Canterbury
District Health Board
Te Poari Hauora ō Waitaha

Management Guidelines for Common Medical Conditions

Eleventh Edition 2005

Internal Medicine Services

Date of Issue: 1 December 2005
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Canterbury DHB

Management Guidelines for Common Medical Conditions

11th Edition 2005

INTERNAL MEDICINE SERVICES

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OBSOLETE

Introduction - Eleventh Edition 2005

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 standardised treatment guidelines to improve medical care. Through the vision and energy of Dr Mike Beard, this was achieved and the first edition of the Blue Book was produced in 1983 with the help of Dr Derek Hart. Each subspecialty continues to produce recommendations with each new edition. This handbook has proven very popular among RMOs and specialists alike, not only in Canterbury, but nationwide. This is the eleventh 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. In several areas, we refer to National and International guidelines. If our guidelines differ, then this reflects the current practice at the CDHB. **Our guidelines 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. This edition has been produced by Streamliners NZ Ltd (www.streamliners.co.nz) in the appropriate format for hard copy, intranet, and PDA versions. We would like to acknowledge the financial support of Canterbury District Health Board.

Note: *These Guidelines are now on the Canterbury DHB Intranet and can also be downloaded onto handheld computers. Please refer to the CDHB intranet for instructions on how to download the PDA version.*

For feedback/ correspondence/ comments, please contact: john.thwaites@cdhb.govt.nz

Contents

1. Abbreviations

General

- **Alb:** Albumin
- **Alk. Phos:** Alkaline Phosphatase
- **ALT:** Alanine Aminotransferase
- **AST:** Aspartate Aminotransferase
- **Bili:** Bilirubin
- **Ca:** Calcium
- **CBC + Diff:** Hb, PCV, MCV, WBC and differential, platelets
- **Cl:** Chloride
- **CPR:** Cardiopulmonary Resuscitation
- **CXR:** Chest X-ray
- **DIC:** Disseminated Intravascular Coagulation
- **DKA:** Diabetic Ketoacidosis
- **ESR:** Erythrocyte Sedimentation Rate
- **GGT:** Gamma Glutamyltransferase
- **K:** Potassium
- **KCl:** Potassium Chloride
- **LDH:** Lactate Dehydrogenase
- **LP:** Lumbar Puncture
- **MCV:** Mean Cell Volume
- **Mg:** Magnesium
- **MSU:** Mid-stream Urine
- **Na:** Sodium
- **NSAID:** Non-Steroidal Anti-inflammatory Drug
- **PO_4:** Phosphate
- **Sat.O_2:** Haemoglobin Oxygen Saturation (pulse oximetry)

Fluid Prescriptions

- **0.9S:** Normal Saline
- **0.45S:** Half Normal Saline
- **D4S:** 4% Dextrose and Fifth Normal Saline
- **D5W:** 5% Dextrose

Drug Administration and Dosage

- **IV:** Intravenous
- **IM:** Intramuscular
- **SC:** Sub-cutaneous
- **PO:** Oral
- **PR:** Rectal
- **BD/TDS/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** or **ml:** Millilitre
- **L:** Litre
- **mcg:** Microgram
- **mg:** Milligram
- **g:** Gram
- **kg:** Kilogram
- **mmol:** Millimole
- **mcmol:** Micromole
- **IU:** International Unit
- **U:** Units
- **mU:** Milliunits

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 DHB 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
- **Pleural:** <1000
- **Peritoneal:** <300
- **Synovial:** <3000
- **Urine:** <10

These are all expressed as:

- 10^6/L or
- per mL or
- per mm^3

All these are the same! Check which units your laboratory is using and always obtain that laboratory's normal range.

2. General Medicine Services

Main Office

- 1st Floor, Riverside, ☎ 81020, Fax 81025

Inpatient Services: Ward 23, 24, 29, 30, Ward 31, and the Stroke Unit.

Clinical Director:

- Dr Alan Pithie, ☎ 89909

Service Manager:

- Sue Teague, ☎ 80634

Nursing Director:

- Pam Kiesanowski, ☎ 88996

Clerical Supervisor:

- Lyn Clark, ☎ 86010, Fax 81025

Secretaries:

- Mary Simes, ☎ 80155
- Margaret Crossan, ☎ 86009

Physicians:

- Bevan Brownlie, Peter Chapman, David Cole, Barry Colls, John Elliot, Valerie Fletcher, Ben Franks, Ruth Hughes, Penny Hunt, Dave Jardine, Richard Laing, David MacGregor, Roland Meyer, Nigel Millar, Peter Moore, Hani Mustafa, John O'Donnell, Anne Roche, Russell Scott, Andrew Sidwell, Steven Soule, Peter Thornley, John Turner, Adrienne Williamson.

There are twelve acute medical admitting teams; two teams on call each day, rostered 1 in 6, and providing a default medical admitting service, admitting more than 8000 patients per year.

One team does cardiac arrest calls, and the other team does surgical consultations.

Consultation Guidelines

An acute consult service is provided.

Contact the acute medical registrar/consultant via the Hospital Switchboard.

Outpatient Clinics

Currently these are provided by Doctors Cole, Jardine, Sidwell, and Mustafa.

3. Use of Ancillary Services

3.1 Guidelines for Requesting 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 Christchurch Hospital Policy and Procedures Manual Volume A.

3.2 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 80999 to arrange training.

3.3 New Zealand Blood Service

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

3.3.1 Contact Telephone Number

Cross matching ☎ 80310

3.3.2 Specimen Labelling

Hand labelled specimens are the only ones acceptable to Transfusion Medicine and must be accompanied by a completed Transfusion Request form.

Refer to Blood Transfusion Practice including the Management of Transfusion Reactions (see page 40) for further information.

3.4 Canterbury Health Laboratories

3.4.1 Service Provided

- The Core Laboratory functions 24 hours daily providing routine chemistry, haematology, microbiology, and haemostasis testing.
- Biochemistry, Microbiology, 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.
- After hours, most laboratories provide an on-call service for urgent specialised tests not performed in the Core Laboratory.

3.4.2 Contact Telephone Numbers

- Reception, ☎ 80300
- Microbiology, ☎ 80350
- Biochemistry, ☎ 80376
- Anatomical Pathology, ☎ 80580
- Haematology, ☎ 80373

3.4.3 Labelling of Specimens and Forms

Mislabelled or unlabelled specimens will not be processed. Specimens will not be returned for re-labelling or amending.

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.

For further information with regard to the requirements for individual tests and for interactions that may interfere with some assays, see CDHB Intranet under Clinical info > CHLabs > Tests.

Minimum labelling requirements for specimens:

- Patient identifier:
  - a hospital number or
  - DOB plus surname and first name or initials

Minimum labelling requirements for forms:

- Patient identifier:
  - a hospital number or
  - DOB plus surname and first name or initials or
  - a unique code used by approved locations (e.g., STD clinic samples, donor number, etc.)
- Name or unique identifier of physician or person legally authorised to request examinations or use medical information.
- Address for the report. The requestor's address should be provided in addition to the referring laboratory's address - including external clients to CHLabs.

3.5 Radiology

Services provided and working hours at each hospital.

- **Christchurch Hospital** - a fully specialised radiology service 0800-1700 hours with a 24 hour presence of radiographers and a registrar, in the department for acute work. Consultant 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 specialised examinations will need to be sent to Christchurch Hospital. Radiologist visits for limited sessions only.
- **Christchurch Women's Hospital** - General radiology and ultrasound 0800-1600 Monday to Friday. Radiographer and radiologist on call at other times. Patients requiring more specialised examinations will need to be sent to the main Radiology department. 24 hour cover to NICU by radiographer and radiologist.

3.5.1 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 hospital 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. A Consultation form must have a Consultant's name, clearly written. A pager number must be provided wherever possible.

3.5.2 Radiology subspecialty organisation

Radiologists and Radiology Registrars practice under a subspecialty organisation. The subspecialty areas are: emergency, cardiorespiratory, musculoskeletal, abdominal imaging, oncology imaging, paediatric radiology, neuro imaging, interventional radiology and obstetric and gynaecology radiology. The first

point of contact should be the Registrar attached to the subspecialty area. The Radiology department operator (☎ 80770) will transfer enquiries to the relevant Registrar or Consultant.

3.5.3 Radiology examinations during evenings and nights (Acute Service only from 17:00 to 08:00)

Radiology examinations should only be specifically asked for during evenings and nights if the examination is likely to significantly change the patient's management. The Radiologist and Registrar on call must be consulted for acute examinations in specialised areas such as CT, MRI, ultrasound, and DSA.

3.5.4 PACS

Christchurch Hospital and Christchurch Women's Hospital are now filmless hospitals and all x-rays are stored digitally. Burwood Hospital is not yet digital. Cashmere Radiology at the Princess Margaret Hospital will be fully digital by 2006.

3.6 Nuclear Medicine

Refer to *Radiology and Nuclear Medicine Procedures, Volume 14, Policy and Procedure Manual*, for indications and interpretations of scans. This manual is available in all clinical departments and wards. 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.

3.7 Pharmacy Services

Located on Ground floor opposite the Orthopaedic Outpatient Department.

Contact Details: ☎ 80840 and ask for the service required.

Services Provided:

- A comprehensive clinical service to all wards in Christchurch Hospital and a limited service to Christchurch Women's Hospital.
- **Hours:** 0800 - 1700 hours weekdays and 0900-1200 hours Saturdays.
- An on-call pharmacist is available outside of these hours to respond to urgent requests from all Canterbury DHB hospitals.

Clinical Service:

A ward pharmacist:

- Visits each ward and reviews patient charts once or twice daily.
- Is involved with drug information questions, medication reviews, aminoglycoside monitoring and therapeutic drug monitoring.
- Is contactable by pager through the Christchurch Hospital telephone operator.

