlorhexidine and 70% Isopropyl alcohol. Alcohol alone is not adequate. Hirsute arms might need shaving. The antiseptic must remain in contact with the skin for at least 60 seconds before inserting cannula.
  - 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 clean and 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.
    - 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 and time must be written in the clinical notes **and** on the dressing. Convenient GREEN LABELS are available for this and should be used.

IV Cannula
Insertion date / /
Time

OBSOLETE

- **Failure to insert an IV line**
  - It is important to recognise that on occasions you will find it difficult or impossible to insert an IV line. Under these circumstances make a **maximum of 2-3 attempts** and then seek help and advice from a more senior / experienced member of your medical team.
- **Care of Catheter**
  - Examine daily. Replace routinely every 48-72 hours, if not required earlier. Never leave longer than 72 hours. Clinical examination detects only some infected catheters. Septic thrombophlebitis causes continuing bacteraemia after removal of the catheter, and may 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 giving set.
  - Swab the cannula exit site as above and leave for 60 seconds.
  - Remove the catheter and cut off subcutaneous portion 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.

OBSOLETE

6.2 Intravenous Line Sepsis

Infusion therapy and intravascular devices carry a substantial and often unappreciated risk for producing iatrogenic harm. Risks include severe metastatic infections such as endocarditis, prosthesis infections, septic arthritis, and endophthalmitis. These complications can be prevented by good insertion technique and subsequent care.

6.2.1 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).
- Failure to seek help when you have had 2 or at the most 3 attempts to insert an IV line.

6.3 Insertion of Central Venous Lines (CVL)

These require special expertise in their insertion and ongoing care. In general, we recommend that CVLs are placed in ICU, Anaesthesia or Radiology. Seek advice from your consultant.

Remember that peripherally placed long lines may obviate the need for a CVL and are also of value for patients facing long term (more than 10 days) IV therapy. Peripherally Inserted Central Catheters (PICC) and midline catheters are placed during normal working hours by Radiology staff. It is essential to call the PICC Insertion Team to discuss which catheter is required and the time of insertion (Ext 80770).

6.4 Chest Aspiration

Diagnostic pleurocentesis may be undertaken by medical staff with appropriate experience. We recommend that you see one performed and then do one yourself under supervision, before attempting a chest aspiration on your own. A lateral decubitus CXR or ultrasound will allow identification of free fluid. Pleurocentesis may be performed safely if 10mm width free fluid is identified on a lateral decubitus CXR or if loculated fluid is identified and can be reached with ultrasound guidance. Use a 20 mL syringe with a 22G needle, using sterile technique. Local anaesthetic may reduce discomfort. It is essential to have the assistance of a nurse when performing a chest aspiration.

OBSOLETE

Diagnostic Pleurocentesis - Method

- Explain the procedure to the patient. The only common complication is pneumothorax which occurs in approximately 5%. Obtain verbal consent.
- Obtain the most recent x-ray.
- Position the patient in an upright position, with arms and head resting forward on a pillow, exposing the posterior chest.
- Using percussion and vocal resonance, locate the upper limit of the effusion, and the area of maximal dullness overlying the known location of the effusion. If ultrasound (U/S) has been performed, the area of maximal fluid should have been marked with an indelible pen. Always position the patient in the same way as for the U/S.
- Using aseptic technique:
  - Decide whether to give local anaesthetic, then using a 20mL syringe with a 22G, 38mm needle, enter the pleural space by progressively advancing then aspirating. This needle is usually of sufficient length to reach the pleural space.

- Aspirate 20mL of pleural fluid.
- Remove the syringe and needle then cover the puncture site with simple adhesive dressing.
- Put three equal specimens into sterile pottles. Leave 2 mL in the syringe and cap it with a bung from a blood gas kit. The syringe will need to be sent directly to the laboratory for assessment of pH. The specimens will need to be processed immediately.
  See Pleural Effusion (Page 244 in the Respiratory Section) for advice on which tests to do on the pleural fluid obtained.

Note: - when to use Ultrasound

- If you are unable to obtain fluid with a 22G 38mm needle.
  It is unwise to use a longer or larger gauge needle without further imaging.
- If the effusion is difficult to locate through clinical examination. This is especially important if the effusion appears loculated.

OBSOLETE

Therapeutic Pleurocentesis - Method

Explain the procedure to the patient. The most common complication is pneumothorax. Obtain verbal consent.

- Follow the same initial steps as described in "Diagnostic Pleurocentesis" as above. A local anaesthetic should be used.
- Insert a 14 – 16 G 50mm intravenous cannula into the pleural space. Following partial removal of the needle (to prevent lung puncture), the catheter should be advanced and secured. **The catheter should be held at all times during the procedure.**
- The needle should be removed, and the catheter attached to a giving set. The distal end of the giving set is attached to a catheter bag, which is placed on the floor. The giving set clamp should then be released and the fluid allowed to flow freely into the bag. Sometimes fluid does not immediately flow, in which case a 50mL syringe with 20G needle should be put into the rubber giving port in the giving set, and 50mL aspirated. This will allow flow to start, in a siphoning fashion.
- Aspiration should be continued until either:
  - 1000 – 2000mL has been removed, depending on the patient's size. Removal of greater than this quantity in one sitting risks re-expansion pulmonary oedema.
  - The patient feels new chest discomfort or persistent coughing, indicating mediastinal shift.

- Repeat chest x-ray to check for pneumothorax.

Note: It is essential in both diagnostic and therapeutic pleurocentesis that the time and date of the procedure, the volume of fluid removed and any difficulties experienced are written in the clinical notes.

6.5 Insertion of Intercostal Tubes

The insertion and management of intercostal tubes is a complex and specialised area. Patients requiring chest tube management should normally be cared for by specialist respiratory or thoracic surgical teams.

Unless it is an emergency, intercostal tubes are best inserted or supervised by experienced staff from the respiratory or thoracic surgical unit. In selected cases tubes may best be inserted by a radiologist under ultrasound guidance.

OBSOLETE

6.6 Joint Aspiration

(See Rheumatology Section Page 250)

Explain what you propose to do to the patient and obtain verbal consent.

We recommend that joint aspiration be performed by a registrar and that house surgeons gain experience by watching the procedure.

If the joint is obviously swollen use a 22G needle and aspirate from the most swollen area. If you are unsure of your technique, seek advice from either Rheumatology or Orthopaedic Services. Record full details of the procedure carried out in the clinical notes.

6.7 Lumbar Puncture

(See also pages 131 and 163)

Make sure you have considered the key points given below before undertaking a lumbar puncture.

It is essential for you to also consult the lumbar puncture protocols in the department in which you are working. The recommendations given here **do not** cover the administration of drugs intrathecally.

The pre-packaged lumbar puncture sets on the wards may not contain a pencil point needle. However, 22G pencil-point lumbar puncture needles are available on the wards and should be used.

Explain the procedure, the indications and possible complications to the patient, and obtain verbal consent. The patient may wish to use the toilet prior to the procedure. Complications include headache, around 5% **if using a pencil point needle**, nerve root injury 2%, infection less than 1%. Severe persisting headache is a rare consequence of lumbar puncture and may indicate continued leakage of CSF. There is specific treatment (application of a blood patch) which is highly effective.

Key Points

- Always ask yourself at least two questions before the lumbar puncture is performed.
  Does CT/MRI need to be done first? In patients with clinical evidence of raised intracranial pressure (raised BP, decreased pulse, decreased level of consciousness), seizures, papilloedema, focal neurological signs, sinus or ear infection – obtain CT/MRI head scan urgently before doing lumbar puncture.
  Is the patient likely to bleed? Check platelets, INR and APTT and review history and examination from this perspective.
- An RMO should either follow the guidelines established by the department they are working in at the time, or if none are available follow the guidelines given here.
- We recommend the use of 22G pencil-point lumbar puncture needles.
- RMOs should observe 2-3 lumbar punctures then perform 2-3 under direct supervision before attempting to do a lumbar puncture on their own.
- After one or at the most two failures an RMO should seek help from a more senior RMO or their consultant.

OBSOLETE

Assist with positioning the patient on their side, head flexed, knees tucked under their chin to help widen intravertebral spaces and assist in locating the intrathecal space for tapping. Place flat pillow between knees to aid correct positioning. Decide before you start whether pressure measurement is required. The use of an atraumatic 22G (pencil-point) needle rather than a quinke 22G (cutting) needle reduces the incidence of headache from up to 25% to 5%. Aim for the L3-4 or L4-5 disc spaces. Strict aseptic technique must be used and chlorhexidine/alcohol should be used for skin sterilisation. Local anaesthetic (lignocaine 1%) is recommended. Insert the needle through the skin and continue advancing the needle until there is decreased resistance (having traversed ligamentum flavum) or the needle has been inserted to half its length; then remove the stylet. If no CSF is obtained replace the stylet and advance the needle about 1mm. Wait at least 30 seconds for CSF to appear in the hub. Rotating the needle through 90-180 degrees may allow CSF to flow. Advance 1-2mm at a time if no CSF has appeared. If no CSF is obtained when the bone is contacted or the needle is fully

inserted, or when you think it has been advanced far enough, withdraw the needle very slowly until CSF flows or the needle is almost removed. Then re-insert the stylet, re-check the patient's position and needle orientation and repeat the procedure.

Encourage oral fluids afterwards. Some practitioners prefer their patients to lie flat in bed for four hours afterwards, although there is no definite evidence that this is of benefit.

When CSF flows – collect samples of CSF into 3 plain sterile tubes and label 1, 2 and 3 in the order in which you fill them.

The following approximate volumes are required for:

Microbiology	Culture, Gram stain, cell count and antigens	1.0 mL
Biochemistry	Protein, glucose	0.5 mL
Virology	Culture and PCR for HSV	0.5 mL
Cytology	If abnormal cells suspected (request cytospin)	0.5 mL

Note:

- Microbiology takes precedence if only a limited amount of CSF is available.
- If oligoclonal band analysis is required, 5 mL of CSF is needed for this as well as a simultaneous venous blood sample – 5 mL clotted sample.

OBSOLETE

If CSF pressure is to be measured it can be done at this stage. The hips should be partly unflexed since any pressure on the abdomen may falsely elevate CSF pressure.

Analgesia should be given for headache. If severe headache occurs or the headache persists then there may be ongoing CSF leakage at the puncture site. Lying flat in bed, good hydration and in particular caffeine containing drinks such as coffee, tea, and coke are helpful in the relief of established headache. Lying flat in bed also helps to relieve the pain which may be aggravated by an upright position. Use of a blood patch should be considered and discussed with the on-call anaesthetist.

Note: It is essential to record the time and date of the procedure, the CSF pressure if taken, the volume of CSF removed and any difficulties experienced in the clinical notes.

7. Clinical Pharmacology

Main Office Ground Floor, Parkside ☎ 89670 Fax 80902

Staff
- Assoc. Prof. Evan Begg ☎ 81055
- Dr Murray Barclay ☎ 80074
- Carl Kirkpatrick ☎ 80901

Consultation and On-call Service

Consultation available on request; services included interpretation of drug concentrations.

Other Services

Drug Information Service ☎ 80900 Fax 80902

Staff Sharon Gardiner and Murray Whitteker

The Drug Information Service is available to answer patient related drug information enquiries. Therapeutic drug monitoring services are also offered, e.g. aminoglycosides.

Office hours - 0800-1700.

After hours - contact on-call Pharmacist.

Drug Utilization Review Service ☎ 89971 Fax 80902

Staff Jane Vella-Brincat

Main Office
- Preferred Medicines List
- Antibiotic Guidelines
- Assistance with guidelines for high use, high cost medicines

OBSOLETE

7.1 Preferred Medicines List (The Pink Book)

Christchurch Hospital's Preferred Medicines List (PML), Antibiotic Guidelines and Pharmacology Guidelines.

The PML is available to all hospital doctors, trainee interns, pharmacists and senior nursing staff. The content reflects local prescribing practice and includes common daily dose ranges, costs and advice about prescribing.

- Drugs should be chosen from the PML unless there is a compelling reason for an alternative choice.
- 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 that may be prescribed only with the approval of a consultant are noted ('Spec').

The Pink Book also contains:

- Antibiotic guidelines (including prophylaxis)
- Antibiotic sensitivities
- Pharmacology guidelines
- Drug profiles

The Pharmacology guidelines contain useful information about:

- Cytochrome p450 drug metabolism and interactions
- Adverse drug reaction assessment / reporting
- Gentamicin/tobramycin dosing guidelines
- Vancomycin dosing guidelines
- Drug use in renal impairment
- Lithium interactions
- QT interval prolongation

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7.2 Drug Information Service

The Drug Information Service (Extension 80900) is available to answer any drug-related questions that cannot be answered readily in 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 drug-related questions
- We can help particularly with: dosing in renal and liver disease, the elderly, pregnancy and lactation and drug interactions.

7.3 Prescribing Advice

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? (See Drug Profiles in Preferred Medicines List – PML)
- When should the effect of the drug be reviewed?
- How long should the patient remain on the drug at this dose?
- Are any drug interactions likely? (See Drug Profiles in Preferred Medicines List – PML)
- What major and minor side effects might occur and should the patient be informed about 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**?
- Is there a patient information leaflet on this drug?

7.4 Dose Alteration in Renal Impairment

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

This list includes:

Table 3: Fraction excreted unchanged in the urine of some medications.

Drug	Fu (fraction excreted unchanged in the urine)
ACE inhibitors	
- enalapril, cilazapril, quinapril	0.9 (metabolite)
- captopril	0.5
Aminoglycosides	0.9
Digoxin	0.8
Allopurinol	0.8 (metabolite)
Lithium	1.0
Metformin	0.9
Methotrexate	0.8
Vancomycin	0.9
(Other drugs – Look up Drug Profiles in PML or phone Clinical Pharmacology Ext: 80900)	

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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 (D_{normal})
- Calculate patient's creatinine clearance (CrCl). Normal is > 1.5 ml/sec

Table 4: Calculation of Creatinine Clearance

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

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

- Calculate dose-rate for this patient ($D_{patient}$)

Table 5: Calculation of Dose Rate

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

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- Decide whether you should decrease the dose itself or prolong the dose-interval (usually prolong the dose-interval). Aim for once or twice daily as this will maximise compliance.

7.5 Dose Alteration in Liver Impairment

- Serum albumin and the INR (reflecting prothrombin activity) best reflect liver metabolic capacity. An albumin concentration <30 g/L or an INR of >1.2 indicate severe liver dysfunction, **provided** there is no other explanation of these abnormal laboratory results.
- Decide on appropriate daily dose for a patient with normal liver function.
- In patients with severe liver dysfunction (defined above) reduce dose by 50% for drugs whose metabolism is affected by liver blood flow (flow-dependent drugs), and 25% for drugs metabolised but not affected by liver blood flow.
  Flow-dependent drugs include - *phenothiazines, tricyclic antidepressants, SSRIs, nitrates, many narcotics, many β-blockers, lignocaine, calcium antagonist drugs and statins.*
- Check with Drug Information Service (Ext. 80900) and the drug package insert if unsure.

7.6 Dose Alteration in the Elderly

- Adjust dosing for renally eliminated drugs (See PML).
- 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.

7.7 Therapeutic Drug Monitoring

A therapeutic drug monitoring service is provided at Christchurch Hospital by Clinical Pharmacology/Pharmacy. The team assesses concentration results outside the therapeutic range for *lithium, phenytoin, digoxin, carbamazepine, theophylline, phenobarbitone and valproate*, and visits wards with these results each afternoon. The Drug Information Service (Ext. 80900) can be contacted with any queries.

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

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Table 6: Therapeutic Drug Monitoring

Essential	Important	Sometimes useful	Useful in overdoses
aminoglycosides lithium	carbamazepine cyclosporin digoxin perhexiline phenobarbitone phenytoin theophylline vancomycin	amiodarone antidepressants clozapine ethosuximide 5-fluorocytosine fluoxetine itraconazole lamotrigine lignocaine methadone methotrexate paroxetine platinum compounds teicoplanin valproic acid	ethanol ethylene glycol methanol paracetamol salicylate

Table 7: Pharmacokinetic Data for some commonly used drugs.

Drug	Half-life	Time to steady state after dose change	Ideal time for blood sample and type of tube
Carbamazepine	Adults: 12-17 hrs Children: 8-14 hrs	Adults: 2-4 days Children: 2-3 days	Just before dose Green tube (or red)
Cyclosporin	6-24 hrs	5 days	Just before dose purple tube only
Digoxin	38 hrs (longer if ↓ renal function	7 days, longer if ↓ renal function unless loading dose given	Just before dose or >8 hrs after dose Red tube (or green)
Lithium	18-24 hrs	5 days	Just before dose or >12 hrs after dose Red tube only
Phenobarbitone	Adults: 53-140hrs Children: 37-72 hrs	Adults: up to 1 month Children: 15 days	Just before dose Green tube (or red)
Phenytoin	Dose dependent Usually 20-30 hrs	5 days	Just before dose Green tube (or red)
Theophylline	6-9 hrs	1-2 days	Oral - just before dose IV - after >12 hrs Green tube (or red)
Valproate	Adults: 8-17 hrs Children: 4-14 hrs	3-4 days	Just before dose Green tube (or red)
Vancomycin	Adults: 5-11 hrs Children: 2-3 hrs	Adults: 2 days Children: 1 day	Just before dose (usually 3rd or 4th dose) Red tube (or green)
Gentamicin/ Tobramycin	Refer to PML, page 125, for guidelines 2 samples required Write exact times on form		Red tube (or green)

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7.8 Advice for Specific Drugs

7.8.1 Aminoglycosides

- Gentamicin is the aminoglycoside of choice except in special circumstances. i.e. tobramycin for pseudomonas infection. Consider amikacin for organisms resistant to gentamicin and tobramycin.
- All patients should have once daily dosing unless they are being treated for endocarditis. Once daily dosing has better efficacy, less nephrotoxicity and greater cost efficiency than multiple dosing.
- All dose predictions for aminoglycosides (both once daily and multiple

daily) should be performed by the Drug Information Service (Ext: 80900 or beep 264), or by the pharmacist on call after hours.

7.8.2 Once daily protocol for gentamicin and tobramycin

- Calculate patient's creatinine clearance *(see page 44)*.
- Calculate the first dose from the following table.

Table 8: Calculation Gentamicin & Tobramycin

Calc. CrCl (mL/sec)	Dose in mg/kg	Time of second blood sample (hours)
>1.1	5-7¦ depending on the	6-14
0.91-1.1	5-6¦ severity of infection	8-16
0.71-0.9	5	10-18
0.51-0.7	4	12-20
0.35-0.5	3	14-22
<0.35	consider using another antibiotic	

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- Give the calculated dose once daily by IV infusion over 30 minutes in 100 mL 0.9S. **Record exact times of starting and finishing the infusion.**
- Take two blood samples for drug concentrations:
  - At approximately 30 minutes after the end of the infusion.
  - At between 6 and 22 hours after the infusion depending on renal function as in Table 6.

  Record exact times the blood samples were taken.
- Contact the ward pharmacist or Clinical Pharmacology (Ext: 80900) during normal working hours, or the pharmacist on call after hours, with the drug concentration results to get a dose prediction for the next dose.
- Repeat the 30 minute and 6-22 hour samples every 3 days or prn depending on the clinical situation.
- Monitor renal function, hearing and vestibular function as you would normally for patients on these drugs.
- If calculated CrCl is deteriorating dose alteration will be required. Refer to Clinical Pharmacology (80900).

7.8.3 Vancomycin

- Calculate the patients creatinine clearance *(see page 44)*
- The first dose of vancomycin should be 15mg/kg **lean body weight** (approx. 1g) - irrespective of renal function.
- Give vancomycin by an IV infusion over at least 1 hour (a longer infusion

time may be necessary to avoid 'Red Man Syndrome' which occurs when given too rapidly. It is caused by histamine release leading to hypotension and a red appearance.)

Record exact times of starting and finishing the infusion.

- For patients with CrCl > 0.5 ml/sec take one trough sample at the end of the second maintenance dose.

  Record exact time of blood samples.

- If calculated CrCl < 0.5 ml/sec then take two blood samples for vancomycin concentration following the loading dose:

  1. at approximately 30 minutes after the end of the infusion (peak).
  2. just before the next dose is given (trough).

  Record exact time of blood samples.

- **Contact** the **Ward Pharmacist** or **Clinical Pharmacology** (Ext 80900) during normal working hours, or the **pharmacist on call** after hours, for a dose prediction. **Trough concentrations should usually be between 5 and 10mg/L.**

When vancomycin concentrations are **not available** prior to the next dose, the next dose should be given as follows based on lean body weight.

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Renal Function	Dose (Lean body weight)	Interval	Total daily dose
>1.5mL/s	7.5mg/kg	6 hours	30mg/kg/day
1.21 – 1.5mL/s	7.5mg/kg	8 hours	22.5mg/kg/day
0.81 – 1.2 mL/s	7.5mg/kg	12 hours	15mg/kg/day
0.41 – 0.8mL/s	7.5mg/kg	18 hours	10mg/kg/day
0.21 – 0.4mL/s	7.5mg/kg	24 hours	7.5mg/kg/day
≤ 0.2mL/s	Contact Clinical Pharmacology		

Take further blood samples every 3 days if required depending on the clinical situation.

Monitor renal function, hearing and vestibular function as you would normally for patients on vancomycin.

If calculated CrCl is deteriorating dose alteration will be required. Refer to Clinical Pharmacology (Ext 80900).

7.8.4 Lithium

- Therapeutic range (0.4-0.8 mmol/L).
- Adverse effects correlate with serum concentrations:

<1.5 mmol/L	nausea, diarrhoea, polyuria/polydipsia, tremor.
1.5-2.0 mmol/L	increased GI and neurotoxic effects, e.g. slurred speech.
> 2.0 mmol/L	arrhythmias, convulsions, drowsiness.

- Interactions:

↑ [lithium]:	ACE inhibitors, NSAIDs, diuretics, volume depletion, low Na^+ diets, renal impairment, hypothyroidism can decrease lithium clearance.
↓ [lithium]:	Theophylline, additive neurotoxicity: SSRIs, methyldopa, benzodiazepines.

- Monitor at least 8 hr and usually at 12 hr after an oral dose, and at steady-state (4-7 days after dose alteration).
- Manage toxicity with saline infusions. Haemodialysis is useful in overdose (see Poisonings section).

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7.8.5 Sampling

- Aminoglycosides and lithium 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 is given.
- Times of dosing, duration of dosing, and times of sampling should be recorded accurately for adequate interpretation.
- Specimen required - one full 7 ml heparinised tube (green top), except for antibiotics and lithium 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.

8. Alcohol Related Problems

8.1 Alcohol Withdrawal

- During alcohol withdrawal the following sequence of events may be seen:
  - tremor
  - hallucinations
  - seizures
  - delirium tremens (autonomic hyperactivity, disorientation and hallucinations).
- Administer thiamine 100 mg IM **before** glucose given. 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.
- Attention to fluids, electrolytes, hypoxia, sepsis.
- Early withdrawal - diazepam 5 - 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.
- Alcoholic hallucinations - Haloperidol 5 mg IM then 1-3 mg BD maintenance. Oral therapy when appropriate. **Note**: Haloperidol may provoke fits or hypertension.
- Delirium tremens:
  - Diazepam 2.5-10 mg in 100 ml 0.9S by 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 recommend phenytoin or sodium valproate as alternatives. To treat seizures use diazepam and see section on epilepsy.

Note: Maintenance anticonvulsant treatment is often contraindicated in patients with alcohol withdrawal fits as non-compliance favours further seizures.

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8.2 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 Beep 811). This service is restricted to patients attending Christchurch Hospital or their family.
- **Alcohol misuse is a common preventable cause of health and social problems:**
  - **80% of New Zealander adults take alcohol, but 10% of these drink 60% of the alcohol.**
  - **10 - 20% of drinkers have problems with alcohol sometime in their lives.**
  - **Concomitant major illness can be an important stimulus to behaviour change.**
- The essence of recognition lies in thinking "could alcohol be contributing to this patient's problems?"
  Some pointers to harmful drinking -
  - gastrointestinal problems
  - symptoms of 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 hazardous drinking or even alcohol dependence.
  It is 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.

- **Hazardous drinking is suggested by the following:**

Males	Over 35 standard drinks per week.
Females	Over 21 standard drinks per week.
Male & Female	Over 6 standard drinks on any one occasion.

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- **Units**
  For routine use it is easier to express intake in units of alcohol, where one unit roughly equates to the standard New Zealand drink. A unit contains 7 - 11 gram of alcohol. In making calculations, due account needs to be taken with unusually high and low concentration drinks e.g. low alcohol beers, wine coolers and departures from standard volumes per drink.

Table 9: Standard NZ drink = one unit

	Beer	Table Wine	Sherry	Spirits
Volume	200 mL 7 oz	120 mL 4 oz	60 mL 2 oz	20 mL hotel nip
Alcohol Content	7g	11g	9g	7g

- **Diagnostic Criteria for Alcohol Dependence**
  (adapted from Diagnostic and Statistical Manual of Mental Disorders. 4th Edition1994)
  - Alcohol is often taken in larger amounts or over a longer period than intended.
  - Persistant desire or unsuccessful attempts to cut down or control alcohol use.
  - A great deal of time spent in alcohol-related activities.
  - Important social, occupational or recreational activities given up or reduced because of alcohol use.
  - Acquired tolerance.
  - Withdrawal symptoms or relief use.
- **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?
  - 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?
  - How often during the last year have you failed to do what was normally expected from you because of your drinking?

  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

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provide them with some frank 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 next step is to offer support and/or intervention.

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

- **Facilities available to help with alcohol related problem**

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City Mission

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

Community & Alcohol Drug Service

Ph: 365-0839 258 Armagh Street
(Assessment, Outpatient)

Odyssey House

Ph: 358-7791 98 Greers Road
(Residential - 40 beds for chronic alcohol / other drug users. Long term therapeutic community.)

Kennedy Villa

Ph: 339-1139 Sunnyside Hospital
(Detoxification)

Salvation Army—Bridge Programme

Ph: 338-4436 35 Collins Street
(Inpatient Programme and outpatient services, Half way house accommodation)

Nova Lodge

Ph: 349-2053 Kirk Road, Templeton
(61 bed residential/long term for chronic alcoholics)

Te Rito Arahi
Ph: 379-5709 194 Madras Street
(Maori Alcohol & Drug Resource)

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

Womens Recovery and Residential Service
Ph: 365-6601 276 Hereford St
(also open to client's children, also provides a day programme for outpatients)

Thorpe House
Ph 379-1682 275 Hereford Street
Non-medical detox

Vincentian Centre
Ph 379 9338 222 Wilson's Road
(inpatient programme for Alcohol dependent males)

Alcoholics Anonymous
Ph 379 0860

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An excellent resource text for alcohol problems is:

"Alcohol and Drug Problems"
by John O'Hagan, Geoffrey Robinson and Edwin Whiteside (1993)

A limited number of free copies are available from the
Alcohol Advisory Council of New Zealand
PO Box 2688
Christchurch.

9. Cardiology

Main Office 2nd Floor, Parkside ☎ 81138 Fax 81120

Inpatient Services Five inpatient teams on Wards 12, 26, & CCU

- Bridgman
- Crozier
- Ikram
- Richards/Elliott
- Smyth

Consultation and On-call Service

24 hours a day, seven days a week. Contact Cardiology registrar or consultant on-call through the Operator on 364 0640. For consults, fax the referral to 364 1120

Outpatient Consultations

Secretary	☎ 81110
Nurse	☎ 81190
On-call Registrar/Consultant	☎ 364 0640

Consultation Guidelines

Chest pain, dyspnoea on exertion, documented angina, myocardial infarction, right and left heart failure, hypertension, valvular heart disease, palpitations and arrhythmias, syncope, congenital heart disease, unexplained cardiomegaly, hyperlipidaemia.

Other Services

Cardiology Day Ward (Cardiac catheters, stress echo, trans oesophageal echo, electrophysiology studies, radiofrequency ablation for tachyarrhythmia, coronary angioplasty, pacemaker/cardioverter/defibrillater implantation)	☎ 81070	Fax 81127
X-ray Control Room	☎ 81075	
Echocardiography Laboratory (consultations)		Fax 81449
Tilt Table Testing		Fax 81120
ECG Department (12 lead ECG, exercise tests, Holter monitor tests)		Fax 80681
Coronary Care Unit	☎ 81099	Fax 81128
Education & Rehabilitation Service	☎ 80640	Beep 262
Cardioendocrine Research Group (clinical research, basic research)	☎ 81116	
Cardiology Research Unit (clinical research studies)	☎ 88095	

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9.1 Heart Failure

9.1.1 Definition

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

9.1.2 Management requires each of the following

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

9.1.3 Aetiology

Primary disease processes

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

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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:
  - β blockers } negative inotropes
  - Calcium antagonists }
  - Most antiarrhythmics }
  - Withdrawal of diuretics, ACE inhibitors, or digoxin, or poor compliance.
  - Fluid retention: steroids, NSAIDs, liquorice, De-Nol.
- Anaemia.
- Thyrotoxicosis - particularly in the elderly.
- Infections (especially endocarditis and pulmonary infections).
- Pulmonary embolism.
- Fluid overload - e.g. transfusion, renal failure.

9.1.4 Investigations

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

- CXR (pulmonary venous congestion/oedema, cardiac size, pulmonary infiltrates).
- 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.
- Echocardiography (urgent if tamponade or bacterial endocarditis suspected).
- Radionuclide scan is useful to document degree of LV dysfunction.
- Thyroid function tests.
- Plasma BNP (where there is doubt over cardiac vs non-cardiac cause of symptoms).

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9.1.5 Therapy

Correct any contributing factor such as arrhythmias, infection etc.

- Acute pulmonary congestion, pulmonary oedema:
  - Sit patient upright.
  - Oxygen at 4-6 L/min to maintain SaO_2 >90%.
  - Glyceryl trinitrate - 0.6 mg tablet sublingual or nitrate spray under tongue. Repeat doses of nitrate every 5 minutes while the blood pressure remains high.
  - Morphine 2.5-5.0 mg IV slowly over 3-5 minutes, count respiratory rate every 5 minutes. 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 treatment then nitrate infusion *(see page 63)*; continuous positive airway pressure by face mask (CPAP); and haemodynamic monitoring in ICU or CCU should all be considered.

- CPAP is useful if hypoxia persists after initial treatment and may avert the need for intubation and mechanical ventilation. It is best started before the patient becomes severely fatigued. If prolonged therapy with high O_2 concentrations is required, consider other ventilatory supports.

- Compromised myocardial function: Low output states can be managed by increasing myocardial contractility (inotropic support) or reducing the cardiac workload (pre load and 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 heart failure with sinus rhythm. The trials indicating benefit from digoxin are not confined to "refactory" heart failure. Initial dose (if not already on maintenance treatment) of 0.5 mg (IV or oral) then 0.25-0.5 mg at 4 and 8 hours to complete a loading dose of 1.0-1.5 mg. Maintenance dose 0.25 mg per day usually given at night. In renal failure and the elderly reduce the dose.

Table 10: Digoxin

- Therapeutic range (1.0 - 2.5 nmol/L)
- Toxicity increases significantly at concentrations > 2.6 nmol/L
- Toxicity more likely in the presence of:
  - potassium <3.5 or > 5 mmol/L
  - renal impairment
  - age > 60 yrs
  - Hypercalcaemia, hypothyroidism or acidosis
- Take concentrations **at least 8 hours post dose**. Trough preferable.
- Maintenance dose adjustment is necessary in renal impairment according to the creatinine clearance (CrCl) using the Cockcroft and Gault formula. A normal serum creatinine may not indicate a normal CrCl.
- If digoxin toxic, stop drug for appropriate number of half-lives to achieve target concentration. $t_{1/2}$ in normal renal function = 36 hr. It is prolonged in impaired renal function.

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- Intravenous adrenergic agonists are useful as short term emergency treatment in patients with severe heart failure on the basis of diminished myocardial function with low output and /or refactory congestion. They require ECG monitoring for arrhythmias and can be done within the CCU,

Wards 12, 14, or ICU as necessary.

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. 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. Doses up to 10-15 mcg/kg/min can be used.

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.

***Caution:** At Christchurch Hospital, the infusion details given here are used in CCU, but differ from those recommended by ICU.

- **Pre load reduction**:
  - Nitrates, diuretics, morphine
- **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 inhibitors but benefit will usually outweigh risk in established heart failure.
  - 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 63 for IV glyceryl trinitrate instructions).*
  - ACE inhibitor dosing

    Although the substantive evidence base for use of ACE inhibitors in heart failure pertains to captopril and enalapril, these agents are no longer subsidised for the newly diagnosed patient with heart failure. Start with either quinapril (Accupril) or cilazipril (Inhibace). The dosage of quinapril is given in the table below. For the Cockroft and Gault creatinine clearance formula, *(see page 44).* The starting dose of cilazipril for a patient with normal renal function is 1.25 mg/day.

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Table 11: Quinapril Dosage

Creatinine Clearance	Dose	
(mL/sec)	Starting	Maximum
>1.5	5-10 mg	30 mg q24h
0.8-1.5	5 mg	20 mg q24h
0.4-0.8	2.5-5 mg	10 mg q24h
0.2-0.4	2.5 mg	5 mg q24h
<0.2	2.5 mg	2.5 mg q24h

Side effects of ACE inhibitors include:

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

Hyperkalaemia — The need for K supplements is usually reduced. Spironolactone (Aldactone) in low dosage (12.5-25 mg/day) has proven to be of benefit in heart failure refractory to ace inhibitors and loop diurectics (refer to the "RALES" trial). Careful monitoring of potassium is required. Similarly, amiloride or triamterene are sometimes necessary to counter refractory congestion or loop diuretic induced potassium depletion. Monitoring is required. Do not give multiple potassium sparing diuretics.

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- **Further Management**
  - Daily weigh. Fluid balance for the 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 if dyspnoea and/or clinical features fail to respond. Consider ECHO if cardiomegaly present (? Pericardial effusion).
  - Low molecular weight Heparin such as enoxaparine (Clexane) 20-40 mg SC q24h. Start on admission. Consider full heparinization then warfarin in those with marked left ventricular impairment, increased atrial size or chronic atrial fibrillation - consult Cardiologist.
  - Potassium supplements will be needed with most diuretics. Requirements may be reduced or unnecessary in renal failure, with 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 aggravating factors. This may include cardiac catheterization in selected cases.
  - β Blocker drugs do not have any role in the management of acute heart failure. However carefully titrated administration of β blockers is of proven benefit in stable chronic heart failure associated with systolic dysfunction.

9.2 Myocardial Infarction

9.2.1 Definition

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.

9.2.2 Causes

Acute Coronary occlusion due to:

- Coronary artery plaque rupture and thrombosis.
- Emboli (rare).
- Spasm (Prinzmetal's angina).

9.2.3 Clinical features

A history of severe crushing retrosternal chest pain radiating to neck and arms is typical. However, atypical presentations are very common. 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 in 2-4 hours.

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9.2.4 Investigations

- ECG daily for 3 days. If coronary artery 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: A Troponin (T or I) and Creatinine Kinase (CK) should be done on admission and at 12 and 24 hours after the onset of symptoms. For patients admitted more than 24 hours after the onset of pain, the above enzymes should be measured on three consecutive days. Worldwide, most laboratories are measuring TNI but the choice in your hospital may be dictated by the equipment available in the Pathology Laboratory.
- CXR can usually wait until normal working hours or prior to discharge. Indications for urgent X-ray:
  - Suspicion of aortic dissection (widened mediastinum " separation of calcified intima).
  - Moderate or severe cardiac failure.
- CBC + diff.
- Na, K, Ca, PO_4, creatinine, glucose.
- Total cholesterol, HDL cholesterol and triglycerides on admission and repeat at 3 months.

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).

9.2.5 Management

- **"Time is muscle"** - expedite treatment and assess suitability for thrombolysis urgently.
- **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 contact the CCU or ICU registrar on call.
- **IV Access** - IV insertion on admission. Flush 4-6 hourly with 0.9S.
- **Oxygen** - should be administered to all patients with MI or unstable angina for the first 12 hours unless there is a strong contra indication.
- **Pain relief** - continuing pain suggests ongoing ischaemia which should be treated with nitrates, β-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 or prochlorperazine (Stemetil) 12.5 mg given IV at the same time may reduce nausea and vomiting.
- **Aspirin** 300 mg sublingual stat then 150 mg orally daily if no contra-indications.
- **Thrombolytic Therapy** - streptokinase (SK) is the most widely used thrombolytic agent. Tissue plasminogen activator (tPA) is also available and should be considered if: SK has been used before; if there has been a definite recent streptococcal infection; if SK provokes unacceptable side effects; there has been a large anterior infarct. Consultation with a cardiologist is essential if there is any doubt as to which agent to use.
- **Emergency coronary angiogram and primary angioplasty** may be the treatment of choice if the patient is in cardiogenic shock; has a large anterior

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infarct; has absolute contraindications to thrombolysis; has recurrent pain and ST elevation soon after thrombolysis; has had coronary stent implantation within the last ten days.

Contact the Cardiology Registrar or Cardiologist immediately to discuss transfer to the Catheter Laboratory. Remember "time is muscle", "time is life".

- **The role of balloon angioplasty / stenting post infarction** - if in-hospital cardiac catheterization demonstrates a critical stenosis and/or slow flow in one coronary artery, then balloon angioplasty and/or stenting should be performed. This will effectively treat angina and should permit early discharge. If the angiogram demonstrates multi-vessel disease, then bypass surgery will be considered.
  If a patient on the waiting list for angioplasty presents with unstable angina, please contact their Cardiologist to expedite in hospital angioplasty. This will shorten hospital stay.
- **Nitrates** may be helpful for continuing pain (patch, isosorbide dinitrate or mononitrate tablets). IV infusion may be preferred to oral nitrates if the patient has unremitting angina, is haemodynamically unstable and to help reduce preload in pulmonary oedema. Remember that IV infusion for more than 24 hours may result in nitrate tolerance. Start other anti-anginals during the first 24 hours of nitrate infusion. 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).