4. Common Emergency Presentations

4.1 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, (Professor of Emergency Medicine)
  - Dr D Richards (Director of Training)
  - Doctors J Bone, C Dillon, P Gee, S Inglis, R Ojala, S Pearson, S Peddie, M Than.
- Secretary, ☎ 89614
- Triage Nurse, ☎ 80274
- Consultant on Duty, ☎ 81900

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.

4.2 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.

4.3 Early Care of Trauma

Trauma web site: http://www.trauma.co.nz.

- The principles of the Emergency Management of Severe Trauma (EMST) course form the basis for evaluation and treatment guidelines.
- Care of injured patients requires a process of rapid assessment and resuscitation followed by thorough examination and appropriate definitive therapy (or transfer). The sequence of evaluation and treatment is therefore:
  - **Primary Survey**: a rapid assessment.
  - **Resuscitation**: immediate therapy for life-threatening injuries and physiological abnormalities detected in the Primary Survey.
  - **Secondary Survey**: a thorough 'top to toe and front to back' examination of the patient.
  - **Definitive Care**: the treatment to 'fix' the injury.

4.3.1 Primary Survey (ABCDE)

Airway (with c-spine control)

1. Assess the airway.
2. Create or maintain an airway by:
   - Suction
   - Chin lift or jaw thrust
   - Oro/nasopharyngeal airway
   - Oro/nasotracheal intubation
   - Cricothyroidotomy
3. Recognise the potential for cervical spine injury and maintain the spine in a safe neutral position until clinical examination and radiological findings exclude injury.

Breathing

1. Assess the chest clinically.
2. Consider chest drain where appropriate.
3. Administer high flow oxygen.

Circulation

1. Assess circulation.
2. Arrest external haemorrhage by local pressure.
3. Insert at least 2 large bore IV cannulae. If no IV access, consider venous cutdown. Take blood for CBC + Diff, cross-match, biochemistry, and ethanol.
4. Begin infusion with crystalloid resuscitation fluid. This should be warmed if possible. Patients with exsanguinating haemorrhage should be resuscitated with blood as soon as it is available (see Collection of Blood from Blood Bank on page 40).
5. Monitor the patient with an ECG and BP monitor and a pulse oximeter.

Disability

1. Determine the level of consciousness. Is the patient:
   - **A**wake?
   - Responding to **V**erbal stimuli?
   - Responding to **P**ainful stimuli?
   - **U**nresponsive?
2. Assess the pupillary size and response.

Exposure/Environmental Control

1. Expose the patient so that an adequate complete examination can be performed.
2. However, prevent the patient becoming hypothermic.

4.3.2 Resuscitation and Monitoring

Ongoing resuscitation of physiological abnormalities detected in the Primary Source is very important.

Monitoring of the progress of this resuscitation requires consideration of the following:

1. Respiratory rate.
2. Pulse (ECG monitor).
3. Perfusion.
4. Blood pressure.
5. Oxygen saturation (ABGs, pulse oximetry).
6. Urine output (urethral catheter should be inserted if not contraindicated).

4.3.3 Radiology

Unstable patients should never leave the Emergency Department for radiological investigation.

In general, only 3 x-rays are appropriate in the resuscitation room:

1. **Chest x-ray**: This is the only x-ray justified in an unresuscitated patient. If a pneumothorax is obviously present it is not necessary to wait for a chest x-ray. Have confidence in the clinical assessment. Insert a chest drain, and x-ray later.
2. **Pelvic x-ray**: A pelvic fracture not clinically obvious can be the site of unexplained blood loss. A dislocated hip can be missed in a patient with multiple injuries, especially if unconscious.
3. **Lateral cervical spine**: This should be done on any patient with any history of loss of consciousness, injury above the clavicle, or signs or symptoms of spinal injury. In these patients a spinal injury should be assumed to be present. A lateral c-spine x-ray may allow an injury to be confirmed early in the assessment process but exclusion requires a 3 view series.

4.3.4 Focused Assessment by Sonography in Trauma (FAST)

FAST may be performed in trauma patients who show signs of abdominal injury. This should be performed only by clinicians trained in the technique.

4.3.5 Secondary Survey

This assessment is a complete examination of the patient from 'top to toe and front to back'. Take a thorough history from the patient, bystanders, or ambulance staff, so that you have as clear an idea as possible of what happened to the patient. This will allow you to prioritise your suspicions and examine the patient with a clear view of what the most likely injuries are. Where possible this is also an appropriate time to record the other aspects of an **AMPLE** history.

- **A** **A**llergies.
- **M** **M**edications (cardiovascular medications and anticoagulants are particularly important).
- **P** **P**revious medical / surgical history.
- **L** Time of **L**ast meal.
- **E** **E**vents/Environment surrounding the injury.

Examination of the Patient

Head and face

- Inspect the whole head and face and palpate the region with gloved fingers.
- Check the pupils again.
- If the patient is cooperative, obtain a rough assessment of visual acuity.

Neck

- Maintain in-line immobilisation of the cervical spine and remove the front of the semi-rigid collar. Inspect the neck and palpate posteriorly.
- If not already performed, ask for a cross-table lateral cervical spine x-ray. Regardless of the result, keep the collar on.

Chest

- Re-evaluate the chest as in the Primary Survey.
- Ask for a CXR. This should be supine in the first instance unless there is no likelihood of any spinal injury.

Abdomen

- Inspect, palpate, percuss and auscultate the abdomen as you would in any other assessment of an acute abdomen.
- Perform a rectal examination. No urinary catheter should be inserted in any male patient before this is undertaken and confirmed as normal. Inspect the perineum and scrotum at the same time.
- If the patient has an abnormal level of consciousness he or she may need a CT abdomen.

Back

- With 4 assistants it is possible to safely log roll the patient and examine the back. Inspection and palpation are the crucial aspects and it may be possible to perform rectal examination at this time.

Extremities

- Carefully inspect and palpate each limb for tenderness, crepitation, or abnormal movement. If the patient is cooperative ask him or her to move the limbs in response to command in preference to passive movement in the first instance.
- Adequately splint any injuries.

Neurological examination

- Assess the Glasgow Coma Scale (see page 22).
- Look for any localising signs.
- Re-evaluate the pupils.

4.4 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).

B - Breathing Impairment

- Recognition:
  - Altered level of consciousness (cause and effect).
  - Hypoxia:
    - Pulse oximetry / arterial blood gases
    - Cyanosis.
  - Hypercapnia - arterial blood gases.
  - Tachypnoea or bradypnoea.
  - 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.
    - CPAP, BiPAP.
    - 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).

4.5 Altered Level of Consciousness

4.5.1 Initial assessment and resuscitation

See also: Stupor and Coma on page 162.

- 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 at 2-3 minute intervals as necessary. If no response and narcotic overdose suspected give naloxone up to a maximum of 4 mg. Higher doses can be given (see page 212), but in this situation, review the diagnosis of narcotic overdose before giving more than 4 mg. (2 mg may be required to reverse methadone overdose.) Flumazenil is available for benzodiazepine reversal but is rarely indicated in the emergency setting.

4.5.2 Complete assessment

History

- Patient, relative, ambulance, GP etc.

Examination

- The Glasgow Coma Scale (see page 22).
- 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.

4.5.3 Definitive Management

According to the cause (see page 162).

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/MRI head scan 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 head scan should be performed.
- Structural
  - Mass lesion, 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.*

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**

4.6 Shock

Definition - inadequate delivery and utilization of oxygen by vital organs due to a problem with the circulation.

- The inadequacy may originate in the pump, the outflow from the pump, the location the blood travels to, the volume of blood, or a combination.
- Assessment of the degree of shock can be difficult, as signs and symptoms will vary with the cause, the speed of onset, the patient's 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.
- If not already instituted, apply oxygen and establish secure IV access. Trendelenberg position. Patient should be managed in an area capable of monitoring and with resuscitation capability. An IDUC should be inserted and urine output monitored.
- Invasive monitoring of the circulation (CVP or Swan Ganz catheter) provides useful objective information but requires expertise in application and interpretation. The change in CVP in response to fluid challenges is more useful than the exact numbers. A low CVP 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 trauma or other unwell patients). Other more unusual causes of high CVP include tricuspid stenosis, tricuspid regurgitation, constrictive pericarditis, pericardial effusion, or SVC obstruction.

Cardiogenic Shock

- Arrhythmias, myocardial dysfunction, acute valvular dysfunction, ventricular or septal rupture, etc.
- Fluid therapy may occasionally be useful to increase filling pressure but more often specific therapy is necessary e.g., anti-arrhythmic agents, DC shock, inotropic agents etc.

Obstructive Shock

- Tension pneumothorax (obstructs venous return), pericardial tamponade or constriction, obstructive valvular disease (aortic or mitral), pulmonary hypertension, massive pulmonary emboli, cardiac tumours, etc.
- 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, etc.
- Skin is warm and pink.
- Relative hypovolaemia due to expanded vascular space.
- Fluid resuscitation and specific treatment is required.

Hypovolaemic Shock

- Blood loss, third spacing etc.
- Urgent surgical consult is necessary should haemorrhagic shock be suspected.
- CVP may be useful (see above), pulmonary capillary wedge pressure measurements provide the ultimate measure of volume status but are only practicable in ICU, CCU, or theatre.
- The urine output is a useful objective measure of renal perfusion assuming no diuretics have been given.

Table 2: Classification of Haemorrhagic Shock

Class I Shock
▪ Blood loss up to 15% blood volume (750 mL) ▪ CNS, Skin, Urine, Pulse, BP: no discernible abnormality
Class II Shock
▪ Blood loss up to 15 - 30% blood volume (750 - 1500 mL) ▪ CNS: agitated ▪ Skin: cool, pale ▪ Urine: decreased ▪ Pulse: tachycardia (>100 bpm) ▪ BP: normal (reduced pulse pressure)
Class III Shock
▪ Blood loss up to 30 - 40% blood volume (1500 - 2000 mL) ▪ CNS: agitated to confused ▪ Skin: cool, pale ▪ Urine: decreased ▪ Pulse: tachycardia (>120 bpm) ▪ BP: falling
Class IV Shock
▪ Blood loss in excess of 40% of blood volume (>2000 mL) ▪ CNS: confused (unconscious by 50%) ▪ Skin: white and cold ▪ Urine: nil ▪ Pulse: >140 bpm, peripheral pulses lost by 40%, central pulses lost by 50% ▪ BP: very low (absent by 50%)

- Therefore, haemorrhagic shock with hypotension suggests 1500-2000 mL of blood loss and demands rapid infusion of 2000 mL of crystalloid.
- Crystalloid 'splints' the circulation temporarily before extravasating, therefore more will usually be required (another 2000 mL).
- For Class III or Class IV shock, transfusion of blood will invariably be required.
- If fluids do not restore satisfactory circulation, then blood transfusion is urgent and should occur prior to cross-match using Type O negative blood (available from the Blood Bank, Lower Ground Floor, Christchurch Hospital). Type specific blood may be available from the blood bank prior to full cross-match.
- Don't forget localised control (pressure on external bleeding, surgery for internal bleeding).

Note: *The elderly and those on drugs such as beta-blockers are less able to compensate and therefore will become hypotensive earlier.*

Note: *There is a greater blood volume in advanced pregnancy and an ability to shunt blood from the placental circulation (at the fetus' expense); therefore shock manifests later in the mother (but earlier in the fetus).*

4.7 Syncope

Definition - a transient loss of consciousness.

4.7.1 Initial assessment and resuscitation

- Airway
- Breathing
- Circulation

Consider telemetry if you are concerned that an arrhythmia is a likely cause of syncope, e.g., if the patient has a known cardiac condition, cardiac symptoms, multiple episodes of syncope, syncope in the horizontal position, drug overdose/toxicity, or an electrolyte disorder. Refer to Telemetry Guidelines on page 83.

4.7.2 Complete assessment

History

The most important part of the assessment is a detailed history, which often requires talking to a witness.

Medications

Current medications, particularly hypotensive drugs.

Examination

- The examination should include careful palpation of pulse, rhythm, volume and character. The JVP should be measured in order to assess volume status.
- BP lying and standing with the heart rate response if there is a fall in blood pressure.
- The heart should be auscultated for murmurs, particularly the ejection murmur of aortic stenosis and hypertrophic obstructive cardiomyopathy.
- Look for focal neurology.

Investigations

- 12 lead ECG
- Random blood sugar
- Na, K and creatinine
- CBC + Diff

4.7.3 Possible causes

Possible causes, in order of prevalence:

- Vasovagal syncope

  Usually occurs when the torso is upright and may be triggered by needlestick phobia, standing in warm crowded rooms, or postural hypotension (particularly in the elderly).

- Cardiac syncope

  Classically occurs during exertion and may be preceded by angina or palpitations. It is usually rapid in onset and offset. It should be considered in anyone with a cardiac condition. It carries a poor prognosis, so if suspected, the patient should be admitted and monitored.

- Epilepsy

  These patients tend to fall and injure themselves. There may be a preceding aura. There is often post-ictal confusion and drowsiness for one to two hours.

- Miscellaneous causes:
  - Hypoglycaemia
  - Alcohol
  - Psychogenic
  - Remember that vasovagal syncope can occur in patients who are in a low output state because of a serious underlying condition, e.g., pulmonary embolus, septic shock, GI bleeding, etc.
  - TIA/stroke is an exceedingly rare cause of syncope and can be clinically excluded if there is no focal neurology.

4.8 Vertigo

A patient with vertigo is experiencing an hallucination of motion. In the acute situation it is whirling rotation of the environment. The cause is usually peripheral but can be central. There may be nausea and vomiting. There **must** be nystagmus. Magnification (Frenzel glasses or 20 diopter lenses) enhances observation of nystagmus and removes patient optic fixation. Any vertiginous patient without nystagmus in the sitting position **must** have a provocative positional test.

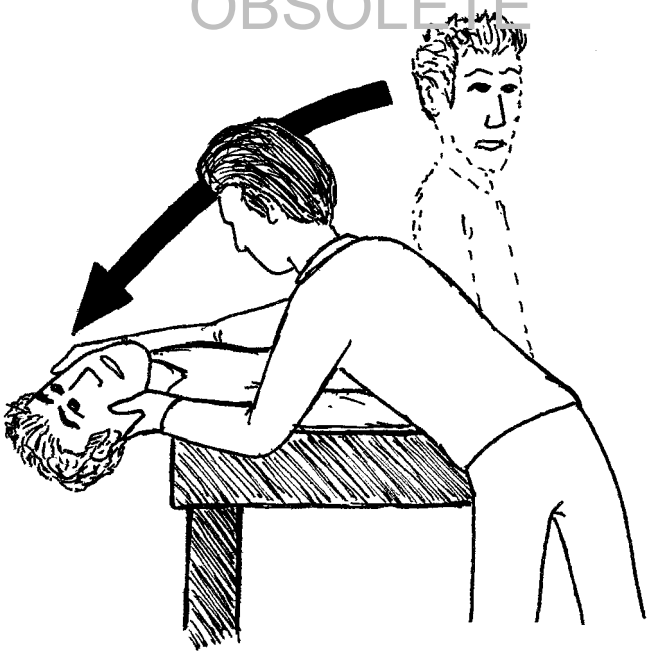
4.8.1 Peripheral causes

Benign Positional Vertigo (BPV)

- Brief (<30 seconds) vertigo induced by a change in head position (turning in bed, looking up). Onset may be dramatic and frightening. Due to dislodged otoconia moving in a semi-circular canal. There is no nystagmus when the patient is upright.
- Diagnosis is by the Dix Hallpike positional test (see below). This is a simple diagnostic procedure.
- In posterior canal BPV the nystagmus is torsional towards the undermost ear. In horizontal canal BPV the nystagmus is horizontal and reverses direction as the head is turned from side to side.
- Posterior canal BPV is the most common and treated by "repositioning" of the otoconia by the Epley Repositioning Procedure (see page 27).
- Most common in the middle aged and elderly. In younger adults it may follow head injury or vestibular neuritis.

Diagnosis of BPV

This is best established by the Dix Hallpike test. This is shown diagrammatically as follows:



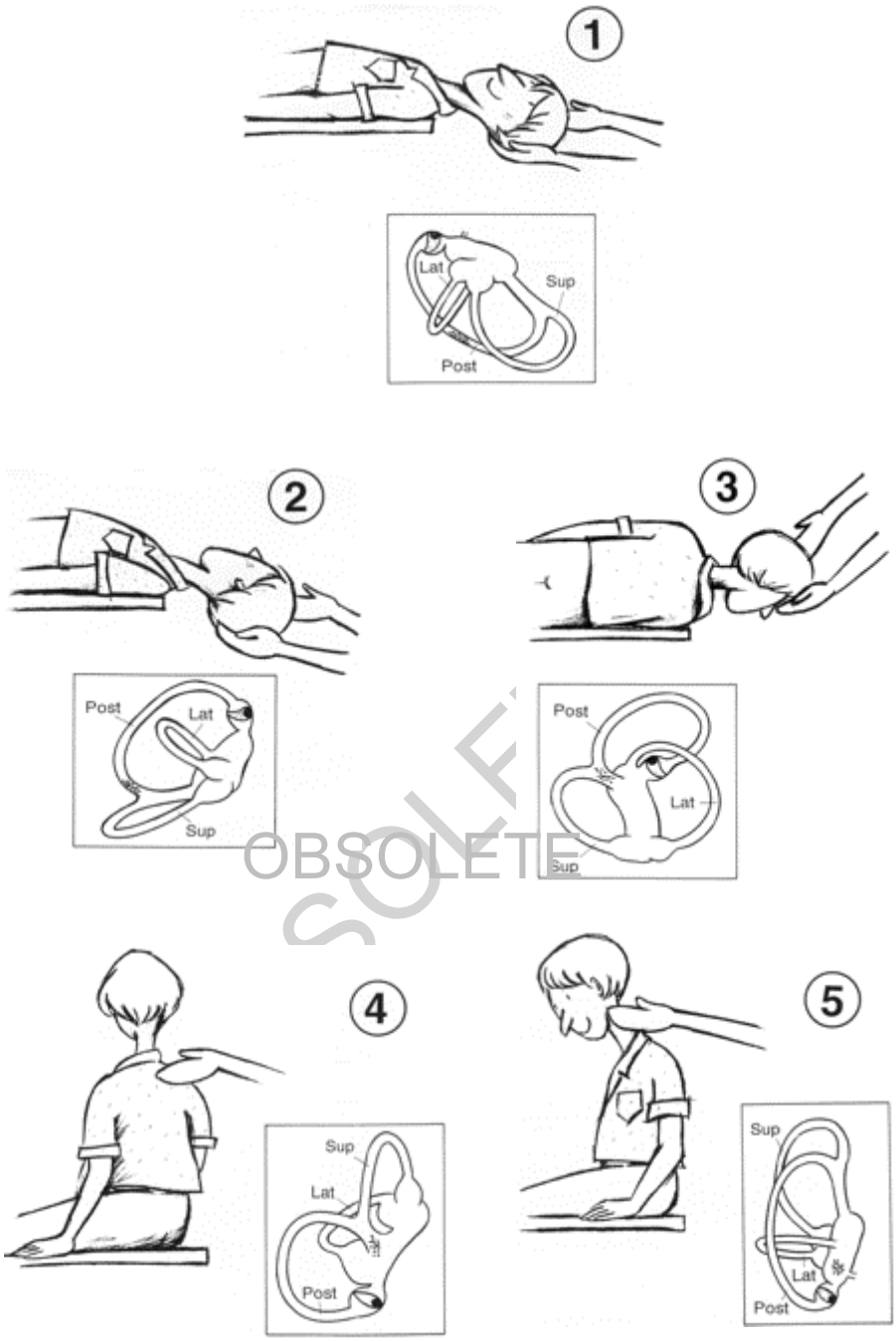
The patient sits with legs extended on the examination table. Turn the head 30° to 45° toward one side and quickly lie the patient back so their head hangs over the end of the table. Look for nystagmus.