The risk of recurrence of angina due to restenosis is decreasing with the use of stents and new anti-platelet agents including IIb/IIIa inhibitors.

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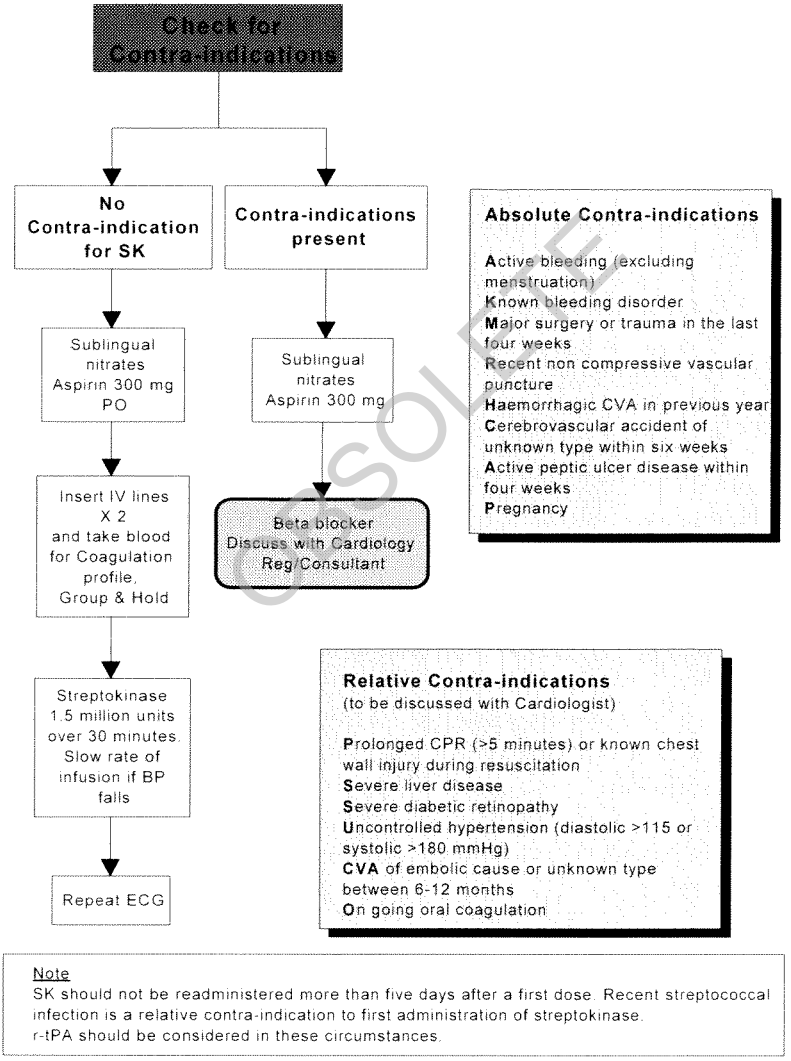
9.2.6 Current Indications for SK Therapy

- 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 proven 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.

Figure 1: Use of Streptokinase Therapy



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- **Administration of streptokinase**
  - Add 1,500,000U of streptokinase to 100 ml 0.9S and infuse over 30 minutes.
- **Monitoring requirements for thrombolytic therapy**
  - Continuous ECG monitoring with senior nurse and doctor in attendance.
  - 15 min pulse, BP and temperature for 12 hours, then hourly for 4 hours, then as needed.
  - Maintain availability of drugs (**see below**) 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 β-blocker (atenolol 25-50 mg PO daily, may need 100 mg) as well as aspirin and nitrates.
- Inferior infarct, check right sided leads for ST elevation (i.e. look actively for right ventricular infarction). **If MI strongly suspected give thrombolysis.**
- Move to CCU **early**.
- Err on side of **excess consultation** with cardiology registrar/cardiologist, acute medical registrar/general physician
- Failed thrombolysis may be an indication for emergency angiography/angioplasty

- **Complications**
  - Hypotension associated with SK infusions:
    - Slow or stop SK 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 metaraminol (Aramine) IV 0.5-1 mg, adrenaline **0.1 mg** (1 ml 1:10,000) 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.**

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- Allergic or febrile reactions which vary in severity from rigors to typical anaphylaxis. Give hydrocortisone 100 mg IV and/or promethazine (Phenergan) 12.5-25 mg IV stat. For severe anaphylaxis consider adrenaline SC/IV.
- Haemorrhage - apply local pressure over all IV access and arterial puncture sites before starting thrombolysis. If significant bleeding occurs 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**

- If thrombolytic therapy is not given, use low molecular weight heparin e.g. enoxaparine (Clexane) 1 mg/Kg q12h with a maximum of 100 mg per dose. If there is renal impairment or if reversal of the heparin effect is likely to be needed, use a continuous infusion of unfractionated heparin and monitor by APTT.
- **Hypnotics** if sleep disturbed.
- **Beta-Blockers** - continue if patient is already on them and no contraindication exists. β-blockers improve medium term prognosis and unless contraindications are present β-blockers such as atenolol (25-50 mg PO daily), metoprolol or timolol, should be commenced on admission, and given IV if continuing pain/arrhythmias. They should be continued for at least 2 years. Avoid in the first few hours after an **inferior** MI unless sinus tachycardia present.
- **Amiodarone** may be indicated for some atrial and ventricular arrhythmias. Discuss with cardiologist.
- **Continuing chest pain** in spite of appropriate morphine IV and sublingual nitrates. Consider β-blocker therapy but remember that patients may benefit from early intervention. Seek Cardiology advice without delay. Nitrate infusion *(see page 63)* or full heparinization may also be helpful.

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9.3 Cardiogenic Shock

9.3.1 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 59 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 *(see page 59)*. 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, repeated if necessary). This should only be done with Swan-Ganz catheter monitoring. CVP monitoring alone in cardiogenic shock is insufficient.
- All patients with cardiogenic shock should be considered for management in CCU or ICU. Call the CCU/ICU registrar for advice. If shock occurs in the hours following an MI, aortic balloon counter pulsation and coronary angioplasty is the treatment of choice.
- Treat any arrhythmias.

In Hospital Management Following Myocardial Infarction

- Mobilization protocols - these protocols are available in CCU and Cardiology Ward. Some patients can be discharged as early as three days after admission.
- Investigation after myocardial infarction:
  - 2D echocardiography should be considered in all patients to assess left ventricular function for prognostic reasons and review the need for on-going therapy with ACE inhibitors. Priority should be given to those with anterior MI, left ventricular failure or hypotension.
  - Sub maximal exercise testing should be considered if there is recurrent pain of uncertain origin during in-hospital mobilization, or if the patient needs reassurance as to their exercise capabilities prior to discharge.
  - Coronary angiography should be considered if patients experience recurrent pain of similar character to that experienced at the time of myocardial infarction. Coronary angiography should also be considered if there is evidence of poor left ventricular function or congestive cardiac failure.
  - Medical therapy should be tailored to each individual patient, but should include aspirin unless it is contra-indicated, beta blockers unless contra-indicated and ACE inhibitors if there is evidence of left ventricular dysfunction. Nitrates are appropriate for control of symptoms. There is little evidence that calcium antagonists improve prognosis following myocardial infarction. However, the use of diltiazem or verapamil could be considered in patients who have contra-indications to beta blocker therapy and have good left ventricular function without clinical evidence of congestive failure.

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- Action to reduce the effects of any risk factor present – smoking cessation, cholesterol lowering agents, control of hypertension, diet if overweight.

9.4 Unstable Angina

The following syndromes may be defined as unstable angina:

- Angina of recent origin (<1 month) which is severe and/or frequent.
- Severe prolonged or more frequent angina superimposed on previous stable angina.
- Angina developing at rest or with minimal exertion.

9.4.1 Definition

The pain experienced with unstable angina is similar to stable angina, though often more intense and of longer duration. It may also be associated with other signs such as sweating and nausea. Very often it is difficult to distinguish between unstable angina and acute myocardial infarction during the initial assessment of the patient. Thus, management in the first few hours will often be similar to that for myocardial infarction (see above).

OBSOLETE

9.4.2 Causes

- Coronary artery disease, often with intracoronary thrombus at the site of a ruptured plaque.
- Coronary artery spasm.

9.4.3 Investigations and Managements

These are similar to the treatment of acute myocardial infarction except that thrombolysis is not indicated. A Cardiologist should be consulted.

- Elevation of Troponin T or I (without Creatinine Kinase (CK) increase) indicates high risk.
- ECG changes such as ST depression or T wave inversion or any serial change over the first 24 hours suggest a poorer prognosis.
- Daily ECG and cardiac enzymes on at least two occasions are mandatory, as is assessment of cardiac risk factors including lipids.
- If ECG changes are present, oxygen, ECG monitoring, aspirin and heparin therapy should be initiated.
- There is increasing evidence that low molecular weight heparins are as effective as standard intravenous heparin and are much more convenient for the patient. Start aspirin, nitrate and at least one β-blocker or calcium antagonist. If patient has presented with unstable angina on anti-anginal therapy, plan to discharge on increased doses or add another anti-anginal. Patients should not be discharged on the same therapy as they were on at admission. Remember to investigate for anaemia, hyperthyroidism, heart

failure and arrhythmias as precipitants for angina. Plasma lipids and body weight should be assessed and treated appropriate.

- Exercise testing should be considered prior to discharge.
- Patients with prolonged episodes of chest pain (>20 min per episode) or with persistent abnormalities of sub-endocardial ischaemia (evolving T wave changes, reversible ST depression) are at very high risk of a major cardiac event during short term follow up. These patients should be considered for inhospital cardiac catheterization to define their coronary anatomy and plan further treatment.
- All patients leaving hospital with the discharge diagnosis of unstable angina should be reviewed at least once in Cardiology Outpatients to assess symptom status and review risk factor assessment.

9.5 Cardiac Arrhythmias

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

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9.5.1 Classification

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

9.5.2 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.

9.5.3 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.

9.5.4 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. Consult Cardiology.

- Check for abnormalities of K, Mg, Ca; acidosis and hypoxia. Metabolic factors may contribute to the initiation/perpetuation of the arrhythmia.
- Thyroid function tests.

9.5.5 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. Doses of beta blockers, calcium antagonists and digoxin should be reduced.
  - 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 progress to 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 requires monitoring in CCU or by telemetry. If stable with regular ventricular escape rhythm and satisfactory blood pressure, may be observed overnight. Be prepared to use isoprenaline (**see dosing instructions below**) to maintain rate if atropine alone is not effective. Discuss with Cardiologist. Symptomatic A-V block not associated with infarction usually merits placement of a permanent rather than temporary pacemaker. A temporary pacemaker may be required if there is recurrent syncope, nonsustained ventricular tachycardia, or severe bradycardia (<30/min).

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Bradyarrhythmias

- Sinus Bradycardia - check for excessive β-blockade. Common after myocardial infarction. Treat with atropine 0.6 mg IV if heart rate <40, if symptomatic or hypotensive. 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 b/min.
- 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 permanent pacing if symptomatic.

Note:

Inferior infarcts are associated with a wide range of rhythms which rarely have much adverse effect on myocardial performance. A-V block is common. 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.

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Supraventricular Tachycardia

- Always perform a 12 lead ECG.
- **Sinus** - slow onset, rate usually below 150/min, slows gradually 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). Do not perform if carotid vascular disease present; check for bruit.

  Monitor the effect of these manoeuvres with ECG, as this may induce 2:1 block.

- Adenosine given as a rapid bolus IV into a large vein, 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 β-blockers:
  - Verapamil 5 mg by slow bolus (5 minutes) followed by 1 mg/min to a total of 15 mg. ECG monitoring required, 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 β-blockers and no structural cardiac disease present use flecainide 2 mg/kg (max 150 mg) over 10 minutes IV or consider further β-blockade (make sure patient is not asthmatic).
  - If unsuccessful, proceed to cardioversion. The patient should be managed in the resuscitation room of the Emergency Department, CCU or ICU. An experienced doctor with anaesthetic skills should be present.
    When anaesthetised, 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 carotid sinus massage has not been performed 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 given below. If spontaneous reversion to sinus rhythm does not occur within 24 hours, the patient should be referred for cardioversion.
- **Atrial Fibrillation**
  New onset atrial fibrillation with rapid ventricular rate:
  - CBC, creatinine, Na, K, thyroid function tests
  - Digitalize:
    - Give 0.5 mg digoxin initially (IV if in heart failure or nauseated).
    - Give a further 0.25-0.5 mg at 4 and 8 hours to complete a loading dose of 1.0-1.5 mg.
  - Other options include:
    - Intravenous β-blocker (not if already on a calcium antagonist).
    - Oral β-blocker (atenolol 25-50 mg/day, sotalol 40-80 mg BD).
    - Oral calcium antagonist (diltiazem 60 mg TDS, verapamil 80 mg TDS).

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  - Most patients without other heart disease and with recent onset atrial fibrillation, will revert to sinus rhythm within 24 hours. Chemical cardioversion may be attempted in patients without other heart disease. Give Flecainide 200 mg PO stat, then 100 mg in 2 hours, then 100 mg BD until conversion.

- Heparin:
  - All patients should be treated with full dose low molecular weight heparin subcutaneous, unless there are contra-indications. Warfarin may not be required if sinus rhythm achieved within 48 hours and there is no left atrial enlargement or major mitral valve abnormality.

Or

- Electrical cardioversion:
  - Indicated if there is cardiac compromise with hypotension, angina or impaired cerebral function or persistant atrial fibrillation. Consult cardiologist.

OBSOLETE

♦ **Chronic atrial fibrillation on digoxin with rapid ventricular rate:**

- Exclude aggravating causes (ischaemia, heart failure, volume depletion, infection, alcohol).
- Check digoxin concentration.
- Add oral β-blocker or calcium antagonist as above.
- Add aspirin if heart structurally normal on echocardiogram.
- Consider warfarin if left atrium dilated or mitral valve abnormal, or age >70 years.

Ventricular Arrhythmias

♦ **Idioventricular** (rate < 100/min,) – this is common after myocardial infarction and no treatment is required.

♦ **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 by cardioversion. Lignocaine 100 mg IV can be used (and repeated x 1) but can precipitate hypotension or VF. 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.

- **QT prolongation**
  - **Acquired long QT**
    - Generally, QT prolongation is acquired and is associated with bradycardias, myocardial ischaemia, metabolic disturbances or drugs.
    - Causes of Acquired QT Prolongation
      - ~ Drugs Macrolides e.g.(erythromycin)
        Antihistamines e.g. (terfenadine)
        Antiarrhythmics
        Class 1 quinidine, flecainamide, disopyramide
        Class 2 amiodarone, sotalol.
        Psychoactive drugs (lithium, tricyclics, haloperidol)
        Other drugs including glibenclamide and theophylline
      - ~ Note: Drug Interactions:
        The following may increase the concentration of the above drugs. Fluconazole, grapefruit juice, metronidazole, macrolides, SSRIs, diltiazem and many others.
      - ~ Other factors - Myocardial ischaemia, bradycardia, low K, low Mg.
    - Check for possible causes and withhold any drugs that may be potentially responsible.
      Correct all metabolic disturbances and treat ischaemia.
  - Congenital long QT
    Usually presents in patients younger than 40 years.
    - Withhold all QT lengthening drugs.
    - Check and correct any metabolic disturbances.
    - β blockers may help suppress recurrent episodes.
    - Refer to Cardiologist for long term management.
- **Torsade de pointes**
  This polymorphic ventricular tachycardia is due to QT prolongation, either congenital or acquired. It may revert spontaneously, otherwise it may require immediate cardioversion.
  If recurrent, IV magnesium 2 – 4 g IV may be tried, otherwise temporary pacing at 90 beats/minute may suppress this arrhythmia. Consult Cardiology.
- **Ventricular Fibrillation** (VF) - D.C. shock (see below).

Amiodarone

- Intravenous amiodarone is very effective in the acute treatment of atrial and ventricular arrhythmias. However, there may be difficulties in switching to oral therapy and potentially important side effects may occur with long term therapy.

OBSOLETE

- Amiodarone has less effect on myocardial contractility than other anti-arrhythmics. Therefore, intravenous amiodarone may be the treatment of choice for arrhythmias if there is known severe left ventricular impairment or concurrent left ventricular failure.
- Give 5 mg/kg [or 150 mg to 300 mg] dissolved in 250 mls of D5W over 30 to 60 minutes intravenously. Continue with 10 mg/kg or 900 mg / 500 mls / 24 hours
- Because of risk of chemical thrombophlebitis, amiodarone should be given into a proximal arm vein. Consider a central line if planning to give more than 24 hours intravenous infusion.
- Patients receiving intravenous amiodarone should be on continuous ECG monitoring.

9.6 Cardiac Arrest

- Commence basic life support - using the ABCs of CPR. Call for the Cardiac Arrest Team and trolley. Precordial thump if witnessed.

REMEMBER:

- External cardiac compression at 100/min.
- Ventilate once every 5 beats for 2 person CPR and twice every 15 beats for 1 person CPR.
- Use oropharyngeal airway with ambu bag and face mask, 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.

OBSOLETE

9.6.1 Identify the Cardiac Rhythm

- **Ventricular fibrillation (VF):**
  - Defibrillate immediately using 200 Joules.
  - Check rhythm; if still In VF, repeat defibrillation using 200 Joules.
  - If still in VF, repeat defibrillation using 360 Joules x2.
  - If unsuccessful give adrenaline **1.0 mg** IV. This may be repeated every 3 minutes.

- If still in VF defibrillate using 360 x3 Joules.
- If still in VF give lignocaine 50-100 mg IV bolus and repeat defibrillation using 360 Joules.
- If further resistant VF give 1.0 – 2.0 mg metoprolol IV and defibrillate.
- If still in VF consult Cardiologist regarding the use of amiodarone or other agents.

♦ **Ventricular asystole:**

Note: Exclude the possibility of monitor failure resulting in apparent asystole. Always attempt thump pacing. Check for evolved QRS and pulse for effectiveness.

- Give adrenaline **1 mg** IV. This may be repeated frequently.
- If still asystole give atropine 3 mg IV.
- Consider an adrenaline infusion. Add 1.0 mg 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:**

- "Thump pacing" may be effective in inducing ventricular depolarization and an adequate cardiac output.
- Atropine 0.6 mg IV and repeat if necessary.
- Adrenaline infusion as described under asystole.
- Definitive management is by pacemaker, transcutaneous (temporary) or transvenous.

♦ **Electromechanical dissociation**, i.e. organised electrical activity on ECG but failure of effective myocardial contraction:

- Consider and treat possible causes including hypovolaemia, tension pneumothorax, cardiac tamponade, pulmonary embolism, overdose, anaphylaxis. Urgent echocardiography may be useful.
- In absence of other specific therapy give adrenaline **1 mg** IV. This may be repeated every 3-5 minutes.

9.6.2 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.

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9.7 Hypertension

9.7.1 Classification

- Primary — Idiopathic, "essential".
- Secondary — Renal, endocrine or neurological disease, diabetes mellitus, coarctation of the aorta. 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.

9.7.2 Aetiology

- Renal — Acute nephritis, renal impairment (acute or chronic), renovascular and volume overload (especially dialysis patients).
- Endocrine — Cushing's Syndrome, phaeochromocytoma, Conn's, hyperparathyroidism, hyperthyroidism, hypothyroidism, acromegaly.
- Neurological — Raised intracranial pressure, autonomic neuropathy.
- Diabetes Mellitis — Both Type I and II patients are commonly hypertensive.
- Coarctation of the Aorta
- Respiratory — Obstructive sleep apnoea.
- Drugs — Presence or absence (clonidine withdrawal). NSAIDs, steroids, sympathomimetics (including non prescription drugs), alcohol, liquorice, cocaine, erythropoietin, cyclosporin.

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9.7.3 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 (diptest for proteinuria/haematuria, microscopy for cells and casts).

- Plasma, Na, K, Cl, creatinine, Ca. Haemolysis of samples may obscure hypokalaemia.
- 24 hour urine collection for protein (plain container).
- 24 hour urine for creatinine clearance, Na, K, VMA, metanephrines and free catecholamines (acidified container).
- Renin and aldosterone plasma levels if Conn's Syndrome possible.

9.7.4 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 and days 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** – A calcium antagonist (e.g. felodipine 2.5 mg), or an α antagonist (e.g. doxazasin 1 mg) or a β blocker can be used. Alternatively captopril 6.25 mg PO may be used but should be avoided in the presence of hyponatraemia. Labetalol gives combined α and β-blockade and may be used if no contraindications to β-blockade (200 mg PO stat then repeat as required up to 1200 mg daily). Avoid a β blocker alone if phaeochromocytoma is a possibility. In this situation, labetalol is generally a good choice.
- **IV therapy** - for true acute hypertensive encephalopathy, i.e. sudden severe rise in diastolic blood pressure, give labetalol 50 mg IV over 1 minute followed by further slow IV push to total 300 mg. An effective 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 is best which will result in a slower reduction in blood pressure and preserve cerebral autoregulation.
- If hypertension is associated with acute LVF or volume overload IV frusemide should be used along with an ACE inhibitor or an A II α antagonist (e.g. losartan).
- Phaeochromocytoma, if suspected, requires α-blockade (phenoxybenzamine) or the combination of α plus β-blockade (e.g.

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labetalol). Avoid β-blocker monotherapy as it may cause paradoxical hypertensive crisis via unopposed alpha 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 β-blocker alone. Stopping clonidine may induce a phaeo-like state which is exacerbated by giving a β-blocker. Labetalol is recommended as it provides α and β-blockade.

9.8 Aortic Dissection

9.8.1 Clinical Features

This diagnosis should be specifically considered in all cases of acute chest pain. Pain is sudden and severe, most often in the interscapular region. It can mimic angina. Pain, hypovolemic shock and an abnormal mediastinum on chest X-ray suggest aortic dissection. Seek urgent advice from the cardiologist on call. The immediate priority is to determine the presence and type of dissection urgently since this will influence management and prognosis.

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9.8.2 Aetiology

- Cystic medial necrosis.
- Marfan's Syndrome.
- Atherosclerosis.
- Hypertension.

9.8.3 Investigations

*See Table 12 – **Diagnosis of Aortic Dissection** on page 80.*

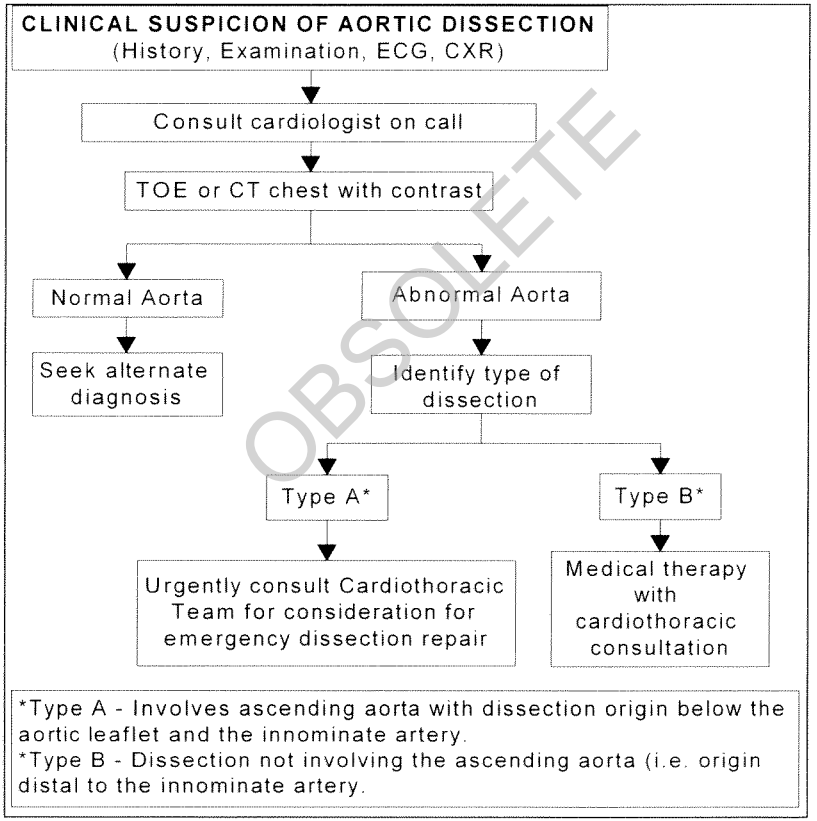
- CXR Widened mediastinum, pleural effusion mainly left sided, calcified intimal flap separated from aortic outline, high aortic arch.
- ECG - dissection involving the aortic root may occlude the coronary arteries, more often the right coronary to produce a myocardial infarction. LVH may be present from long standing hypertension.
- CT scan with contrast or transoesophageal echocardiogram (TOE).
- Crossmatch blood.

9.8.4 Treatment

- Aim to reduce systolic pressure to 100-120 mm Hg and reduce contractility of left ventricle.

- Monitor BP and urine output.
- Give intravenous and later oral β-blockers (e.g. labetolol) unless contraindicated; cardiac failure, bradycardia <60/min, heart block, and obstructive airways disease, **and** start a glyceryl trinitrate infusion *(see page 63)* or a sodium nitroprusside infusion.
- Analgesia, morphine 10-15 mg. Give prochlorperazine (Stemetil) 12.5 mg IV to prevent vomiting.
- Seek advice urgently from cardiologist or cardiothoracic surgeon.

Table 12: Diagnosis of Aortic Dissection



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9.9 Bacterial Endocarditis

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

9.9.1 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.
- CXR.
- ECG.
- MSU x 2 before therapy.
- Na, K, Ca, glucose, creatinine, Bili, ALP, AST.
- CBC + diff.
- Urgent echocardiogram.

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9.9.2 Treatment

- Initial Therapy - benzylpenicillin 2 mega units q4h IV, plus gentamicin. Flucloxacillin should be added if Staphylococcal sepsis suspected (e.g. IV drug user).
- Gentamicin dose of 1 mg/kg not exceeding 80 mg IV q8h for 48 hours. Seek advice about subsequent dosage.
- 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.

9.10 Infective Endocarditis Prophylaxis

The following tables are taken from the Heart Foundation Technical Report "Prevention of Infectious Endocarditis asociated with Dental Treatment and other Medical Interventions. (July 1999)" The Heart Foundation has given permission for sections of this report to be reproduced here. You are encouraged to obtain copies of the full report from:

The National Heart Foundation of New Zealand
PO Box 17160
Greenlane
Auckland 1130

9.10.1 Cardiac conditions and endocarditis prophylaxis

Endocartitis prophylaxis recommended

High risk category

All patients with a previous episode of endocarditis.[a]

Prosthetic cardiac valves, including bioprosthetic and homograft valves.[b]

Complex cyanotic congenital heart disease (e.g. tetralogy of Fallot, tricuspid atresia, complex anomalies with functional single ventricle, or transposition of the great arteries).

All major left-sided valve anomalies.

Surgically constructed systemic-pulmonary shunts, or conduits from the heart to the great arteries.[c]

Moderate risk category

With the exception of those listed in the "not recommended" category in the following section, most other congenital cardiac malformations carry a moderate risk. These include:

All high-pressure (left sided) congenital anomalies even if minor, including supravalvular, valvular and subvalvular aortic stenosis, coarctation of the aorta, and ventricular septal defect.

All acquired valvular dysfunction, e.g. rheumatic heart disease.

Hypertrophic cardiomyopathy.

Mitral valve prolapse with valvular regurgitation and/or thickened leaflets and dysplastic myxomatous valves.[d]

Major congenital right-sided lesions, e.g. Ebstein's anomaly of the tricuspid valve and significant pulmonary stenosis.

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*Endocarditis prophylaxis **NOT** recommended*

Isolated secundum atrial septal defect.

Complete surgical or device closure of atrial septal defect, ventricular septal defect, or patent ductus arteriosus (beyond six months after repair).[e]

Previous coronary artery bypass graft surgery.

Mitral valve prolapse without valvular regurgitation or dysplasia.

Previous Kawasaki disease without valvular dysfunction.

Previous rheumatic fever without valvular dysfunction.[f]

Cardiac pacemakers (intravascular and epicardial) and implanted defibrillators.

Physiological, functional or innocent murmurs.[g]

Notes

a Even if the underlying lesion is minor, a previous attack of endocarditis demonstrates risk.

b The risk of endocarditis remains high after replacement of the native valve with any prosthesis.

c All surgical conduits carry a high risk, particularly as the wall becomes irregular and thickened.

d A degree of mitral valve prolapse is very common. Dysplastic, myxomatous mitral valves are associated with connective tissue anomalies, such as Marfan's syndrome, and with increasing age. Sometimes both these types of valves can leak with exercise, but an increased risk of endocarditis has not been shown unless valvular regurgitation is present at rest, or valve structure is very distorted.

e Six months allows sealing of minute leaks around the periphery of the closure, and endothelialisation of surfaces. The same period is advised for these lesions treated by percutaneous placement of a mechanical device. In the small number of patients with a residual leak, long-term prophylaxis may be recommended.

f Prophylaxis is recommended when subclinical, echocardiography-demonstrated mitral or aortic regurgitation are present after acute rheumatic fever.

g A systolic murmur (often associated with a fever) can be recorded in well over 50% of young children. Most of these are "benign systolic murmurs" where the heart is normal. This diagnosis is established by:

- Exclusion of any cardiac symptoms and any associated signs including reduced femoral pulses.
- Recognising the signs that are typical of the "benign murmur", i.e. a grade 1-2/4 low-pitched, mid-systolic vibratory murmur, maximal around the 3rd to 4th left interspace and not radiating prominently to the suprasternal notch – unlike an aortic murmur, which is softer in the sitting than in the lying position.

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9.10.2 Dental Procedures and endocarditis prophylaxis

Endocarditis prophylaxis recommended

In general, any procedure that causes bleeding from the gingiva, mucosa or bone.

Periodontal procedures including probing, scaling, root planing and surgery.

Endodontic instrumentation or surgery beyond **the apex.**

Application of matrix bands **below** the gingival margin.

Subgingival placement of gingival retraction cord/strips.

Placement of orthodontic bands, but not brackets.

Intraligamentary local anaesthetic injections.

Reimplantation of avulsed teeth and repositioning of teeth after trauma.

Oral surgical procedures including biopsy procedures and raising of mucosal flaps.

Surgical drainage of dental abscesses.

Extraction of teeth.

Endocarditis prophylaxis NOT recommended

Natural shedding of primary deciduous teeth.

Dental examination, **other than periodontal probing.**

Radiographic examination.

Local anaesthetic injections, **unless intraligamentary.**

Restorative dentistry where the procedure is above the gingiva.

Impressions, construction and placement of removable prosthodontic/orthodontic appliances.

Adjustment of orthodontic appliances.

Placement of rubber dam, **other than subgingival manipulation.**

Postoperative suture removal.

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9.10.3 Other procedures and endocarditis prophylaxis

Endocarditis prophylaxis recommended

Respiratory tract

Tonsillectomy and/or adenoidectomy.

Surgical operations that involve the respiratory mucosa.

Bronchoscopy with a rigid bronchoscope (with or without biopsy).

Genitourinary tract

Prostatic surgery, transrectal prostatic biopsy, cystoscopy, or urethral dilatation (even in the absence of infection).

Surgical procedures **in the presence of infection**, e.g. urethral catheterisation, uterine dilatation and curettage, therapeutic abortion, sterilisation procedures, insertion and removal of intrauterine devices, circumcision.

Gastrointestinal tract

Sclerotherapy for oesophageal varices or oesophageal stricture dilatation.

Endoscopic retrograde cholangiography and biliary tract surgery.

Surgical operations involving the intestinal mucosa (other than endoscopic biopsy and percutaneous endoscopic gastrostomy).

Other sites

Incision and drainage of focal sepsis, e.g. subcutaneous abscess. (Note that prophylaxis here will often necessarily be part of more prolonged antibacterial treatment).

Endocarditis prophylaxis NOT recommended

Respiratory tract

Endotracheal intubation.

Bronchoscopy with a flexible bronchoscope, with or without biopsy.

Tympanostomy tube insertion.

Genitourinary tract

Vaginal delivery, Caesarean section, vaginal hysterectomy.

Surgical procedures in the absence of infection, e.g. urethral catheterisation, uterine dilatation and curettage, therapeutic abortion, sterilisation procedures, insertion and removal of intrauterine devices, circumcision.

Gastrointestinal tract

Transoesophageal echocardiography.

Endoscopy with or without biopsy.

Percutaneous endoscopic gastrostomy.

Other sites

Procedures through surgically prepared skin, e.g. liver or kidney biopsy, dermatological procedures.

Cardiac catheterisation including balloon angioplasty.

Implantation of cardiac pacemakers, defibrillators and coronary stents.

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Table 13: Antibacterial Recommendations for Dental, Oral, Respiratory Tract or Oesophageal Procedures

	Moderate cardiac risk[a]	High cardiac risk[a]
Standard [b]	• Oral amoxycillin 2.0 g one hour before procedure and oral amoxycillin 1.0 g six hours later.	• Oral/intravenous amoxycillin 2.0 g plus intravenous/ intramuscular gentamicin (2.0 mg/kg, not > 120 mg) within 30 minutes of procedure. No subsequent dose recommended.
Penicillin allergy [b,c]	• Oral cefuroxime axetil 1.0 g one hour before procedure and oral cefuroxime axetil 1.0 g six hours later. • Oral clindamycin 300 mg one hour before procedure and oral clindamycin 150 mg six hours later. • Oral clarithromycin 500 mg one hour before procedure. No subsequent dose recommended.	• Intravenous cefuroxime 750 mg plus intravenous or intramuscular gentamicin (2.0 mg/kg, not > 120 mg) within 30 minutes of procedure. No subsequent dose recommended. • Intravenous clindamycin 300 mg within 30 minutes of procedure and intravenous or oral clindamycin 150 mg six hours later. • Intravenous vancomycin 1.0 g infused over 60-90 minutes ending within 30 minutes of procedure. No subsequent dose recommended.
Notes		
a	Some international guidelines now recommend the same regimen for those with high and moderate cardiac risks. The options listed for high-risk cases are those with theoretically maximal preventative activity.	
b	Those who have received a beta-lactam (either a penicillin or cephalosporin) within two weeks of the procedure, or are on long-term penicillin prophylaxis for rheumatic fever, need a clindamycin or clarithromycin regimen from the moderate-risk category or any of the high-risk category options. In some of the latter, the synergistic killing of the combined beta-lactam plus aminoglycoside overrides the possible reduced beta-lactam susceptibility from prior beta-lactam treatment.	
c	The oral cefuroxime axetil and intravenous cefuroxime/gentamicin regimens are options for patients whose penicillin allergy was not anaphylaxis or a rapid-onset skin reaction.	

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Table 14: Antibacterial Recommendations for Genitourinary Tract and Gastrointestinal Tract (excluding oesophageal) procedures

	Moderate cardiac risk[a]	High cardiac risk[a]
Standard [a]	• Oral amoxycillin 2.0 g one hour before procedure and oral amoxycillin 1.0 g six hours later.	• Intravenous amoxycillin 2.0 g plus intravenous or intramuscular gentamicin 2.0 mg/kg within 30 minutes of procedure and oral or intravenous amoxycillin 1.0 g six hours later.
Pencillin allergy [a,b]	• Intravenous vancomycin 1.0 g infused over 60-90 minutes ending within 30 minutes of procedure. No subsequent dose recommended.	• Intravenous vancomycin 1.0 g infused over 60-90 minutes ending within 30 minutes of procedure plus intravenous or intramuscular gentamicin (2.0 mg/kg, not > 120 mg). No subsequent dose recommended.
Notes		
a	Prior or continuing penicillin treatment does not affect these regimens.	
b	There is no oral option for those with penicillin allergy.	

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10. Endocrinology / Metabolic Disorders

10.1 Endocrinology

Main Office 2nd Floor, Riverside ☎ 80927 Fax 81159

Inpatient Services Ward 26

- Prof E. Espiner
- Dr W. Inder

Consultation and On-call Service Daily.

Contact Endocrinologist or Endocrine registrar. For consults fax referral to 81159.

Consultation Guidelines

Adults

- Pituitary, adrenal, gonadal, corticosteroid use or requirement, calcium and electrolyte problems including unexplained hypoglycaemia. Disorders of growth and/or puberty, amenorrhoea, hypogonadism, hirsutism, 'endocrine' hypertension.
- Infertility and gynaecological endocrinology - in association with the Gynaecology Outpatient Service, Christchurch Womens Hospital.

Thyroid Disorders - Nuclear Medicine Department.

- Medical Consultation (Drs Brownlie/Turner). ☎ 80890 Fax 80869

Children (<12 years)

- Endocrine disorders, growth etc through Department of Paediatrics (Associate Professor Abbott) in first instance.

Other Services

Endocrine Laboratory (Endolab). ☎ 80848 Fax 80818

– Laboratory technical consultation test and sampling enquiries.

Medical consultation and patient enquiries. ☎ 80927 Fax 81159

Endocrine special tests. ☎ 80934 Fax 81159

See Endolab Handbook (June 1995) for details on request, logistics and interpretation of endocrine tests.

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10.2 Diabetes Service

Main Office 245 Antigua Street ☎ 80860 Fax 80171

Consultation and On-call Service 24 hours a day, seven days a week.

- Dr H. Lunt ☎ 025 333508
- Dr M.P. Moore ☎ 025 377095
- Dr R.S. Scott ☎ 025 366380
- Dr D. Cole ☎ 025 452416

Consultation Guidelines

Physician input. New Type 1 diabetes mellitus. Admissions with metabolic decompensation as the primary cause of admission. Also consider acute input if glycaemic control is unstable and in younger patients with newly diagnosed Type 2 diabetes or significant diabetes complications.

Other Services

Diabetes Nurse Educator	☎ 80860	Fax	80171
Nurse Maude District Nurse (Maria de Jong)	☎ 025 375903		
(To start insulin in the domiciliary setting)			
Outpatient Appointments (Brenda Pullan)	☎ 80321	Fax	80171
Lipid and Diabetes Research Group (Tracey Hubert)	☎ 80449	Fax	80457

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10.3 Adrenal Insufficiency

10.3.1 Causes

- Primary adrenal failure:
  - Autoimmune.
  - Tuberculosis.
  - Haemorrhage e.g. heparin therapy.
  - Metastases.
  - HIV infection.
- Secondary:
  - ACTH deficiency [pituitary failure].
  - Adrenal suppression, or glucocorticoids stopped or not increased at time of acute stress.

10.3.2 Clinical Features

- Progressive weakness, weight loss, anorexia/nausea.
- Postural hypotension, confusion.
- Symptoms of hypovolaemia [shock] are more prominent in primary failure where skin pigmentation [ACTH effect] is usually also seen.

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10.3.3 Investigations

- Biochemistry - may be normal [hyponatraemia common]. In later phases of primary deficiency, low Na and high K, high urea, lowish glucose.
- CBC and diff - may be eosinophilia and neutropenia.
- CXR - cardiac size may be decreased [hypovolaemia].
- Draw blood for cortisol, ACTH, renin and aldosterone (10 ml into EDTA tubes). Contact Biochemistry for immediate 4°C centrifugation and freezing of plasma. Urgent Synacthen test [plasma cortisol before and one hour after 24 units ACTH - 0.25 mg IM] - may be indicated.
- **Interpretation:** in primary adrenal insufficiency plasma ACTH and renin are markedly raised [low cortisol / ACTH and low aldosterone / renin ratios]. Plasma cortisol can be in the "normal" range but there is a diminished response to Synacthen. In pituitary failure [ACTH deficiency], plasma cortisol is usually inappropriately low for the clinical status and usually the cortisol response to Synacthen is also diminished - but can be normal. Successive 0800 hr plasma cortisol levels may be indicated, and/or stress tests [insulin tolerance test (ITT) etc]. Consult Endocrine Team.

10.3.4 Treatment

- If hypovolaemic; especially if primary adrenal insufficiency, 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. Amounts of potassium infused [if any] based on plasma levels. Consider antibiotics in cases of profound shock [e.g. IV cefuroxime and gentamicin].
- Hydrocortisone 50-100 mg IV then 50 mg 12 hourly for no more than 24 hours, then reduce daily dose of hydrocortisone (e.g. daily dose rapidly reduced to 50-75, 30-50 mg/day on successive days depending on metabolic status) then gradually down to a long term maintenance level of 20-30 mg per day. **If** primary adrenal insufficiency, 9α-fludrohydrocortisone 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 suppression of the hypothalamic-pituitary-adrenal axis. Such patients receiving long term glucocorticoids [e.g. more than 5-7.5 mg prednisone/day] for conditions other than cortisol deficient states, who are admitted with acute illness, sepsis etc, may require a doubling of the dose [e.g. 20 mg prednisone/day for 1-2 days] then reduce rapidly to normal maintenance doses. If unable to take oral steroids, consider parenteral hydrocortisone e.g. 50-100 mg/24 hr for 1-2 days and monitor electrolytes, mental status, blood pressure. Reduce steroid dose rapidly as clinical state allows to maintenance levels. These patients are usually much less sensitive to acute stress than those with intrinsic endocrine disease of the pituitary or adrenal.

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10.3.5 Management of stress situations in patients with steroid deficiency on maintenance steroids.

- Patients with intrinsic lack of ACTH or with primary adrenal insufficiency are especially sensitive to acute stress illness. Careful monitoring needed.
- 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.
  - 50 mg hydrocortisone IM or IV 30 minutes preoperatively. Then 50 mg over the 4 hours of surgery, thereafter 25 mg q6h and reduce as above, after 24 hours.
  - Check electrolytes daily looking particularly for hyperglycaemia and hypokalaemia.

10.4 Diabetes – General Comments

- **Diabetes terminology** - The preferred terminology is Type 1 and Type 2 diabetes instead of insulin dependent and non insulin dependent diabetes.
- **Unstable blood glucose** - Patients with diabetes, who are admitted to hospital for reasons other than diabetic control, often experience unstable blood glucose results. Any sustained increase in blood glucose will lead to a delay in wound healing and slow the resolution of infection.
- **Does a hospitalised patient with high glucose values have diabetes?** Inpatients with no previous history of diabetes may have a temporary elevation in glucose, in response to stress and medications (e.g. corticosteroids). However many of these patients will have long standing undiagnosed diabetes. A glycated haemoglobin assay may help distinguish transient impairment of glucose tolerance from undiagnosed diabetes. If in doubt, arrange GP follow-up after discharge.
  - The preferred screening test for **well** patients is two fasting laboratory plasma glucose values.
  - A diagnosis of diabetes can be made on two fasting results ≥ 7 mmol/L.
- **Opportunistic complications screening**
  - A small percentage of patients with known diabetes do not see a health care practitioner on a regular basis for diabetes complications screening.
  - Opportunistic screening should be undertaken if these patients are admitted to hospital.
  - Retinal photography can be arranged through the Eye Department.
  - Foot examination and a laboratory urinary microalbumin assay should also be performed.
- **Changes in inpatient insulin requirements.**
  - Some patients who were previously well controlled on diet and tablets, 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.
  - If insulin is needed an "average" starting regimen would be Penmix 20 60% in the morning and 40% at teatime, at a total dose of 0.3 u/kg per 24 hrs. For example a 100 kg patient might be prescribed Penmix 20 18 units before breakfast and 12 units before tea. Supplemental Actrapid or lispro insulin could be prescribed before or with meals, e.g. 6 units if blood glucose ≥ 15 mmol/L. This starting dose is likely to be insufficient for most patients and **will need daily adjustment**. (A small percentage of patients will experience hypoglycaemia – this mandates **immediate** adjustment of the regimen.)

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Table 15: Description of insulins currently available in New Zealand

Type of Insulin	Brand Names	Description of Action	Duration of Activity *		Can it be used in a pen injector?	Common outpatient use
			Peak (hours after injection)	Time to disappearance (hours after injection)		
Lispro	Humalog	Fast acting	1.5	6	Yes	t.d.s. with food – requires the addition of a once or twice a day intermediate or long-acting insulin.
Neutral (soluble)	Actrapid Humulin R	Short Acting	2 – 4	10	Yes	t.d.s. half an hour before food in addition to a bedtime intermediate or long-acting insulin.
Premixed** insulin e.g. 30% neutral 70% isophane	Penmix30** Humulin 70/30**	Biphasic	As for component insulins	24	Yes	Half an hour before breakfast and the evening meal.
Isophane (NPH)	Protaphane Humulin N	Intermediate acting	3 – 8	24	Yes	Background (basal) insulin – often given at bedtime and used in conjunction with fast/short acting insulins or with oral anti-diabetic agents.
Insulin zinc suspension	Monotard Humulin L	Intermediate acting	4 – 10	24	No	Background (basal) insulin in patients prepared to use syringes. May result in less nocturnal hypoglycaemia than NPH, when injected at bedtime.
	Ultratard Humulin U	Long acting	4 - 14	28		

*Insulin activity varies between injections (i.e. within patient variablilty) and from patient to patient (i.e. between patient variability). This table of duration of action therefore represents an approximate guideline only.

**Other mixes with a different percentage of neutral and isophane insulin are also available e.g. 10/90, 20/80, 50/50.

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- **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 hospitalisation.
    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 stabilised, four times a day testing is usually adequate.
  - Patients on Actrapid or Penmix (or equivalent) should be tested before main meals and at bedtime.
  - 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.
- **Hyperglycaemia induced hyponatraemia**
  - Mild hyponatraemia is common in well hydrated patients with hyperglycaemia. Hyperglycaemia is associated with a shift in water from intracellular to extracellular fluid and this causes a compensatory dilutional hyponatraemia.
  - For each 10 mmol rise in serum glucose, an approximately 3.0 mmol fall in serum sodium is expected.
  - Correction of the elevated glucose will usually result in normalisation of the serum sodium.
- **Metformin - induced lactic acidosis**
  - Lactic acidosis is a rare but potentially fatal complication of metformin treatment.
  - Metformin should be avoided in patients who are at increased risk of lactic acidosis.
  - This includes patients with renal impairment [serum creatinine >0.16 mmol/L], cardiac failure, hepatic impairment, hypoxia and severe dehydration.
  - Patients with a severe intercurrent illness will require temporary cessation of metformin.
- **Intravenous insulin infusion post acute myocardial infarction in patients with diabetes**
  - Both short and long term cardiovascular mortality is reduced in patients with newly diagnosed as well as known diabetes, if an intravenous insulin

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infusion is used in the immediate post myocardial infarction period. For further advice consult with the on call Cardiology and Diabetes teams.

- **Pre-discharge planning**
  This should be undertaken at least 24 hours before patients on insulin leave 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 stabilise, after discharge?
  - Have you prescribed the right sort of insulin? (Most patients use 3 mL cartridges – some patients use 10 mL vials.)
  - Have you prescribed pen injector needles and insulin syringes, if required?
  - Have you prescribed the right sort of glucose test strips for the patient's blood glucose meter?

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

OBSOLETE

10.5 Diabetic Ketoacidosis

10.5.1 General Principles and Precautions.

- DKA is associated with significant mortality, particularly in the older patient with an underlying acute medical condition precipitating ketoacidosis. Death from DKA in young, otherwise healthy patients, is 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.
- Can the patient be safely managed as an outpatient?
  Some patients with Type I 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 hyperglycaemic hyperosmolar non ketotic syndrome (see below). **Discuss the management of these patients with the Diabetes Physician on call.**

10.5.2 Common Causes for DKA

- Insulin withdrawal or reduction.
- Myocardial infarction, stroke, trauma or other medical stress.
- Infection such as pneumonia, gastroenteritis, influenza, UTI, meningitis.

10.5.3 Baseline Investigations

- Glucose.
- K, Na, urea and creatinine. (Creatinine may be falsely elevated if ketones are high due to interference with the assay). Measurement of ketone bodies e.g. plasma hydroxybutyrate.
- 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.

10.5.4 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.

- **Monitoring**
  - 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 closely 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 a 3-way connector.

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- **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 5-10 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 be required within two hours of commencing insulin, or sooner if baseline K is low.

OBSOLETE

Table 16: Potassium Replacement in DKA

Serum Potassium	Potassium via Infusion pump (mmol/kg/hr)
<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 about a 2 hour overlap from IV to SC insulin. The time required for overlap following commencement of subcutaneous lispro injection is half an hour.
  - If the patient has newly diagnosed Type I diabetes, 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 Penmix 20 before breakfast and tea, or lispro with main meals and Humulin N at bedtime.

- **Use of sliding scale subcutaneous Actrapid on its own is inappropriate and is likely to delay stabilisation 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.
  - 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.

OBSOLETE

10.6 Hyperglycaemic Hyperosmolar Non Ketotic Syndrome

- Differentiated from patients with DKA by:
  - Absence of significant ketosis (there may be a lactic acidosis).
  - High blood glucose and plasma osmolality, for example a serum osmolality of >320 mosm/kg and a serum glucose of >33 mmol/L
  - Profound dehydration.
- These patients are often drowsy, confused or comatose, due to cerebral intracellular dehydration. This syndrome tends to occur in older patients with Type 2 diabetes. Precipitating causes include infection, diuretic therapy and myocardial infarction. It is associated with mortality rates of up to 40%.

10.6.1 Investigations

as for DKA but include plasma osmolality.

10.6.2 Management

- General Principles
  - **The key to adequate management is appropriate fluid replacement.**
  - The correct choice of fluid replacement and speed of administration are critical.
  - The management plan should 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.
- These guidelines may need to be modified for patients with mild diabetic hyperosmolar syndromes [osmolality 320-340 mosm/kg, glucose <50 mmol/L], as they are less likely to require ICU admission, CVP monitoring and hypotonic saline.
- 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**

- 1L 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).
- 2-3L 0.45S at 500 ml/hr. The rate of subsequent 0.45S infusions will depend on the patient's clinical state.
- Run D5W in addition to 0.45S when the blood glucose is <15 mmol/L and Na <150 mmol/L. If glucose is <15 mmol/l but Na >150 mmol/L, change to D4S or D5W only with no 0.45S.
- 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 5-10 units/hr, initially.
- Once the glucose has reached 15 mmol/L, decrease the rate to 1-2 units/hr.
- 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.

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- **Prevention of venous thrombosis**
  - These severely dehydrated, comatose patients are at particular risk of DVT. Consider the use of prophylactic low molecular weight heparin.

10.7 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 unit/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.

OBSOLETE

10.8 Management of the newly diagnosed patient with Diabetes

- **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 or metformin 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. Use of sulphonylureas also has implications for vocational drivers.
- **The obese patient**
  Weight reduction and exercise are the cornerstones of management. Many patients will also require metformin. 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**
  They 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.

10.9 Hypoglycaemia

This is common in patients on insulin or a sulphonylurea

- **Management**
  - If the patient is unconscious deal with the airway, breathing and 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 (short acting carbohydrate followed by long-acting carbohydrate).
  - If the patient is hypoglycaemic due to a long-acting sulphonylurea, the hypoglycaemia may recur up to 48 hours after initial presentation and regular capillary glucose checks are needed over this period. Management with a 10% dextrose drip may be 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.
- **What precipitated hypoglycaemia?**
  - 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.
- If hypoglycaemia is suspected or diagnosed but the patient is not known to be on treatment for diabetes i.e. if insulinoma or inappropriate ingestion of a sulphonylurea (venous glucose less than 3.0 mmol/L), draw blood for repeat glucose, insulin and c-peptide (10 mL blood into EDTA tubes and contact Biochemistry for immediate 4°C centrifugation and freezing of plasma), and consult the Endocrine team.

OBSOLETE

10.10 Hyponatraemia

10.10.1 Symptoms - likely if sodium is 125 mmol/l or less.

- Weakness, lassitude, headache, nausea.
- Confusion, convulsions, coma.
- Some patients may have no symptoms.

10.10.2 Causes

- These are many and varied *(see Table on page 102)*.
- Remember to consider factitious causes:
  - Laboratory error - check anion gap and calculate osmolarity.

- Drip arm specimen.
- Pseudohyponatraemia (hyperlipidaemia or hyperproteinaemia). You may need to get a direct reading of Na (contact biochemistry).

10.10.3 Approach to Hyponatraemia

Always try to assess whether the patient is **volume deficient, normal or volume expanded.** A good history from the patient [or the spouse etc] is important in assessing the likelihood of plasma volume depletion [e.g. history of poor salt intake, nausea, vomiting and diarrhoea].

Table 17: Guide to Assessment of Plasma Volume Status in Hyponatraemia

	Volume Deficient	Normal or Volume Expanded
History	• Renal or GI losses • Burns • Third space losses • Diuretic use • Cortisol deficiency • Cerebral salt wasting • History of heart failure • Cirrhosis	• Excess water ingestion • Potomania (excess beer drinking) • Recent surgery / trauma / pain • Hypothyroidism • Renal failure • SIADH – pulmonary – neoplastic – drugs - DDAVP, oxytocin, tricyclics, vincristine, NSAID's, carbamazepine, ACE inhibitors
Examination	Volume contraction with low JVP and postural hypotension, or signs of congestive heart failure or cirrhosis	Normovolaemic clinically. No postural BP fall. JVP not low. Sometimes subcutaneous tissues feel "spongy".
Laboratory	• Hypo-osmolar plasma • Hypo or hyperosmolar urine • Urine Na <20 mmol/L **NOT** - if recent diuretics - if tubular disorders - if cortisol deficient • Normal or raised uric acid, urea, creatinine	• Hypo-osmolar plasma • Hyperosmolar urine • Urine Na >20 mmol/L **NOT** - if water restricted • Reduced uric acid, urea, creatinine

Note: Congestive heart failure and cirrhosis etc are considered in the volume deficient category as the "effective arterial blood volume", a marker of renal perfusion pressure, is reduced causing altered renal handling of sodium and water.

OBSOLETE

10.10.4 Management

- Withdraw inappropriate drugs.
- Exclude deficiencies of thyroid or adrenal function.
- Whatever the cause, some treatment or monitoring is needed if plasma Na <130 mmol/L.
- If **volume deficient** give 0.9S IV provided CHF/cirrhosis excluded.
- If **not volume deficient**, main treatment is water restriction (500-1000 ml/day; allow water intake equal to urine output). Ensure adequate Na and K intake (IV saline may be needed especially if plasma sodium <120 mmol/L).
- In all cases aim to restore plasma Na to 125 mmol/L but monitor levels so that the rise is not more than 8 mmol/L per day. Central pontine myelinolysis has been associated with rapid correction of plasma Na, therefore correct slowly, especially if hyponatraemia is likely to have been chronic.
- Severe hyponatraemia may be life threatening (e.g. coma or convulsions) and may require hypertonic saline - consult before use.
- Investigate and treat underlying cause.

OBSOLETE

10.11 Hypercalcaemia

If marked (>3.5 mmol/L), this is a medical emergency – usually symptomatic if calcium is >3 mmol/L.

10.11.1 Causes

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

10.11.2 Symptoms

May be none. Nausea, vomiting, constipation, abdominal pain, thirst, polyuria, confusion, coma.

10.11.3 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, alb, 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.
- Parathyroid hormone [PTH] levels (contact Endocrine Laboratory). Draw samples for PTH **and** calcium before giving hypocalcaemic drugs.
- 25OH Vitamin D (contact steroid lab).
- 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 if alkaline phosphatase is raised, (best site for hyperparathyroidism). Consider bone scan.
- Ultrasound examination of kidneys + plain abdominal X-ray (calculi).
- 24 hour urine collection - calcium excretion, creatinine clearance. Ca/Cr ratio on a random urine will often suffice.
- 1-25 diOH vitamin D assay (contact Steroid Laboratory) and PTH related peptide may all be helpful, the latter if malignancy suspected. In general these more complex investigations should only be done following Endocrine consultation.

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

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Table 18: Calcium Correction Formula

Corrected calcium = observed calcium + {(40 - albumin g/L) x 0.02 mmol/L}

10.11.4 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 immobilisation, and establishing its cause. A marked elevation is a medical emergency especially if nausea and vomiting, and/or patient is volume depleted. If hypercalcaemia is causing significant symptoms and active treatment is 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 litre 0.9S 6-8 hourly and reassess at 18-24 hours. Potassium supplements 10-20 mmol KCl per 500 ml may be required.
- APD (aminopropylidine diphosphonate).
  - 30 mg in 0.5 litre 0.9S IV over 4 hours. Ensure no extravasation occurs (irritant to tissues). Fever may occur. Plasma calcium falls progressively with nadir at 3-5 days. Repeat doses may be necessary.
- Prednisone - if sarcoidosis or vitamin D toxicity is proven, prednisone in a dose of 20- 40 mg daily may be effective.
- 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.

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10.12 Hypocalcaemia

Check albumin and if necessary adjust the calcium level (**see above**). If <2.0 mmol/L needs investigation - provided chronic renal failure is not present. Symptoms may not be prominent if problem is long standing. (Check Chvostek and Trousseau signs, and history of fits, tetany, cataracts and previous thyroid surgery).

10.12.1 Causes

- Hypoparathyroidism or resistance to parathyroid hormone.
- Renal failure.

- Vitamin D deficiency (fasting morning phosphate level may be low unless there is an associated renal impairment).
- Low magnesium states.
- Pancreatitis, rhabdomyolysis.

10.12.2 Investigations

- Fasting morning Ca, PO_4, creatinine, Mg, and parathyroid hormone levels.
- May also need to check for 25 hydroxy - Vitamin D level and assessment for osteomalacia etc. Consider malabsorption and lack of other fat soluble vitamins, (A, E, & K).
- Calcium / creatinine ratio on a random urine.

10.12.3 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. Doses of 15 mg/kg of elemental calcium over 24 hours may be needed with half of this given in the first 6 hours.
  - Consider magnesium replacement if hypomagnesaemic.
  - If the cause is hypoparathyroidism, also start 1,25 dihydroxy Vitamin D (calcitriol) e.g. 0.25 mcg/day and monitor daily serum calcium. Consult Endocrinologists.

OBSOLETE

10.13 Hypertriglyceridaemia

- Levels >10 mmol/L require immediate medical attention : the major risk is pancreatitis.

- Triglyceride levels may be as high as 50-100 mmol/L. The serum is typically lipaemic and examination of the retinal vessels reveals lipaemia retinalis. Patients may present with eruptive xanthomata.

10.13.1 Causes

- Familial syndromes/primary hyperlipidaemias.

- Alcohol.
- Diabetes Mellitus.
- Drugs (thiazides, steroids, oestrogens).
- Hypothyroidism.

10.13.2 Treatment

- Diet
  - Elimination of alcohol and refined sugars.
  - Reduction of total fat and calorie intake with weight loss.
- Drugs
  - Stop contributory drugs.
  - Treatment with omega 3 fatty acids, nicotinic acids and fibrates has been disappointing.
- Treat diabetes to normalise glucose.

10.13.3 Follow Up

- Specific dietary advice (dietician) and medical follow up should be arranged.

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10.14 Acidosis / Alkalosis

Interpretation of blood gas results

When determining the status of 'metabolic' acidosis or alkalosis allowance must be made for the influence of **respiratory** abnormalities of pH and HCO_3-. *(See page 219 - interpretation of blood gases.)*

- The observed pH and HCO_3 values can be adjusted if the $PaCO_2$ deviates significantly from 40 but in practice this is rarely helpful.

A diagnosis of **respiratory acid/base disturbance** reflecting abnormal pulmonary function should be investigated and managed as discussed in the respiratory section.

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 advanced renal failure.

10.14.1 Causes of Metabolic Acidosis

Table 19: Calculate Anion Gap

Anion Gap = $[K^+] + [Na^+] - [Cl^-] - [HCO^{3-}]$
Normal = 8-16 mmol/L

Increased anion gap

- Increased acid production:
  - Ketoacidosis
    - diabetes.
    - starvation.
    - alcoholism.
  - Lactic acidosis
    - respiratory/circulatory failure (including anaemia, carbon monoxide, shock).
    - neoplastic disease.
    - liver failure (decreased metabolism of lactate).
    - drugs/toxins.
  - Poisoning
    - salicylate, ethylene glycol, methanol.
- Renal failure.
- Hyperchloraemia e.g. excess 0.9S infusion.

Normal anion gap (hyperchloraemia increase matches bicarbonate decrease)

- Renal tubular dysfunction:
  - Renal tubular acidosis.
  - Hypoaldosteronism.
  - Potassium-sparing diuretics.
- Loss of alkali:
  - Diarrhoea.
  - Ureterosigmoidostomy.
  - Carbonic anhydrase inhibitors (acetazolamide).
- Acid intake:
  - Ammonium chloride, cationic amino acids.

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10.14.2 Investigations

- Na, K, Ca, PO_4, creatinine, urea, chloride.
- Arterial blood gases.
- Toxicology - as appropriate.
- Ketones if indicated.
- Lactate.

10.14.3 Treatment

- **Treat underlying cause.**
- Recent evidence suggests that in most forms of metabolic acidosis, the use of $NaHCO_3$ offers no benefit and may even be harmful.
- 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.

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10.14.4 Causes of Metabolic Alkalosis

- Volume deficit - Na conservation is coupled to HCO_3 reabsorption and therefore metabolic alkalosis is sustained.
  - Vomiting, gastric suction.
  - Diuretics (not acetazolamide, K-sparing).
- Mineralocorticoid excess:
  - Cushing's Syndrome.
  - Primary hyperaldosteronism (Conn's Syndrome).
  - Bartter's syndrome (decreased NaCl absorption in kidney leading to increased renin/aldosterone).
- Severe K depletion.
- Milk-alkali syndrome - chronic excess soluble calcium salts plus alkali cause a nephropathy which impairs HCO_3 excretion.
- Post-hypercapnic (one of the most common in general medical setting).
  - High bicarbonate due to metabolic compensation of respiratory acidosis. When ventilation improves an alkalosis may result which will resolve spontaneously as long as patient is not volume deficient.
- Gastric outlet obstruction.

10.14.5 Investigations

- ABG.
- Na, K, Cl, Creatinine.
- Urine:
  - Chloride low in volume depletion (<10 mmol/L).

10.14.6 Treatment

- Treat the underlying cause.

OBSOLETE

11. Gastroenterology

Main Office **Investigative Unit** ☎ 80920 / 80965 Fax 80419
4th Floor, Riverside

Inpatient Services Ward 29 ☎ 89290

- Dr B. Cook
- Dr B. Chapman
- Dr M. Burt
- Dr M. Barclay
- Dr A Ross

Consultation and On-call Service

Liver and GI tract disorders. 24 hour a day, seven days a week. Contact Gastroenterologist through the Operator.

Gastrointestinal Investigative Unit

Diagnostic and therapeutic upper GI endoscopy, colonoscopy & ERCP, gastrostomy tube placement, oesophageal stent insertion, motility investigations (oesophageal, ano-rectal, biliary), oesophageal pH studies.

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11.1 Haematemesis

11.1.1 Causes

- Ulceration/oesophagitis.
- Upper GI tract 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.

11.1.2 Management

Resuscitation takes precedence over diagnostic investigations. Gastroscopy should normally be performed within the first 24 hours. Early consultation, if therapeutic procedures such as injection of bleeding ulcers, sclerotherapy, or banding are likely to be required. A patient who continues to bleed heavily may require immediate surgery without other investigation unless varices suspected.

- Assess degree of blood loss *(see Section on Shock page 21)*:
  - History often unreliable.

- Useful signs include:
  - Resting tachycardia.
  - Hypotension.
  - Postural drop >15 mmHg.

- Stabilize patient and monitor:
  - Give 0.9S IV, then blood when available.
  - Use Group O Rh negative blood in an emergency.
- Initial investigations:
  - Crossmatch 6 units of resuspended red cells.
  - CBC and diff.
  - Coagulation profile.
  - Na, K, creatinine

Urgent surgical consultation if:

- More than 3 units of blood need to be transfused.
- Continuing or prolonged bleeding.
- Perforation suspected.

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Gastroenterology consultation

- Urgent consultation in all patients over 60 as they tolerate bleeding poorly. Endoscopic therapy may improve outcome in this group.
- Gastroscopy should be considered and done urgently if varices are suspected as they may require injection or ligation. Otherwise it should be done within 24 hours.

11.1.3 Therapy

- **Varices:**
  - Urgent sclerotherapy or variceal ligation.
  - IV infusion of octreotide using a 50 mcg bolus followed by 50 mcg/hr in 0.9S or D5W for up to 48 hours.
  - Sengstaken-Blakemore or Linton tube and transfer to ICU. (Consider endotracheal intubation first to reduce the risk of aspiration if level of consciousness is impaired.)
- **Acute stress ulceration**:
  - Liquid antacids (30 ml Aludrox PO 1-2 hourly).
  - IV infusion of H_2 receptor blockers (e.g. ranitidine 25 mg/hr for 2 hours

and repeat 6-8 hourly).

- **Peptic ulceration**
- **Acute bleeding** from a peptic ulcer. Omeprazole 40 mg 6 hourly IV or PO has been shown to reduce further bleeding episodes.
- Eradication therapy for **Helicobacter pylori** when this has been identified. The following is recommended:
  - **Either** omeprazole 20 mg BD. Amoxycillin 1 g BD. Clarithromycin 500 mg BD for 7 days. If penicillin allergy substitute metronidazole for amoxycillin
  - **Or** Helidac [combined pack] for 14 days. This consists of De-Nol, metronidazole and tetracycline.

Other regimens are available for treatment failures. Consult Gastroenterology.

11.2 Vomiting

11.2.1 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.

11.2.2 Complications

- Aspiration pneumonia.
- Haematemesis (Mallory Weiss tear).
- Oesophageal perforation (pain is a prominent feature).
- Malnutrition/dehydration.
- Electrolyte/volume depletion.

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- Hypochloraemic alkalosis.

11.2.3 Treatment

Determine and treat the **underlying cause**. If antiemetics are indicated:

- Dopamine antagonists:
  - Metoclopramide 10 mg q8h PO, IM, IV, PR but higher doses may be required.
  - Domperidone 10-20 mg QID PO.
- Peripheral Cholinergics:
  - Cisapride 10 mg PO TID.
- Phenothiazines:
  - Prochlorperazine 12.5 mg q8h PO, IM, PR.
- Anticholinergics:
  - Cyclizine 50 mg q8h PO, IM, IV.
- Sedatives and hypnotics may be used.
- Ondansetron (for approved indications).

Note: for vomiting in malignancy - *see page 182.*

OBSOLETE

11.3 Acute Diarrhoea

(e.g. less than 2 weeks)

11.3.1 History

- Try to assess whether this has an infectious basis.
- Initial history is important. Include severity of diarrhoea, fever, 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. Ask about similar symptoms in relatives or friends.

11.3.2 Examination

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

11.3.3 Investigations

- An urgent erect and supine 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	Parasites (microsporidia, 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. Giardia antigen - request specifically for this antigen if required. Fresh specimen needed.

11.3.4 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 infective 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 page 267).*
  - 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 7-10 days. Alternatives - vancomycin 125 mg PO QID, or cholestyramine 9 g PO in divided doses 10-14 days.
  - 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 intracellulare, Kaposi's sarcoma, lymphoma etc. *(see Section on HIV and AIDS on page 137).*
  - Amoebic dysentery - metronidazole 800 mg PO, TID for 10 days.

OBSOLETE

- **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 Mesalazine commencing at 2-3 g 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.

OBSOLETE

11.4 Constipation

11.4.1 General Measures

- PR examination (a plain abdominal x-ray may be required).
- Look for treatable causes - pregnancy, cancer, hypothyroidism, hypercalcaemia.
- Avoid constipating drugs (e.g. codeine, opiates, tricyclics, anticholinergics, calcium channel blockers, aluminium hydroxide).
- Dietary control e.g. increase fluid, fibre, fruit.

11.4.2 Specific Measures

- Increase fluid intake.
- Bulking agents (e.g. metamucil, granocol, isogel). If no response then consider:
  - Lactulose has an osmotic effect but may cause excess flatulence.
  - 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.
  - Bowel washout with Golytely may be needed.
  - Glycerine suppositories/manual evacuation for faecal impaction.

11.5 Jaundice

- If bilirubin unconjugated consider Gilbert's or haemolysis.
- If bilirubin increase is both conjugated and unconjugated - liver disease, cholestasis.

11.5.1 Obstructive Jaundice (Cholestasis)

- Ultrasound is investigation of choice to exclude duct dilatation.
- Check coagulation and if necessary correct with vitamin K.
- If intrahepatic cholestasis, i.e. no duct dilatation, consider drug toxicity, primary biliary cirrhosis, or primary sclerosing cholangitis.
- If extra hepatic cholestasis (dilated ducts), consider common bile duct stones, stricture and tumors. Appropriate investigations would include endoscopic retrograde cholangio pancreatography (ERCP), magnetic resonance cholangio pancreatography.
  Consult a surgeon.

OBSOLETE

11.5.2 Hepatic Jaundice

- Infectious causes - hepatitis A, B, C, Delta, EBV, CMV, other viruses including Hepatitis E.
- Acute alcoholic hepatitis.
- Chronic liver disease - alcohol, autoimmune hepatitis, primary biliary cirrhosis, sclerosing cholangitis, Wilson's Disease.
- Drugs, toxins.

11.6 Ascites

In general ascitic fluid should be tested for the following

- WBC and differential.
- Albumin.
- Culture - fluid placed in blood culture bottles.
- Amylase.
- Cytology.
- Request TB culture if this infection is suspected.

The serum albumin/ascitic gradient [the serum albumin minus the albumin level in the ascitic fluid] is very useful. If >11 this makes portal hypertension the likely cause.

Spontaneous bacterial peritonitis is likely with a white count of >500 x 10^6/L with neutrophils predominant. The initial treatment for proven or suspected bacterial peritonitis is cefotaxime 1 g q6h IV.

Management of ascites should consist of a low salt diet, spironolactone 50-200 mg daily with or without frusemide and the removal of ascitic fluid by peritoneal tap, if necessary combined with IV albumin infusion. Give 8 g albumin for every litre of ascitic fluid removed.

11.7 Liver Failure

Where this is suspected commence treatment early.

11.7.1 Clinical and Biochemical Features

- Jaundice.
- Coagulation defects (check prothrombin and Echis ratios).
- Hypoalbuminaemia.
- Encephalopathy (confusion, apraxia, asterixis).
- Ascites.

OBSOLETE

11.7.2 Causes / Precipitants

- **Acute severe hepatic necrosis**:
  - Drugs - paracetamol.
  - Alcohol.
  - Autoimmune - submassive necrosis.
  - Fatty liver of pregnancy.
  - Viral - hepatitis B ± Delta superinfection.
  - Idiopathic.
- **Chronic liver disease with acute deterioration**:
  - GI haemorrhage.
  - Sepsis (especially Gram-ve).
  - Spontaneous bacterial peritonitis **[see above under ascitic fluid],** consider cefotaxime 1g q6h IV until culture available.
  - Drugs (especially alcohol, benzodiazepines).
  - Electrolyte disturbance and volume depletion (diuretics, hypokalaemia).
  - Hepatocellular carcinoma.

11.7.3 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.
- If cause not obvious consider smooth muscle and antinuclear antibodies.

11.7.4 Treatment

- Treat any underlying cause (e.g. bleeding varices, sepsis).
- Stop all offending drugs.
- Correct hypokalaemia, hypotension, hypoglycaemia.
- If ascites present aspirate for diagnostic purposes **[see above]**.
- Correct coagulation defects with vitamin K 10 mg IV slowly and fresh frozen plasma as indicated.

11.7.5 If encephalopathysuspected

- Give high carbohydrate/low protein diet.
- Gut sterilisation with neomycin 1 g q4h PO
- Purge with lactulose 10-30 ml TID adjusted to produce three loose stool per day.
- Watch for alcohol withdrawal *(see page 50)*.
- Consult gastroenterologist promptly.

OBSOLETE

11.8 Acute Pancreatitis

11.8.1 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.

11.8.2 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.

11.8.3 Aetiology

- Biliary tract disease (especially gallstones).
- Alcohol.
- Idiopathic.
- Drugs.
- Types I and V hyperlipidaemia.

11.8.4 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.
- CXR.

OBSOLETE

11.8.5 Management

- Treatment of shock *(see page 21).*
- 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.

The following are associated with a poor prognosis:

Table 20: Prognostic Factors in Acute Pancreatitis

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

11.9 Acute Hepatocellular Dysfunction (Hepatitis)

11.9.1 History

- Ask about recent medicines and other drug and alcohol history, IV drug use, previous Hepatitis, blood transfusions, tattoos, and recent overseas travel.

11.9.2 Investigations

- U/S scan of the liver and biliary tract.
- INR, and Echis ratio if INR prolonged.
- Tissue auto antibodies, including ANA. Serum protein electrophoresis.
- IgM anti Hepatitis A serology.
- HbsAg.
- Anti Hepatitis C antibody.
- Hepatitis C PCR if indicated.
- EBV & CMV serology in selected cases.

OBSOLETE

12. Haematology

Main Office Ground Floor, Laboratories ☎ 80300 Fax 81067

Consultants

- Dr David Heaton
- Dr Ruth Spearing
- Dr Steve Gibbons
- Dr Nigel Patton
- Dr Peter Ganly

Inpatient Services

Clinical Haematology Unit, (BMTU) Lower ground Floor, Riverside Block

Consultation and On-call Service

24 hours per day, seven days per week. Fax referrals to 80750. Please also put in internal mail. To speak directly to the consultant, phone 80300 (**not switchboard**) and ask for the 'consult consultant'. Registrars available on Pagers 314, 316 and 507. Please make it clear whether a full consultation is required or just a bone marrow examination.

Consultation Guidelines

Cytopenias, bleeding or thrombotic problems, haematological malignancies including lymphoma and myeloma.

Other Services

Haematology Laboratory and ask for laboratory consultant or registrar. ☎ 80300

OBSOLETE

12.1 Management of Haemorrhagic Disorders

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

12.1.1 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 defects which may result in abnormal bleeding. Consultation with the Coagulation Laboratory is strongly recommended (Ext: 80374).

12.1.2 Treatment

- This is entirely dependent on the results of the initial tests obtained. If a severe **thrombocytopenia** ($<20 \times 10^9$/L) is present then this constitutes a medical emergency. An accurate diagnosis is necessary and this will often require bone marrow examination. These patients may need platelet transfusions.
- 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 **haemophilia A** life threatening bleeding requires immediate Factor VIII infusions, with concentrated freeze-dried preparations. A rough guide is given by the following formula.

Table 21: Factor VIII Infusion

Units of Factor VIII required	=	$\frac{\text{weight in kilograms} \times \% \text{ rise desired}}{2}$
Currently each CSL ampoule contains 250U. Recombinant Factor VIII (Kogenate or Recombinate) is also available		

OBSOLETE

- 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 CSL 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 IV over 20 minutes. If given subcut, DDAVP still needs to be given slowly over 15 – 20 minutes in a volume of 10 – 20mL.
- 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.

12.2 Management of Severe Anaemia

The following investigations are suggested for anaemia 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, and bone marrow infiltration.

12.2.1 Investigations

- CBC + film and reticulocyte count.
- MCV <80 fl - probable iron deficiency. Request iron, transferrin and ferritin. Check CRP, as an anflammatory anaemia may be present.
- MCV >100 fl - could merely reflect an increased reticulocyte count (haemolysis/blood loss). If retics normal do B_{12} and folate levels. Consider B_{12} and folate deficiencies, alcoholism, liver disease.
- 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.

OBSOLETE

12.2.2 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 ferrogradumet 325mg PO daily, 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 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).

12.3 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.

12.3.1 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 should be given immediately together with intravenous hydration. If the site of infection is obvious this will influence the choice of antibiotics (see Septicaemia page 100). Do not await the results of cultures in this situation. Suitable dosages for the above antibiotics in this situation are:
  - **Gentamicin** - use 24 hour dosage *(see page 46)*.
  - **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.

OBSOLETE

13. Health Care of the Elderly

Main Office	Princess Margaret Hospital	☎ 66808 Fax 66923
Consultants	• Dr N. Millar	• Dr T. Wilkinson
	• Dr J. Elliot	• Dr J. Kidd
	• Dr N. Gilchrist	• Dr C. Hanger
	• Prof R. Sainsbury	• Dr J. Thwaites

Health Care of the Elderly is situated at the Princess Margaret Hospital. It is responsible for the assessment, treatment and rehabilitation of elderly patients / patients with age related conditions. This is achieved through a multi-disciplinary approach with the involvement of physicians and psychogeriatricians. These patients are managed as inpatients, outpatients, through the Day Hospital or in the Community.