Sit the patient upright for a few minutes to recover from the vertigo, then repeat the procedure with the head turned in the opposite direction. Again, record any nystagmus.

This test requires some experience to perform efficiently and safely. Seek advice from a more senior colleague with expertise in this procedure if you feel it is indicated.

Treatment of BPV

The Epley Canalith Repositioning procedure should be performed if a diagnosis of BPV has been made. Once again, this procedure requires experience. Seek advice.



The repositioning procedure is designed to remove the debris out of the canal. From position 1, the head is rotated 30 degrees to the right (2). Then the head and body are rotated 135 degrees (3). With the head to the right, the patient is sat up and then chin down (4, 5). The cycle is repeated until no nystagmus occurs. Patients are advised to sleep propped up on two pillows for two nights. Special care is required for patients with back and neck problems.

Vestibular Neuritis (Neurolabyrinthitis)

Acute vertigo is the only symptom, due to sudden unilateral vestibular failure (probably viral) of the superior and/or inferior nerve. The nystagmus is always undirectional and rotatory-horizontal with the fast phase away from the affected side. The patient is usually able to stand, but prefers to lie. The head impulse test is abnormal on the affected side and normal on the other side. (This is a test of the vestibular-ocular reflex (VOR). The patient, who is sitting up, is asked to fix their gaze on a distant object. The examiner suddenly moves the head 20-30° to the right, and then to the left. If the VOR is normal, on the side of the direction of the movement, the eyes will remain on the visual target.) The acute vertigo can last up to a week; balance recovery may take a month, and longer in older individuals. Benign positional vertigo may follow. The main differential diagnosis is cerebellar infarction.

Meniere's Disease

Inner ear disorder with attacks of vertigo (<12 hours) usually accompanied by deafness, tinnitus and aural fullness (blocked feeling) in the affected ear. Nystagmus is rotatory-horizontal, often initially towards the symptomatic ear and later away from it. Occurs in middle aged and older adults. Diagnosis is established by electrocochleography.

4.8.2 Central Causes

Usually associated with other symptoms such as headache, ataxia, diplopia, hemiparesis.

Migraine Vertigo

In some patients with migraine headaches vertigo can occur as a migraine aura accompanying some or all of their headaches.

Cerebellar Infarction

Vertigo, ataxia. Patients usually unable to stand. Nystagmus may be bi-directional or vertical, and not suppressed by optic fixation. If the head impulse test (see above) is positive the patient has vestibular neuritis. If the head impulse test is negative the patient may have a cerebellar infarct and an early MRI scan is required.

Multiple Sclerosis

Frequent early presentation is disturbance of balance and gait. A demyelinating lesion at the 8th nerve root entry zone can cause an attack of vertigo, which is initially indistinguishable from vestibular neuritis.

Vertebrobasilar Ischaemia

A large proportion of patients with vertebrobasilar distribution infarcts have preceding dizziness and vertigo. Ischaemia of brainstem nuclei and cerebellum cause abnormal perceptions of tilt and lateropulsion (falling). Ischaemia of the vascular supply to the ear can cause brief vertigo. Vascular ischaemic vertigo is typically **brief** and lasts **minutes**.

Acoustic Neuroma

Schwannoma of the superior vestibular nerve. Presents with tinnitus, hearing loss in one ear and subtle deterioration of balance, but occasionally with acute vertigo. Differential diagnosis is Meniere's disease and other central causes.

4.8.3 Management of Vertigo

Benign Positional Vertigo

Repositioning treatment (see page 27). Absence of vertigo on positional test, non-response to treatment or non-typical nystagmus should alert to a possible central cause.

Acute Spontaneous Attack

- Prochlorperazine IM or under top lip (Buccastem), suppositories if continued for days. An alternative drug is cyclizine.
- For migraine: sumatriptan or non-steroidal anti-inflammatory.

Vertigo Prophylaxis

- For Meniere's disease: salt restriction, betahistine
- For Migraine: beta-blocker, sodium valproate

Investigation

The three most relevant investigations are MRI (8^{th} nerve pathology, infarction/ischaemia, demyelination, tumour), pure tone audiogram, and electrocochleography for Meniere's disease.

4.9 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 pruritus
- **Respiratory:** Airway spasm/ oedema/ feeling of pharyngeal constriction.
- **Cardiovascular:** Shock
- **Gastrointestinal:** Cramps and diarrhoea

4.9.1 Immediate Management

- Secure airway and give high flow oxygen.
- Circulation: nurse head down, legs elevated to ensure cardiac and cerebral blood flow maintained.
- **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 with circulatory collapse, and/or the patient is unresponsive to the above initial treatment. Cardiovascular monitoring must be available. Begin with **0.5-1 ml of 1:10,000 (0.05 mg to 0.1 mg)** 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).

Note: If the patient is taking beta-blockers or tricyclic antidepressants, halve the initial dose of adrenaline, however patients on beta-blockers may respond poorly to adrenaline and may require additional doses of adrenaline, or glucagon in large amounts, or atropine. Ref BMJ 327.1332-1335. 2003.

Note: Ensure one person is in charge of proceedings and that multiple doses of adrenaline are not being given through different routes at the same time.

- IV fluids: secure access and infuse normal saline to maintain blood pressure.
- Antihistamine: promethazine 25 - 50 mg IM or diluted in 10 ml normal saline and given by slow IV injection.
- Hydrocortisone: 200 mg IV stat.
- Contact ICU if patient is failing to respond.

4.9.2 Further Management

- Admit for observation once stable.
- Start prednisone 40 mg orally daily and continue promethazine or change to loratidine.
- Tryptase: obtain serum sample noting time from onset of symptoms. Serum tryptase is a marker of mast cell activation and release, however its appearance in serum is delayed for 2 - 3 hours after mast cell release.
- If at high risk of recurrence, e.g., multiple previous episodes, strong suspicion of peanut and/or nut allergy, history of significant asthma, or lack of ready access to emergency care for logistical reasons:
  - Instruct in the self-administration of adrenaline.

  Note: at the time of writing, the only preloaded adrenaline device for IM injection is the EpiPen. Adult EpiPen contains 0.3 mg adrenaline. These devices currently cost approximately $150 and are not subsidised. Some patients may be unable to afford this cost. The only alternative is to instruct the patient in drawing up adrenaline from an ampoule, which requires significant time in education.
  - Refer the patient to the Immunology Service (see page 243).

  Note: at the time of writing, the waiting list for review in Immunology outpatients is 12 - 18 months, however each referral is prioritised on a case-by-case basis.

4.9.3 On Discharge

- On discharge, if the attack has been difficult to manage:
  - Prednisone 40 mg orally daily for 3 days.
  - Loratidine 10 mg orally daily for 3 days.
- If the reaction was associated with a drug, food, contrast media, or contact allergen (e.g., latex), **ensure the adverse drug reaction is appropriately documented and reported**:
  - ADR reporting guidelines (http://intraweb.chhlth.govt.nz/cph/pml/intro_04_adr.html).
  - CARM (national pharmacovigilance) ADR reporting form (http://intraweb.chhlth.govt.nz/ADR/adr.htm).

4.10 Head Pain

4.10.1 Initial assessment and resuscitation

- Airways, Breathing, Circulation: brief appraisal

4.10.2 Complete assessment

History, Examination, Investigations: directed according to the differential problem list

4.10.3 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 urgent investigations. CT scan should precede lumbar puncture if focal signs, papilloedema, or an impaired level of consciousness is present.*

4.10.4 Important to exclude

- Subarachnoid haemorrhage
- Space occupying lesions
- Meningitis

4.11 Chest Pain

4.11.1 Initial assessment and resuscitation

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

4.11.2 Complete assessment

History, Examination, Investigations: directed according to the differential problem list

Investigations usually include CXR and ECG but may require pulse oximetry/arterial blood gases and markers of myocardial damage. Myoglobin or rapid Troponin T 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 and Creatine Kinase (CK) at 6 - 12 hours from the onset of symptoms. Other investigations that may be required include a ventilation/perfusion scan, abdominal ultrasound, aortography, CT chest scan, echocardiography.

4.11.3 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.

4.11.4 Important to exclude

- Ischaemic heart disease
- Pulmonary embolism
- Pneumothorax
- Pneumonia.

4.12 Abdominal Pain

4.12.1 Initial assessment and resuscitation

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

4.12.2 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.

Investigations

Guided by findings above - not all are routinely indicated.

- Dipstick urinalysis for protein, blood. Urine microscopy and culture.
- Pregnancy test - either urine or blood.
- CBC + Diff.
- Urea, creatinine, Na, K, Ca, glucose, amylase, bili, AST, GGT, alk. phos.
- Erect CXR (?perforation). Supine/erect abdominal x-ray (?obstruction).
- Ultrasound - liver, biliary system, pancreas, kidneys, ureters, pelvis, aorta.
- Other radiology - contrast studies, CT scan.

4.12.3 Definitive management

According to the cause. Possible causes:

- Gastrointestinal
- Hepatobiliary
- Pancreatic
- Urological
- Gynaecological - complications of pregnancy (eg. ectopic)
- Musculoskeletal
- Respiratory
- Vascular
- Metabolic

4.12.4 Important to exclude

- Abdominal aortic aneurysm [may present like renal colic]
- Ischaemic bowel [non tender abdomen initially but pain may be out of proportion for clinical signs]
- Ectopic pregnancy
- Torsion of testicle.