Consultation and On-call Service

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 the acute admitting, medical, surgical and orthopaedic wards.

Consultation Guidelines

- Specialist opinion about an elderly patient.
- Assessment of patients for transfer to the ARU, ongoing treatment in the Day Hospital or 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.
- The medical and surgical wards have a Health Care of the Elderly physician responsible to them. Please use this consultant and don't hesitate to telephone them.

OBSOLETE

13.1.1 Attitudes

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

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 diagnosis and prompt treatment.

13.1.2 History

- 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. Place of domicile, level of home supports, presence of caregiver, should be recorded. Also level of functioning with respect to ADL's, mobility and use of walking aids. Always end admission clerking with a diagnosis or problem list.
- Medications - each admission should be used as an opportunity to review patient's medications. Medications that are no longer necessary should be stopped. The doses and dose frequency of the other medications should be reviewed. Always consider the side effects of medications as potential causes of patient's presenting symptoms.
- As well as the standard systems examination, the specific Health Care of the Elderly checklist should be completed.

13.2 Health Care of the Elderly Check List

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- 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 -
  - Tongue.
  - Dentition and fitting of dentures.
  - Angles of the mouth for nutritional deficiencies.
- MSQ:
  - Check the standard 10 point MSQ *(see page 157).*
- Bladder and bowels:
  - Is the bladder palpable?
  - Should a continence chart be used?
  - Is vaginal examination required?
  - Is the descending colon palpable?
  - Rectal examination.
- Temperature:
  - Infection may be present with a normal temperature.
  - If you suspect that the patient is hypothermic, check rectal 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.

13.3 Altered Presentation of Illness

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

Falls, confusion, failure to manage, "gone off legs", are common presentations. These patients need meticulous examination and work up as there is often an underlying medical condition that has contributed to their decompensation.

OBSOLETE

13.4 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). *(See page 213)*

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 *(see page 157)*.
- 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.

- 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, which usually 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.

13.5 Incontinence Problems

Urinary Incontinence

- Attempt to ascertain whether the patient has stress or urge incontinence.
- Check the medication list to see whether drugs are contributing.
- Check MSU.
- Abdominal, rectal " vaginal 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.

OBSOLETE

Faecal Incontinence

- Faecal impaction with overflow is the leading cause in the elderly.
- Abdominal and rectal examination (± abdominal x-ray) to exclude faecal impaction, painful rectal and anal conditions.
- Check the medication list for offending drugs.
- 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.

14. Infectious Diseases

Main Office 3rd Floor, Riverside ☎ 80951 Fax 80952

Inpatient Services Ward 30

- Associate Professor Steve Chambers
- Dr Alan Pithie
- Registrar

Consultation and On-call Service

A service is available on a restricted basis seven days a week. Contact the Infectious Diseases Physician through the operator.

Consultation Guidelines

Any patient with sepsis from viral, bacterial, fungal or parasitic causes, HIV/AIDS, atypical infections including tuberculosis. Advice on antibiotics and arranging microbiological testing. Travel related infections. Investigation of pyrexia of unknown origin.

OBSOLETE

14.1 Meningitis

14.1.1 Clinical Features

- Fever, headache, photophobia, neck stiffness and impaired sensorium. The latter may be the only sign in the elderly.

14.1.2 Causes

- N. meningitidis; S. pneumoniae; H. influenzae (usually paediatric). Listeria monocytogenes (immunosuppressed, elderly 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.

14.1.3 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.
- CSF shunts.

14.1.4 Investigations

- Blood cultures - 2 sets before antibiotics given.
- Lumbar puncture. Caution - see below before doing a lumbar puncture. Collect 2 mL of CSF into each of three numbered vials. Send to Microbiology. Routine tests done are cell counts, glucose, protein, culture and gram stain. Antigen detection tests and viral culture should be done if WBC count is $>5 \times 10^6$ cells/L of CSF.
- 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 tests needed for cryptococcus, TB, amoeba, viruses - consult Microbiologists, if indicated.

OBSOLETE

14.1.5 Management

- Lumbar puncture *(see page 38 for advice on lumbar puncture technique).* In patients with clinical evidence of raised intracranial pressure (raised BP, decreased pulse, decreased level of consciousness), seizures, papilloedema, focal neurological signs, sinus or ear infections - obtain CT/MRI head scan urgently before doing lumbar puncture.
  Note: If a lumbar puncture can't be done immediately make sure that appropriate antibiotics are given at once. If antibiotics were given before the patient reached hospital make sure the correct dose and type of antibiotic was used and if necessary give supplementary doses.
- **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. Transfer patient to ICU.
- **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.

Table 22: Usual CSF Patterns in Meningitis

	Pyogenic	Tuberculous	Aseptic
Predominant Cells	Neutrophils	Mononuclear	Mononuclear
Numbers of WBC	>1000x10^6/L	10-350x10^6/L	50-1500x10^6/L
Glucose	< 2/3 plasma	< 2/3 plasma	> 2/3 plasma
Protein	>1.0 g/L	>1.5 g/L	<1.5 g/L

14.1.6 Antibiotic Therapy

The spread of pneumococcal strains which are resistant to penicillin and ceftriaxone has lead to changes in recommendations for empiric treatment of meningitis in some centres. The recommendations given here may need to change, depending on the local prevalence of these organisms.

OBSOLETE

- Proven or presumed pyogenic menginitis:
  - These recommendations apply to all patients over 15 years. The same treatment is given to previously well patients, and to those with complicating pre-existing illness, such as ear or sinus disease, immunosuppresion, or recent pregnancy.
  - **Treatment as soon as CSF taken** (if not already started):
    - ceftriaxone 2g IV q12h **and** amoxycillin 300mg/kg/day, up to 12 gm IV daily in 4-6 divided doses.
    - If Gram stain or antigen testing is suggestive of pneumococcal disease, then add vancomycin 1 gm IV q12h over 90 minutes via a pump. **Consult Infectious Diseases Service.**
    - If severely penicillin or cephalosporin allergic chloramphenicol 80-100 mg/kg IV/day up to 4 g/daily in 4 divided doses should be given.
    - When cultures are available, modify the treatment according to the organisms isolated. Intravenous benzylpenicillin is the preferred treatment if the organism is sensitive.
  - **Steroids**
    No clear benefit has been demonstrated **in adults** with meningitis due to Stretococcus pneumoniae or N. meningitidis. Because of the increasing evidence of pneumococci with reduced sensitivity to penicillin, steroids

are not recommended as they decrease penetration of antibiotics into the CSF.

- Meningococci must be cleared from the nasopharynx before the end of the treatment period. Give rifampicin 600mg BD PO for 2 days or ciprofloxacin 500 mg as a single oral dose. This is not necessary if the patient has received ceftriaxone therapy.
- Close household contacts of patients with meningococcal meningitis should be given rifampicin prophylaxis as above as soon as diagnosis made. Throat swab not necessary. If pregnant give ceftriaxone 250 mg single dose IM or IV.
- Notify MOH if applicable – *See page 267.*
- Under 15 years seek paediatric advice.
- **Tuberculous meningitis:**
  - PCR for M.tuberculosis is available in some Centres. 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 (or gastric washings), EMU x3 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 25 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 clinical state. Check HIV status.
  - Consult Infectious Diseases Service.
- **Aseptic meningitis:**
  - Most often due to viruses - mumps, and enteroviruses (PCR test available). 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 (PCR test available), leptospirosis, neoplasia, drugs, cyst related, Mollaret's, SLE, Bechet's, sarcoidosis, and others. Accurate cytology essential.
  - Consult Infectious Diseases Service.

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14.2 Septicaemia

This is life threatening. 30-50% of patients will die despite appropriate therapy. Early diagnosis and treatment are vital. Those who are apparently well may deteriorate rapidly. Patients are usually toxic and febrile. The patient may be in shock or just look unwell. Those with chronic renal failure or advanced age may have no fever or be hypothermic. Systemic steroids may mask the symptoms and signs. The various stages that patients with severe sepsis may go through has recently been defined (see Harrisons's "Principles

of Internal Medicine" 14th Edition 1998 p776). The systemic inflammatory response syndrome (SIRS) is defined as being present when two or more of the following criteria are met:

Temperature >38°C or <36°C.

Heart rate >90/min.

Respiratory rate >20/min or a $PaCO_2$ of <32 mm Hg.

WBC >12 x 10^9/L or <4 x 10^9/L or the presence of > 0.1 x 10^9/L immature neutrophils.

These criteria need to be interpreted in the context of the individual patient and any co-morbidities that may be present.

Prompt treatment with broad spectrum antibiotics is vital if the patient satisfies the above criteria for SIRS even though a definite diagnosis of bacterial sepsis has not been established. Make sure the appropriate cultures have been taken.

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14.2.1 Clinical Situations which may predispose to Septicaemia

- IV lines (especially if there is local inflammation).
- Urinary catheters.
- Local sepsis.
- Steroid therapy.
- Advanced age and debilitation.
- Drug addiction and alcoholism.
- Diabetes mellitus.
- Chronic renal failure.
- Post surgical or obstetric procedures.
- Splenectomy.
- Malignancy - leukaemia, myeloma etc.
- Immunosuppressive therapy - neutropenia etc.

14.2.2 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 spp.
- Multiple pulmonary infiltrates with rapid cavitation - S. aureus.

14.2.3 Investigations

- Blood cultures.
- The diagnosis is based on culturing organisms from the blood so good technique is essential.
- 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 may need to be taken at antibiotic trough times
- Using BacTAlert bottles: Wash hands and select vein. Remove and discard plastic cap of BacTAlert bottle. Clean patient's skin and rubber seal of BacTAlert bottle with an antiseptic solution and allow to dry. We recommend 1% chlorhexidine and 70% isopropyl alcohol. Avoid an iodine based solution. Draw 20 mL of blood into syringe. Do **not** change needle on syringe. Inoculate one BacTAlert set (one bottle for aerobes and one for anaerobes) with 10 mL blood into each bottle (i.e. 20 mL per set). Inoculate blood culture bottles before other blood tubes. 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

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for more than an hour.

- CBC + diff, coagulation profile for DIC screen.
- Na, K, creatinine, glucose, AST, GGT, alk. phos., Bili.
- CXR.
- Arterial blood gases.

14.2.4 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 21)*. If you are not competent to place a CVP line do not attempt it *(see page 36)*.
  - If patient remains hypotensive (systolic <80 mmHg) despite adequate hydration then dopamine infusion may be needed *(see page 59)*.
  - Fluid management in septic shock can be difficult and CVP readings may be misleading. Swan Ganz catheterisation is frequently required.
- **Oxygen therapy** *(see page 226)*.
- **Monitoring:**
  - Urine output - If patient hypotensive/shocked a catheter may be needed but avoid if possible.
  - Daily creatinine.
  - Arterial blood gases and pulse oximetry - ARDS is common and the patient who has progressive hypoxaemia may need ventilatory support.
  - Severe acidosis secondary to inadequate tissue perfusion may require partial correction *(see page 109)*.
  - 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. 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 the likely source of sepsis and the common organisms associated with sepsis from this site. The sections on endocarditis, pneumonia, urinary tract infections, meningitis, cellulitis, bone and joint infections etc, will give guidance as to which drugs to use initially.
  - In hospitalised patients organisms may have been previously isolated and

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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 + metronidazole.
  - Intra-abdominal sepsis:
    - ~ Amoxycillin + gentamicin + metronidazole.
    - ~ Ceftriaxone + metronidazole.
  - Cellulitis: Flucloxacillin *(see page 146)*.
  - Pseudomonas sepsis:
    - ~ Piperacillin + tobramycin.
    - ~ Ceftazidime + tobramycin.
  - Urinary tract:
    - ~ Gentamicin.
    - ~ Ceftriaxone.
  - Neutropenia:
    - ~ Moderate (neutrophils >0.5 x 10^9/L) gentamicin and cefuroxime.
    - ~ Severe (neutrophils <0.5 x 10^9/L) gentamicin and piperacillin.

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14.3 HIV and AIDS

Managing these patients is complicated and requires close cooperation with Infectious Diseases and Microbiology. The indications for treatment with antiviral drugs require expert advice. The infections that have been found in association with HIV constitute a huge and expanding list and are often unusual.

14.3.1 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 to be transmitted if splashed onto inflamed or broken skin or on to 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 should be worn 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 make the lesion bleed and wash with soap or detergent. Obtain a blood sample from the patient unless known to be HIV positive. Contact Microbiology immediately. Prophylactic therapy may be indicated and should be administered urgently.
- Follow protocol for needle stick injury. This is available in all wards and departments.

14.3.2 Antibody Testing

- Counselling and explanation. Consult with Infectious Diseases if you are uncertain.
- Obtain consent to test for HIV antibody.
- Tell patient of the limitations 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 letters of first name.
  - M or F (Sex).
  - Date of birth.

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14.3.3 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.

14.3.4 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. If HIV is strongly suspected, testing for viral RNA can be done. Seek advice since treatment in the acute phase may be indicated.

- 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.

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14.3.5 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.

- 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/SaO_2 - hypoxemia and desaturation (>5%) on exercise are common.
  - CXR - diffuse interstitial infiltration but CXR may be normal in up to 5% of cases.
  - Induced sputum **in a side room** as TB may also be present. Use 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 Services.

- Treatment:
  - Begin treatment for presumed PCP with co-trimoxazole when one induced sputum specimen has been 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 in drug insert e.g. 320 mg in 500 ml. Change to oral after 5 days if patient improving. Nausea is very common but often responds to prochlorperazine. Folinic acid 15 mg/day orally if platelet count $<100 \times 10^9$/L or neutrophil count $<1.5 \times 10^9$/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 daily. Hypotension is common. Hypoglycaemia unusual but unpredictable and can be intractable. Check BM stix QID.

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♦ **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**.

- 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 this is a diagnosis of exclusion. CMV, HSV, lymphoma or atypical mycobacteria should be sought.
- Meningitis - Usually Cryptococcus neoformans. Headache universal, lethargy, fatigue, fever and weight loss are 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 head scan. If this shows:

  - Diffuse atrophy then HIV encephalopathy is likely. Perform lumbar puncture.
  - Ring enhancing lesion - assume due to toxoplasmosis if seropositive and treat. Seek advice.
  - Solid lesion - consider brain biopsy urgently.
  - Low density lesions without enhancement - most likely progressive multifocal leucoencephalopathy.
- Lumbar puncture:
  - CSF culture for bacteria fungi, mycobacteria and viruses.
  - CSF for cryptococcal antigen.
  - India ink stain for cryptococcus.

  Note: CSF may have normal WBC, protein and glucose in cryptococcal meningitis.
- Blood and Urine: Cryptococcal antigen.

- Treatment - Seek advice.

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♦ **Retinitis**

Most often due to cytomegalovirus. This infection may progress to **blindness** very rapidly.

Investigations:

- Ophthalmology opinion.
- Serology for CMV titres.

Treatment:

- Begin ganciclovir 5 mg/kg IV q12h as soon as possible.

♦ **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 q24h, itraconazole 200 mg PO q24h. or fluconazole 200 mg PO bd.
- Diarrhoea can be due to many causes.
  - Initial investigations include stool samples for ova and parasites, routine bacterial culture, stains for cryptosporidia, microsporidia and mycobacterium intracellulare.
  - Treatment - appropriate for the pathogen isolated, but note that there is no satisfactory therapy for cryptosporidia.

- **Constitutional Disease**
  Systemic symptoms - fever, weight loss >10%, sweats, fatigue
  If fever is documented but no localising symptoms begin systematic search as follows.
  - 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.
  - Consider bone marrow culture and special stains, lymph node biopsy, CT scan abdomen and lumbar puncture.

All patients will need thorough work up for other STDs, and decisions made about appropriate use of antiretroviral drugs and prophylactic antibiotic regimens.

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14.4 Malaria

14.4.1 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, Papua-New Guinea and Sub Saharan Africa.

Chloroquine resistance in P.vivax has recently been reported from Papua-New Guinea and the Solomon Islands and is becoming more widespread. Consult Infectious Diseases Service early. It is reasonable to assume Chloroquin resistance in all cases of P.falciparum until proven otherwise.

14.4.2 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, but may be longer if prophlaxis has been taken..
- 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.
  - Pulmonary oedema.
  - Hypoglycaemia especially during pregnancy, in children and quinine therapy.

Note:
The fever pattern may be suggestive of malaria but often does not follow the classical pattern. Lymphadenopathy, muscle tenderness, and joint effusions DO NOT occur in malaria. Look for another cause.

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14.4.3 Investigations

- Thick and thin blood films. If negative repeat examination daily. Antigen tests are very accurate for P.falciparum and P.vivax, but the gold standard remains thick and thin blood films.
- CBC + diff and film.
- Creatinine, AST, GGT, alk. phos., Bili, glucose.
- CXR.
- Arterial blood gases.
- Blood culture.
- Urinalysis.

14.4.4 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. Quick screening tests are available. If normal then primaquine is safe. If G6PD deficient check with Infectious Diseases.
- 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.
  - Oral mefloquine may be appropriate in some patients with mild/moderate disease. Give Mefloquine 750 mg (3 x 250 mg tab) PO THEN, 500 mg after 6 hours THEN, 250 mg after 12 hours if >60 kg.
- **IV therapy**: Quinine dihydrochloride 20 mg/kg in D5W by infusion over 4 hrs. **Do not give as a bolus.** Then give 10 mg/kg over 4 hrs by IV infusion q8h until patient can swallow.
- Cerebral malaria - give phenobarbitone prophylactically 3.5 mg/kg IM stat.

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

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14.4.5 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.

14.5 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).

14.5.1 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. Respiratory manifestations may predominate.
- CNS - confusion, seizures, psychotic behaviour.
- Skin - rose spots, 2-4 mm maculopapular lesions blanch on pressure occur in a minority of patients.

14.5.2 Investigations

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

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14.5.3 Complications

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

14.5.4 Treatment

Blood cultures will be reported as positive for Gram negative bacilli. If typhoid fever is a possible diagnosis begin ceftriaxone 2 g IV q24h or ciprofloxacin 400 mg q12h IV, or 500 mg BD PO, until sensitivites are known. Aminoglycosides are ineffective.

14.6 Penicillin Allergy

- It is unwise to give penicillins to patients who have a history of definite and moderate to severe allergy to penicillin. It may be unavoidable in some situations e.g. enterococcal endocarditis 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 a true allergy. 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. Cephalosporins are usually 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.

14.7 Cellulitis and Erysipelas

- Definitions:
  - Cellulitis is an infection of the skin and soft tissues, most commonly caused by Group A Streptococci and Staphylococcus aureus.
  - Erysipelas is a form of cellulitis with rapid onset, clearly demarked margins and is almost exclusively caused by Group A Streptococci.
- Predisposing Factors:
  - Cellulitis/erysipelas may follow minor, sub-clinical skin trauma, or may arise in an area where there is dermatophyte infection, eczema, psoriasis, a traumatic or surgical wound or other break in the skin barrier.
  - Patients with venous insufficiency, oedema, lymphatic obstruction, previous cellulitis/erysipelas, diabetes, alcoholism and cerebrovascular disease have a high incidence of cellulitis and are prone to relapse.
- Investigations:
  - Blood cultures.
  - Aspirate any skin blisters or fluctuant areas and swab any skin lesions, ulcers or wounds in the area of the infection.
  - CBC, glucose, creatinine. Check immunoglobulins in older patients and those with recurrent cellulitis.
  - Consider subcutaneous aspirate or skin biopsy in patients who are immunocompromised or who are not responding to standard treatment.
- Antibiotics:
  - **Erysipelas** and cellulitis thought to be caused by Group A Streptococci can be treated by penicillin alone. Give benzylpenicillin 2 mega U IV q4-6h.
  - **Uncomplicated cellulitis of uncertain aetiology:**
    - Use flucloxacillin 1-2 g IV q6h, until defervescence of fever and improved clinical appearance, followed by flucloxacillin 0.5-1.0 g QID orally for 7-10 days.

    Note:

    Additional treatment with penicillin is not required.

    - If mild penicillin allergy (e.g. rash) then use cephazolin 1 g IV q8h.

    - If significant penicillin allergy (e.g. anaphylaxis, angioedema) then use vancomycin, erythromycin or clindamycin.

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Note: Cellulitis commonly appears to worsen in the first few days but this should not automatically lead to a change in antibiotics unless the patients overall condition (e.g. fever, pulse, BP) deteriorates.

- **Complicated cellulitis:**
  - Cellulitis associated with burns, chronic ankle or decubitus ulcers, wounds, or in patients with diabetes, vascular insufficiency or immunocompromise. These infections are often polymicrobial; predominantly Group A Streptococci and Staphylococcus aureus but Gram negative enteric bacilli and anaerobes may also be present.
  - Consider underlying osteomyelitis in those with diabetes, vascular insufficiency, or chronic ankle or decubitus ulcers.
  - Remember antitetanus prophylaxis for traumatic wounds.
  - While awaiting results of appropriate swabs aspirates or surgical debridement, the following antibiotic combinations are recommended: Augmentin 1.2 g IV q8h with or without gentamicin *(see page 46 for dosage guidelines)* **or** flucloxacillin + gentamicin + metronidazole **or** cefuroxime + metronidazole
- **Necrotising skin and soft tissue infections**:
  - If there is severe pain, and/or disproportionate systemic toxicity; or there is evidence of gangrene, necrotic change or gas formation, then the following antibiotics should be given: **high dose (20-24 mega U/day) IV benzylpenicillin + IV clindamycin + gentamicin**. If penicillin allergy, give **ceftriaxone 2 g q12h + clindamycin.**
  - Early and aggressive debridement of involved tissue is essential. Urgent surgical referral.
  - Tissue specimens sent to the Microbiology Laboratory for Gram stain and culture.
  - Consult Infectious Diseases urgently. Notify Microbiology Laboratory of incoming specimen.

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15. Nephrology

Main Office 3rd Floor, Parkside West ☎ 80655 Fax 80941
Inpatient Care Ward 14
- Dr K. Lynn
- Dr M. Searle
- Dr R. Robson

Consultation and On-call Service
24 hours a day, seven days a week. Registrar and consultant on call - contact operator. Fax consults to 80941.

Consultation Guidelines
Acute and chronic renal failure, drug-induced renal disease, urinary tract infections, renal hypertension, systemic diseases involving the kidney (including diabetic nephropathy), electrolyte disturbances

Acute Dialysis Unit 3rd Floor, Parkside West ☎ 89108 Fax 89109
Home Dialysis Training Unit 278 Antigua Street ☎ 80610

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15.1 Renal Failure - Acute

Acute renal failure (ARF) is defined by an elevation of plasma urea and creatinine concentrations. 30% of patients are not oliguric and some may be polyuric. The following are important aspects of the management of acute renal failure.

- Controlling hyperkalaemia.
- Recognition and correction of dehydration.
- Recognition and relief of urinary tract obstruction.

15.1.1 Causes

- Pre-renal:
  - Hypovolaemia or hypotension
- Renal:
  - Nephrotoxins including drugs and chemicals.
  - Acute interstitial nephritis.
  - Acute glomerulonephritis.
  - Systemic vasculitides.
  - Haemolytic - uraemic syndrome.
  - Acute-on-chronic renal failure, e.g. in patients with polycystic disease, glomerulonephritis, 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 hypertrophy or cancer.

15.1.2 Investigations

Evaluation of the state of hydration is crucial in the management of patients with renal failure. Initially 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, red cells including their morphology, white cells and casts. Urine culture, Urinary Na, K concentrations and osmolality may sometimes be helpful. Ward nursing staff 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 need checking more often.
- Urinary tract ultrasonography to exclude obstruction and calculi and to assess kidney size and echo texture. Renal area tomography may be indicated. Discuss with Radiologist.
- If you suspect Goodpasture's Syndrome or a systemic vasculitis such as Wegener's granulomatosis - a rapid serological test for anti-GBM, antiproteinase 3 and anti-myeloperoxidase antibodies is available from Immunology.
- Urgent renal biopsy may be indicated, particularly when the urine evaluation suggests an aggressive glomerulonephritis.

OBSOLETE

15.1.3 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 frusemide 40-80 mg IV and give double the dose in 2 hours if no response. If still no response, give no further frusemide and seek advice.
- Ureteric obstruction - consult Urology team urgently.
- 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.

Table 23: Treatment of Hyperkalaemia

Plasma Potassium	Treatment
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 of 50% dextrose IV plus 12 units actrapid insulin IV. Salbutamol 20 mg by nebuliser or 0.5 mg by IV infusion over 15 minutes may also be used.
>7.5 mmol/L	As above but also give 10-30 ml 10% calcium gluconate IV. Consult re immediate dialysis

- Indications for urgent dialysis:
  - Markedly raised urea and creatinine concentration – no absolute figures can be given.
  - K >7.5 mmol/L.
  - Pericarditis.
  - Cardiac failure or fluid overload.

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Note:
Care should be taken with IV line insertion - veins may be required for subsequent AV fistula formation. Where possible try to use the dominant arm and avoid forearm veins. Avoid radial and brachial artery for blood gas sampling from the non-dominant arm.

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.

Table 24: Urine Chemistry in 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 $\frac{\text{U/P Na}}{\text{U/P creatinine}} \times 100\%$	<1*	>1

* Also in acute glomerulonephritis

15.2 Renal Function and Drug Dosage

- 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 152)*. This is particularly important for drugs such as the aminoglycosides, cephalosporins, cimeidine, ranitidine, digoxin, procainamide, the ACE inhibitors and some of the β-blockers. Drugs which are metabolised extensively do not usually require dose adjustment in renal insufficiency unless an active metabolite or toxic metabolite is excreted through the kidneys.
- Some drugs should be avoided completely 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.

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The **ACE inhibitors** are used widely 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 are 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 generally be used in combination with potassium-sparing diuretics, potassium supplements, or NSAIDs. *(see page 59 for dosage recommendations)*

15.3 Renal Function – Assessment

- The plasma creatinine concentration alone is not a sufficiently accurate predictor of 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 concentration, body weight, sex and age. The formula is as follows:

Table 25: Creatinine Clearance Calculation

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

- 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.

OBSOLETE

15.4 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 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 women should be screened in each trimester for (asymptomatic) bacteriuria. Only about one-half of pregnant asymptomatic patients will have pyuria ($>10 \times 10^6$ WBC/L) indicating urinary tract inflammation. The prevalence is 5-6% in caucasian women and 15-18% in Maori/Polynesian women. These women 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.

15.4.1 Diagnosis

- The diagnosis is confirmed by culturing **either** a 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.
- Interpretation of the number of bacteria forming units/litre of urine (cfu/L) of any uropathogen needs to take into account the presence/absence of pyuria and symptoms regardless of the patient's gender.

15.4.2 Investigations

- Always consider the question - **"Is this infection a pointer to some underlying abnormality in the urinary tract?"**
- In general the indications in adult men with urinary tract infection are no different from those for women. A urine flow rate measurement may be appropriate in males with any prostatic symptoms.
- Adults with a UTI only require organ imaging of the urinary tract (usually urinary tract ultrasonography)if:
  - They had urinary tract infections/symptoms prior to the commencement of sexual activity.
  - They have acute pyelonephritis that has an atypical clinical course.
  - The infections have become closely-spaced.
  - Proteus species or an unusual organism is present.
  - Microscopic haematuria persists, or
  - Single dose therapy has failed.
- Cystoscopy should be considered for most older males and some post menopausal women. Consult if uncertain.

15.4.3 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:

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Table 26: Drug Guidelines for Cystitis

Single dose	3 day course
Trimethoprim 600 mg*	Trimethoprim 300 mg q24h
Norfloxacin 800 mg	Nitrofurantoin 50 mg q8h (ineffective for Proteus spp) Norfloxacin 400 mg q12h Amoxycillin 250 mg q8h (for enterococcus faecalis)

* Trimethoprim is just as effective as co-trimoxazole in the urinary tract and has a lower incidence of side effects.

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

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Prophylactic Treatment for Patients with Recurrent Urinary Tract Infections

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

  - 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 can be used for prophylaxis.
- Prophylactic treatment should be started only 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 for 6-12 months, although the patient may wish to continue for longer.
- Nitrofurantoin prophylaxis - 0.5% of patients treated will get a pulmonary reaction. Warn the patient to report any new respiratory symptoms.
- A prophylactic antibiotic on alternate nights, 3 nights a week or after

intercourse may be equally efficacious.

- In post menopausal women, an atrophic vaginitis should be considered and treated appropriately, e.g. intravaginal oestrogens.

15.5 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.
- 10-15% will have a bacteraemia.

15.5.1 Causes

- Acute pyelonephritis may occur in a structurally normal urinary tract (**uncomplicated**) or as a complication of some underlying urinary tract structural or functional disorder (**complicated**).

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15.5.2 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.

  It is difficult to eliminate the problem of contamination of voided urine samples, urine may be obtained 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 examinations should be done if indicated.
- CBC.
- Na, K and creatinine concentrations.
- Blood culture - 2 sets.
- Patients with acute pyelonephritis who follow an atypical course (?obstruction, kidney stone) should have an ultrasound examination of the urinary tract and possibly a plain abdominal x-ray (for kidneys, ureter,

bladder). A CT urogram is probably the best test if a urinary stone is suspected.

- A cystoscopy may very occasionally be indicated.

15.5.3 Management

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

Parenteral antimicrobial therapy usually consists of a single intravenous dose of antibiotic (e.g. gentamicin) with oral therapy possible on the second day of treatment. Treatment should be continued for 5 days.

The choices of **parenteral** agents are:

- Gentamicin *(see page 35 for dosage guidelines).*
- Ciprofloxacin 200 mg q12h.
- Ceftriaxone 2 g q24h.

Note:

- The aminoglycosides and quinolones are the drugs of choice.
- Check local sensitivity patterns for trimethoprim.
- 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. Augmentin should also be avoided because of its slow clinical response, low cure rate and high incidence of side effects.

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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 (e.g. ciprofloxacin, norfloxacin) 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 10-14 days after completion of therapy.

16. Neurology

Main Office 3rd Floor, Riverside ☎ 80940 Fax 81226

Inpatient Services These are provided by medical teams headed by:

- Assoc. Prof I. Donaldson
- Dr S. Avery
- Dr T. Anderson
- Dr P. Parkin

Consultation and On-call Service

These are provided on a 24 hour per day, seven days per week rotational basis. For consultations, fax the referral to 81226, or contact the Neurology Department (80940). Out of hours contact through operator.

Other Services

Neurophysiology Section - this is situated within the Department of Neurology and provides inpatient and outpatient EMG, nerve conduction studies, evoked potentials, EEG and other neurophysiological investigations. Routine requests for investigation should be sent directly to the Department. Requests for urgent investigation should be made through direct telephone contact via 80940.

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16.1 Neurological Examination

Some points to remember are:

Dilatation of the pupils by mydriatic drops should be avoided in neurology patients, particularly those who are ill.

A quick routine test of mental function such as the Mental Status Quotient, may be useful in the elderly but is not sensitive enough in most younger patients. If there is any doubt about mentation in this latter group, more specific tests of mental function will be needed, including tests for dysphasia, dysgraphia and the like.

Mental Status Quotient (MSQ)

- 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.

Reflexes

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

Biceps jerk	C5, C6 (Musculocutaneous nerve)
Brachioradialis reflex	C5, C6 (Radial nerve)
Triceps jerk	C7, C8 (Radial nerve)
Knee jerk	L2, L3, L4 (Femoral nerve)
Ankle jerk	S1, S2 (Tibial nerve)

Segmental Innervation

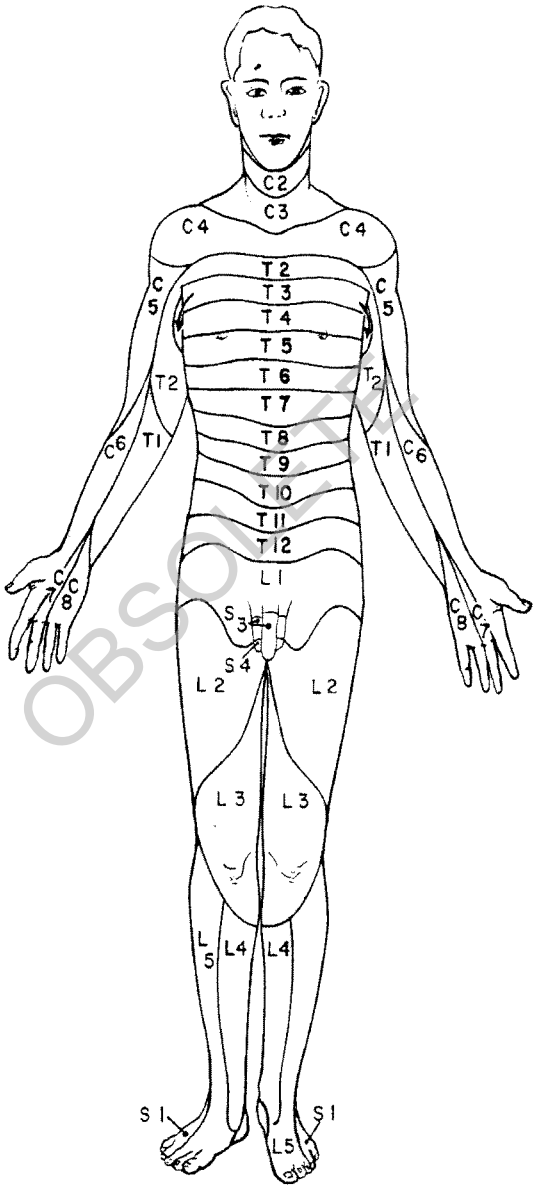
- The segmental innervation of the skin is illustrated overleaf. This can be more readily recalled by remembering certain "key" dermatomal levels e.g.:

C5	Skin over deltoid muscle	T10	Umbilicus
C6	Thumb	L1	Groin
C7	Middle finger	L3	Knee
C8	Little finger	L5	Anterolateral calf and dorsum of foot.
		S1	Lateral foot and little toe

OBSOLETE

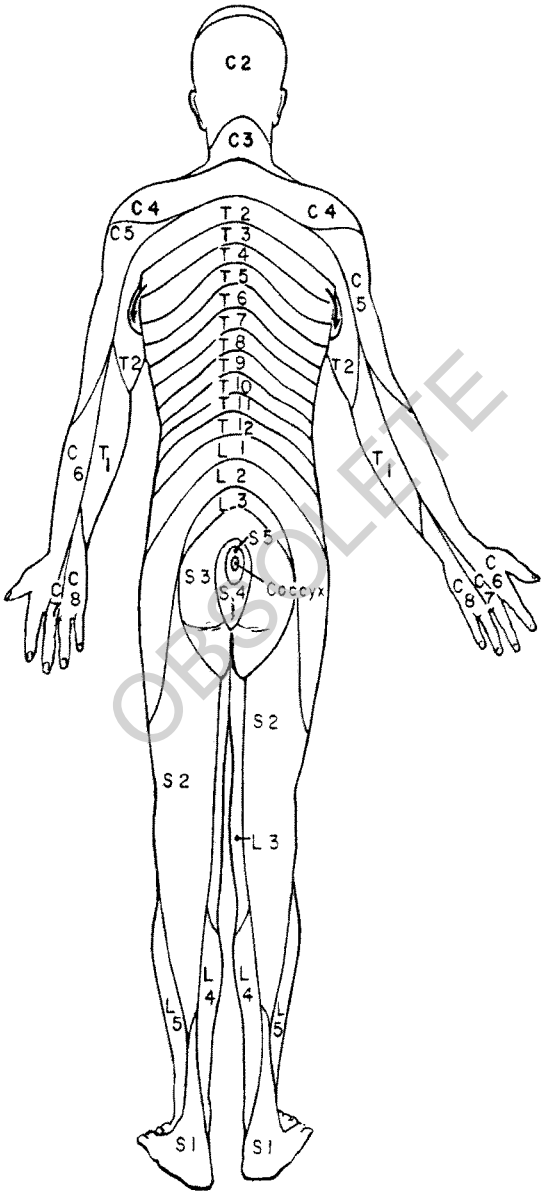
16.1.1 Dermatome Distribution

Figure 2: Dermatomes - anterior



OBSOLETE

Figure 3: Dermatomes – posterior



OBSOLETE

16.2 Stroke

16.2.1 Causes

- **Infarction (85%)**:
  - Thrombotic.
  - Artery - artery embolism (from atherosclerotic plaque).
  - Cardioembolic (atrial fibrillation is the most common).
  - Lacunar (common in chronic hypertension).
- **Haemorrhage (15%)**:
  - Hypertension, coagulation disorders, haemorrhagic infarction, A-V malformation, berry aneurysm.

Note:
Carotid or vertebral artery dissection may cause cerebral ischaemia. Suspect in younger patient and in those with retro-orbital or neck pain.

16.2.2 Investigations

- Include:
  - CBC (polycythaemia, thrombocytosis).
  - ESR (arteritis).
  - Na, glucose, Ca, albumin, creatinine, lipids.
  - Chest X-ray.
  - ECG.
  - CT head scan.

May include

- Syphilis serology.
- ANA.
- Coagulation profile. In younger patients consider investigations for a prothrombotic state, either congenital (Factor V Leiden; hyperhomocysteinaemia) or acquired (antiphospholipid antibodies.) Seek advice with regard to the exact deficiency states to test for.
- Echocardiogram (transthoracic or transoesophageal).
- Duplex carotid ultrasonography.
- Angiography.

Note: Cerebral haemorrhage cannot usually be distinguished from cerebral infarction on clinical grounds. A CT head scan must be obtained to exclude haemorrhage before anticoagulation.

16.2.3 Management

- **Thrombotic or artery-artery embolic stroke**:
  - Completed stroke.
    - Aspirin 300 mg daily.

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- Stroke in evolution. (Exclude other causes, e.g. tumour, subdural haematoma.)
  - Anticoagulation - heparin.

♦ **TIA's** - a neurological deficit which resolves completely within 24 hours. Serial hemispheric TIAs must be investigated urgently.
- Treat hypertension if moderate or severe. Avoid large/sudden changes in blood pressure.
- Aspirin 300 mg daily.
- Vertebrobasilar territory - exclude subclavian steal. Take blood pressure in both arms.
- Carotid territory - semi-urgent carotid ultrasonography if patient potentially suitable for endarterectomy, with incomplete residual deficit. A "complete" occlusion on ultrasound may be due to an almost complete stenosis and should still be considered for angiography.

♦ **Cardioembolic stroke**:
- Anticoagulation (heparin followed by warfarin) unless contraindicated or the infarct is large.
- If infarct is large, a delay in anticoagulation for 2-10 days may be indicated (consult neurologist), as the exact risks of re-embolism versus bleeding into the infarct are uncertain.

♦ **Cerebral Haemorrhage:**
- Stop any anticoagulants, including aspirin.
- If life threatening, surgical evacuation of haematoma should be considered (especially if superficial intracerebral or intracerebellar) – consult a neurosurgeon urgently.

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16.3 Subarachnoid Haemorrhage

16.3.1 Causes

♦ Intracranial aneurysm. – 80 %.

♦ "No cause found" - usually associated with systemic hypertension and with negative intracranial angiography. – 14 %.

♦ Intracranial arterio-venous malformation. (AVM/angioma) – 5 %.

♦ Haemorrhage from intracranial tumour, coagulation disorder (usually iatrogenic) – 1 %.

Mortality

♦ Mortality from the first bleed of an intracranial aneurysm is approximately 30%.

♦ Mortality of an early rebleed is at least 40%.

♦ 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.

16.3.2 Specific Investigations

- CT head scan as soon as possible. CT scan within 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.
- If CT scan is negative for presence of blood and there is no evidence of an intracranial mass lesion, then a diagnostic lumbar puncture should be performed.

  Note: Lumbar puncture is **contra-indicated** if the patient has an impaired conscious level, or has significant lateralizing neurological signs. In such patients CT scan **must be obtained** as likelihood of intracerebral haematoma is high and a lumbar puncture could prove fatal.

  Remember: There are only three ways to diagnose a subarachnoid haemorrhage: lumbar puncture, cranial imaging (CT or MRI scan) or post mortem.
- 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.

OBSOLETE

16.3.3 Treatment Guidelines

- Complete bedrest.
- Adequate analgesia (paracetamol, narcotics e.g. morphine 5-7.5 mg IM).
- Intravenous fluids to ensure adequate hydration – with a minimum of 2 litres of IV fluids per day assuming that there is also a normal oral intake, and no increased risk of fluid overload.
- Start nimodipine to reduce risk of ischaemic stroke due to arterial spasm. Dose is 5 mL/hr (1 mg/hr) for 2 hours, then increase slowly to 10 mL/hr (2 mg/hr) over several hours if there is no associated hypotension.
  IV nimodipine is usually administered for 14 days after initial haemorrhage. After 14 days intravenous administration can be changed to oral treatment (60mg q4h). Oral treatment is usually continued for a further 7 days.
- Prevent vomiting – antiemetics. Avoid straining. Stool softeners if needed.
- Raised blood pressure should only be treated if:
  - 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.

- The patient was already on anti-hypertensive drugs before the haemorrhage. Continue the current therapy but beware of hypotension which may occur in conjunction with the intravenous nimodipine – such hypotension may seriously impair cerebral blood flow.

16.3.4 Christchurch Hospital Admission Arrangements

- Patients in whom there is a strong clinical suspicion of subarachnoid haemorrhage (SAH) should be admitted to Neurology or Neurosurgery.

16.4 Status Epilepticus

16.4.1 Definition

- Prolonged seizures or failure to regain consciousness between seizures.
- Up to one-third of cases may have non-convulsive status and manifest clinically as acute onset confusion or delirium.

16.4.2 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 or benzodiazepine withdrawal, hypoglycaemia and infection.

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16.4.3 Investigations

- CBC and diff.
- Na, K, glucose, LFT, Ca, toxicology.
- Anticonvulsant concentrations.
- Arterial blood gases.
- ECG.
- EEG if there is not a prompt response to treatment, or cause uncertain.

16.4.4 Management

- Ensure adequate airway and oxygenation - monitor. Nurse in lateral position with an oral airway. Respiration is impaired by continued seizures, and by many anticonvulsants. If status is not stopped promptly (i.e. within minutes) patient should be transferred to ICU for intubation.
- Insert IV line and take bloods (above). Give diazepam 2 mg/min until seizure stops or 20 mg max. May repeat 20 mg once more but stop if respiratory depression occurs.
- If no past history of epilepsy consider thiamine 100 mg IM followed by an IV bolus of 50 ml 50% dextrose.

- If patient is not already receiving anticonvulsants start phenytoin.
  Place 11 mg/kg phenytoin In 100 mL 0.9S and administer at a rate no faster than 25 mg per minute.
  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.
  Be aware that the oral dosage via NG tube should be increased by at least 50 % if the patient is also receiving enteral or parenteral nutrition.
  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.

Table 27: Phenytoin

- Therapeutic range (40-80 µmol/L). Concentrations as low as 20 µmol/L may give seizure control.
- Saturable kinetics. At the higher end of the therapeutic range, small dose increases may result in large plasma concentration increases.
- Highly protein bound. This makes interpretation of total plasma concentrations difficult in hypoalbuminaemia or severe renal impairment. Free concentrations are advised if albumin < 30 g/L, or if there is a known displacing drug (eg *valproate, aspirin*)
- CYP2C19 inducing drugs (eg *rifampicin, carbamazepine)* may ↓ phenytoin concentration.
- CYP2C19 is subject to genetic polymorphism. 20% Asians and <5% Caucasians are poor metabolisers resulting in ↑[phenytoin] in "standard doses".
- Drugs metabolised by CYP3A4, 1A2, 2C9 (see PML for lists) may have decreased concentrations if co-administered with phenytoin.
- Measure trough concentrations and wait at least 5 days to ensure steady-state.

OBSOLETE

- If seizures persist while awaiting a therapeutic response from phenytoin give diazepam infusion, 50 mg in 500 ml 0.9S q4-6h (i.e. 8-12 mg/hr) or depending on response. Alternatively, Epilim IV 10 mg per/kg over three to five minutes followed by continuous or repeated infusion bolus to a maximum of 2.5 g/day (0.6 mg/kg/hr). (Reconstituted Valproate = 95 mg/ml).

- If the patient is already known to be on anticonvulsants, obtain urgent blood concentration. 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, carbamazepine, sodium valproate or IM phenobarbitone.
- Rarely, toxic levels of anticonvulsant can precipitate status. In such patients, cover with diazepam (see above), allow anticonvulsant plasma levels to fall into therapeutic range, and restart the anticonvulsant.
- Sedation and respiratory depression may result. Early ICU consultation and transfer may be necessary.

16.4.5 Further Points

- Epilim may be the most effective drug in non convulsive status.
- 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 the IM route for diazepam or 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 concentration, before discounting a drug as ineffective.

OBSOLETE

16.5 Epilepsy: Patients Presenting with their First Seizure

16.5.1 Diagnosis

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

16.5.2 Investigations

- CBC + diff, ESR.
- Glucose, Na, Ca.
- CXR.
- Cranial imaging by MRI. A CT head scan may be done if an MRI is not readily available or if an intracranial haemorrhage is suspected.
- EEG useful if diagnosis is in doubt or delayed recovery. It may give a clue to the type of epilepsy (e.g. distinguishes the "absence" of temporal lobe epilepsy from that of Petit Mal) and may show a lateralised abnormality or assist prognosis for recurrence.

16.5.3 Treatment

- Most patients who have recovered and are well following a single seizure, do not require hospital admission.
- If delay in return to normal mental status, consider urgent medical / neurological admission. Also EEG to exclude non-convulsive status.
- Generally, do not commence anticonvulsant therapy following a single seizure unless there is a structural abnormality 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 seizure.
- Phenytoin, carbamazepine or sodium valproate are the drugs of first choice for tonic clonic or for partial (focal) seizures.
- All patients should be advised against driving a motor vehicle even after a single seizure for twelve months. Patients should also be advised of potential risk of swimming alone, working at heights and other high risk activities. Document in the notes that this advice has been given.
- Phenytoin has dose-dependent kinetics with a rapid rise in level after small increases in dosage. Therefore use 30-50 mg daily dosage increments when blood level is near the therapeutic range. Adult doses around 300 mg/day may be given in a single dose, provided adequate blood levels are present just before next dose is due.

OBSOLETE

16.6 Raised Intracranial Pressure (ICP)

16.6.1 Causes

- Include intracranial mass lesion, obstruction to the flow of CSF (hydrocephalus), and brain swelling.

16.6.2 Investigations

- CT or MRI head scan is mandatory to establish the cause. Note that papilloedema may be a late sign of raised ICP or may never appear.
- Remember that many patients with raised ICP sufficient to cause death **will not have or will never** develop papilloedema.
- Do not dilate pupils.

16.6.3 Management

- Consult Neurosurgeon/Neurologist.
- Close observation with neurological recordings every 15-30 minutes will 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.

- Consider dexamethasone 4 mg IV/IM/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 1g/kg IV over 20-30 minutes (500 mL of 15% Mannitol contains 75 g).
- Carefully assess adequacy of the airway. The prime reason for intubation is to ensure a safe airway and adequate oxygenation.
- In an acute situation transfer to ICU while awaiting neurosurgical intervention.

Note:

Lumbar puncture - never perform a lumbar puncture if a patient may have raised intracranial pressure without obtaining a CT/MRI 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.

OBSOLETE

16.7 Encephalitis

16.7.1 Clinical Features

- These usually include fever, meningism and signs of cerebral dysfunction such as altered conscious levels/confusion, seizures, myoclonus, papilloedema or focal signs such as aphasia or weakness

16.7.2 Causes

- **Viral**
  - **Herpes Simplex Virus (HSV)** - 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 infecting agents** - many severe viral and other encephalitides are transmitted by biting insects.
- **Post Viral**

  One of the most common causes. MRI shows diffuse, multifocal changes.
- **Non-Viral**

  Bacterial Endocarditis, Tb, syphilis, listeria, cat scratch, malaria, nocardia (with or without abscess), toxoplasmosis, systemic lupus, sarcoid and neoplasia.

16.7.3 Investigations

Important differential diagnoses include meningitis, severe sepsis, cerebral neoplasia, toxic metabolic encephalopathy. *(see page 171, Stupor and Coma)*

- MRI head - to exclude other diseases mimicking encephalitis and to help establish a diagnosis of HSV encephalitis. MRI is also abnormal in post viral 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. An additional 0.5 ml CSF is required for HSV culture and PCR.
- 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, serology for EBV.
- 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.

OBSOLETE

16.7.4 Treatment

- If HSV suspected treatment is urgent. Give acyclovir 10 mg/kg IV q8h. Consult Infectious Diseases regarding duration of acyclovir treatment (often 14 days). Adjust dose for reduced renal function. Outcome correlates with level of consciousness at commencement of therapy.
- Dexamethasone may be appropriate for selected cases of both herpetic and non-herpetic encephalitis where there is evidence of raised ICP.
- Anticonvulsant therapy will be necessary in some patients.
- Close neurological observation to detect signs of increasing intracranial pressure.

16.8 Spinal Cord Compression

16.8.1 Causes

- Trauma.
- Tumour - extrinsic/intrinsic.
- Haemorrhage.
- Extra-dural abscess.
- Disc prolapse / degenerative changes / narrow spinal canal.

16.8.2 Investigations and Management

- **Remember that quick action may avoid irreversible damage – tetraplegia, tetraparesis, paraplegia, paraparesis.**
- Catheterise if urinary retention present.
- 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, obtain immediate **(i.e. at once)** neurological/neurosurgical consultations and MRI.
- CBC + diff, ESR, Na, glucose, K, Ca, creatinine, AST, GGT, alk. Phos., Bili, albumin. Serum protein electrophoresis and prostate specific antigen may be indicated. In selected patients a search for a specific malignancy, e.g. multiple myeloma or lymphoma should be carried out.
- Remember that in some tumours, e.g. myeloma, secondary deposits, radiotherapy and/or chemotherapy may be the treatment of choice. Urgent consultation with a Haematologist or Oncologist is recommended.
- 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.

OBSOLETE

16.9 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 localising 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 haematoma.
- Diagnostic errors are common. Most frequent misdiagnosis is stroke.
- Younger patients tend to present with raised intracranial pressure/headaches and clouding of consciousness, whereas older patients tend to present with a progressive neurological dificit e.g. hemiparesis rather than raised intracranial pressure. Both groups of course eventually die of raised intracranial pressure if left untreated.

16.9.1 Investigation and Management

- CT/MRI head scan.
- Commence neurologic recordings and consult Neurosurgeon for further advice on management.
- CBC + diff and coagulation profile.

16.10 Stupor and Coma

Coma or stupor should be regarded as a potentially life threatening emergency until:

1. Vital functions are stabilised.
2. The cause of coma/stupor is diagnosed.
3. Reversible causes are corrected.

This section is concerned with the diagnosis and management of the patient with stupor or coma of uncertain cause.

OBSOLETE

16.10.1 Emergency Management / Resuscitation

Emergency Management/Resuscitation. *(see page 16)*

16.10.2 Causes

The causes of coma are:

Drug overdoses	30%
Intracranial lesions, haemorrhage, infarction, tumour	34 %
Metabolic	36%

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

♦ **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.

- **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).
- **Other**
  - Head Injury.
  - Epilepsy/post-ictal.
  - Hysteria / hypnosis.
  - Subarachnoid 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.

OBSOLETE

16.10.3 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**

  The neurological examination is directed at:
  - Detecting meningeal irritation.
  - Defining the level of consciousness.

- Assessing brainstem function.
- Looking for focal/lateralising features.

- **Meningism**
  In all but the deepest coma, meningeal irritation (from meningitis or subarachnoid haemorrhage) will cause resistance to passive neck flexion (but not neck extension or rotation). Resistance in all planes of neck movement is usually due to generalised muscular rigidity (e.g. neuroleptic toxicity) or cervical spine disorder. Kernig's sign (resistance to hip flexion) is usually positive in association with neck stiffness in diffuse meningeal irritation from meningitis or subarachnoid haemorrhage. Kernig's sign is usually negative with neck stiffness from pressure coning due to temporal lobe or cerebellar herniation.
- **The level of Consciousness**
  The Glasgow Coma Scale *(see page 19)* is the best hierarchical assessment of the level of consciousness. The response to commands, calling the patient's first name and painful stimuli are recorded for eye opening, limb movement and vocalisation. Suitable painful stimuli include supraorbital pressure (applied with the thumb) for central stimulation and nailbed pressure (applied with the shaft of a pen) for peripheral stimulation. All four limbs are tested individually for movement and the best response scored, but note should be made of any asymmetry. The level of coma should be made serially. If the level of coma is improving there is no necessity for urgent management decisions, but if there is deterioration urgent action is required.
- **Brainstem Function**
  The brainstem reflexes are important in identifying lesions which may be affecting the reticular activating system (a region important in maintaining consciousness), explaining the reason for coma and determining the viability of the patient. The reflexes used relate to the pupils, ocular movement and respiratory pattern.
  - **Pupil size and reactivity**
    If the pupils are of normal size and reaction then the midbrain is intact and the cause of coma is likely to be metabolic. Important exceptions to this rule include the following:
    - Severe barbiturate intoxication can cause midsized unreactive pupils.
    - Opiates can produce pinpoint pupils with constriction to light too small to see.
    - Atropine and tricyclic poisoning can produce dilated and fixed pupils.
    - Enlarged (>5mm) and unreactive pupil(s) suggest : a tectal midbrain lesion (intrinsic or secondary to compression), or ipsilateral III nerve lesion, or mydriatic eye drops, or anticholinergic drugs, or orbital trauma.

OBSOLETE

- Bilateral pinpoint pupils (<1mm) suggest: bilateral pontine lesions, or opiate overdose, or miotic eyedrops for glaucoma, or bilateral Horner's syndrome.
- Midposition fixed pupils suggest: midbrain lesion, or bilateral cavernous sinus lesions, or deep barbiturate intoxication.
- Small reactive pupils suggest: diencephalic lesion, or metabolic cause.

Reminder

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

- **Eye Movements**

The oculomotor examination comprises observation of:
Eye deviation.
Spontaneous eye movements.
Caloric testing.
Oculocephalic reflex.

- *Eye Deviation:*
  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.
  Conjugate horizontal (lateral) eye deviation is due to either a large ipsilateral hemisphere lesion or contralateral pontine lesion.
  Conjugate downwards deviation is usually due to brainstem lesions (mostly from tectal compression), but may be seen in hepatic coma.
  Downwards and converged eyes are seen in thalamic and subthalamic lesions.
  Conjugate upwards deviation is poorly localising.
- *Spontaneous Eye movements:*
  Spontaneous, conjugate, roving movements suggest midbrain and pons intact and favours bilateral hemisphere dysfunction or metabolic/toxic cause.

  Nystagmus in a comatose patient suggests an irritative or epilectic supratentorial focus.

  Ocular bobbing, rapid conjugate downward jerks of the eyes with slow return, is usually due to acute pontine lesions, and less commonly cerebellar haematomata, or hydrocephalus.

OBSOLETE

Ocular dipping, slow downwards conjugate movement of the eyes with rapid return, is usually due to diffuse cerebral damage.

Ping pong gaze, repetitive or periodic alterating horizontal conjugate gaze has uncertain cause.

~ *Caloric Testing (see page 176):*

Caloric testing is easily done by instilling at least 20 mL (usually 50-200mL is required) of ice cold into the external auditory meati (water irrigation of ears). It is important to perform auroscopy first to exclude tympanic perforation which is a contraindication to caloric testing. A 20mL or 50mL syringe is adequate.

The interpretation of caloric testing is depicted in Figure 4 *(see page 176)*.

The expected response in an (intact) unconscious person to ice cold water is tonic conjugate deviation of the eyes towards the syringed ear. Each ear is irrigated in turn. Warm water irrigation (e.g. 44°C) induces conjugate deviation away from the syringed ear.

Usually it is not necessary to use warm irrigations in addition to cold, but this can be useful in confirming a peripheral (i.e. labyrintine or VIIIth nerve) lesion or if cold water testing is inconclusive.

If horizontal nystamus is induced the implication is that the patient is conscious.

~ *Oculocephalic (Doll's eye) response):*

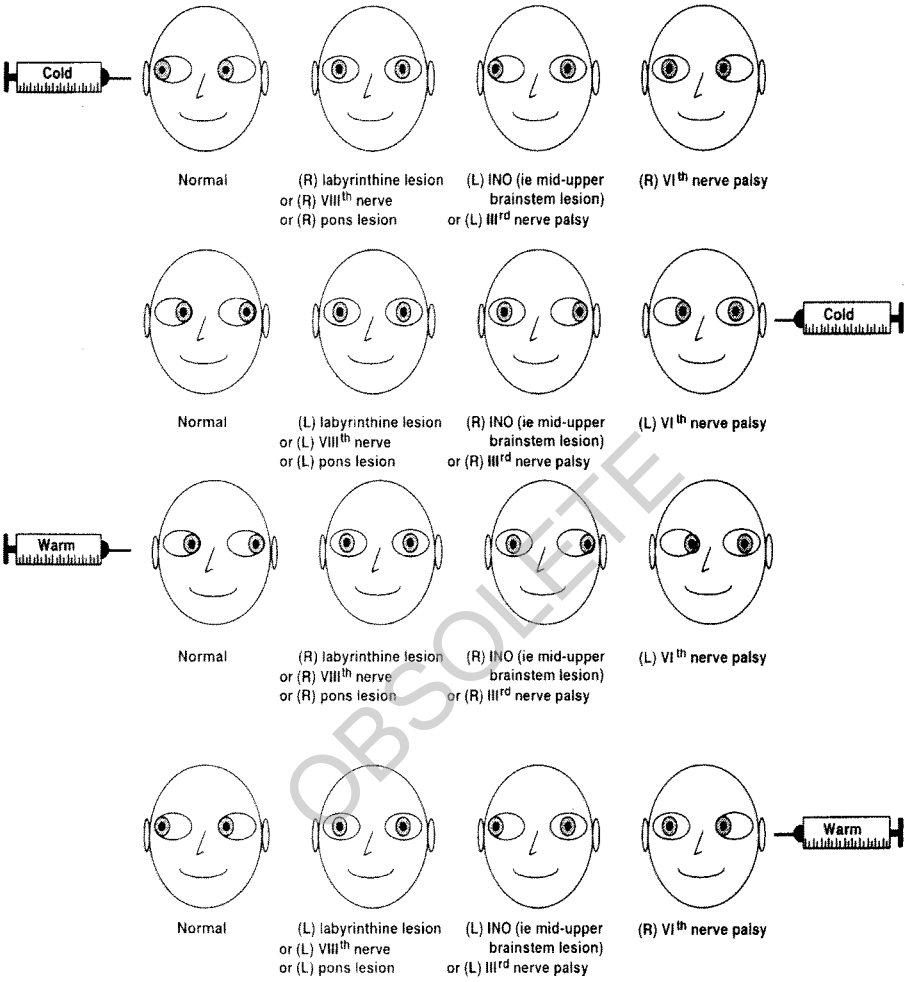
This reflex is tested by sudden passive rotation of the head laterally whilst observing the movement of the eyes. In coma with an intact brainstem the eyes will move conjugately and in a direction opposite to head movement. The types of possible response parallel the ocular responses cold water irrigation of the ear contralateral to the direction of head rotation. For example, rotation of the head to the left induces the same eye movements as cold irrigation of the right ear *(See Figure 4)*. Indeed, the oculocephalic manoeuvre can be employed to enhance the caloric test if the responses to the latter are weak or indeterminate.

Oculocephalic (and caloric) responses are generally normal in hemisphere lesions.

Note: Do not attempt the oculocephalic manoeuvre if neck injury is suspected.

OBSOLETE

Figure 4: Caloric Responses in Coma



OBSOLETE

– *Respiratory Pattern:*

The pattern of respiration has less localising value than the neuro-opthalmic changes detailed above but may give useful additional information.

Cheyne-Stokes respiration (slow oscillation between hyperventilation and hypoventilation) suggest bilateral cerebral hemisphere dysfunction and if stable, usually imply a relatively good prognosis.

Central neurogenic type (40-70 breathes per minute) due to a pontine lesion is rare. Mostly hyperventilation is due to pulmonary congestion (aspiration, infection, neurogenic pulmonary oedema) or acidosis.

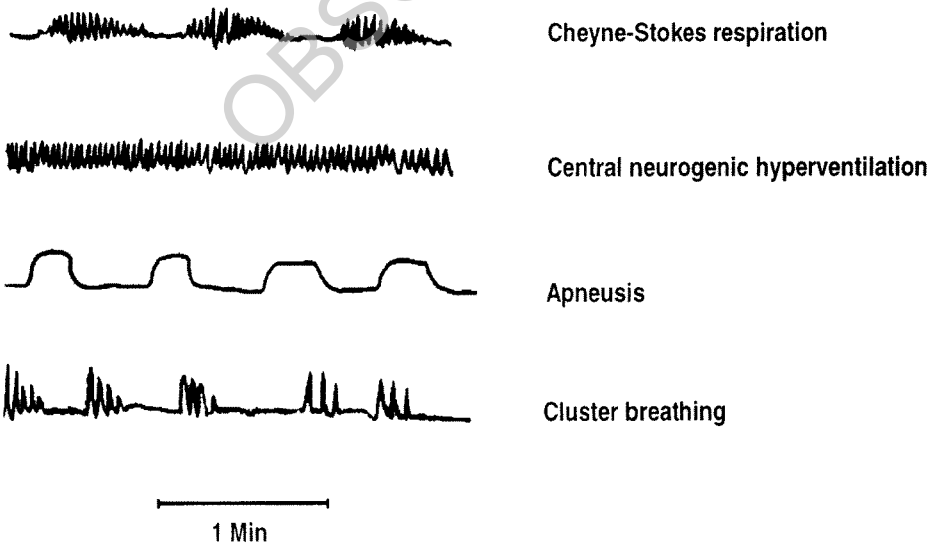
Apneustic breathing (prolonged inspiratory gasp with end-inspiratory pause) generally accompanies lower pontine lesions.

Cluster breathing (periodic respirations that are irregular in frequency and amplitude, with variable pauses between clusters of breaths) results from high medullary lesions.

Ataxic breathing (irregular in rate and rhythm) is usually due to a low medullary lesion. Ataxic breathing and bilateral VIth nerve palsy may be a warning sign of expanding lesion in the posterior fossa.

OBSOLETE

Figure 5: Respiratory Patterns in Coma



- **Motor Function**
  - Observe responses to noxious stimuli applied to nailbeds, sternum or suprorbital 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 Reponses* include several stereotyped postures of limbs:
    - **Decorticate posturing /rigidity** (flexion of elbows and wrists, leg extension) decorticate posturing generally carries a less serious prognosis and is associated with more rostral supratentorial lesions.
    - **Decerebrate posturing/rigidity** (extension of arms and legs). Decerebrate posturing is often associated with brainstem or diencephatlic injury. Note that these patterns are often incomplete, variable and can interchange. Both may accompany hypoxic or hypoglycaemic 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 partial (focal) seizures generally indicates a focal cause of coma, though some metabolic causes, especially hypoglycaemia, can produce focal seizures.
  - The presence of multifocal myclonus or generalised seizures raises possibility of metabolic or ischaemic-hypoxic aetiology.

OBSOLETE

16.10.4 Investigations

- CBC + diff.
- Glucose, Na, K, osmolality, Ca, AST, GGT, alk phos., Bili.
- Arterial blood gases.
- Blood cultures – 2 sets.
- Drug levels (consider gastric lavage).
- CT scan if structural lesion suspected.
- If meningitis a possibility give antibiotics, do CT, then lumbar puncture if safe to do so *(see page 38)*.
- EEG may be considered to identify psychogenic unresponsiveness or partial complex epileticus.

17. Oncology

17.1 Oncology

Main Office Oncology Building ☎ 80020 Fax 80759

Inpatient Services Three inpatient teams on Ward 27

- Assoc. Prof. B. Robinson / Dr C. Atkinson / Dr K Grundy (Palliative Care Physician)
- Dr B. Fitzharris / Dr A. Abdelaal / Dr C Wynne
- Dr M Jeffery / Dr B Hickey

Consultation and On-call Service

For non urgent consults fax referral to 80759. Some specialization exists and will be referred to the appropriate consultant. For urgent consult phone 80023. For urgent (medical or radiation oncology) consults after hours, contact on call oncologist.

Other Services

Radiation Therapy

- Planning ☎ 80098
- Other enquiries ☎ 80020
- Charge Radiation Therapist (Felicity Woodham) ☎ 80754

Outpatient Service

- Appointments / Enquiries ☎ 80023 Fax 80759
- Nurse Specialist (Jan McRae) ☎ 80391

Palliative Care ☎ 81473

OBSOLETE

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.

17.2 Potentially Curable Malignancies

- Early discussion or referral to an Oncologist, Paediatric Oncologist, Haematologist or other appropriate specialist 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 younger patients.
- Any malignancy in children 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.

17.3 Superior Vena Cava 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.

OBSOLETE

17.4 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 103)* 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.

17.5 Inappropriate Secretion of ADH

There are a number of causes of this condition *(see page 102)* for a list of the causes and recommended treatments). In malignant disease the most common cause of this and the other paraneoplastic syndromes is lung cancer, particularly small cell lung cancer.

17.6 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 169)*.

17.7 Neutropenic Sepsis

This may occur in patients receiving chemotherapy or radiation 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 124)*, and the section on Septicaemia *(page 133)*.

OBSOLETE

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

17.9 Lymph Node or Other Tissue Biopies

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 or other haemopoietic malignancy is a possibility, fresh tissue may be needed for surface marker, cytogenetic and 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 Surface Marker Laboratory (Ext: 80917) 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 the Laboratory next morning.

Sarcoma Biopsies

Biopsies of suspected sarcomas of bone and soft tissue, should be deferred until after full staging investigations, including plain x-rays, CT scans, bone scan, and MRI. It is advisable to discuss the patient with the Sarcoma Clinic (Ext: 80023) before biopsy to avoid prejudicing future surgery or radiation options.

17.10 Management of Pain in Cancer Patients

See page 187 in the Pain Section.

17.11 Management of Nausea and Vomiting

Hypercalcaemia, electrolyte imbalance, opiate use, constipation, bowel obstruction and increased intracranial pressure can all be associated with nausea and vomiting and it is important to screen for these problems before commencing treatment.

Check that Ca, creatinine, Na and K have been measured recently. Give intravenous fluids if dehydrated. Use specific treatment if cause identified e.g. dexamethasone for cerebral metastases, hydration for hypercalcaemia.

OBSOLETE

17.11.1 Chemotherapy - Associated Nausea and Vomiting

- Standard antiemetic regimen:
  - Ondansetron 8 mg PO, before chemotherapy, then 12 hourly, 2 more doses if required; or
  - Tropisetron 5 mg PO before chemotherapy, then 1 more dose within 24 hours if required.
- For highly emetogenic chemotherapy:
  - Ondansetron 8 mg PO/IV 8 hourly for 24 hours; or
    ± Dexamethasone 8-16 mg IV [in 100 ml normal saline];
- For persisting emesis, more than 24 hours after chemotherapy:
  - dexamethasone PO 8 mg BID for 2 days, then
    4 mg BID for 2 days

Some patients may be controlled with metoclopramide 10-20 mg IV for mildly emetogenic chemotherapy, e.g. 5FU.

17.11.2 Other Antiemetics for Oncology patients

Metoclopramide	10 mg PO, IV, SC or PR	4-6 hourly
Prochlorperazine	5-10 mg PO	6 hourly or
	25 mg PR	8 hourly
Haloperidol	0.5-3 mg PO, SC or IV	6-12 hourly, or nocte
Cyclizine	25-50 mg PO, SC or IV	8-12 hourly
Dexamethasone	2-4 mg PO	daily
Lorazepam	0.5-1 mg PO or SC	6-8 hourly

Note:

- Avoid metoclopramide in bowel obstruction; haloperidol, prochlorperazine or cyclizine preferred
- Combinations of 2 or 3 agents may be more successful than single agent.
- Haloperidol very helpful for opiate nausea.
- Dexamethasone can be useful for liver metastases.
- If nausea and vomiting remains a problem consult Oncology for further advice.

OBSOLETE

17.12 NSAID and Corticosteroids in Oncology

- Avoid the combination of NSAIDs and corticosteroids in oncology patients if possible.
- Recommend misoprostol or omeprazole prophylaxis if increased risk of upper GI tract ulceration or bleeding.

18. Pain Management

18.1 Principles

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

- Type of pain.
- Cause.
- 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.

Drug metabolism and excretion may be reduced in liver and renal disease and in the elderly. 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.

OBSOLETE

18.2 Severe Acute Pain

Opiates (morphine, pethidine) are usually the drugs of choice. Morphine is the gold standard. Pethidine should only be used where there is an intolerance of morphine. The metabolite of pethidine, norpethidine, is neurotoxic. Dosage of opiates should be sufficient to relieve pain without producing problems such as excessive respiratory depression. Give IV initially (especially if the patient is hypovolaemic or shocked) and titrate in increments against the patient's pain, level of consciousness and respiratory rate. If patients are intolerant of either morphine or pethidine then consider fentanyl 1 mg/kg IM. 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 is used to manage severe acute pain. These include:

- Patient controlled analgesia [PCA].
- Epidural infusions.
- Regional nerve blocks.

The Acute Pain Management Service [APMS], available on Beep 114 or via the on-call anaesthetist, can advise on the appropriate technique.

18.2.1 Doctor's Responsibilities

- Initial assessment of patient:
  - Magnitude and cause of pain.
  - Existence of factors that might affect the patient's 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.

18.2.2 Nurse's Responsibilities

- Assess opiate dosing levels at regular intervals.
- 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 Table 28 and Table 29)*.
- Manage appropriately any untoward effects *(see Table 30)*.

OBSOLETE

Table 28: Dosage Guidelines for Systemic Opiates in Acute Pain

Drug	Route	Dose For Adults	Notes & Dose Intervals
Morphine	Intramuscular	0.15 mg/kg/dose i.e. 10 mg/dose/70kg	3-6 hourly
	Intravenous over one minute	0.03 mg/kg/dose i.e. 2 mg/dose/70kg	Can be repeated at 5 min intervals until desired effect achieved and respiration and sedation are satisfactory
	Intravenous infusion	0.02 - 0.04 mg/kg/hr i.e. 2-3 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 over one minute	0.3 mg/kg/dose i.e. 20 mg/70kg	Can be repeated at 4 min intervals until desired effect achieved and repeated as required
	Intravenous infusion	0.3 mg/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 paediatric guidelines.

Table 29: Frequency of observation during Acute pain Medication

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

Table 30: Pain and Sedation Scores

Pain Scores	Sedation Scores
0 = 'No Pain'	**0** = None, Alert
1 = 'Mild Discomfort'	**1** = Mild. Occasionally drowsy, 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
5 = Worst imaginable pain	attempt must be made to rouse patient)

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Table 31: Management of Severe Complications

Complication	Management
Respiratory depression	
Life threatening	• stimulate patient • support ventilation and airway - bag and mask • oxygen by mask • stop opiate administration • give naloxone *
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 • consider other causes.

*Naloxone 0.2-0.4 mg IV injection repeated every 5 minutes until desired effect (maximum 2.0 mg). May need further naloxone as its half-life (~ 1h) is shorter than most opiates.

18.2.3 Adjuncts to Opiates for severe Acute Pain

- Other agents can be useful along with opiates e.g. a non-steroidal anti-inflammatory agent (NSAID) PO or by suppository, or paracetamol.
- 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).

18.3 Chronic Pain

This is broadly classified into two categories - cancer and non-cancer pain.

18.3.1 Cancer associated Chronic Pain

- It is important to establish the cause 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.
- **For severe pain use morphine**.
- **Guidelines for starting morphine for palliative therapy:**
  - Commence using morphine elixir (1 mg/mL, 5 mg/mL or 10 mg/mL).
  - Starting dose; 10 mg 3-4 hourly, throughout 24 hours. Extra doses must be available for severe pain.
  - Reduce dose if elderly.
  - Increase dose if already on codeine regularly. 5 mg morphine is approximately equivalent to 60 mg codeine.
  - Give dose 6-8 hourly if impaired liver or renal function.
  - If pain is not controlled increase dose of morphine elixir by 100% (up to 20 mg), 50-100% (up to 50 mg), 25-50% (up to 100 mg) or 25% (>100 mg).

Starting sustained release morphine, including LA Morph, MST Continus 10, 30, 60 , 100 or 200 mg tabs or Kapanol 10, 20, 50 or 100 mg caps.

- Add up morphine doses over 24 hours during which pain was controlled, and divide by 2 to get the 12 hourly MST dose. Kapanol can be given once or twice daily.

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Patients may be started directly on sustained release morphine in a 20-100 mg daily dose with morphine elixir for breakthrough pain. Caution required and safe dose difficult to predict.

Breakthrough Pain:

- Morphine elixir 3 to 4 hourly, using doses up to 20% of total daily morphine dose.

If several doses needed per day, increase MST dose.

Incident Pain:

Use morphine elixir, 20% of total daily morphine dose, before activity which causes pain. (Do not adjust MST dose).

- Pain is a physiological antagonist to morphine induced respiratory depression. Morphine doses can be increased until pain is controlled.
- 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. Consultation with Palliative Care or with Pain Management Centre, Burwood Hospital may be helpful.

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Table 32: Management of Opiate side effects

Nausea and vomiting	
Metoclopramide	5-10 mg PO q4-8h
Prochlorperazine	5-10 mg PO q8h, 3 mg buccal q12h, 25 mg pr q12h
Haloperidol	0.5-1.5 mg PO q8h
Cyclizine	50 mg PO q6-12h
Nausea and vomiting due to opiates tend to subside over the first week. Therefore reassess need for anti-emetics.	
Metoclopramide has theoretical advantages in the presence of constipation as it stimulates peristalsis. However it is contraindicated if obstruction likely.	
Ondansetron 4 – 8 mg PO q12h or 4 mg IV q2-4h for severe nausea & vomiting which has not responded to the first line antiemetics.	
Constipation - regular stool softeners or bulking agents (e.g. Metamucil, coloxyl, lactulose) with intermittent use of stimulants (e.g. Bisacodyl).	

18.3.2 Non Cancer Chronic 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 in 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.
- It is potentially harmful to use oral opioids on a long term basis for non cancer pain.
- Where severe pain persists, other analgesics should be considered (DHC, nefopam, tramadol). Tramadol is an expensive new oral parenteral analgesic. It has both μ opioid, noradrenergic and serotonergic receptor mechanisms of analgesic action. Side effects are similar to opiates but with slightly less constipation. Efficiacy is similar to pethidine. There is a low potential for addiction and it is therefore a non controlled medication. However it should not be considered a first line analgesic.
- Tricyclic antidepressants or anti-convulsant drugs may be useful for neuralgic type pain. Any problems in this area may be referred to the Pain Clinic, Department of Anaesthesia.

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19. Poisoning / Drug Overdose

19.1 Management Priorities

1 The Airway, Breathing and Circulation

The first priority in all unwell patients is attention to:

- Airway
- Breathing *(see page 16)*
- Circulation

Many patients after overdose have potential A, B or C problems which should be anticipated.

Central nervous system depression or convulsions causing problems with A and B, and cardiovascular depression or arrhythmias, are common consequences of poisoning.

2 Gastrointestinal Tract Decontamination

After attention to A, B, and C, gastrointestinal decontamination measures should be considered.

The options include:

i. Activated charcoal - first choice if any is used
ii. Gastric lavage (stomach washout).
iii. Induction of emesis with syrup of ipecac - no longer recommended.
iv. Whole bowel irrigation - occasional indications for this.

Note: Usually gut decontamination is not required.