4.13 Shortness of Breath

4.13.1 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
  - May need to check ABG in COPD patients to assess ventilation.
  - Refer to pulse oximetry (see page 224).

4.13.2 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.

4.13.3 Definitive management

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.

4.14 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.

4.14.1 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. The "Bair 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.
  - Overhead warming device e.g., Fisher & Paykel.
- 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.

4.14.2 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, Investigations: for traumatic injuries, underlying disease and complications of cold.

4.14.3 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.

4.15 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.

4.15.1 Initial assessment and resuscitation

- Airway, Breathing, Circulation: Supplemental oxygen
  - 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.*

4.15.2 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.

4.15.3 Definitive management

Usually cooling/monitoring continues in ICU including management of the many potential complications.

4.16 Renal Colic

4.16.1 History

- Pain: severe loin to groin radiation (50% of patients giving this history will not have a kidney stone).

4.16.2 Examination

- If there is a high fever >38°C + significant renal tenderness, infection may be present.
- May be tender over the affected kidney.

4.16.3 Differential Diagnosis

The following must be excluded in anyone with suspected renal colic, especially the elderly:

- Aortic and iliac aneurysms.
- Pyelonephritis.
- Peritonitis, including appendicitis and diverticulitis.
- Biliary colic.
- Renovascular compromise, including renal artery or vein thrombosis.
- Cancer, especially renal.
- Endometriosis.
- Ovarian torsion.

4.16.4 Investigations

- MSU: haematuria is present in only 85% of patients with renal colic. If there are white cells or bacteria in the urine, consider infected stone (may need antibiotics).
- CBC + Diff, creatinine, electrolytes, calcium, phosphate, uric acid (the white cell count is often raised even when there is no infection).
- Blood cultures if infection suspected.
- CT urogram and plain x-ray (KUB) for kidney, ureter and bladder (see page 37).

4.16.5 Management

- IV access for analgesia + fluids.
- Adequate analgesia with paracetamol or NSAIDs (or opioids if not controlled by simple analgesia).
- If vomiting consider suppositories.

Patients with infection and obstructed kidneys may develop urosepsis. Use gentamicin, initial dose 3-5 mg/kg IV. Take levels if gentamicin is to be continued for more than 48 hours.

4.16.6 Further Investigation

In the young, healthy patient in whom the diagnosis of renal colic is clinically not in question, the pain has completely settled and there is no suspicion of any complication, there is no need to obtain immediate diagnostic imaging but it should be arranged prior to discharge. If pain is severe and ongoing, the diagnosis is in doubt, another condition is suspected, or if the patient is elderly, some diagnostic imaging is essential.

1. **CT urogram**:
   - Is the first line of imaging.
   - Advantages: sensitivity 95-97% and specificity 96-98% in detection of renal stones. Faster than IVU, avoids intravenous contrast.
   - Limitations: will diagnose other conditions such as AAA and GI tract disease but is not as sensitive or as specific as CT with contrast.
2. **Plain x-ray KUB**:
   - 90% of renal stones are radio-opaque but the sensitivity is only up to 52-58% and the specificity 69-74%. Negative predictive value is only 23%.
   - In patients in whom the diagnosis is already established, plain x-ray is useful in following the passage of a radio-opaque stone.
3. **Intravenous urogram (IVU)**:
   - Comparable to CT in sensitivity and specificity for stone but also shows renal function.
   - Contraindications: serum creatinine >0.2 mmol/L, history of adverse (allergic) reaction to contrast, Metformin.
   - Contrast can be nephrotoxic in the following conditions: pre-existing renal insufficiency, diabetes, dehydrated patients, hypotension, age >60 years, multiple myeloma, hypertension, hyperuricemia, use of diuretics for cardiovascular system, history of IV radiocontrast within 72h.
4. **Ultrasound**:
   - When IVU or CT is contraindicated (e.g., pregnancy) or when there is no haematuria.
   - Will detect larger (>5mm) stones, particularly in the proximal and distal ureter but only poorly visualises midureteric stones.
   - Very sensitive for hydronephrosis (98%) but 22% of hydronephroses detected on ultrasound do not represent obstruction.
   - Advantages: non-invasive, no contrast, no radiation, no side effects. Can give clues to other pathology (such as AAA).

4.16.7 Subsequent Management

The decision to admit or discharge the patient must be taken by the consultant concerned.

Admission is required in the following situations:

- Fever >38°C, or septic, as may require a nephrostomy.
- Severe ongoing pain that does not settle with IV opioid and NSAIDs.
- Recurrent attacks of colic with repeated visits to the Emergency Department.
- Ureteric stones >6 mm in diameter (these are unlikely to pass).
- Any stone in a solitary kidney.

- Creatinine >0.2 mmol/L.
- Admission may be required in other circumstances. Discuss with consultant.

When discharged:

- Send a referral to the Urology Outpatient's Clinic. The patient will be seen in 4 weeks with an updated KUB film unless the stone is radiolucent when a limited IVU or non-contrast CT will be done.
- Advise patient to strain urine for a stone and to return if they develop a fever.
- Give the patient a prescription for diclofenac unless there is a contraindication to this drug.

4.17 Acute Urinary Retention

4.17.1 History

- Past symptoms of outflow obstruction and its duration.
- Any previous episodes of retention/haematuria.

4.17.2 Examination

- Presence of palpable bladder.
- Rectal examination to assess prostate size and consistency.

4.17.3 Investigation

- CBC + Diff, creatinine, electrolytes, PSA.

4.17.4 Management

- Catheterise patient (see page 53): urine specimen to laboratory.
- Do not have more than 2 attempts to pass a urethral catheter.
- If unsuccessful a supra-pubic catheter (SPC) may need to be inserted.
- If they drain more than 1.5 L, monitor fluid and electrolyte balance.

4.18 Macroscopic Haematuria

4.18.1 History

- Is it associated with renal or bladder pain or painful urination (dysuria)?
- Where does it occur in urinary stream: initial, total or terminal?
- Is it bright red blood, old dark blood or contain clots?

4.18.2 Examination

- Signs of hypovolaemia or anaemia.
- Presence of palpable renal mass or palpable bladder.
- Rectal examination to assess prostate.

4.18.3 Investigations

- MSU to confirm that the red urine is in fact blood.
- CBC + Diff, microscopy, creatinine, Na, K.
- Request "CT Haematuria".
- Cystoscopy (flexible as outpatient).

4.18.4 Management

- Discuss need for hospital admission with consultant.
- If there is macroscopic haematuria with clot retention:
  - Admit and investigate as for urinary retention.
  - Catheterise with large bore catheter (24 Fr) using 3-way Foley catheter.
  - Irrigate the bladder with a bladder syringe to remove clot and set up through and through irrigation.

4.19 Microscopic Haematuria

Microscopic haematuria without an infective cause should be investigated in Outpatients with:

- MSU, cytology, creatinine.
- Request "CT Haematuria".
- Flexible cystoscopy.
- Urinary cytology.

5. Blood Transfusion Practice

5.1 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 Blood Bank (Crossmatch and Tissue Typing Laboratory) is on the Lower Ground Floor of Christchurch Hospital.

- Blood Bank ☎ 80310 24 hours.
- Transfusion Medicine specialist - contact via the Blood Bank.

Product information and clinical guidelines are available on the Transfusion Medicine Guidelines site on the Canterbury District Health Board Intranet. See also the **Transfusion Medicine Handbook 2003** available from the New Zealand Blood Service.

5.2 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. For group and hold or cross-match, send 6 ml blood in EDTA tube.
- A sample of the patient's blood must accompany the requisition form. 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.
- A Christchurch audit revealed that 25% of patients were transfused to an excessively high haemoglobin level. If the haemoglobin is >110 g/L, a transfusion is rarely justified.
- In an adult, 1 unit of blood will raise the haemoglobin by 10 g/L.

5.3 Collection of Blood from Blood Bank

- During laboratory hours at Ashburton, Christchurch, Christchurch Womens, The Princess Margaret and Burwood Hospitals, blood is issued by laboratory staff on production of Transfusion Form QMR022A, properly completed.
- When blood is collected, the particulars on the container (ie. the patient's full name, patient identification number and group) must be checked against Form QMR022A.
- 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 (NZBS) or his/her deputy must be informed.**

5.4 Administration of Blood

- Written consent must be obtained from patients or their guardians before transfusing blood or blood products. In an emergency, a medical officer can take legal responsibility to transfuse without consent. Blood must not be collected until it is needed for transfusion. Half an hour is the maximum interval permitted between collection and administration. The transfusion must be completed within 4 hours of collecting the blood from the Blood Bank. 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.
- A record of the transfusion should be kept in the patient's notes.

5.5 Transfusion Reactions

- Reactions occurring during blood transfusion are extremely variable. Mild febrile reactions, temperatures <1.5°C increase from baseline, and transient skin rashes are common. Since a serious haemolytic reaction may initially present with such mild symptoms, any reactions occurring during blood transfusion must be immediately reported to the doctor responsible.

  Refer to Table 3: Guidelines for Management of Mild Adverse Transfusion Reactions (see page 42) and Table 4: Management of Moderate and Severe Adverse Transfusion Reactions (see page 43).

- 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, e.g., Yersinia enterocolitica. 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 regimen is meropenem 1 gm IV q8h plus gentamicin 5 mg/kg IV, loading dose.
  - If, after testing (see page 43), no cause can be found for a moderately severe reaction, it may be presumed to have an allergic basis. The transfusion can then be restarted following the administration of either paracetamol or hydrocortisone. As all blood is now leucodepleted by NZBS, these reactions are likely to be due to antibodies reacting against donor derived protein, or other allergens.

Table 3: Guidelines for Management of Mild Adverse Transfusion Reactions

FIRST MILD REACTION

- **Mild febrile reaction**
  - Temp up: <1.5°C from baseline.
  - Haemodynamically stable.
  - No respiratory distress.
  - **and** no other symptoms.