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19.1.1 Activated Charcoal

- First choice for gastrointestinal decontamination, if any is indicated.
- Provided as 50 g in 300 ml - give as a single dose, either orally, via a nasogastric tube, or via orogastric tube after gastric lavage.
- Repeated doses are indicated with some poisonings to interrupt the enterohepatic circulation and possibly as enteral dialysis (e.g. theophylline). Do not use repeated doses of activated charcoal with sorbitol as sorbitol will cause fluid, sodium and potassium loss.
- The following substances are not adsorbed well to activated charcoal and therefore, alternative decontamination methods should be considered - ethanol, methanol, ethylene glycol, hydrocarbons such as petroleum distillates and essential oils, lithium, iron, potassium and lead.
- The dose of activated charcoal is not well established but doses of ten times the gram weight of the poison or 1 g/kg body weight have been suggested.
- The efficacy of activated charcoal beyond one hour of the ingestion is not proven. It should not be considered routine beyond 1 hour. Activated charcoal may cause vomiting, and if aspirated, will cause a lung injury. It

should not be used for trivial overdoses. For overdoses greater than 1 hour since ingestion, it should be given only if the perceived benefits outweigh the risks. It should not be given to a drowsy patient without airway protection. Nasogastric administration is only rarely indicated because of the increased risk of aspiration associated with this route.

19.1.2 Gastric Lavage

- The indications for gastric lavage are limited.
- Less than a third of the ingested toxin is removed in experimental models and no clinical benefit over charcoal has been demonstrated in clinical trials.
- Gastric lavage is indicated if:
  - The poisoning is life threatening **and**
  - Removing less than a third of the ingested dose would significantly reduce the likelihood of toxicity **and**
  - The ingestion occurred within 1 hour (2 hours for drugs that delay gastric emptying, e.g. tricyclics, or form concretions e.g. aspirin).
- Gastric lavage is not indicated:
  - If the patient has been vomiting **or**
  - As a punitive measure **or**
  - In poisonings which are not life threatening with good supportive care (e.g. benzodiazepines).
  - Gastric lavage has a small but significant risk of iatrogenic injury, should only be performed under the direct supervision of someone skilled in the procedure and should be preceded by endo-tracheal intubation if the patient is very drowsy.

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19.1.3 Induction of Emesis with Syrup of Ipecac

- No longer available.
- Inappropriate for adult ingestion (variable effect, delayed action, dangerous if patient becomes drowsy or convulses), contraindicated with corrosives and volatile substances.

19.1.4 Whole Bowel Irrigation

- Occasional indications which include:
  - Significant overdoses with sustained release preparations (e.g. theophylline, calcium channel blockers).
  - Significant overdoses with substances not well bound by activated charcoal (e.g. iron, lithium, lead).

- Use Golytely (polyethylene glycol solution) at approximately 2 L/hr orally until the rectal effluent is clear (usually 4-6 hours).
- Contraindications: ileus, perforation, obstruction.
- Cautions:
  - Golytely will displace drug from activated charcoal.

  Diarrhoea may be prolonged. Na and K levels should be checked regularly and IV fluids may be required to replace losses.

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).
- Poisons CD database - 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 (Mon to Fri 0900-1700) 03 479-1200.
- Various texts:
  - The New Ethicals Compendium.
  - Handbook of Poisoning - Dreishbach, Lange Publications.
  - Clinical Management of Poisonings and Overdose - Haddad and Winchester.

  (All three are available in the Emergency Department).

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19.1.5 Investigation

- Determining the poison ingested is derived mainly from the history. Occasionally a toxic 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. A toxic screen 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 only performed 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. As paracetamol rarely produces clinical symptoms of toxicity in the early stages, a paracetamol level may be indicated if paracetamol ingestion cannot be excluded on history.

- Other investigations may be indicated e.g. arterial blood gases, ECG.

ecific Management of Some Poisonings / Overdoses

19.2 Paracetamol

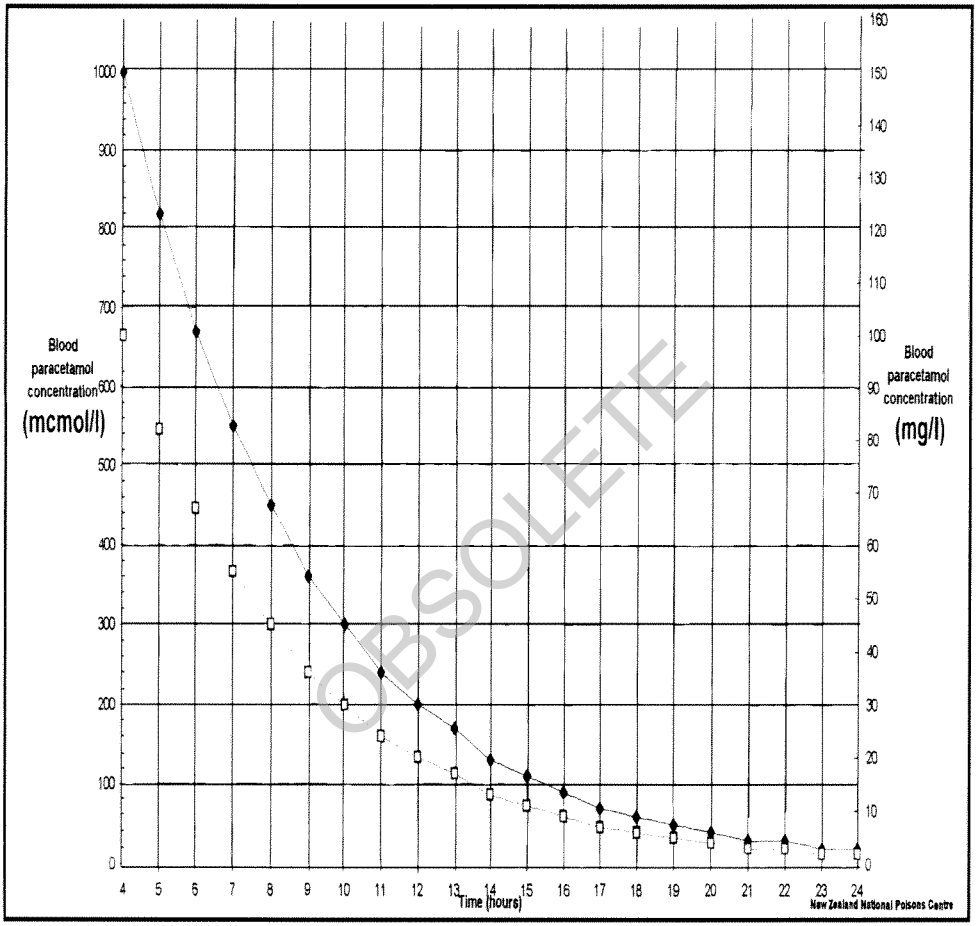
- 140 mg/kg approximates a toxic dose, although as little as 100 mg/kg has caused toxicity.
  Note: Chronic alcohol use, phenytoin, phenobarbitone, antihistamines, and other drugs that induce the cytochrome P450 enzyme system can increase toxicity for the same dose or blood level by shunting more paracetamol through the pathway that produces the toxic metabolite.
- Blood levels direct treatment, but should not be taken until approximately 4 hours after the ingestion.

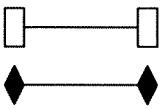
  Note: Pre school children who drink paracetamol elixir can tolerate a higher toxic dose (approx. 200 mg/kg) and should have blood levels taken at 2 hours as the elixir is absorbed more quickly.
- Treatment is effective if begun within 6 – 8 hours. Therefore the N-acetylcysteine infusion may await the result of the 4 hour blood test. Those with massive ingestion presenting after 6 – 8 hours, especially if symptomatic, should have N-acetylcysteine without waiting for the blood level result.
- Clinical toxicity follows four approximate stages:
  - ½- 24 hours — Nausea, vomiting, malaise (or asymptomatic).
  - 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 on page 194.

OBSOLETE

Figure 6: *Modified Rumack-Matthew Paracetamol Nomogram (μmol/L and mg/L)*





Treat above the lower line if patient has risk factors for toxicity (e.g. chronic alcoholism, enzyme inducing drugs, fasting, etc).

Treat all patients with levels above the upper line.

Table 33: Paracetamol Toxicity Treatment

N-acetylcysteine (Parvolex)	INITIALLY	150 mg/kg in 200 mL D5W over 60 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 most effective when given within 15 hours of ingestion but there is increasing evidence that even beyond 24 hours, it may be beneficial.
- 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.
- If rash or urticaria develop during the N-acetylcysteine infusion, slow or stop the infusion, treat with promethazine 25 mg IV and hydrocortisone 200 mg IV and then restart the infusion at a slower rate. For more significant anaphylaxis, treat with adrenaline. Rarely, for significant allergy consider using methionine instead of N-acetylcysteine.

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.

OBSOLETE

19.3 Tricyclic Antidepressants

- Numerous receptor effects (sodium channel, calcium channel, muscarinic and α-receptor blockades, some sympathetic agonist effects).
- Large volume of distribution, liver metabolism with enterohepatic circulation.
- The main toxic effects are usually apparent within six hours of ingestion:
  - Anticholinergic effects.
  - CNS depression, irritation, convulsions.
  - Cardiovascular depression/arrhythmias.

Note: After initial tachycardia, the main cardiac effects are slowing of 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. 10-20 mg/kg is associated with toxicity and 30-40 mg/kg is likely

to be fatal in adults.

- Sodium bicarbonate has both prophylactic and therapeutic effects on cardiac toxicity for two possible reasons. Alkalinization of the blood to a pH of 7.5 seems to be cardio-protective. Sodium bicarbonate also provides a bolus of sodium and the sodium channel blocking effect seems to be a relevant factor in causing cardiac toxicity. Give 50-100 mmol $NaHCO_3$ over 5-10 minutes.
- Correction of hypoventilation by IPPV, will also help, by raising the blood pH.
- The use of physostigmine is discouraged as the response is unpredictable.
- When treating arrhythmias do as little as is necessary. All drugs that prolong repolarization are contraindicated. Phenytoin and lignocaine are considered safe but their efficacy is debated and lignocaine may aggravate seizure tendency. Pacing, cardioversion and defibrillation are relatively safe.
- Suxamethonium may raise parasympathetic tone which can increase heart block, however, its use may be unavoidable if urgent airway management is required.
- Treat convulsions with diazepam and phenytoin if required.
- Patients with a widened QRS or any altered level of consciousness should be referred to ICU. Patients who have no signs or symptoms at 6 hours post ingestion can be discharged after psychiatric assessment.

Note: - beware of the serotonin syndrome *(see page 198)*

OBSOLETE

19.4 Benzodiazepines

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

Note:

- T½ flumazenil is one hour, i.e. sedation can recur.
- It may be helpful if trying to establish the cause of an altered level of consciousness (although sedation due to alcohol and hepatic encephalopathy may also respond).
- It is useful in reversing paradoxical agitation caused by benzodiazepines, seen occasionally in children and the elderly. Other indications are rare.
- Use of flumazenil in mixed overdoses (e.g. benzodiazepines and tricyclic antidepressants) may induce seizures.

19.5 Phenothiazines

- Dopamine receptor blockade can produce symptoms like dystonia, akathisia, Parkinsonism, tardive dyskinesia and neuroleptic malignant syndrome.
- Seizures and arrhythmias can occur via mechanisms similar to tricyclic poisoning especially with thioridazine. Treatment in such cases is similar.
- Treatment is generally supportive. Dystonic reactions can be treated with benztropine 2 mg IM or IV.
- Neuroleptic malignant syndrome requires identification and aggressive treatment prior to and after transfer to the Intensive Care Unit.

19.6 Monoamine Oxidase Inhibitors

- More than 2-3 mg/kg is potentially life threatening.
- Examples of MAO-I drugs currently available include phenelzine, moclobemide and tranylcypromine.
- Effects of an overdose include CNS stimulation, tachycardia, hypertension, hyperpyrexia and tachypnoea. In severe overdoses there is CNS and cardiovascular depression.
- Blood levels for MAO-I are not done routinely. Diagnosis and assessment of severity is clinical.
- Treat hyperthermia with aggressive external cooling and sedation. May need neuromuscular paralysis. Dantrolene may also be useful.
- Control BP and tachycardia with labetalol (combined α and β-blockade may be better than β alone). Phentolamine or sodium nitroprusside may also be used.
- Convulsions should be treated with diazepam with or without phenytoin.
- Treatment is otherwise supportive.

Note:

- Because of persistence of MAO-inhibition, all dietary intake and drug therapy should be monitored for potential interactions for the next 7-10 days. Similarly other antidepressant drugs should not be commenced during this time.
- Be alert to the serotonin syndrome *(see page 198)*.

OBSOLETE

19.7 Fluoxetine/Paroxetine

(Selective Serotonin Re-uptake Inhibitors, SSRIs)

- To date overdoses with these drugs have been mostly benign although at least one death has occurred and seizures have been reported.
- Serious toxicity and death can occur when taken with MAO-Is and they may increase the toxicity of any tricyclics ingested at the same time. The serotoninergic syndrome may result consisting of confusion, restlessness, myoclonus, hyperreflexia, hyperpyrexia, sweating, shivering, tremor, diarrhoea, incoordination, seizures and coma.
- In most cases CNS depression will predominate.
- Treatment is supportive.

19.8 Serotonin Syndrome

- May occur with combination of drugs such as MAOIs with SSRIs, clomipramine, other tricyclic antidepressants, lithium or pethidine.
- Diagnosis requires such a combination, or the increase in dosage of serotoninergic drug plus any three of the following clinical signs - agitation, diaphoresis, diarrhoea, fever, hyper-reflexia, mental status changes, myoclonus, shivering, tremor, incoordination in the absence of any recent addition or increase in dosage of a neuroleptic agent.
- Treatment is supportive, which may need to be aggressive. Diazepam for agitation. Chlorpromazine 50-100 mg IV, has anti-serotonin activity and should not be used in the neuroleptic malignant syndrome. [Cyproheptadine 4 mg 4 hourly or methysergide 2 mg are alternatives which may be of use].

OBSOLETE

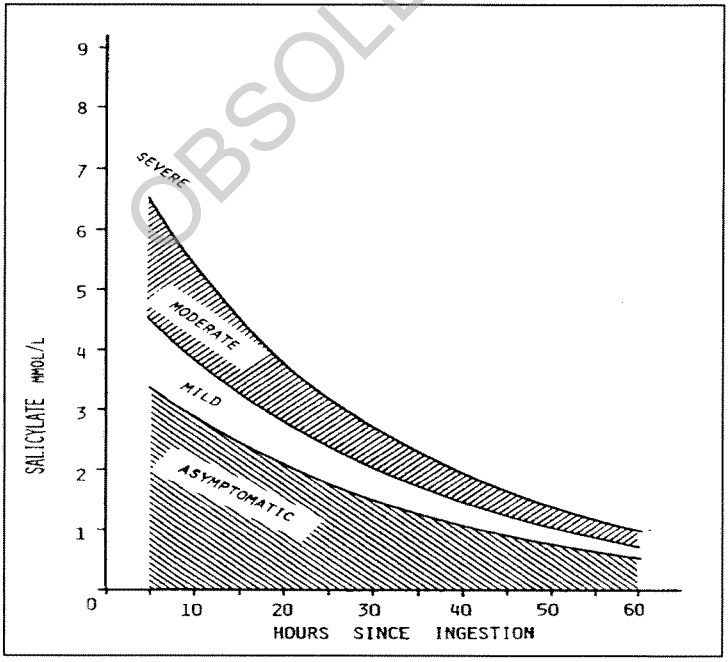
19.9 Aspirin (and other salicylates)

- 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.
- Blood concentration - nomogram helpful in acute poisoning. Measure serum salicylate concentration 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.
- An abnormal pH may be a better indication of toxicity than salicylate levels, particularly if the time since ingestion is uncertain.

OBSOLETE

Figure 7: Salicylate Toxicity



Note: Salicylates are present in a number of over the counter preparations, e.g. methyl salicylate in oil of Wintergreen.

Treatment

- If 6 hours post ingestion, concentration low and symptoms absent then patient may be discharged after appropriate psychiatric intervention.
- Consider gastric decontamination if less than 1-2 hours post-ingestion and >150 mg/kg of aspirin has been taken. Admit for observation. Monitor salicylate levels and pH between 8-12 hours to detect delayed toxicity.
- IV fluids - all patients with significant salicylate poisoning are dehydrated.
- Monitor blood glucose, electrolytes (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 IV 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.

  Note: urine output [aim for 2-3 ml/kg/hr], pH, and serum potassium need to be monitored closely [every 1-2 hours].
- If salicylate level greater than 6.0 mmol/L or if very unwell, then consider haemodialysis (Contact Nephrologist on call). Transfer to ICU.

OBSOLETE

19.10 Opiates

- Altered level of consciousness, respiratory depression (typically a very slow respiratory rate with a maintained tidal volume), miosis.
- 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 (especially for long acting narcotics like methadone). 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.

19.11 Theophyllines

- Greater than 10 mg/kg may be toxic. Chronic overdosage is more dangerous than acute for any given concentration.
- Arrhythmias - atrial/ventricular ectopics and tachyarrhythmias.
- CNS irritation/convulsions.

- Nausea and vomiting.
- Potassium falls with increasing acute toxicity
- Slow release preparation will have delayed effects.
- Treatment:
  - Consider repeated doses of charcoal (50 g) orally or via NG tube every 4 hours. (Do not give repeat doses of charcoal with sorbitol.)
  - Charcoal haemoperfusion:
    - Effective and may be life saving - consult Nephrologist on call.
    - Indications:
      - Intractable seizures or persistent unstable haemodynamics.
      - 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.
  - 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.

  Note: Bronchospasm may ensue - consider risk benefit in asthmatics.
  - Hypotension:
    - IV fluids.

      Propranolol as above (hypotension is due to excessive β-adrenergic stimulation). Caution in asthmatics.

OBSOLETE

19.12 Beta Blockers

- Bradycardia plus hypotension may initially be treated with atropine followed by isoprenaline and dopamine if required. In severe poisoning, consider the use of Glucagon (3-10 mg by IV push over one minute, or infusion at 2-5 mg/hr). Calcium (calcium chloride 10% 5-10 ml IV) can also be given.
- Monitor blood glucose levels.
- Bronchospasm may be treated with inhaled beta-agonist, adrenaline SC, aminophylline IV.
- Consider cardiac pacing in severe bradycardia.

19.13 Calcium Channel Blockers

For symptomatic bradycardia, the following agents (in order of preference) may be used:

- Atropine 0.6 mg IV and repeat as necessary.
- Calcium chloride 10% 5-10 ml IV (which may be repeated according to clinical response. Large doses are occasionally required).
- Glucagon (3-10 mg by IV push over one minute, or infusion at 2-5 mg/hr).
- Cardiac pacing.
- IV fluids for hypotension.
- Consider inotropes (dopamine and/or dobutamine) for hypotension which persists despite adequate hydration and resolution of bradycardia.

19.14 Digoxin

OBSOLETE

- Acute poisoning:
  - Measure plasma concentration.
  - Plasma potassium 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. Agents that could be used:
    - Lignocaine 1.5 mg/kg IV.
    - Phenytoin 15 mg/kg IV infusion at no more than 50 mg/min.
    - Magnesium sulphate 2-4 g IV.
  - Cardioversion:
    - May precipitate ventricular tachycardia/ventricular fibrillation, or asystole.
    - Use low energy (10-25 joules).
  - Hyperkalaemia:
    - Standard treatment *(see page 150)*, but avoid calcium chloride/calcium gluconate as calcium may potentiate digoxin cardiotoxicity.
- Fab fragments (Digibind - kept in ICU and CCU):

- Indications:
  - Life threatening arrhythmias due to digoxin toxicity such as VT, VF, severe bradycardia not responding to atropine.
  - Severe hyperkalaemia refractory to insulin/glucose therapy.
- Dosage for cardiac arrest or severe haemodynamic compromise:
  - 5 ampoules initially over 20 minutes in 100 ml 0.9S.
  - If no response repeat dose (in adults).
  - If still no response in adults, (or after first dose in children), then give more according to the calculated dosage requirement.
- For dosage in less urgent clinical situations, see Digibind Drug insert.

Beware:

- Potassium levels can drop rapidly, check regularly.
- Cardiac output may fall.
- Ventricular rate may increase.

NOTE: All patients should be monitored preferably in ICU/CCU if Fab fragments are used.

OBSOLETE

19.15 Lithium

- Well absorbed orally. Low protein/tissue binding. Eliminated solely by the kidneys.
- CNS depression or stimulation. CVS stimulation progressing to conduction defects and block. Nausea, vomiting, confusion and coma.
- Toxic dose approximately 40 mg/kg.
- Serum levels useful. Greater than 1.5 mmol/L considered toxic. Chronic overdosage is more severe than acute for any given concentration.
- Activated charcoal doesn't bind lithium.
- Normal saline infusion to induce volume diuresis which aids renal elimination.
- Haemodialysis useful if significant symptoms, deteriorating, not improving, level >4 mmol/L or if urine output inadequate - significant toxicity will usually require ICU monitoring. Consult Nephrologist on call.

19.16 Carbon Monoxide

- Carbon monoxide (CO) is the most common non-medicinal poison in Australasia.
- There is a poor understanding of the neurotoxicity of CO. Severity of poisoning does **not** correlate with the admission carboxyhaemoglobin level, which is, therefore a poor guide to management.
- There remains controversy over the role of hyperbaric oxygen (HBO) due to lack of well-designed clinical trials. Nevertheless where it is readily available, as in Christchurch, it should be used.
- If time allows do carboxyhaemoglobin, CBC, Na, K, creatinine, and glucose on admission; ECG in elderly; ABGs.

Treatment

- 100% oxygen via a breathing system with a reservoir bag for at least 6 hours. This also hastens the elimination of CO.
- HBO indications (referral is URGENT).
  - Loss of consciousness at any stage **OR**
  - Any neurological symptoms or signs (including cognitive, behavioural or psychological) except headache and/or nausea alone **OR**
  - Delayed deterioration. This may occur in 8-30% of patients. Therefore daily follow up (on outpatient basis if appropriate) is required **in all cases** for at least 4 days, plus instructions to return for assessment if problems develop subsequent to this period. Delayed symptoms may respond well to HBO.

Note: No correlation between severity of presentation and development or severity of delayed sequelae.

To arrange HBO or for advice follow the procedure given on page 271.

OBSOLETE

19.17 Ethanol

- Intoxication:
  - Supportive care.
  - Nurse in recovery position to avoid aspiration in the event of vomiting.
  - Measure blood glucose if obtunded.
  - Blood or breath levels may be of diagnostic value.
  - Very rarely haemodialysis may be considered e.g. if >500 mg %.

Table 34: Ethanol - Formula to convert units

To convert mg % to mmol/L
multiply by 0.22
i.e. 100 mg/% (mg/100 mL) = 22 mmol/L.

Note: The chronic alcoholic with altered mental status should be given thiamine 100 mg IM. Glucose infusion in thiamine deficiency can precipitate Wernicke's encephalopathy therefore always give thiamine to cover glucose infusion.

19.18 Methanol

19.18.1 Accidental ingestion of methanol in the non alcoholic

- 30 ml of pure methanol is potentially fatal. Methanol is metabolized to formic acid in the body.
- 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, do not gastric lavage beyond 1 hour.
  - Not bound by activated charcoal.
  - Sodium bicarbonate - often large amounts required to keep pH >7.2 (also sodium bicarbonate helps formate excretion).
  - Ethanol IV or orally if methanol concentration >6mmol/L or visual symptoms or severe acidosis.
    - Loading dose oral or via NG tube of 1 ml/kg 96% ethanol in 200 ml orange juice OR IV loading of 1 ml/kg 100% ethanol diluted in 500 ml D5W over 30 minutes.
    - Maintenance dose 0.3 ml/kg 96% ethanol/kg q2h in orange juice or 0.15 ml/kg 100% ethanol/hr IV oral or NG tube. Aim to maintain an ethanol concentration of 22 mmol/L.
  - Haemodialysis if methanol >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.

19.18.2 Management of the chronic alcoholic / meths drinker

- In the community

OBSOLETE

- Individuals who have symptoms that are unlikely to be explained by ethanol excess alone should be assessed medically.
- Individuals who stop ethanol intake either voluntarily or of necessity, need to be monitored closely. Any unusual features will need medical review.

In either instance, medical advice could be sought from Sunnyside or other Healthlink South detoxification unit, or the Emergency Department, Christchurch Hospital. If the latter, ask for the triage nurse in the first instance.

- Medical problems in these patients are likely to be:
  - If methanol toxicity - shortness of breath, visual problems, headache, drowsiness, abdominal pain.
  - If ethanol toxicity - inebriation, agitation, tremor, DTs, alcohol withdrawal fits.

OBSOLETE

♦ **In the Emergency Department**

- When the patient presents, make an attempt to distinguish simple ethanol excess from other causes of general dysfunction.
- Clinical assessment with further investigations as dictated by the history and examination.

  Laboratory assessment should include blood gases for pH, CBC + diff, Na, K, creatinine, bilirubin, alkaline phosphatase, gamma GT, AST, and ethanol and methanol levels.

The decision to admit is difficult because one cannot always predict which meths drinker is going to run into severe toxicity.

♦ **The decision to admit**

- The severely ill ± acidotic patient needs admission to ICU before the ethanol/methanol levels are available. Discuss with ICU.
- Patients with complicating medical/surgical problems, eg pneumonia, should be admitted to the appropriate service promptly.
- Other patients, who are reasonably well but have elevated methanol/ethanol levels, and are not acidaemic, present a difficult problem.

Recommendations in this situation include:

- Review the reasons for their attendance at the Emergency Department.
- If the pH is greater than 7.35 and the **ethanol level is higher than the methanol level**, advise them to stop drinking meths but to continue drinking ethanol, and ask them to return for evaluation (repeat levels

and pH) the next day. Give them a card with the time/day/place. Under these circumstances contact the City Mission for assistance with follow up. The City Mission reception telephone number is 365 0635 or fax 366 7100. However, if a patient has come from Thorpe House, please contact Thorpe House for follow up, telephone 379 1682.

- If the pH is greater than 7.35 but the **methanol level is higher than the ethanol level** then admit to the Emergency Observation Area (or to a medical ward) for repeat bloods at 6 and 12 hours. No specific treatment at this stage.
- If the pH level is less than 7.35 then admit medically to closely monitor pH and to keep ethanol level over 100mg/100ml (22mmol/l). Initially get pH by arterial puncture - subsequently pH by venous blood samples should be sufficient.

♦ **Inpatients**

- ICU treatment including ethanol, thiamine, folic acid, dialysis as dictated by ICU protocols.
- General Medicine & Other Specialty Wards:
  - Evaluate the patient as above and discuss management with the consultant in charge.
  - Treat any intercurrent condition, eg pneumonia.
  - If, on more detailed assessment, there are no signs of possible methanol toxicity and no acidosis, then observe closely, repeat methanol/ethanol levels at 6 and 12 hourly. Do not give ethanol. Patients who are well with no complicating medical illness and whose methanol levels are falling, may be discharged after 24 - 48 hours observation, regardless of their methanol level.
  - All other patients who are symptomatic and/or acidotic should be given ethanol, oral or IV (as above). Treat any intercurrent illnesses.

Note: All patients who are not chronic alcoholics / meths drinkers but who have ingested methanol, should be managed as described in section [1] above.

OBSOLETE

19.19 Ethylene Glycol

♦ 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 (do not perform gastric lavage beyond 1 hour).
- Activated charcoal doesn't bind ethylene glycol.
- Sodium bicarbonate as for methanol.
- If hypocalcaemic, give calcium chloride 10% 5-10 ml and reassess.
- Thiamine and pyridoxine (act as co-factors for glyoxylate metabolism).
- Intravenous fluid to maintain good urine output.
- Ethanol as for methanol (see above).
- Haemodialysis if significant intoxication.

19.20 Lead

♦ CNS toxicity, PNS toxicity, anaemia, nephrotoxicity.

♦ Levels useful (discuss with Toxicology).

♦ For paediatric poisoning - see Paediatric Handbook.

♦ Mild to moderate poisoning is not considered here - reference to more detailed literature is appropriate.

♦ Lead encephalopathy may require urgent treatment:
- British anti-Lewisite (BAL or dimercaprol) 4 mg/kg/dose by deep injection every 4 hours for 5-7 days.
- Calcium Disodium edetate given with the second dose of BAL. Give 25 mg/kg/dose as a 2 hour infusion every 12 hours with 10 doses in total. (Do not use a solution stronger than 0.5%). Ensure a urine output of at least 0.5 ml/kg/hour.

OBSOLETE

19.21 Iron

♦ Four stages of toxicity described:
i. GI tract stage - nausea, vomiting, haematemesis which may progress to shock (first few hours).
ii. False reassurance stage - symptoms may abate (within 12 hours).
iii. Shock, hepatic, renal and cardiac dysfunction (beyond 12 hours).
iv. Days/weeks later - small bowel obstruction or gastric outlet obstruction due to stricture.

♦ Concentration - useful at 4 hours but serum iron levels are not an entirely reliable guide to the amount of iron taken:

- Potentially toxic if serum iron level >60 mcmol/L.
- If level greater than TIBC.

NOTE: Concentration can be low due to tissue distribution and TIBC is artificially raised after overdosage.

- Toxic dose >20 mg/kg (lethal dose 200-300mg/kg).
- Begin treatment if:
  - Vomiting.
  - Diarrhoea.
  - Toxic concentration (>90 mcmol/L or >60 mcmol/L if symptomatic).
  - If concentration greater than TIBC.
  - Dose greater than 20 mg/kg of elemental iron (ferrous sulphate is 32% elemental iron - look up the amount in the preparation ingested).
- Treatment:
  - Golytely whole bowel irrigation may be warranted.
  - Abdominal x-ray may be useful to determine the success of gastrointestinal decontamination.
  - Desferrioxamine by slow IV infusion at a maximum rate of 15 mg/kg/hr
  - If performing gastric lavage, leave 5 g of desferrioxamine in the stomach.
  - Continue infusion until levels fall or clinically improved. Slow the infusion if hypotension develops. Desferrioxamine itself may be toxic if infused for more than 24 hours.

OBSOLETE

19.22 Other Heavy Metals

- e.g. arsenic, mercury.
- Treatment - British anti-Lewisite (BAL or dimercaprol).
- May be very toxic even after the ingestion of small amounts. Seek advice.

19.23 Organophosphates

- Bind cholinesterase causing cholinergic toxicity.
- Plasma cholinesterase level will guide treatment (red cell levels are probably better but less readily available).
- Cholinesterase levels (% of normal):

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

- Treatment:
  - For skin contact rapidly wash with soap and water.

- Wear gloves when handling the patient and ensure the room is well ventilated.
- Suxamethonium will have very prolonged effects, so avoid if possible.
- Atropine 1 mg test dose. If atropinized by this dose (i.e. dilated pupils, dry mouth, flushed skin and decreased pulmonary secretions on auscultation) then the diagnosis is wrong. If not atropinized then repeat 2 mg every 10-15 minutes until desired effect (massive doses may be required). An infusion of 0.5 mg/kg/hr may be required.
- Pralidoxime:
  - Regenerates 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.

19.24 Paraquat

OBSOLETE

- Small amounts can be fatal (1-6 g).
- GI tract absorption is both slow and prolonged.
- After initial oropharyngeal burning, the onset of systemic symptoms is often delayed
- Actively taken up by lungs and lung injury will often prove fatal if early renal failure does not.
- Treatment:
  - Avoid O_2 (oxygen worsens pulmonary toxicity) unless profoundly hypoxic.
  - Aggressive attempts to decontaminate the GI tract are the most useful intervention.
  - Begin with Fullers Earth 60 g in 200 ml of water taken orally.
  - Perform gastric lavage and then instill Fullers Earth 70 g in 100 ml of glycerol made up to 1 litre of water (use the same dose of Bentonite if Fullers Earth not available) **and** mannitol 20% 3 ml/kg - pour this into the stomach before removing the orogastric tube.
  - Irrigate eyes and skin if contaminated.
  - Give further doses of Fullers Earth, as made up above, 500 ml at 4 and 8 hours.
  - Transfer to ICU and commence whole bowel irrigation.

20. Psychiatry

Disordered behaviour occurring in a non-psychiatric hospital may arise in the following circumstances

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).

As a severe disagreement or misunderstanding in staff/patient relations, sometimes augmented by alcohol or other drug abuse.

Occasionally as a factitious disorder, feigning craziness for some obscure advantage.

20.1 Delirium

20.1.1 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 hyperosmolar 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 multi infarct dementia; TIA; subarachnoid haemorrhage; subdural haematoma; hypertensive encephalopathy; SLE.
  - Epilepsy: post-ictal; psychomotor; petit mal status.

OBSOLETE

20.1.2 Characteristic Features

- Rapid onset.
- Fluctuating course.
- Night-time worsening.
- Impairment of consciousness - usually mild but patient may be barely rousable; a 'clouding' of consciousness, diminished attention, reversed sleep/wake cycle.
- Disorientation in:
  - Time - often the first noticed.
  - Place - especially in unfamiliar surroundings
  - Person - seldom unless 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 or 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).

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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).

Prompt and accurate diagnosis is therefore the key to management.

Do not confuse delirium and dementia

Table 35: 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	++	-

Key

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

OBSOLETE

20.1.3 Management

- Seek, identify and treat underlying cause.
- Symptomatic treatment involves:
  - Optimal nursing care:
    - Close, sympathetic surveillance.
    - Humane restraint - see guidelines.
    - 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), 5-10 mg

(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. Control usually achieved within 45 minutes.
Maximum daily dosage 80-100 mg. Extra pyramidal side effects rare with IV use. With oral haloperidol, benztropine 2 mg IV/PO may be required.

- **Benzodiazepines** are useful adjuncts to enhance sedation. Use short acting in delirious states (e.g. midazolam) and long acting in alcohol or drug withdrawal states (e.g. diazepam).
- **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 elderly, and those underweight will need cautious dosage.
- Do not stop habitual benzodiazepines abruptly and censoriously (especially the short-acting).
- Benzodiazepines are rarely indicated.
- Monitor treatment closely, adding and subtracting according to response. Try to have patient free of psychotropic drugs by discharge.

Note:
A time lag of a few days (sometimes weeks) commonly occurs between the apparent resolution of an organic cause and the resolution of a delirium.

OBSOLETE

20.2 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.

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 aggression, 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.

20.3 Some Oral Parenteral Antipsychotic Drugs

Range of dosage suggested

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Table 36: Oral Parenteral Psychotic Drugs

Drug	Acute (mg)		Daily
	IM	Oral	
Haloperidol	1 - 10	5 - 15	5 - 80
Chlorpromazine	50 - 100	75 - 150	50 - 1200
Thioridazine	N/A	75 - 150	50 - 800
Risperidone	N/A	1 - 2	4 - 6
Olanzepine	N/A	5 - 10	5 - 20

Common side effects

- **Low potency** (chlorpromazine, thioridazine).
  - Sedation
  - Hypotension.
  - Marked anticholinergic effects; dry mouth; constipation; urinary retention.
- **High potency** (haloperidol).
  - Extrapyramidal effects (especially oral dosage in range 5-20 mg/day).
  - Dystonia (torticollis, opisthotonus, oculogyric crisis).
  - Dyskinesia.
  - Akathisia.

- **Anti-parkinsonian agents for extra pyramidal side effects**

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)
Procyclidine	5-10 mg (PO)

- Atypical Neuroleptics (risperidone, olanzepine)

  Side Effects

  - Weight gain
  - Agitation (initially)
  - Less extrapyrimidal

Some practical management guidelines

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

OBSOLETE

20.4 Major Depressive Disorder

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

- Chance association with other disorders.
- 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, anhedonia, depressive delusions) are more discriminative.

20.4.1 Management

- Beware of sensitivity to the side effects of antidepressant medications, particularly the tricyclics.
- Selective serotonin re-uptake inhibitors (fluoxetine, paroxetine, citalopram) and reversible inhibitors of monoamines (moclobemide) may be better tolerated than tricyclics. These two groups of drugs should not be used together and a suitable lag time should be allowed when changing from one to another (Consult Drug Information Ext: 80900).
- If rapid response is critical, ?methylphenidate (but urgent consult recommended in this setting, Ext 80900)
- ECT should not be forgotten as an option. Consult.
- The physically ill should not be allowed to suffer an untreated depressive disorder.

OBSOLETE

20.5 Drug Withdrawal Management

- Benzodiazepine withdrawal syndrome.
  - Occurs from 1-2 days (short acting) to weeks (long acting) after cessation
  - Enhanced anxiety, anorexia, tremor, seizures, delirium.
  - Diazepam 10-20 mg PO hourly until control then reduce dose by 5-10% daily.
- Opioid Intoxication.
  - Naloxone 0.2 mg IV and repeat in 5 minutes. May need up to 4 mg IV in this situation. Monitoring essential as the effect of naloxone can wear off before that of the opioid.
- Opioid withdrawal syndrome.
  - Not life threatening but very unpleasant.
  - 2-3 days post last dosage, but duration varies with the opioid.
  - 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 problems and initiate therapy.

21. Respiratory Medicine

Main Office 2nd Floor, Riverside ☎ 80280 Fax 80914

Inpatient Services Three inpatient teams on Ward 25:

- Resp 1 - Dr P. Thornley / Dr I.Town / Dr L. Beckert
- Resp 2 - Dr C. Drennan / Dr J. Gillies
- Resp 3 - Dr R Meyer

Consultation and On-call Service

24 hours a day, seven days a week, on a rotational basis. For consultations fax the referral to 80914. For urgent problems contact the Acute Respiratory Registrar or the Acute Respiratory Physician through the hospital operator.

Consultation Guidelines

Respiratory failure, sleep apnoea, complicated pneumonia, severe COPD/asthma, pleural effusion of unknown cause, pulmonary mass lesions or infiltrates, pneumothorax, bronchiectasis or suspected TB.

Other Services

Respiratory Laboratory	☎ 80874	Fax 80878
– For spirometry, allergy skin tests and blood gases		
– For other tests contact Maureen Swanney, Technical Director	☎ 80924	
– Some tests require Respiratory Physician approval		
Outreach Service	☎ 80167	Fax 80849
– For home-based service and education		
– Domiciliary oxygen service		
– Respiratory rehabilitation programme		
– Maori Respiratory Educator		
– Smokescreen Educator - Prue Fraser	☎ 88029	026 358 1591 (pager)
Education Service		
– Glenys Martin, Respiratory Education Nurse	☎ 81140	Fax 81260
Respiratory Outpatients		
– Enquiries and appointments	☎ 80280	Fax 80914
– Clinic Nurse	☎ 80463	Fax 81260
Respiratory Research Group		
– Virginia Hewat, Research Manager	☎ 81157	Fax 81184
Sleep Unit		
– Bryn Sparks, Technical Director	☎ 81089	Fax 81089
Burwood Respiratory Rehabilitation Unit (Ward 7)	☎ 9778	Fax 9847

OBSOLETE

21.1 Respiratory Failure

21.1.1 Definition

Respiratory failure is defined as occurring with PaO_2 <60 mmHg, or $PaCO_2$ >50 mmHg in a patient at rest breathing air. Respiratory failure is **not a disease** but reflects the inability of the lungs to maintain normal gas exchange.

21.1.2 Classification

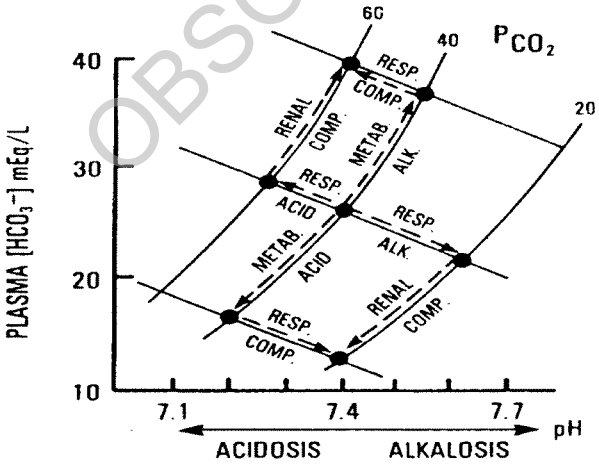
- Type I Respiratory Failure (gas exchange/hypoxaemic) - causes include pulmonary oedema, infections, inflammatory lung disease and pulmonary embolism.
- Type II Respiratory Failure (ventilatory/hypercapnic) - causes include COPD, asthma, massive obesity, kyphoscoliosis, CNS depression due to drugs, neuromuscular disease and pneumothorax.

Both types of respiratory failure may be **acute** or **chronic.**

Interpretation of Blood Gases This is not covered in this section. *Please refer to medical texts. See also the Endocrine Metabolic Section (Page 107).*
The following diagram may assist:

OBSOLETE

Figure 8: Blood Gas Interpretation



Patient assessment
The **underlying cause** for the respiratory failure must be determined to enable appropriate treatment in each case.

21.1.3 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. Unless prompt recovery is anticipated, such patients should be managed in ICU. An oropharyngeal airway should be used pending recovery or intubation.
- **Reversal of Precipitating Cause**
  - Always consider the possible contribution of infection, cardiac failure and bronchospasm. These may not be the primary cause of the respiratory failure but are readily treatable.
  - Drug induced – 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.
  - CPAP may be useful in LVF, COPD and some other pulmonary conditions. Patients who need CPAP should be managed in ICU. Discuss with ICU/Respiratory Physician.
- **Clearance of endobronchial secretions**
  This may improve ventilation and help prevent atelectasis and infection. Patients may require regular chest physiotherapy to:
  - Encourage effective coughing.
  - Maximize inspiratory effort.
  - Facilitate postural drainage.
- **Oxygen Therapy** *(see page 226)*
- **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 since they may be able to assist in the detection of subsequent changes in the patient's clinical status.
  - Indications for mechanical ventilation:
    - Severe hypoxia (<50 mmHg) despite high (>50%) inspired oxygen concentration.

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- Significant hypoxia (<60 mmHg) and or hypercapnia (>45 mmHg) along with
  - Diminished/ing level of consciousness
  - Diminished/ing chest expansion.
  - Evidence of respiratory muscle fatigue.
  - Sputum retention.
  - Thoracic cage trauma/lung contusion.

21.2 Obstructive Sleep Apnoea (OSA)

- Classically presents with day time hypersomnolence, and nocturnal snoring and/or apnoea[s]. It is a common and frequently missed diagnosis. Consider OSA in patients who have:
  - Unexplained respiratory or right sided 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.

OBSOLETE

21.3 Chronic Obstructive Pulmonary Disease (COPD)

21.3.1 Definition

- COPD is not a disease process but a clinical syndrome and consists of various admixtures of:
  - Chronic bronchitis.
  - Emphysema.
  - Small airways disease.
  - Bronchial hypersensitivity/asthma.

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

21.3.2 Aetiology

- Smoking.
- Asthma.
- Bronchiectasis.
- Occupational exposures (cadmium, silica dusts).
- Cystic fibrosis.
- Alpha-1 antitrypsin deficiency.

Causes of acute deterioration

- Bronchitis (viral or bacterial).
- Increased bronchial irritability.
- Pneumonia.
- Pneumothorax.
- Pulmonary embolism.
- Left ventricular failure.
- Sepsis.
- Drugs (e.g. β-blockers, NSAIDs).
- Acute abdomen.
- Chest pain (e.g. trauma, osteoporosis).
- Post-operative sedation / retention of secretions.

OBSOLETE

21.3.3 Investigations

- CBC, Na, K, creatinine.
- Peak expiratory flow rate (PEFR/spirometry).
- Arterial blood gases (not oximetry alone).
- Sputum culture.
- CXR.
- ECG.
- Theophylline concentration.
- BNP [brain natriuretic peptide] - to assess possible contribution of LVF.

21.3.4 Severity Assessment in COPD

Make an immediate assessment of severity (see table) and initiate treatment accordingly. Confirm the diagnosis, identifying precipitating factor(s) and estimate the degree of functional impairment, **referring to the old case notes** for information about previous functional status, spirometry and blood gas analyses.

Table 37: Severity Assessment in COPD

Emergency: respiratory arrest, unconscious patient, upper airway compromise			
Other Categories:-	**Mild**	**Moderate**	**Severe**
Speech	Sentences	Phrases	Words only
Respiratory rate (per minute)	Normal	18-25	>25 or <12
Pulse rate (per minute)	<100	100-120	>120
PaO_2 (related to steady state level)	Normal	<60 (on air)	<60 (on O_2)
$PaCO_2$ * (related to steady state level)	Normal or reduced	>45 (on air)	>50 (on air or O_2)
pH	Normal	Close to normal	Falling (<7.3)

* If the HCO_3 level is raised and pH normal this suggests chronic CO_2 retention.

21.3.5 Management

- **Emergency Action**
  - Call ICU immediately.
  - Prepare for emergency intubation and assisted ventilation.
  - Initiate 'urgent action" (see below).
  - Notify Respiratory Physician or General Physician on call.
- **Severe exacerbation**
  - Commence controlled oxygen therapy, aiming to maintain a PaO_2 >60 mmHg or SpO_2 (haemoglobin oxygen saturation) >90% (see section on Oxygen Therapy).
  - Monitor for rising $PaCO_2$ (see Section on Oxygen Therapy). If pH <7.3 or failure to respond to initial therapy, notify Respiratory Physician or General Physician on call.
  - Nebulized combivent or salbutamol 5 mg + ipratropium 0.5 mg stat and 2-6 hourly according to clinical response. Use compressed air if $PaCO_2$ elevated.
  - Insert IV line for either salbutamol infusion (refer to section on Asthma) or theophylline (refer to table below).
  - Consider hydrocortisone 200 mg IV q6h.
  - Consider the use of CPAP if intubation inappropriate.

OBSOLETE

- Consider IV antibiotics if the patient has two out of three of the following:
  - Purulent sputum
  - Increased sputum production
  - Increasing dyspnoea
- The choice of antibiotics is essentially the same as recommended for Community Acquired Pneumonia. *(see page 237)*
- Consider chest physiotherapy if concerns about sputum retention.

Table 38: Theophylline Therapy

General Comments
♦ Therapeutic range (55-100 µmol/L). ♦ Some patients respond at lower concentrations. ♦ Factors that ↑ [theophylline]: hepatic dysfunction, severe cardiac failure, older age, febrile illness, drugs inhibiting CYP1A2 (e.g. *erythromycin, clarithromycin, ciprofloxacin, cimetidine, amiodarone*). ♦ Factors that ↓ [theophylline]: cigarette smoke (↓ theophylline by 50%), cystic fibrosis, high carbohydrate diet, CYP1A2 inducers (e.g. *carbamazepine, phenytoin, rifampicin, charbroiled foods*). ♦ IV Aminophylline dose = oral theophylline dose ÷ 0.8. ♦ Measure concentrations 8-12 hr after initiation of infusion, then 12-24 hr after any modification of the dose. When taken orally, measure concentration 12 hours after the last dose. ♦ Haemoperfusion useful in severe toxicity.
Intravenous Aminophylline Therapy
Loading Dose 5 mg/kg based on ideal body weight. Mix with 100 mL of 0.9S and give over 20 minutes. **Omit loading dose if on oral theophylline.**
Maintenance Dose Add 500 mg aminophylline to 500 mL of 0.9S (1 mg/mL). Infuse at between 0.3 and 0.9 mg/kg per hour. Monitor concentration daily aiming for 55-100 µmol/L. Doses recommended for intravenous aminophylline: Calculated (mg/kg/hr) Non smoker 0.5-0.7 Smoker 0.9 macrolide/ciprofloxacin/rifampicin/SSRI 0.3-0.4 Cor pulmonale or hepatic insufficiency 0.25

OBSOLETE

- **Mild or Moderate exacerbation**
  - Oxygen *(see Section on Oxygen Therapy Page 226)*
  - Nebulized combivent (or salbutamol 5 mg + ipratropium 0.5 mg). Repeat 4-6 hourly according to clinical response. Use compressed air if $PaCO_2$ elevated.
  - Oral prednisone 40 mg stat; then 40 mg mane until clinical response adequate; then 20 mg mane for an equal number of days; then stop or reduce to usual maintenance dose.
  - Oral antibiotics may be appropriate.
  - Consider chest physiotherapy.
- **Monitor Progress**
  - Oxygen therapy:
    - Monitor SpO_2 and aim to maintain SpO_2 >90%.
    - Monitor for hypercapnia (symptoms of drowsiness and / or confusion).
    - Perform ABG if evidence of falling SpO_2 or clinical deterioration.
  - Clinical monitoring:
    - Check for fatigue – beware respiratory paradox.
    - Pulse rate.
    - Sputum volume and appearance.
    - PEFR/spirometry.
  - Adjustment of treatment:- individual patient needs may change during the course of treatment including the frequency and dose of nebulized bronchodilator, intravenous therapy (particularly aminophylline according to blood concentration), fluid and electrolyte requirements and bronchial secretions (chest physiotherapy for retained bronchial secretion). Commence oral therapy as soon as condition stabilizes. Bronchodilators should be given by Metered Dose Inhaler (MDI). Use a spacer if patient has any problem with MDI use.

- **Discharge Planning**
  - At Christchurch Hospital consider transfer to Burwood Respiratory Ward 7 for ongoing treatment and rehabilitation if indicated. Contact the General Medicine Co-ordinator, Beep 492 to discuss transfer.
  - Most patients will be ready for discharge when their functional status has returned to near their pre exacerbation state. When this has been achieved, contact should be made with the patient's usual family practitioner and other health professionals as appropriate (see below).

OBSOLETE

- It is helpful to obtain spirometry and ABG at discharge.
- A COPD Action Plan should be considered for all patients. A COPD discharge check list is available and is useful to ensure all relevant actions have been considered – contact Respiratory Outreach for copies of the Action Plan and discharge form.
- Outpatient Management
  - Support services:
    - Respiratory Outreach.
    - Domiciliary Services.
  - Preventive measures:
    - Rehabilitation programme.
    - COPD education and Action Plan.
    - Smoking cessation (Smokescreen educator at Christchurch Hospital).
    - Nutritional advice and supplements.
    - Influenza vaccine each autumn.
    - Pneumococcal vaccine (revaccinate every 5 years).
  - Medical surveillance:
    - Family practitioner - regular follow up.
    - Respiratory Outreach - long term follow up for high risk patients.
    - Specialist review, as required, with spirometry. Recommended for those with severe disease [FEV_1 <30% predicted or PaO_2 <55 mmHg].

OBSOLETE

21.4 Oxygen Therapy

AIM - to prevent important tissue hypoxia and thereby reduce morbidity and mortality. There is virtually no evidence based data on the therapeutic use of oxygen in most acute clinical situations.

21.4.1 Background

Tissue oxygenation depends on two factors:

- Tissue perfusion - affected by cardiac output and peripheral vascular resistance.
- Arterial oxygen content - this is determined by the haemoglobin content and haemoglobin oxygen saturation.
  The latter is the only factor affected by oxygen administration.

21.4.2 Indications

- PaO_2 less than 60 mmHg or SpO2 <90%.
- Conditions such as myocardial infarction, CO poisoning, acute/severe anaemia where marginal increases in arterial oxygen content may be beneficial.
- At risk of hypoxia such as post-op, LVF etc.

21.4.3 Pulse Oximetry

- This is very useful for determining haemoglobin oxygen saturation (SpO_2) i.e. oxygenation. However, it does **not** assess haemoglobin level, ventilation (CO_2) problems, cardiac output or tissue perfusion. It's useful for monitoring but is not a substitute for arterial blood gases.
  Remember that changes in PaO_2 above 100 mmHg will not change the haemoglobin oxygen saturation.
  Oxygen therapy is indicated primarily to relieve hypoxia **not** dyspnoea.

21.4.4 Administration

OBSOLETE

- Oxygen is a drug and must be prescribed on the drug administration chart indicating flow rate and device.
- Do not withhold oxygen in severely hypoxaemic patients merely to get a "baseline blood gas estimation."
- Do monitor oxygen administration carefully according to the clinical circumstances.
- **Nasal cannulae:** 0.5-4 L/min, provide an inspired oxygen concentration of 24% to 40% depending on the flow. Remember that this is uncontrolled oxygen therapy and it is not possible to accurately predict the inspired oxygen concentration (FIO_2). Most 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. They allow oral intake, communication and the easy use of nebulisers. They do not cause the sense of suffocation some patients have with a face mask. For a flow rate of 0.5 L/min you will need a low flow oxygen meter.
- **Variable concentration mask:**
  - Use initially in COPD patients during the acute phase.
  - Use 24% initially when there is a possibility of CO_2 retention (check previous case notes).
- **Standard mask**
  This is also uncontrolled oxygen therapy. 6-10 L/min, provides about 50% oxygen depending on the patient's ventilation levels. The initial method of choice in acutely hypoxic patients i.e. acute asthma, pneumonia, LVF and

pulmonary embolism. Don't use these at flow rates less than 6 L/min as CO_2 retention can occur through rebreathing. A reservoir bag can further increase the percentage oxygen.

- **High flow humidified**
  Used for long term therapy where drying of the bronchial secretions needs to be avoided. It is only indicated in special circumstances but can provide more accurate inspired oxygen concentrations than other methods. Contact ICU or Respiratory Ward 25.

21.4.5 Adjusting the Dose

- Do the ABGs show evidence of chronic CO_2 retention? i.e. a compensated respiratory acidosis (elevated HCO_3 level), together with chronic hypoxaemia. If so, take care to avoid CO_2 retention.
- Using a pulse oximeter as a monitor, adjust the flow rates:
  - For nasal cannulae in 0.5 - 1 L/min steps.
  - For variable concentration masks in percentage increments.
  - For standard masks in 2 L/min steps.
- Get the haemoglobin oxygen saturation (SpO_2) to about 90%, wait about five minutes at each step for those with COPD.
- Once stable, if there is any risk of CO_2 retention, check the blood gases about 30 minutes later.

The predicted oxygen percentages supplied by masks and nasal cannulae are not precise.

OBSOLETE

21.4.6 Monitoring

- **Pulse oximetry provides an estimate of capillary haemoglobin oxygen saturation. It does not assess the adequacy of ventilation nor the gas exchange status.**
- Arterial blood gas analysis must be performed on admission and in many cases at regular intervals to assess response to treatment.
- 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 $PaCO_2$ may rise 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.

21.5 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 (pollen, animal dander, viral infection) or non specific. Typical symptoms include dyspnoea, wheeze, chest tightness and cough. They vary from being almost undetectable to severe, unremitting and sometimes life threatening.

The aims of hospital 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.

Diagnosis of Acute Asthma

Assess Severity

Immediate Treatment

Subsequent Management

OBSOLETE

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

All patients should have the following measured:

- PEFR/spirometry.
- Respiratory rate.
- Pulse, blood pressure, temperature.
- Pulse oximetry/arterial blood gases.

At Christchurch Hospital the Asthma Admission Form should be used for ALL admissions.

21.5.1 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 clinical judgement and the following guidelines:

Table 39: Severity Assessment in Acute Asthma

	Severity		
	Mild	**Moderate**	**Severe**
Speech	Sentences	Phrases	Words
PEFR (% of predicted or previous best)	>60%	40-60%	Less than 40% or less than 150 L/min if best peak flow unknown
FEV_1 (% Predicted)	>60%	40-60%	<40% or absolute value less than 1.0 L
Respiratory rate	Normal	18-25	>25, <10
Pulse rate	< 100	100-120	>120
Oximetry	>94%	90-94%	<90%
PaO_2	Test not necessary	<80 mmHg	<60 mmHg
$PaCO_2$	Test not necessary	<40 mmHg	≥40 mmHg

OBSOLETE

DANGER SIGNS:

Exhaustion, confusion, cyanosis, bradycardia, unconsciousness, silent chest on auscultation, signs of respiratory muscle fatigue (indrawing of lower costal margin, abdominal paradox).

21.5.2 Immediate Management

Specific treatment is dependent on severity. All patients should be treated with nebulised bronchodilator in the first instance. Other therapy is added depending on the response and reassessment of severity. Table 40 gives guidelines for the management of patients with asthma according to severity.

Table 40: Management of Asthma

Severity	Management		Monitoring
Mild	Nebulized salbutamol 5 mg q4h + prn.* Prednisone 40 mg orally stat then daily		PEFR after initial treatment then QID. Pulse, respiratory rate QID
Moderate	Nebulized salbutamol 5 mg q4h + prn* Prednisone 40 mg orally stat then daily		PEFR 2-4 hourly. Pulse oximetry.** Pulse, respiratory rate, BP QID. Monitor for hypokalaemia which may be exacerbated by β-agonist therapy
	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 †	
Severe	Increase	Nebulized salbutamol 5 mg up to 2 hourly. Nebulized ipratropium 0.5 mg q4h. Oxygen 8L/min by Hudson mask. Adjust to maintain O_2 sat >95%	ICU or high dependency unit. Oximetry / arterial blood gases Continuous ECG. Pulse, respiratory rate, BP 2 hourly. Special nurse. Serum potassium 12 hourly
	Add	Intravenous access. IV hydrocortisone 200 mg stat then q6h (for 24 hours). Fluids 0.9S 1L 6 hourly initially. IV bronchodilator if not responding to nebulized bronchodilator ††	
	Contact	Respiratory Physician	
	Perform	CXR in all cases †	
Danger Signs Present	Increase	Oxygen to high flow system	Resuscitation or Intensive Care Unit. Nurse and doctor to stay with patient at all times
	Contact	ICU team + Respiratory Physician	
	Add	IV salbutamol 250 mcg loading dose then salbutamol infusion (5 mg/100 ml) at 10-30 ml/hr. See ††	

OBSOLETE

Notes:

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

** Pulse oximetry is very useful in assessing the adequacy of tissue oxygenation in

patients with 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.

† Patients with life threatening asthma, or severe asthma not responding to initial treatment, and patients in whom there is any suspicion of a complication require a CXR. Complications which might be identified 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.

†† Intravenous bronchodilators:

Salbutamol	Loading dose 250 mcg IV or IM. Prepare intravenous infusion by adding salbutamol 5mg/5mL and make up to 100 mL with D5W. Infuse at 10-30 mL/hr.
or	
Aminophylline	*(see page 224)*

Table 41: Key Points - Acute Severe Asthma in Adults

21.5.3	**Life Threatening Features** • FEV_1 or PEFR <33% predicted (or of usual best). • Silent chest, cyanosis, or feeble respiratory effort. • Bradycardia or hypotension. • Exhaustion, confusion or coma.
21.5.4	**Management** • High Flow Oxygen (40 – 60%). • Salbutamol + Atrovent via oxygen driven nebuliser (initially continuous). • Loading Dose IV salbutamol 250 mcg with subsequent infusion (5mg/5mL salbutamol made up to 100 mL with D5W, infuse at 10 – 30 mL/hr). • CXR to exclude pneumothorax. • ICU or Respiratory team review.
21.5.5	**Important Points** • Pulse oximetry does not assess adequacy of ventilation – ABG must be measured. • Patients with life threatening asthma may not be distressed. • A normal CO_2 in an asthma attack is a marker of severe disease.

OBSOLETE

21.5.6 Subsequent Management

Depends on the severity of the attack and the patient's response to initial treatment.

- **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 PEFR (or FEV_1) 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 $PaCO_2$ high normal or raised.
    - The patient's condition deteriorates.

    Measure and record heart rate and respiratory rate, at least QID. Theophylline concentration should be measured daily if IV aminophylline is continued for more than 24 hours. Serum K and glucose daily.

- **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).

- **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 0.5 mg q6h.
    - Consider using an intravenous bronchodilator.

OBSOLETE

- Consider the possibility 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.

♦ **Unhelpful Treatments**

- Sedatives are usually contra-indicated.
- Antibiotics are not indicated unless there is evidence of bacterial infection (fever, purulent sputum, CXR opacity).
- Percussive physiotherapy.

♦ **Indications for Intensive Care**

Patients with the following features usually require observation and management in ICU:

- Hypoxia: PaO_2 <60 mm Hg despite receiving high flow oxygen.
- Hypercapnia: $PaCO_2$ >50 mmHg or rising.
- Increasing fatigue.
- Confusion, drowsiness, impaired level of consciousness.
- Respiratory arrest.

OBSOLETE

♦ **Management During Recovery and Following Discharge**

- Once the acute episode has been brought under control, attention must be directed towards:
  - Interval asthma control.
  - Severity assessment - what is the risk of severe asthma recurring?
  - Self-management skills.
- Interval asthma control should be assessed by specific questioning directed at the following features:
  - Nocturnal waking and morning chest tightness.
  - Interference with exercise.
  - Use of rescue bronchodilator.

- Peak flow values.
- Days off work or school.
- Use of corticosteroids and nebulizer for exacerbations.
- Compliance with preventer therapy.

Note:

- These features are itemized on the Asthma Admission Form used at Christchurch Hospital. Copies are available from the Department of General Medicine or Respiratory Ward 25.
- Patients with unstable features or poor compliance should be referred **to a Respiratory Physician, preferably while in hospital.**

21.5.7 Severity Assessment

- The risk of a severe or fatal asthma attack is associated with:
  - Hospital admission for asthma in the last 12 months.
  - Previous severe asthma requiring ventilation or ICU admission.
  - Frequent attendances to the emergency department.
  - Nocturnal symptoms.
  - Precipitous asthma episodes in the past - severe episodes coming on over less than 3 hours.
  - Frequent requirement for courses of oral steroids.
  - Poor self-management skills.
  - Poor social circumstances.
  - Psychological impairment.

OBSOLETE

21.5.8 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?
  - Was there any 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 respiratory physiotherapist.
- Check 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 Physician.
- Respiratory Nurse Educator - Respiratory Outpatient Unit.
- General Practitioner/Practice Nurse.
- Community Asthma Educator - Canterbury Asthma Society.

OBSOLETE

21.5.9 Treatment on Discharge

♦ This will obviously vary from case to case but usually the patient will receive:

- Inhaled corticosteroid - beclomethasone or budesonide 800-2000 mcg daily or fluticasone 500-1000 mcg daily.
- Prednisone 40 mg mane for 1 week then 20 mg mane for 1 week (longer courses may be required for chronic severe asthma).
- A long acting β_2- agonist may be appropriate in some patients, but is best started once they have recovered from an acute attack. It should be considered in patients with frequent daytime and nocturnal symptoms.
- β_2- agonist inhaler to use as required (**NOT** regularly).
- Advice regarding common side effects of these medications:

β_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, insomnia.
Theophylline	indigestion, insomnia.

Note:
For patients prescribed theophylline arrangements will need to be made for a theophylline concentration to be measured following discharge.
Patients prescribed inhaled corticosteroids in higher doses (>800 mcg) should be encouraged to use a large volume spacer device.

21.6 Community Acquired Pneumonia (CAP)

21.6.1 General Points

- The clinical features and initial investigations do not provide a reliable guide as to the causative organism.
- The initial treatment is therefore usually empiric and based upon assessment of the severity of the pneumonia.

21.6.2 Diagnosis

- Differential diagnosis includes - lung carcinoma, lung infarction, cardiac failure, ARDS and chronic intersitial lung diseases. Don't forget that acute intra-abdominal conditions can mimic pneumonia. e.g. acute pylonephritis.
- The diagnosis is easily missed in the elderly who may present with non specific signs and symptoms.

21.6.3 Assessing the Microbiological Causes

- Gram stain of sputum sometimes provides a guide to initial therapy.
- Streptococcus pneumoniae causes 50-70% of cases.
- Legionella is a common cause of pneumonia in Christchurch.
- With advancing age (>65 years) Gram negative and Staphylococcal pneumonia become relatively more common.
- Seasonal variation occurs - influenza epidemics occur in autumn and early winter, Legionella is more common in summer.
- Patients with chronic lung disease often develop 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 3-4 years.

Note:

- Abnormal liver function tests and gastrointestinal symptoms may occur with any type of pneumonia.
- Patients with mycoplasma pneumonia have often had symptoms for 2-3 weeks and have failed to respond to β-lactam therapy prior to admission. Myalgias are common.

21.6.4 Assessing Severity

- This is essential as it directly influences initial management and patients with severe pneumonia can deteriorate rapidly.

OBSOLETE

- **Indicators of severe pneumonia at or during admission**
  - Respiratory rate >30/min.
  - Diastolic BP <60 mmHg.
  - Confusion (MSQ <8).
  - Blood urea >7.0 mmol/L.

  The presence of two or more of these criteria indicates an increased risk of death. Such patients must be monitored carefully and receive dual or triple antibiotic therapy *(see page 240)*.
- Additional severity indicators include:
  - Age > 60 years.
  - Pre-existing medical conditions (especially cardiorespiratory and neurological).
  - PaO_2 <55 mmHg on air or oxygen.
  - WBC <4 x 10^9/L or >30 x 10^9/L.
  - CXR evidence of multiple or spreading infiltrates.

  Patients with these features should also be carefully monitored for signs of deterioration.

OBSOLETE

21.6.5 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 water prior to collection.
    - Prior antibiotic usage must be recorded.
    - Sputum may be refrigerated (4°C) for up to 24 hrs, but must reach the lab within 4 hrs of warming to room temperature.
    - Consider whether specific tests are indicated:
      - Pneumococcal antigen.
      - Legionella immunofluorescence, PCR.
      - ZN stain and culture for TB.
      - Stains for Pneumocystis carinii.
  - Blood cultures - 1 set prior to antibiotics (10mL in each bottle).
  - Oximetry (or ABGs for severe cases or where there is chronic respiratory or cardiac disese.)

- Serology - acute specimen for the following:
  - Respiratory viruses.
  - Legionella species.
  - Mycoplasma pneumoniae (IgM and IgG).
- Throat and nasopharyngeal swabs for viral antigen detection and culture especially if influenza is suspected.
- Urine and serum for Legionella PCR [contact laboratory].

♦ **Additional Investigations**

- Pleurocentesis: *(See page 36 for advice on how to do this)*
  - Should be performed when a significant [>1 cm on lateral decubitus CXR] parapneumonic effusion is present on CXR. Inexperienced staff must be supervised.
  - Send for Gram stain, culture, total and differential WBC, pH, total protein, glucose, LDH and pneumococcal antigen.
    Note:
    For pH estimation the fluid must be sent in a **capped** ABG syringe. Transfer 2 mL from the specimen pottle as soon as possible after taking.
- Bronchoscopy. Indications include:
  - Immunosuppressed patient.
  - Life threatening pneumonia.
  - Multiple CXR changes.
  - Deterioration despite appropriate initial treatment.

  Contact the Respiratory Physician on-call.

OBSOLETE

21.6.6 Management

♦ Assess severity.
♦ Resuscitate.
♦ Choose antibiotics:
- Usually empiric.
- Depends on:
  - Clues as to likely pathogen.
  - Severity.
  - Initial Gram stain.
- **Antibiotics must be administered without delay in patients with pneumonia.**
- Initial antibiotic MUST cover Streptococcus pneumoniae.
- Obtain blood cultures prior to giving antibiotics.

- Obtain sputum sample as soon as possible - involve a physiotherapist if necessary but do not delay giving antibiotics while awaiting a sputum sample.

21.6.7 Recommendations for Initial Antibiotic Treatment

♦ **Mild-Moderate Community Acquired Pneumonia**

- Those patients without any of the major risk factors for increased mortality *(see page 238).*

Table 42: Antibiotic Treatment for Community Acquired Pneumonia (mild – moderate)

	Targets	First choice	Alternatives
Majority - young, non-smoking, no underlying lung disease	Must cover streptococcal pneumonia	Amoxycillin 1g q8h IV	Benzylpenicillin IV Clarithromycin IV
Older patient COPD Smokers	Must cover H. influenzae and M. catarrhalis	Augmentin 1.2g q8h IV	Cefuroxime IV
Mycoplasma or Legionella suspected		Clarithromycin 500 mg q12h IV	Tetracycline* Oral Ciprofloxacin
Little improvement wih initial therapy	Chlamydia species Legionella species	Clarithromycin 500 mg q12h IV	Tetracycline*

OBSOLETE

*No IV preparation available
Change to oral therapy once patient afebrile for 48 hours and clinically improving.

♦ **Severe Community Acquired Pneumonia**

- Antibiotic choice is generally empiric and should cover most bacterial causes.
  - **Augmentin 1.2 g IV q8h and clarithromycin 500 mg q12h IV.**

  Note:
  - Mix IV clarithromycin in 250 ml of 0.9S and give via a large vein or CVP as phlebitis is a common complication.
  - If mild penicillin allergy suspected give cefuroxime 1.5 g IV q8h instead of Augmentin.
  - If staphylococcal pneumonia suspected - add flucloxacillin 2 g q4h IV.
  - If strongly suspect Legionella infection and patient's condition is deteriorating, add rifampicin 600 mg PO q12h or ciprofloxacin 500 mg PO q12h and contact Respiratory or Infectious Diseases Physician.

- **Life Threatening Pneumonia - Use ceftriaxone 2 g q12h IV, gentamicin *(see page 47)* and clarithromcyin 500 mg q12h IV.**

21.6.8 Initial General Management

- Controlled oxygen therapy - prescribed on basis of arterial blood gases/oximetry *(see page 226)*.
- Insert intravenous line (large vein or PICC if using IV clarithromycin).
- 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 (see above).
- 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). Ensure that nursing staff report any change promptly to medical staff.
- Physiotherapy - indications:
  - May be useful to assist in obtaining a sputum sample.
  - May help with sputum clearance, especially in patients with underlying COPD.

OBSOLETE

21.6.9 Subsequent Management

- Patients must be reviewed regularly to ensure that they are not deteriorating. Beware of tachycardia, tachypnoea, hypotension and SpO_2 <92%.
- All cases of severe CAP should be discussed with:
  - Respiratory Physician/Infectious Diseases 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.
- In patients who remain unwell despite appropriate antibiotic therapy, the possibility of a vasculitic disorder should be considered *(see page 249)*.
- CXR – according to clinical progress. Mild cases usually don't need a

repeat until reviewed as an outpatient or by the GP.

21.6.10 At Discharge

- Appropriate oral antibiotic:
  - Total duration of 7-10 days in uncomplicated pneumonia.
  - Longer (14-21 days) course required for complicated disease (e.g. COPD, severe pneumonia or Legionella).
- Stop smoking - refer for smoking cessation programme.
- Check spirometry in all smokers and alert GP or refer to Respiratory Physician if significantly impaired.
- Instruct patient to contact their GP if they develop fever, chest pain or increasing dyspnoea.
- Follow up appointment either with GP or hospital team at 6 weeks to include:
  - CXR - **this should be arranged by the hospital team prior to discharge**.
  - Convalescent serology if considered relevant.

  Note:
  - CXR may take up to 3 months to clear especially in older patients and those with COPD.
  - Physiotherapy may be needed if sputum retention likely.

OBSOLETE

21.6.11 Common Complications

- Parapneumonic effusion - seen in up to 40% of cases. Should always be aspirated to exclude empyema and complicated para pneumonic effusions.
- Empyema - 1-5% of pneumonia cases. Immediate consult with Respiratory Physician and/or Thoracic Surgeon.

21.6.12 Other Considerations

- Any pneumonia that doesn't resolve at usual rate - consider endobronchial obstruction, tuberculosis, or other diagnoses.
- 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.

21.7 Hospital Acquired Pneumonia (HAP)

- The incidence of HAP is around 0.7% in among adult inpatients at Christchurch Hospital.
- In post-operative patients presentation is usually with fever, deteriorating gas exchange and CXR infiltration.
- Intensive post-operative physiotherapy may help prevent HAP.
- Medical patients may become more unwell very quickly - the diagnosis should be suspected in any medical patient developing a fever.

21.7.1 Investigations

- Sputum sample - involve a physiotherapist if necessary.
- Blood cultures - 2 sets. 10 mL in each bottle.
- WBC + diff.
- CXR.

21.7.2 Management

- Physiotherapy - especially if patient has underlying lung disease.
- Oxygen if indicated.
- Bronchodilators if history of airflow obstruction.
- Antibiotics:
  - **Mild/moderate:**
    Augmentin 1.2 g IV q8h
    Add clarithromycin 500 mg q12h IV if patient immunocompromised (alcoholic, diabetes, steroids, cytotoxics) or failing to respond to initial therapy.
  - **Severe**:
    (Criteria include tachypnoea >30/min, urea >7.0 mmol/L, hypotension, PaO_2 <55 mmHg on oxygen, anyone in ICU).
    ceftriaxone 2 g IV q24h AND
    gentamicin *(see page 35)* AND
    clarithromycin 500 mg q12h IV

  Note: If on a ventilator or pseudomonas suspected substitute piperacillin or ceftazidime or imipenem for ceftriaxone.

OBSOLETE

21.8 Aspiration Pneumonitis

- Chronic occult microaspiration of gastric content 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.

21.8.1 Clinical Diagnosis

- The right upper lobe and the upper segments of both lower lobes are the pulmonary segments most commonly affected. Patients may present with indolent, multi-segmental pneumonia and a low grade fever. Others may present in respiratory failure.

21.8.2 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. Antibiotic therapy must be guided by culture results. There is no recognized standard regimen and pulmonary isolates that are antibiotic resistant are common.
  - Steroids are not helpful.
- **Microaspiration pneumonia** - antibiotics to consider include: Augmentin, penicillin and metronidazole or clindamycin. Attention must be directed towards underlying gastro-oesophageal reflux, and gingival disease.

OBSOLETE

21.9 Pleural Effusion

21.9.1 Classification

The differentiation between **exudates and transudates** is the essential first step in the diagnostic evaluation.

21.9.2 Investigations

- Diagnostic pleurocentesis *(see page 36)*. May be undertaken by medical staff with appropriate experience. A lateral decubitus CXR will allow identification of free fluid. Pleurocentesis may be performed safely if 10 mm width free fluid is identified on a lateral decubitus CXR or if loculated fluid is identified and can be reached with ultrasound guidance. Use a 20 ml syringe with a 22G needle under sterile conditions.
- Measure plasma total protein, glucose and LDH levels for comparison with pleural fluid.

21.9.3 Contraindications

- Unwilling or uncooperative patient.
- Abnormal bleeding tendency. Check history, examination, PR, APTT, and platelets. If in doubt, discuss with Consultant before proceeding.
- Insufficient pleural fluid.
- Chest pyoderma or herpes zoster.

21.9.4 Tests that should be performed on Pleural Fluid

- pH. Accurate pH measurement requires about 2 mL of fresh sample in a capped ABG syringe.
- Glucose.
- LDH.
- Total protein.
- Total and differential WBC.
- Gram stain and culture.
- Cytology.

OBSOLETE

21.9.5 Exudateor Transudate

- 99% of exudates meet one or more of the following criteria:
  - Pleural fluid total protein >30 g/L.
  - Pleural fluid total protein/serum total protein ratio >0.5.
  - Pleural fluid LDH/serum LDH >0.6.
- If transudate - further tests are usually not needed - seek cause, e.g. heart failure, cirrhosis, nephrotic syndrome, acute glomerulonephritis, peritoneal dialysis, myxoedema (but 5% malignant effusions are transudates).
- If exudate - assess the differential white cell count (total WBC is of limited diagnostic value).
  - If lymphocytes predominate consider malignancy, tuberculosis, connective tissue disease.
  - If neutrophils predominate consider parapneumonic effusion, empyema, pulmonary embolus, pancreatitis, subphrenic abscess, early tuberculosis.
- Criteria for parapneumonic effusion and empyema.

- Simple parapneumonic effusion.
  - pH ≥ 7.3, glucose >2.5, LDH <1000.
- Complicated parapneumonic effusion.
  - pH ≤ 7.2 glucose <2.5, LDH >1000.

  Note: pH 7.2-7.3 - observe closely. Repeat CXR and pleural tap if not clinically improving.
- Empyema.
  - Organisms seen on Gram stain or frank pus.

♦ Pseudoexudate. Remember – a fluid/serum cholesterol ratio of < 0.3 may be seen in patients with LVF treated with diuretics.

♦ The diagnosis and management of anything but a simple parapneumonic effusion usually requires referral to a Respiratory Physician.