OR

- **Mild allergic reaction**
  - Occasional urticarial spots.
  - **and** no other symptoms.

Action:

1. Notify medical officer.
2. Check labels on blood/blood product bag/container and recipient identification.
3. Slow transfusion.
4. Consider giving medication:
   - antipyretic for pyrexia, e.g., paracetamol
   - antihistamine for urticaria
5. Continue transfusion at a slower rate with increased monitoring, e.g., blood pressure, pulse, temperature every 15-30 minutes.

SUBSEQUENT TRANSFUSIONS AND:

- **Recurrence of mild allergic reactions**

OR

- **Recurrence of mild febrile reactions**

Action:

1. Consider giving premedication:
   - Febrile reaction - antipyretic (e.g., paracetamol)
   - Urticarial reaction - antihistamine
2. Hydrocortisone - not usually needed.

Table 4: Management of Moderate and Severe Adverse Transfusion Reactions

MODERATE AND SEVERE ADVERSE TRANSFUSION REACTIONS MAY INCLUDE ANY OF THESE:

- Fever: ≥ 1.5°C from baseline; or fever with rigors/chills.
- Unexpected tachycardia.
- Unexpected change of blood pressure.
- Acute breathlessness, stridor or cyanosis; pharyngeal/laryngeal oedema.
- Extensive erythematous or urticarial rash; pain up transfusion arm.
- JVP acutely elevated.
- Loin pain; haemoglobinuria.
- Severe apprehension.

Action if moderate or severe reaction is suspected:

- **Stop** transfusion and review.
- **Call** for medical assessment. Commence appropriate treatment according to the patient's symptoms. This may include resuscitation.
- **Replace** IV set; administer intravenous normal saline to keep vein open and/or maintain blood pressure (**keep blood component / product bag and IV Giving Set**).
- **Check** that blood/blood product bag/container label and recipient identification information is correct.
- **Obtain specimens** (collect away from transfusion site) to recheck group and crossmatch, direct antiglobulin test, and to screen for red cell antibodies.
  - CBC + Diff and Biochemistry for Na, K, creatinine, urea, and 6 ml EDTA tube for blood group serology.

  All specimens are to be handwritten, referring to the patient identification band for patient details. These specimens may be used for subsequent crossmatches.

  And consider need for:
  - **Blood cultures if sepsis suspected.**
  - Blood gases if respiratory distress present.
  - Urine to check for haemoglobinuria.
  - Coagulation screen if bleeding or disseminated intravascular coagulopathy (DIC) suspected.

  Send
  - Adverse Reaction Notification form (111F00901) to Blood Bank
  - Blood product with IV set attached (in plastic bag) to Blood Bank (**NOT** via the Lamson Tube).
  - Blood Specimens to laboratory.

  Notify Blood Bank by phone: discuss urgency of follow-up tests and further transfusion needs.

  Discuss with Transfusion Medicine Specialist if severe reaction present.

  Further Treatment - depends on cause:
  - Septic reaction likely: antibiotics (e.g., gentamicin and meropenem).
  - Anaphylaxis/anaphylactoid reaction: adrenaline subcutaneous/ intramuscular
    Adverse reaction recurs: discuss use of washed cellular products with Transfusion Medicine Specialist/Haematologist.
  - Other: based on clinical state, test results and Transfusion Medicine Specialist consultation.
  - HLA antibodies: Red cell and platelet products are now leucocyte-depleted. HLA antibodies are unlikely to cause clinical reaction.

5.6 Blood Products Available

See also *Blood Components and Blood Products, Volume F - Fluid and Medication Management, Division Wide Manual*.

Table 5: Blood Components

Blood components available include:	
▪ Resuspended Red Cells ($165)	▪ Platelet Pool ($685)
▪ Resuspended Red Cells Neonatal	▪ Platelets Apheresis ($685)
▪ Whole Blood Autologous	▪ Fresh Frozen Plasma($169)
▪ Whole Blood Plasma Reduced	▪ Fresh Frozen Plasma Neonatal
▪ Cryoprecipitate ($318)	
Note: *Cryoprecipitate contains on average 1.3 g of fibrinogen per bag.*	
Note: *The above prices (as at 2004/2005) are approximate.*	

Table 6: Blood Products

Manufactured blood products have a NZBS label and are dispensed by NZBS directly to the requesting area.	
Blood Products include:	
▪ AHF (Factor VIII)	▪ Normal Immunoglobulin
▪ Albumex 20	▪ Prothrombinex HT (Factors II, IX & X)
▪ Albumex 4	▪ Anti-D Immunoglobulin
▪ Fibrogammin P (Factor XIII)	▪ Tetanus Immunoglobulin
▪ Hepatitis B Immunoglobulin	▪ Thrombotrol VF (Antithrombin III)
▪ Intravenous Immunoglobulin	▪ Zoster Immunoglobulin
▪ Monofix-VF (Factor IX)	▪ C1 Esterase Inhibitor
Note: *Recombinant clotting factors are available from Pharmacy.*	

5.7 Micro-Filters and Massive Transfusion

All blood products are now leucodepleted so micro-filters are not required for this patient group.

6. Fluids and Nutrition

6.1 Fluid Management

Fluids

- The body is about 60% water (two-thirds is intracellular, one-third extracellular).
- One-quarter of the extracellular fluid is intravascular and three-quarters is interstitial.
- The main intracellular cation is **potassium** while the main extracellular cation is **sodium**.

Normal daily fluid losses (2,500 ml per day)

- Urinary: 1500 ml
- Stool: 300 ml
- Respiratory tract: 200 ml
- Sweat: 500 ml

Normal daily requirements of fluid and electrolytes

- **Water:** ~2,500 ml
- **Sodium:** 75 mmol (~1 mmol/kg)
- **Potassium:** 50 mmol (~0.5 mmol/kg)

Reasons for increased fluid and electrolyte requirements

- Bleeding
- Vomiting or NG tube drainage: high in Cl, H and K
- Diarrhoea or high output stoma e.g., ileostomy
- Diuresis
- Hyperventilation
- Pyrexia: 200 ml more fluid lost/day for every 1°C increase in body temperature
- Sweating: contains large amounts of sodium

Types of Fluids

1. Crystalloids:
   - **Sodium chloride 0.9%**: 154 mmol/L Na, 154 mmol/L Cl
   - **Standard premix**: 0.9% sodium chloride + 30 mmol/L KCl
   - **Dextrose 5%**
2. Colloids:
   - Gelofusine®
   - Hemohes®
3. Blood Products:
   - RBC
   - Fresh frozen plasma (FFP)

General rules for IV fluids

The elderly, people with a reduced GFR, and people with cardiac dysfunction have difficulty excreting salt (sodium). It is critical to limit the infusion of intravenous fluids, particularly sodium chloride 0.9% in these people unless they have obvious large losses.

There are no magic formulae for predicting the clinical response to fluid therapy. The effects of any fluid prescription should be reviewed regularly. In patients with major fluid deficits receiving large amounts of fluid, hourly clinical assessment (pulse, lying and standing BP, JVP/CVP) may be necessary.

Resuscitation Fluids

1. Isotonic Crystalloids: use sodium chloride 0.9%
   - Large volumes required: 3 times the amount of blood lost.
   - Short half life (Note: only 20% remains in the intravascular space after 2h).
2. Colloid: Gelofusine® or Hemohes®
   - Colloids may be indicated in septic and anaphylactic/cardiogenic shock but use should be discussed with more senior medical staff in the first instance.
3. Blood (see "Blood Transfusion Services" on page 40)

   The only fluid available that will carry oxygen! Indicated if the patient is anaemic and haemodynamically unstable, has persistent hypoxia, or has lost a significant amount of blood (30% of blood volume).
   - Best to use fully typed and cross-matched blood (6 ml EDTA tube; takes 30 min if no antibodies found). Group specific uncross-matched blood takes 10 min.
   - In desperate situations use uncross-matched Group 0 negative blood.
   - Keep blood and patient warm if massive transfusion necessary.

Maintenance fluids

- Ensure patient really needs IV fluids and is unable to manage with oral or NG fluids.
- Sodium chloride 0.9% or 5% dextrose depending on cardiac and renal function and plasma sodium concentration.
- Daily weighing gives accurate assessment of fluid balance.

Replacement fluids

- Sodium chloride 0.9% or sodium chloride 0.9% + 30 mmoL KCl if high potassium losses.
- Usually not necessary to use other types of fluids.
- Use of IV bicarbonate to correct metabolic acidosis not usually appropriate. If you think it may be needed, discuss with consultant.

6.2 Nutrition Support

6.2.1 Introduction

Studies have shown that up to 50% of patients on admission to hospital have evidence of protein energy malnutrition caused by reduced nutrient intake. Nutrition support is the provision of nutrients orally, enterally, or parenterally with therapeutic intent.

Nutritional support is individualised, based on a formal nutritional assessment and concomitant factors such as disease state, organ function, metabolic condition, electrolyte measurements, medication use, and duration of nutritional support proposed.

Parenteral nutrition (PN) consists of a 2 litre bag containing glucose (250 - 350 g), amino acids (80 - 100 g), electrolytes, trace elements and multivitamins, and a 500 ml bottle of either 10 or 20% lipid emulsion providing essential fatty acids and the balance of energy.

PN may be required where enteral nutrition is not possible. This is likely to occur in the following circumstances:

- Inadequate enteral/oral intake over a period of 7 - 10 days, reducing to 5 - 7 days if the patient is catabolic or malnourished.
- Severe pancreatitis.
- Mucositis following chemotherapy.
- Short bowel syndrome.

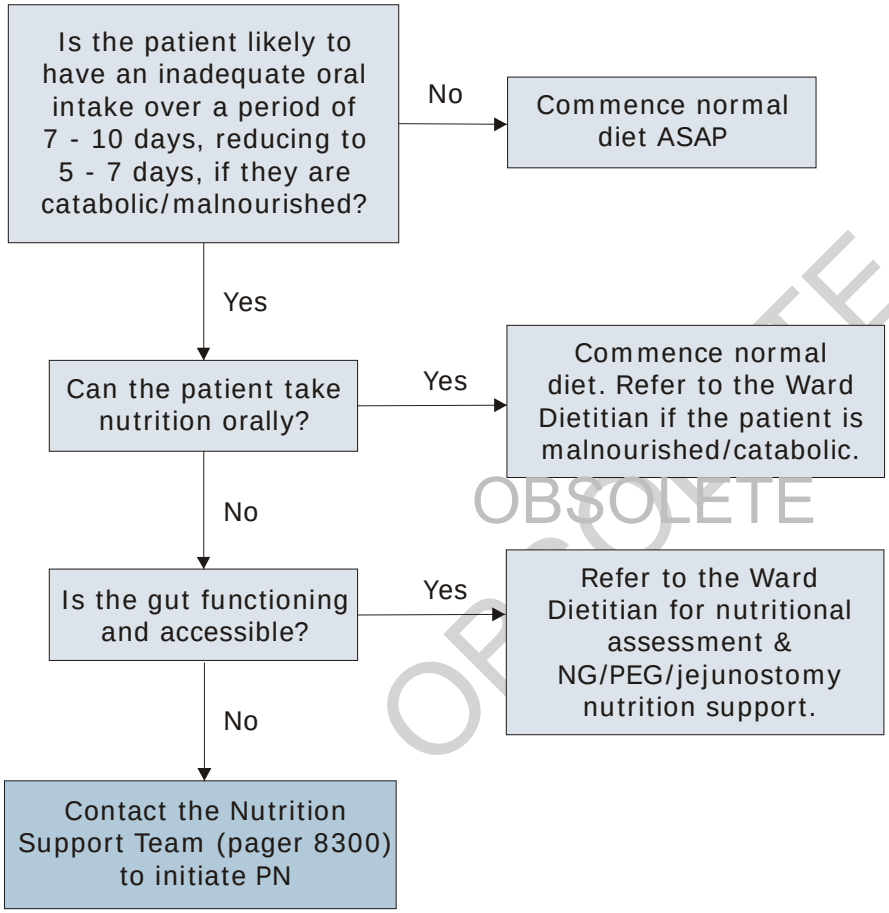
PN of less than 5 days is unlikely to benefit the patient.

PN can be initiated once a peripherally inserted central catheter (PICC) has been placed and the Nutrition Support Team (pager 8300) or the ward dietitian has been consulted. PN is initiated at half rate (42 ml/hr of Premix 5) for the first 24 - 48 hours. Lipid emulsion is commenced at the same time and run over 8 - 12 hours.

Bloods for biochemistry should be taken at approximately 18:00 hours when the PN bag is changed. This expedites the writing of the following day's prescription, which must be faxed to the pharmacy by 11:00 hours.

If in doubt, contact the Nutrition Support Team (pager 8300).

6.2.2 Medical Review of Patient's Nutritional State



Notes:

- PICC line must be in situ to commence PN.
- Baseline bloods are required before commencing PN. These are Na, K, urea, creatinine, glucose, Ca, PO_4, Mg, alb, bili, alk. phos., AST, ALT, GGT, zinc, trigylcerides, CBC + Diff, prothrombin time.
- Iron is not contained in PN and may be required in a separate infusion.
- Vitamin K must be provided via IV line 10 mg 2x/week.
- Patients referred after 11:00 Friday and in the weekend will receive a standard PN without calcium, trace elements, and vitamins. Early referral is encouraged.

7. Medical Procedures

7.1 Introduction

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

A Clinical Skills Laboratory has been established for the teaching of clinical skills. It is on the Christchurch Hospital Campus. Please contact telephone extension 81673 or clinicalskills@cdhb.govt.nz or the Clinical Skills website on the Intranet (under Training).

7.2 Intravenous Cannula Insertion and Care

Any procedure that 'breaks' the protective skin surface has the potential to introduce infection. It is important for RMO's to be skilled in IV line insertion. 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 and documented in the clinical notes.
  - Wash hands. Soap and water is adequate. Non sterile gloves are recommended in all cases and essential if there is significant risk of infection from blood contamination (eg. severe dermatitis, open wounds).
  - Choose an upper limb vein if possible. Avoid antecubital fossa if you can and remember to use a tourniquet or sphygmomanometer.
  - 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. Make sure you can still feel arterial pulsation after the tourniquet has been applied. If veins are poor, warm the limb and use a sphygmomanometer inflated to 70 - 80 mmHg.
  - Insert cannula into vein. Avoid touching puncture site. Obtain flashback and advance further to ensure that the plastic cannula is in the vein. 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 which will stabilize the cannula and act as a dressing.
    - Tape (eg. 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

- **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 IV Cannula**
  - 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 Cannula Infection**
  - Remove giving set.
  - Clean the cannula exit site with antiseptic solution 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.

7.3 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.

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 (eg. by ambulance staff, or in Emergency Department).
- Failure to seek help when you have had 2 or 3 attempts to insert an IV line.

7.4 Peripheral Blood Culture Technique

Using BacTAlert bottles: Wash hands before and after the procedure. Examination gloves should be worn to protect yourself.

Remove and discard plastic cap of BacTAlert bottle.

Clean patient's skin and rubber seal of BacTAlert bottle with an antiseptic solution. Allow to dry as alcohol kills bacteria by dessication. We recommend 1% chlorhexidine and 70% isopropyl alcohol. Avoid iodine-based solutions.

Do **not** palpate the venepuncture site from the time of antiseptic application until after the needle has been removed. Draw 20 mL of blood into the syringe. Do **not** press down on the venepuncture site whilst removing the needles as this may damage the vein. Do **not** allow the cotton wool ball to touch the needle as the needle is withdrawn. Do **not** change the needle.

Inoculate one BacTAlert set (one bottle for aerobes and one bottle 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.

Taking 2 to 3 blood cultures improves the sensitivity of the test. If taking multiple blood cultures, repeat the whole procedure at different sites, preferably a few minutes apart for acute sepsis and an hour or two apart for acute endocarditis. Taking samples before giving antibiotics also improves sensitivity but therapy should never be delayed in an acutely ill patient.

7.5 Insertion of Central Venous Lines

Central Venous Lines (CVL) 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). These should also be considered for patients with difficult peripheral venous access who require ongoing IV therapy.

7.6 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 38mm needle, and sterile technique. Local anaesthetic (1% lignocaine) infiltration of skin and subcutaneous tissue is required. It is essential to have the assistance of a nurse when performing a chest aspiration.

Diagnostic Pleurocentesis - Method

- Explain the procedure to the patient. The only common complication is pneumothorax which occurs in approximately 5%. Obtain verbal consent and document in the clinical notes.
- Obtain the most recent chest 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:
  - Infiltrate with 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. Stop the procedure if you aspirate air or the patient develops pain or coughing. Withdraw the needle immediately and arrange urgent CXR.
  - 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.
    Refer to Pleural Effusion (see page 237) 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.**

Therapeutic Pleurocentesis - Method

Explain the procedure to the patient. The most common complication is pneumothorax. Obtain verbal consent and document in the clinical notes.

- Follow the same initial steps as described in Diagnostic Pleurocentesis (see page 50). 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 stopped when:
  - 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.

7.7 Insertion of Intercostal Tubes

The insertion and management of intercostal tubes is a complex and specialised area. Internal medicine patients requiring chest tube management should be cared for by the specialist respiratory or cardiothoracic surgical team in their respective wards.

The choice of the particular drain and drainage collection system should be discussed with the consultant in charge before the procedure.

Unless it is an emergency, intercostal tubes are inserted or supervised by trained staff only.

See also: Intercostal Tubes on page 241.

7.8 Joint Aspiration

Explain what you propose to do to the patient. Obtain verbal consent and document in the clinical notes.

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 with aseptic technique 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.

See also: Rheumatology on page 243.

7.9 Lumbar Puncture

See also: Meningitis on page 126, and Subarachnoid Haemorrhage on page 154.

- RMOs should observe 2-3 lumbar punctures, then practise on a model in the Clinical Skills Unit and 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 a consultant.

Before performing the lumbar puncture:

- **Always consider**:
  - **Does CT/MRI need to be done first?** Do not perform a lumbar puncture if there is any clinical suspicion of raised intracranial pressure from a space-occupying lesion. If there is 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. Check whether the patient has recently received heparin. Lumbar punctures should not be done within 12 hours of a dose of low molecular weight heparin.
  - **Are there any other non-invasive diagnostic procedures which will give you the information you are looking for?**
- You **must** consult the lumbar puncture protocols in the department in which you are working. If none are available, follow the guidelines given here.

  Note: *The recommendations given here **do not** cover the administration of drugs intrathecally.*
- We recommend the use of 25-26G pencil-point lumbar puncture needles for routine use - 22G pencil point needles are reserved for difficult taps.

  Note: *The pre-packaged lumbar puncture sets on the wards may not contain a pencil point needle - please check.*

Performing the Lumbar Puncture:

- Explain the procedure, the indications, and possible complications to the patient, and obtain written consent. The patient may wish to use the toilet before 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.*
- Assist with positioning the patient on their side, head flexed, knees tucked under their chin to help widen intervertebral spaces and assist in locating the intrathecal space for tapping. Place flat pillow between knees to aid correct positioning.
- Decide before you start whether a pressure measurement is required.
- The use of an atraumatic (pencil-point) needle rather than a Quincke (cutting) needle reduces the incidence of headache from up to 25% to 5%. A 25-26G pencil-point needle may further reduce the risk of complications compared with a larger gauge needle.
- Aim for the L3-4 or L4-5 disc spaces. Use strict aseptic technique and chlorhexidine/alcohol for skin sterilisation. Local anaesthetic (lignocaine 1%) infiltration of skin and subcutaneous tissue is required.
- 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.