♦ Tests which may help elucidate the cause of an exudate:

- Cytology - carcinoma, lymphoma.
- Cell surface markers - to distinguish between reactive and malignant lymphoid proliferation.
- ZN stain and culture - tuberculosis (consider sending fluid for PCR).
- Haematocrit - if >50% of peripheral blood haematocrit = haemothorax.
- Rheumatoid factor - rheumatoid effusion.
- Triglyceride - chylothorax.
- Amylase - pancreatitis, malignancy.

♦ Other investigations and treatment options include:

- Closed pleural biopsy.
- Diagnostic thoracoscopy.
- CT scan.
- Bronchoscopy.
- Intercostal tube drainage.
- Thoracotomy.

  Notes:

  – Do not drain the pleural fluid until the diagnosis is established, unless the patient is very dyspneic. The presence of some pleural fluid is necessary to perform a closed pleural biopsy.

  – If complicated parapneumonic effusion or empyema suspected, **do not delay seeking help.** Early treatment may prevent a thoracotomy.

OBSOLETE

21.10 Pneumothorax

21.10.1 Causes

- Traumatic - usually after chest trauma with rib fracture.
- Spontaneous.
- Chronic obstructive pulmonary disease
- Acute severe asthma - often with pneumomediastinum.
- Iatrogenic - usually after cannulation of the neck/subclavian veins, lung biopsy (FNA or transbronchial) and occasionally after an anaesthetic.

21.10.2 Clinical Signs

- Symptoms vary from mild dyspnoea with or without pleuritic chest pain to tension pneumothorax with cardiovascular compromise.
- Signs may include:
  - Reduced chest wall movement on the affected side.
  - Diminished breath sounds on the affected side.
  - Surgical emphysema in the neck or over chest wall.

  Deviation of the trachea - may be deviated towards the affected side **or** in tension pneumothorax towards the opposite side.

21.10.3 Investigations

- CXR - at the bedside if patient unwell.
  Note: The CXR tends to underestimate the size of the pneumothorax
- Arterial blood gases.

21.10.4 Treatment

- Discuss case with Consultant.
- Treatment is not always required. A small pneumothorax in the absence of underlying lung disease may resolve on its own over 3-10 days.
- Simple aspiration is recommended for a larger spontaneous pneumothorax without underlying lung disease. See protocol in the Respiratory Ward.
- **Intercostal tube drainage is recommended in the following circumstances:**
  - Tension pneumothorax (if life threatening use a 14G IV cannula in the 2nd intercostal space anteriorly and place an intercostal tube thereafter).
  - Respiratory compromise:
    - Traumatic pneumothorax or haemopneumothorax.
    - Failed simple aspiration.
- Tube placement – usually in anterior axillary line, through 5th or 6th intercostal space, directed upwards and posteriorly.
- Inpatient cases with pneumothorax should be managed by either the Respiratory Service on the Respiratory Ward or by the Cardiothoracic Surgical team if traumatic.

OBSOLETE

21.10.5 Follow Up

All patients must have a follow up CXR at 10-14 days to ensure that the pneumothorax has resolved. Recurrent pneumothorax may be an indication for pleurodesis – referral to a Respiratory Physician or Thoracic Surgeon is recommended. Advice should be given about air travel [not advised within 6 weeks] and scuba diving [contra-indicated].

21.11 Intercostal Tubes

The insertion and management of intercostal tubes is a complex and specialised area. Patients requiring chest tube management should normally be cared for by specialist respiratory or thoracic surgical teams.

Unless it is an emergency, intercostal tubes are best inserted or supervised by experienced staff from the respiratory or thoracic surgical unit. In selected cases tubes may be best inserted by a Radiologist under ultrasound guidance. Respiratory Medicine has guidelines to follow.

OBSOLETE

21.11.1 Indications

- Pneumothorax.
- Pleural effusion.
- Parapneumonic effusion/empyema.
- Haemopneumothorax.

21.11.2 Contraindications

- Bronchial obstruction on the affected side.
- Thickening of the visceral pleura.
- Loculated pleural effusion. CT scan advised in this setting.
- Coagulopathy.
- Chest wall infection.

21.11.3 Care of Chest Tubes

Duty medical staff are often asked to assess patients with chest tubes for potential or actual problems. Solving these problems is dependent on knowledge of the patient plus the type of chest drain and water seal chamber being used. At Christchurch Hospital, the nursing staff from the Respiratory Ward have information and knowledge which may be helpful. After hours, contact the Respiratory Physician on call.

- Assessment should include the following:
  - Check the insertion site, all tubes and connections.
  - Check for swinging (movement of the water column during deep breathing).
    Note: A tube on suction will not swing.
  - Check for air leak - air bubbling through the water seal chamber, especially on coughing.

- Obtain a CXR if there is any concern about the patient.

21.11.4 Emergencies

- Acute deterioration in the patient's condition:
  - Check all tube connections and underwater seal system.
  - Administer oxygen.
  - Bedside CXR.
  - Notify the Respiratory Physician on call.
- Development of subcutaneous emphysema:
  - This is most likely to occur in the setting of COPD with an air leak and is precipitated by temporary blockage of the tube.
  - Check all tubes for kinks/blockages.
  - Administer oxygen.
  - Urgent bedside CXR.
  - Notify the Respiratory Physician on call.

21.12 Acute Pulmonary - Renal Syndrome

Patients with pulmonary infiltrates and deteriorating renal function require urgent investigation. The possibility of a vasculitis must be considered. Check the urine for an active sediment, undertake screening tests as indicated below and consult early with Respiratory Physician / Immunologist / Renal Physician.

Table 43: Acute Pulmonary - Renal Syndrome

Suspected diagnosis	Screening tests
A Idiopathic Vasculitis – Wegener's Granulomatosis – Microscopic Polyangiitis – Anti GBM Disease – SLE – Mixed cryoglobulinaemia	 c-ANCA p-ANCA Anti-GBM ANA Cryoglobulins
B Non Vasculitic – Renal vein thrombosis/PE – TTP	 Radiological Blood film
C Infective (a rare cause) – Mycoplasma – Legionella – Mycobacterium – S. pneumoniae	 IgM antibody PCR Sputum ZN stain Sputum Gram stain

OBSOLETE

22. Rheumatology

Main Office 3rd Floor, Riverside ☎ 80953

Inpatient Services Ward 30

- Dr P. Moller ☎ 89670
- Dr P. Chapman ☎ 80955
- Dr J. O'Donnell ☎ 80950

Consultation Guidelines

The clinical focus of the department is on the diagnosis and management of rheumatic diseases, primary immunodeficiency and serious allergic disorders.

Outpatient Services

Rheumatic diseases - Outpatient Department (Loyis)	☎ 80492	Fax 80491
Immunodeficiency and allergic disorders - Chris Martin	☎ 80950	Fax 81241

The department is closely associated with the Immunology Laboratory, Canterbury Health Laboratories.

OBSOLETE

22.1 Acute Swelling of a Single Joint

The cause of the acute swelling must be established before any rational form of treatment can be given.

22.1.1 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.

22.1.2 Investigations

- 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.

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.

OBSOLETE

22.1.3 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 145).*
  - 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 1.2 mg stat, then 0.6 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 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 6 weeks or longer.

- 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 is 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.

OBSOLETE

23. Thrombosis, Embolism and Anticoagulation

23.1 Deep Vein Thrombosis (DVT)

23.1.1 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.

23.1.2 Precipitating Causes

- Surgery. Immobilization including travel. Combined oral contraceptives. Stilboestrol. Malignancy. Pregnancy and puerperium. Polycythaemia. Thrombocytosis.
- **Suspect –**
  - activated protein C (APC) resistance (Factor V Leiden).
  - anti-thrombin III deficiency.
  - protein C or protein S deficiencies.
  - prothrombin mutation.
  - Hyperhomocyteinaemia.
  - antiphospholipid syndrome.
    - If the patient is under 45, has had recurrent thromboses or there is a family history of thrombosis.

OBSOLETE

Table 44: DVT – Predictive "Risk" Factors (Lancet 1997; 350:1795-1798).

Clinical Model to Determine Pre-Test Probability for Deep Vein Thrombosis	
• Active cancer (treatment within previous six months or palliative.	1
• Recent immobilisation >3 days or major surgery within 12 weeks.	1
• Local tenderness along the distribution of the dep venous system.	1
• Entire swollen leg.	1
• Calf swelling 3 cm > asymptomatic side (measured 10 cm below tibial tuberosity).	1
• Pitting oedema confined to symptomatic leg.	1
• Collateral superficial veins (non-varicose).	1
• Alternative diagnosis as likely or greater than that of DVT.	-2
Total Points	
Clinical Probability calculated as follows: **HIGH (3 or greater) MODERATE (1 or 2) LOW (0 or less)**	

23.1.3 Investigations *(See Decision Tree – Page 255)*

- Ultrasound of the femoral and popliteal veins is usually the investigation of choice. A thrombus can be demonstrated and if present the vein will not be compressible. A Doppler ultrasound may also show reduced flow. Ultrasound may sometimes detect calf vein thrombosis. Calf vein thrombi do not cause clinically significant pulmonary emboli but the difficulty lies in being certain that they will not extend proximally.

  The management as outlined in the decision tree is critically dependent on the ability of the ultrasound technique performed to detect thrombi in the femoral venous system. Since no test is infallible in situations where clinical suspicion is moderate or high and the ultrasound is negative, venography should be considered, particularly if the D-dimer test is positive.

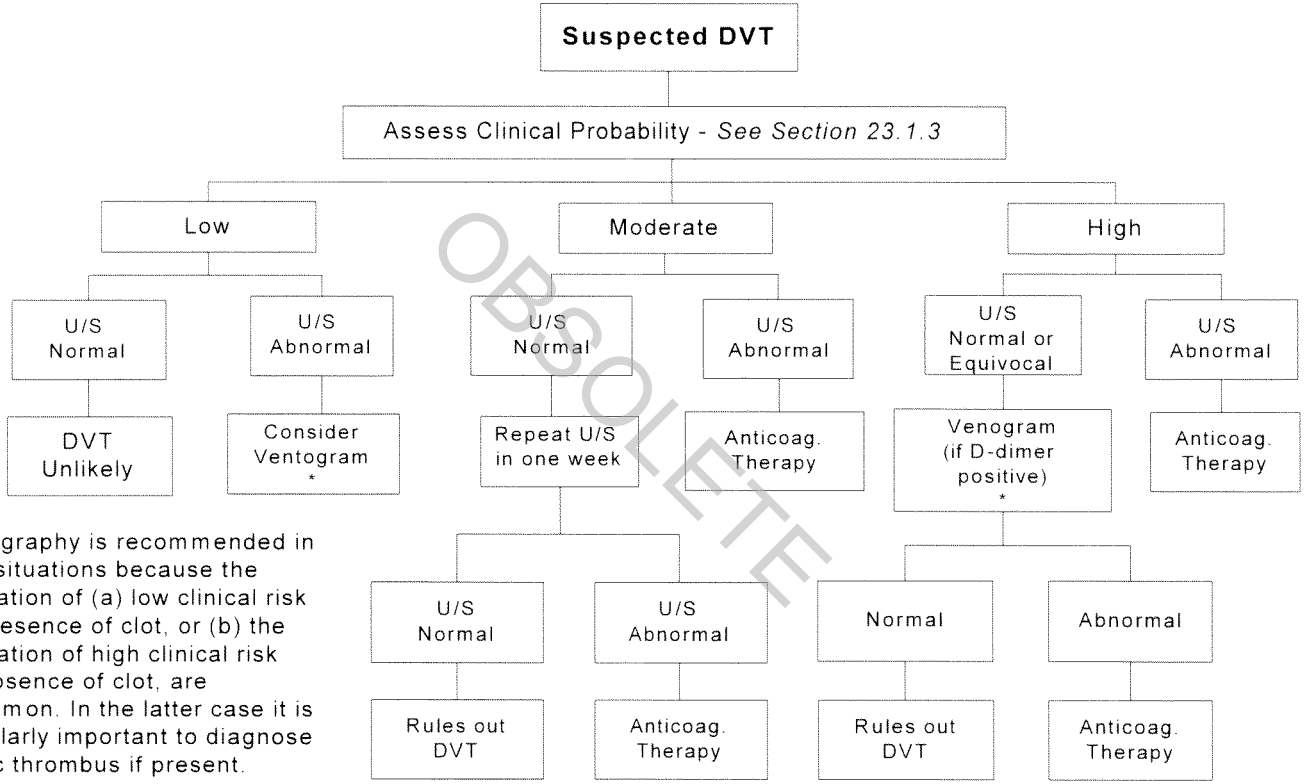
- Other investigations:
  - CBC + diff, (polycythaemia, raised platelets).
  - PT, APTT before starting anticoagulant treatment.
  - If thrombophilia is suspected take an extra 10 mL blood into citrate and request thrombophilia screen.
    Note: - These samples must be taken before starting treatment.
  - If ultrasound negative and a ruptured Baker's cyst is a possibility consider arthrography.

OBSOLETE

23.1.4 Management

- Anticoagulation with heparin and warfarin. *(see page 261).*
- If massive thrombosis and no contraindication consider fibrinolytic therapy. *(see page 263).*

Figure 9: Suspected DVT



* Venography is recommended in these situations because the association of (a) low clinical risk with presence of clot, or (b) the association of high clinical risk with absence of clot, are uncommon. In the latter case it is particularly important to diagnose an iliac thrombus if present.

The D-dimer assay performed by the SimpliRed method has a sensitivity for DVT of 89-100% and negative predictive value of 95-100%.

23.2 "Medical" DVT Prophylaxis

It is known that certain medical conditions such as stroke, myocardial infarction and heart failure increase the risk of DVT. Compared to surgical prophylaxis there are relatively few trials designed to assess this risk and the degree of benefit, if any, associated with prophylactic treatment.

- **Myocardial Infarction**
  Early studies showed an incidence of DVT (fibrinogen uptake) of between 20-30% in the two weeks following myocardial infarction. With the current use of thrombolysis and anticoagulation for acute MI, it is unlikely that additional prophylactic measures are required.

- **Ischaemic Stroke**
  Fibrinogen uptake studies show a DVT incidence of 50 - 60 % following paralytic stroke and one study found death from PE in 5% of such patients. Anticoagulation or thrombolysis is not standard treatment for the cerebral lesion in this country, therefore prophylaxis to prevent DVT needs to be considered. Studies do show that prophylactic heparin for 7 - 14 days reduces the DVT rate to about 20 % with a non-significant reduction in PE and "death from all causes". This is achieved without increased risk of haemorrhagic transformation of the cerebral infarct.

- **Congestive Cardiac Failure and Related Medical Conditions**
  Elderly medical patients, especially those with cardio-respiratory failure, infections and immobility are prone to DVT (rates of 10 - 30 % fibrinogen uptake). Low dose unfractionated heparin, LMW Heparin and warfarin have shown to reduce that risk without increased bleeding complications. Despite a reduction in DVT, large studies have failed to demonstrate a statistically significant reduction in mortality rates though there is a trend in that direction. Prophylaxis should be considered in patients over the age of 40 with the above conditions.

- **Recommended Prophylaxis Schedules**
  These include enoxaparin (Clexane) 40 mg subcutaneous daily or unfractionated heparin 5000 U subcutaneous bd or tds.

OBSOLETE

23.3 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 not very common.

OBSOLETE

23.3.1 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.

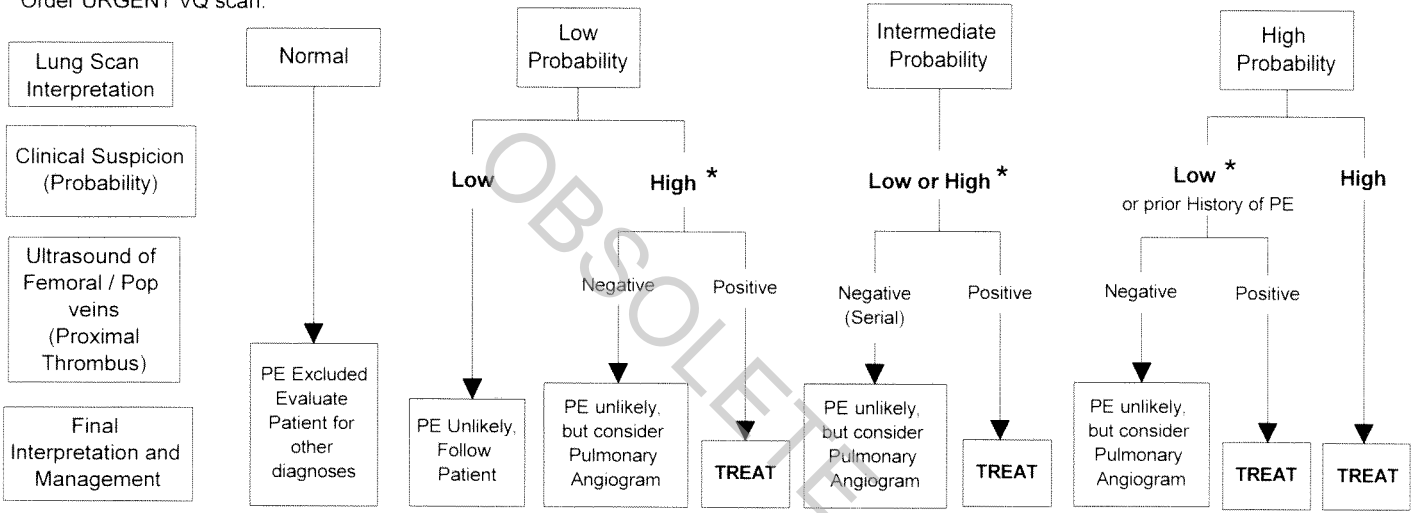
23.3.2 Investigations

(see Decision Tree Page 258)

Pulmonary embolism may have to be treated initially on a presumptive basis. In all cases attempts must be made to establish the diagnosis as treatment can be hazardous.

Figure 10: Assessment of Patient with suspected Pulmonary Embolism

Obtain CXR, ECG, ABGS, and SimpliRED D-dimer test.
If PaO2 >79mm and Resp Rate <21/min and SimpliRED negative, PE is very unlikely.
PE still suspected, give Heparin.
Order URGENT VQ scan.



OBSOLETE

*In these cases, when clinical and lung scan probability are in disagreement, consider CT pulmonary angiogram.
-In cases of suspected massive pulmonary emboli, consider urgent CT pulmonary angiogram or echocardiography for right heart thrombi & pressure measurement.
-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 invasive pulmonary angiography.
-CT pulmonary angiography is available for the diagnosis of PE. Further assessment of its usefulness is awaited.

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 definitive. 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. CT pulmonary angiography is useful in detecting clot in the major pulmonary arteries but is insensitive for peripheral emboli.

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.
- **Chest radiograph** - excludes other pathology such as pneumothorax, but is never diagnostic. The most specific sign is segmental loss of vascular markings. Parenchymal 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.
- **D-dimer test** - D-dimers are breakdown products of fibrin. With reference to pulmonary embolism the test is not specific but is moderately sensitive. Normal levels of D-dimer (a negative SimpliRed test) indicate a low probability of pulmonary embolism or DVT.

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- **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 a Doppler ultrasound examination of the femoral veins is recommended. A negative result lowers the probability of PE. A positive result increases the probability and justifies anticoagulation.

- **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.

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23.4 Management of DVT & PE and the use of Anticoagulants and Related Drugs

This is summarised in the following Table.

Table 45: Treatment of DVT and PE with Heparin and Warfarin

	Drug Therapy	Duration	Monitoring
DVT	**Low molecular weight heparin** [e.g. dalteparin [Fragmin] 200 U/kg q24h **and** commence	5 days - if PT ratio (INR) >2 at day 4.	Not usually required [see next page].
	Warfarin 15 mg PO day 1, 5 mg day 2 then dose according to PT ratio. (If patient <60 kg or pre treatment INR >1.2 a lower initial dose is recommended.	6-12 weeks if reversible risk factors. 3-6 months if idiopathic. Consider indefinite treatment if thrombophilia and rethrombosis risk greater than risk of bleeding from therapy.	PT ratio (INR) range 2.0-3.0. Check daily for 5 days. Then if stable twice weekly for 2 weeks then PRN (maximal interval 1 month).
PE or massive DVT	**Either standard heparin** 5000U bolus IV then continuous infusion initially at 1500 U/hour **or**	7 days	Monitor with APTT at least once daily.
	Low molecular weight heparin [e.g. dalteparin [Fragmin] 120 U/kg SC q12h]. Consider thrombolytic therapy for massive PE or DVT (see below). With all the above treatments give.	7 days	Not usually required [see next page].
	Warfarin as outlined for DVT starting **day 3**.	Give therapy for 12 weeks if reversible risk factors. 6 months if idiopathic. Consider indefinite treatment if thrombophilia, and rethrombosis risk greater than risk of bleeding from therapy.	PT ratio (INR) range 2.0-3.0. Check daily for 5 days. Then if stable twice weekly for 2 weeks then PRN (max interval 1 month)
Recurrent DVT or PE	**Low molecular weight heparin** [e.g. dalteparin [Fragmin] 200 U/kg SC q24h. **and**	5 days (DVT) 7 days (PE)	Not usually required [see next page].
	Warfarin as above	Consider lifelong treatment	PT ratio (INR) range 2.0-3.0 as above. If recurrent **while on warfarin** increase range to 3.0-4.0. Check for underlying cause.

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Note:

- Before starting treatment take blood into citrate for PT/APPT in all patients. In patients under 45, or with recurrent DVT/PE or with a positive family history take 2 citrate tubes for anticardiolipin antibodies, lupus anticoagulant, protein C, protein S, APC resistance and antithrombin III (thrombophilia screen). Consider also prothombin mutation and homocysteine assay.
- **Low molecular weight heparins** [LMWH] have more predictable pharmacokinetics, a longer half life compared to standard (unfractionated) heparin and a lower rate of thrombocytopenia. They cause less bleeding complications. Clinical trials have compared them to standard heparin but not to each other.
  Low molecular weight heparins that are currently available for the treatment of DVT include dalteparin [Fragmin] *(see page 261)*, enoxaparin [Clexane] 1 mg per kg SC q12h, tinzaparin [Innohep] 175 U/kg SC q24h or fraxiparine.
- **If heparin induced thrombocytopenia** occurs **Orgaran** is usually the best form of anticoagulant to use.
- Studies have shown that **outpatient treatment of DVT** with LMW heparin is safe and effective. If you wish to treat as an outpatient, use the admitting form provided and contact the haemostasis nurse.
- Warfarin should be started at least 5 days before LMWH or unfractionated heparin is stopped.

If **massive pulmonary embolism** is suspected and life saving treatment appropriate:

- Immediate heparinization.
- Cardiovascular support with inotropic therapy, oxygen, analgesia.
- Urgent pulmonary angiography and/or echocardiogram.
- Give fibrinolytic therapy (see below) provided no absolute contra-indications exist and no improvement in cardiovascular function is seen over 20-30 minutes. Fibrinolytic therapy carries a higher risk of bleeding than heparin. For contra-indications to fibrinolytic therapy *(see page 263)*.

Note: If pulmonary angiography cannot be obtained, do a perfusion lung scan. 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 senior clinician.

- **Minor pulmonary embolism:**
  - Anticoagulate *(see page 261)*.
  - Oxygen if PaO_2 less than 60 mmHg.
  - Analgesia as required.

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- **Thrombolytic Therapy**

The exact role of thrombolysis and which agent to use in DVT and PE remains controversial.

Thrombolytic agents should be considered in life threatening PE and massive DVT.

Table 46: Thrombolytic agents in PE and DVT

Drug Therapy	Duration	Monitoring
Streptokinase IV infusion 250,000U over 30 minutes. Then 100,000U/hourly. Premed with 50 mg hydrocortisone IV. **then** Heparin and warfarin see treatment table. **If streptokinase resistance** is documented or suspected e.g. previous streptokinase therapy **then use**	24 hours (PE) 24-36 hours (DVT)	Contact Haematology re resistance tests and monitoring
Tissue plasminogen activator (tPA) IV infusion 100 mg over 2 hours **then** Heparin and warfarin (See treatment table)	2 hours	None required

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Note:

- Long term benefits of fibrinolysis for life threatening PE or massive DVT are not yet clearly defined.
- We recommend controlling streptokinase treatment with thrombin times aiming for 2-4 times the control value. Measuring fibrinogen levels is also recommended. 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. A 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.
- Streptokinase 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 61)*. The dosages recommended and the monitoring used are different from those given above.

♦ **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 5 days before it is planned to stop heparin. During this time check PT 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).

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The degree and duration of anticoagulation will vary according to condition being treated. The following is recommended:-

Table 47: Drug Treatment in DVT & PE

	Prothrombin Ratio (INR)	Duration
Pre and perioperative anticoagulation	1.5-2.0	Days
Treatment of DVT	2.0-3.0	6-26 weeks
Treatment of PE or massive DVT	2.0-3.0	12-26 weeks
Treatment of recurrent DVT or PE**	3.0-4.0	? life long
Atrial Fibrillation	2.0-3.0	life long
Prosthetic heart valves	3.0-4.0	life long
Arterial disease	3.0-4.0	life long

** Recurrence despite prothrombin ratio between 2 and 3.

23.4.1 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
Ketoconazole
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

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23.5 Treatment of Anticoagulant Overdosage

23.5.1 Heparin

- Protamine sulphate has been used to reverse overdosage with standard unfractionated heparin. There is less experience with its effect in neutralizing low molecular weight heparins. Reversal is only necessary if there is 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 evaluation is quite variable. Neutralization tests can be performed but are of limited value.

  Caution: Excess protamine sulphate may act as an anticoagulant itself.

23.5.2 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.

23.5.3 Streptokinase

- Abnormal haemorrhage may be very difficult to correct at least for some hours. If fibrinogen level is low, cryoprecipitate may help.

23.6 Peripheral Arterial Embolism

23.6.1 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.

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23.6.2 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.

23.6.3 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.

24. Diseases Notifiable in New Zealand

(Includes suspect cases)

24.1 Information for the Medical Practitioner

The Health Act (1956) places certain obligations upon medical practitioners and these are as follows:

"74 Medical practitioners to give notice of cases of notifiable disease -

(1) Every medical practitioner who has reason to believe that any person professionally attended by him is suffering from a notifiable disease or from any sickness of which the symptoms create a reasonable suspicion that it is a notifiable disease shall -

(a) In the case of notifiable infectious disease, forthwith inform the occupier of the premises and every person nursing or in immediate attendance on the patient of the infectious nature of the disease and the precautions to be taken, and forthwith give notices in the prescribed form [to the Medical Officer of Health, and, except where the disease is specified in Section B of Part I of the First Schedule to this Act, to the local Authority of the district]:

(b) In the case of a notifiable disease other than a notifiable infectious disease, forthwith give notice in the prescribed form to the Medical Officer of Health.

(3) Every medical practitioner who by post-mortem examination or otherwise becomes aware that any deceased person was affected with a notifiable disease shall forthwith give notice in the prescribed form to the Medical Officer of Health.

(4) Every medical practitioner commits an offence against this Act who fails to comply with the requirements of this section."

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24.2 Communicable Diseases and Septic Conditions in Relation to Maternity Patients and Infants.

Note: At the time of preparation of this 8th Edition it seemed likely that the Obstetric Regulations were about to be changed.

Obstetric Regulations 1975 as amended 1986

"6 Medical practitioners *and registered midwives* to notify of septic condition, etc - (1) 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 or registered midwife 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".

24.3 Venereal Diseases

Duties of Medical Practitioner 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

© 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.

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Venereal 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, gonorrhoeal infection affecting any site, chancroid or venereal granuloma in a communicable form, and the patient either -

(a) Fails for 1 week after the date fixed for the purpose by the medical practitioner to attend 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, gonorrhoeal 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).

24.4 Diseases Notifiable in NZ

Table 48: Diseases Notifiable in NZ

DISEASES NOTIFIABLE IN NZ (Include suspect cases)* Notifiable Infectious Diseases Under the Health Act 1956	
Section A - Infectious Diseases Notifiable to Medical Officer of Health and Local Authority	
Acute gastroenteritis ** Cryptosporidosis Giardiasis Legionellosis Meningoencephalitis – primary amoebic Shigellosis Yersiniosis	Campylobacteriosis Cholera Hepatitis A Listeriosis Salmonellosis Typhoid and paratyphoid fever
Section B - Infectious Diseases Notifiable to Medical Officer of Health	
Acquired Immunodeficiency Syndrome (AIDS) Arboviral diseases Diphtheria Hepatitis B Hepatitis (viral) - not otherwise specified Leprosy Malaria *Neisseria meningitidis* invasive disease Pertussis Poliomyelitis Rheumatic Fever Rubella Yellow Fever	Anthrax Brucellosis *Haemophilus Influenzae b* Hepatitis C Hydatid disease Leptospirosis Measles Mumps Plague Rabies Rickettsial diseases Tetanus Viral haemorrhagic fevers Spongiform encephalopathies including *Creutzfeldt-Jakob Disease*

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Diseases Notifiable to Medical Officer of Health (other than Notifiable Infectious Diseases)
Notifiable to the Medical Officer of Health
Cysticercosis Taeniasis Trichinosis Decompression sickness Lead absorption equal to or in excess of 15µg/dl (0.72µmol/L) *** Poisoning arising from chemical contamination of the environment
Notifiable Diseases Under Tuberculosis Act 1948
Notifiable to the Medical Officer of Health
Tuberculosis (all forms)

* During times of increased incidence practitioners may be requested to report, with informed consent to their local Medical Officer of Health of cases of other communicable diseases not on this list.

** Not every case of acute gastroenteritis is necessarily notifiable - only those where there is a suspected common source or from a person in a high risk category, (e.g. food handler, child care worker, etc) **or** single cases of chemical, bacterial, **or** toxic food poisonings such as botulism, toxic shellfish poisoning (any type) and disease caused by verocytotoxic *E.coli*.

*** Blood lead levels to be reported to the Medical Officer of Health (15µg/dl or 0.72 µmol/L) are for environmental exposure. Where occupational exposure is suspected, please notify OSH through the NODS network.

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24.5 Immunisation Information For International Travel.

Information on immunisation for overseas travel is available from the Medical Officer of Health of the District.

25. Treatment and Referral of Patients to Hyperbaric Unit

A double compartment hyperbaric chamber for treatment with hyperbaric oxygen (HBO) is available at Christchurch Hospital. This requires specialist consultation with the Hyperbaric team.

Current Acute Indications for HBO are:

- Decompression sickness.
- Carbon monoxide poisoning (possibly cyanide and H_2S also).
- 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, e.g. necrotising fasciitis. streptococcal fasciitis.
- Crush injury with acute traumatic ischaemia.

Referral for these conditions is **VERY URGENT.** Patients with these conditions will be accepted for assessment and therapy on an emergency basis. An HBO course varies from 1-12 treatments for these conditions.

HBO is also indicated in some other conditions in selected cases:

- Osteoradionecrosis (especially of mandible); soft tissue radionecrosis.
- Refractory osteomyelitis.
- Diabetic ulcer.

These latter conditions require extended treatment courses [4-10weeks]. Referrals will be considered on an individual basis and require specialist **and** budgetary approval.

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25.1 Patient referral (Inpatient and External)

Since these are normally emergency referrals the following procedure will ensure the most rapid response. [Note that on-call staff are only rostered out of normal working hours].

- Ring Christchurch Hospital Operator (Internal '0' or external (03) 3640-640) and ask them to 'ALERT HYPERBARIC TEAM'.
- Give the Operator your name and contact phone number.
- Once a member of the hyperbaric medical staff has been contacted by the Operator they will return the enquirer's call.
- Trying to contact individual clinicians may result in delay. However, in normal working hours, also ring the Department of Anaesthesia, Christchurch Hospital (03) 364-0288 (internal extension: 80288). Less urgent consultation requests should be sent to Dr Michael Davis, C/o Department of Anaesthesia, Christchurch Hospital.

26. Blood Transfusion Practice including the Management of Transfusion Reactions

26.1 Blood Transfusion Services

Blood Transfusion services are provided by the New Zealand Blood Services. In Christchurch they have two sites. The blood donor centre, processing and accreditation facilities are located at 87 Riccarton Road. The crossmatch and tissue typing laboratories are on the 1st floor of Canterbury Health Laboratories.

26.1.1 Ordering of Blood

Note: The majority of transfusion errors are of a clerical nature.

- The same care and consideration must be taken with ordering blood transfusion as for the prescription of a dangerous drug.
- Blood must be ordered on the appropriate blood transfusion request form which must be completed as printed. No forms are acceptable unless they show the full particulars of the patient including surname, first names and patient identification number or date of birth which should be obtained from the identification bracelet.
- A sample of the patient's blood is to accompany the requisition form except during such hours as specimens are routinely taken by the laboratory staff. All samples must be labelled in biro or ink as soon as they are taken, at the patient's bedside with details **from the patient's wristband and must be word and letter perfect. Self adhesive labels are not acceptable on samples for compatibility testing.**
- Orders for non-urgent transfusion must reach the Blood Bank during normal laboratory hours, and in any case at least two hours before the blood is needed. Please state when the blood is needed.
- No more than six units of blood may be ordered at any one time, except by special arrangement with the Blood Bank staff.

26.1.2 Collection of Blood from Blood Bank

- During laboratory hours at Christchurch Womens Hospital, The Princess Margaret Hospital and Burwood blood is issued by laboratory staff on production of Transfusion Form MR22A, properly completed.
- At Christchurch Hospital and in the absence of laboratory staff, RMOs, authorised registered nurses, but no-one else, may issue blood from the Blood Storage Refrigerator inside the main doors of the Operating Theatres.

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- When blood is collected, the particulars on the container (i.e. the patient's full name, patient identification number and group) must be checked against Form MR22A.
- Blood which has not been properly labelled by Blood Bank staff as suitable for the patient in question must not be taken from the Blood Bank. The sole exception to this rule is cases of extreme urgency occurring outside laboratory hours when on the direct order of a senior medical officer, blood of Group O Rhesus negative may be issued, which has not expressly been labelled as suitable for the patient in question. In such cases (which should be rare) the Transfusion Medicine Specialist or his/her deputy must be informed immediately.

26.1.3 Administration of Blood

- Written consent must be obtained from patients or their guardians before transfusing blood or blood products. Blood must not be collected until it is needed for transfusion. Half an hour is the maximum interval permitted between collection and administration. If the blood has been collected and a delay seems likely, the container must at once be returned to the Blood Bank for further refrigeration. Do not store blood in ward or theatre refrigerators, however short the period. Blood which is darker than normal or discoloured may be infected and should not be transfused.
- Any blood product which is prepared by an open method, for example, washed red cells, or reconstituted plasma products, is potentially infected and must be used within 24 hours of preparation.
- Nothing is to be added to blood.
- Before a unit of blood is administered, the particulars on the label must be checked with the particulars on the identification bracelet worn by the patient. The doctor supervising the transfusion has the responsibility to ensure that the blood issued from the laboratory is administered to the correct patient. Any delegation of this responsibility must be indicated in writing in the patient's notes and should clearly state to whom this responsibility has been delegated.
- A record of the transfusion should be kept in the patient's notes.

26.1.4 Transfusion Reactions

- 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 38.5C, and transient skin rashes are common. Since a serious haemolytic reaction may initially present with such mild

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symptoms, any reactions occurring during blood transfusion must be immediately reported to the doctor responsible.

- The transfusion need not be stopped if the patient has a low grade fever and is symptom free. Skin rashes merely require the administration of an antihistamine.
- If more severe reactions occur with fever over 38.5C, chills, nausea, rigors, shock or hypotension, the transfusion is stopped, the laboratory should be alerted and the following procedure should be carried out.

The identity of the recipient and the name of the compatibility label should be checked.

The unit of blood should be inspected for signs of bacterial contamination.

The remains of the unit together with the giving set should be sent to the transfusion medicine laboratory.

7.5 ml blood in EDTA should also be sent to the laboratory. A post- transfusion sample of urine should be checked for haemoglobin.

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 either 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 or caused by transfused cytokines. The transfusion 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 and thereby reduce the likelihood of febrile reactions in repeatedly transfused patients. Filters are available from Central Supplies and are indicated if a patient has two consecutive non haemolytic febrile transfusion reactions despite appropriate premedication.

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26.1.5 Red Cell Preparations Available.

The following general principles will apply in filling requests for blood for transfusion.

- MODIFIED WHOLE BLOOD will be used for neonatal exchange transfusions. Red cells (paediatric) with an haematocrit of 0.7 will be used for neonatal top up transfusions.
- For other purposes, RESUSPENDED RED CELLS with an haematocrit of 0.45-0.65 will be issued. Plasma (250 ml) is removed from whole blood and replaced by an additive fluid (63 ml).
- CONCENTRATED RED CELLS may be required if circulatory overload is a problem. The haematocrit will be above 0.70.
- FRESH FROZEN PLASMA, PLATELETS AND SPECIAL PREPARATIONS are available. Requests should be discussed with the Transfusion Medicine Specialist or his/her deputy.

26.1.6 Micro-Filters and Massive Transfusion

Micro-filters should be reserved for patients with special problems, e.g. those requiring massive transfusions associated with poor respiratory function, elderly patients, ruptured aneurysm etc., or recurrent febrile transfusion reactions. Micro-filters slow the rate of transfusion. They remove platelets and are expensive.

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27. Christchurch Hospital Emergency 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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28. Document Management

The purpose of this Manual is to provide a book containing guidelines for Medical staff at Christchurch Hospital. It is an addition to the formal documentation of the various departments.

Content

The Manual contains guidelines for the management of medical conditions at Christchurch Hospital.

Scope

The Manual is for the use of all Medical Staff.

DocumentControl

The Manual Issue and Expiry dates are documented on the first page.
The cover of the Manual is blue and includes a different coloured stripe for each issue to easily identify the current issue.
The current period of March 2000 – November 2001 the stripe is
GREEN

The Manual will be updated every second year in November at which time owners of the previous year's document must discard the expired copy.

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Distribution

Copies of the Manual are available to all staff.

A List of Persons who have been issued a copy is maintained in the Department of Medicine/RMO Unit.

Updates and Amendments

This Manual will be updated and reprinted in November of each second year.

There will be no amendments during the year.

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