- When CSF flows, collect samples of CSF into three 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.
- 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.

  Note: *Sitting position with patient hunched over 1-2 pillows placed on their thigh could be considered if location of CSF difficult in the lateral position - however, CSF pressure measurements will be uninterpretable.*
- 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.
- Give analgesia 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.
- **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.**

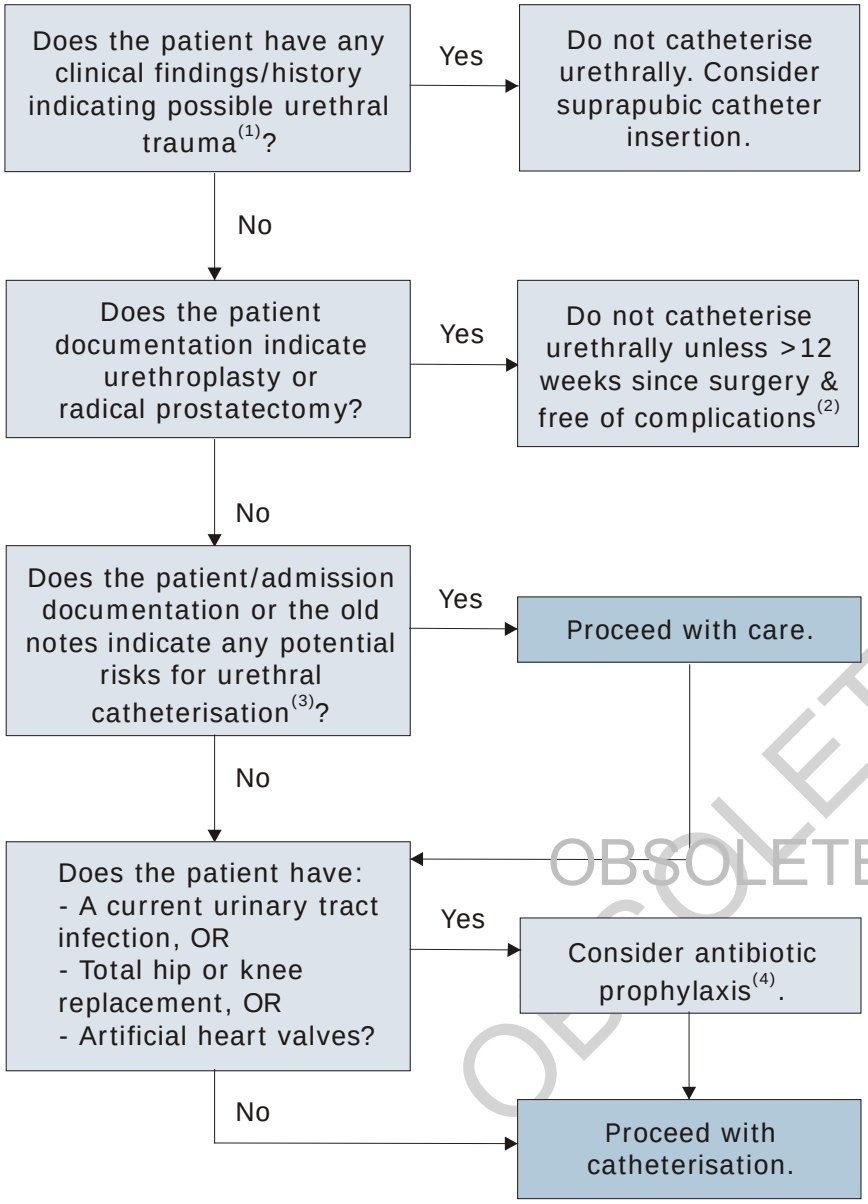
7.10 Urethral Catheterisation

7.10.1 Indications for Urethral Catheterisation

- Acute retention.
- Unconscious or sedated patients unable to void.
- In the operative and peri-operative setting.
- Patients with prolonged epidural anaesthesia e.g., in labour.
- Chronic retention if associated with impaired renal function or infection.
- Incontinence. Note: urethral catheterisation for incontinence needs to be carefully assessed in light of social situation.

7.10.2 Algorithm for Male Urethral Catheterisation

Note: Male urinary catheterisation can be performed by Registered Nurses when they have been trained, audited, and are competent in this procedure.



If the decision has been made that it is safe to proceed with urethral catheterisation by an audited staff member:

- Explain the procedure to the patient.
- Select appropriate catheter.
- Set up equipment following recommended best practice.
- Insert catheter as per recommended best practice.

Refer to Insertion of Urinary Catheter (see page 55).

Notes

For all staff audited for male urethral catheterisation.

(1) Straddle injury (fall, kick, cycle) or fractured pelvis (car accident, fall, crush) accompanied by penile tip blood, lower abdominal pain and inability to pass urine, or perineal haematoma.

(2) History of:
- Urethroplasty or Radical Prostatectomy < 12 weeks

This surgery indicates the presence of a urethral graft or anastomosis. Catheterisation should therefore be performed by a Urology Registrar if available. If unavailable, medical staff to insert a suprapubic catheter.

- Urethroplasty or Radical Prostatectomy > 12 weeks

  Proceed with urethral catheterisation with care using 14g catheter. If unsuccessful, insert a suprapubic catheter.

(3) Potential risks for urethral catheterisations:
- Other prostate or urethral surgery in the last four weeks (e.g. TURP, urethrotomy, bladder neck incision)
- Urethral trauma in the last four weeks
- Known prostate enlargement
- Known urethral stricture
- History of long term difficulty in passing urine (e.g. urinary retention, poor urinary flow)
- History of difficult urethral catheterisation previously

(4) For bacterial endocarditis prophylaxis, refer to Antibacterial Recommendations for Genitourinary Tract and Gastrointestinal Tract (excluding oesophageal) procedures (see page 92). For other situations, seek consultant advice.

7.10.3 Insertion of Urinary Catheter

Choose catheter size and type according to reason for catheterisation. Choose the smallest catheter that will suit the purpose of the catheterisation:

- **Use catheter size 14 - 16**:
  - For uncomplicated urinary retention.
  - To facilitate accurate urine measurements.
  - For urinary incontinence.
- **Use catheter size 20 - 24, 3 way**:
  - For moderate to heavy haematuria with potential for clots e.g., post urological surgery, bladder and/or prostate cancer, renal trauma.
- **Recommended best practice**:
  - **If catheter does not pass along the length of the urethra and into the bladder with ease, do not proceed.** Remove catheter and discuss with medical staff or Urology Charge Nurse
  - **If catheter is being inserted for retention**, ensure volume of urine drained is measured and documented in patient's notes.
  - If catheter enters the bladder and urine begins to drain, **advance the catheter until the Y-connection reaches the meatus before inflating the balloon**. This ensures the balloon is clear of the urethra and within the bladder, preventing trauma on inflation.
  - **If the patient has not been circumcised**, return the foreskin to its natural position after catheterisation.
- **Indications for Suprapubic Catheterisation:**
  - Failed urethral catheterisation
  - Long term management of patients with neuropathic bladders

  If contemplating this always discuss with the Urology Registrar or Consultant.

  Beware of lower abdominal scars from previous surgery: loops of bowel may be under the scar between skin and bladder.

7.10.4 Subsequent Management

Urinary catheters should be removed as soon as possible, but this will vary according to the circumstances. Seek advice - if the catheter is removed prematurely, it may have to be reinserted.

8. Clinical Pharmacology

8.1 Clinical Pharmacology Department Information

- For specific pharmacology information, refer to The Preferred Medicines List (referred to as The Pink Book).
- For all patient-related drug enquiries, contact Drug Information ☎ 80900.

Main Office

- Ground Floor Parkside, Department of Medicine, ☎ 89670 Fax 81003

Consultants

- Prof. Evan Begg
- Dr Murray Barclay

Consultation Service

- For consultations, Fax 81003.
- For urgent consultations, contact the registrar ☎ 80901, or Drug Information ☎ 80900.

Consultation Guidelines

Any pharmacology issues, for example:

- Interpretation of drug concentrations, advice on therapeutic drug monitoring and toxicology.
- Difficult polypharmacy.
- Guideline writing.
- Drug utilization / costs.
- Drug information.

We encourage clinical teams to work closely with the clinical pharmacists.

Clinical Pharmacology Intranet

The Clinical Pharmacology site provides ready access to information about all aspects of the Clinical Pharmacology Service, and contains:

- The "Pink Book".
- MIMS: access to the MIMS formulary online and for PDA.
- Adverse Drug Reactions: on-line reporting.
- Drug Profiles.
- Patient Information Leaflets: can be printed from here.
- Therapeutic Drug Monitoring: detailed drug profiles.
- Electronic BNF.
- PHARMAC link: to the Pharmaceutical Schedule.
- MEDSAFE link: includes drug manufacturers' datasheets.
- Drug information.
- Drug utilisation review: drug expenditure, campaigns.
- Bulletins/Guidelines: clinical pharmacology bulletins/guidelines.

To access this site, go to the CDHB Intranet home page > Clinical Information > Clinical Pharmacology.

8.2 Drug Information Service

Staff

- Bob Buckham
- Judy Dalrymple
- Pam Buffery

☎ 80900, pager 8264, Fax 80902.

Email: druginfo@cdhb.govt.nz

Function

To answer patient-related drug information enquiries from health professionals.

- Verbal answers can usually be provided immediately or as soon as possible.
- Written, referenced answers are provided for more complex questions.

8.3 The Preferred Medicines List (PML), known as 'The Pink Book'

"The Pink Book" reflects local prescribing practice and includes common daily dose ranges, costs and advice about prescribing. It is updated annually, written in conjunction with local specialists. It is a non-restrictive document available in hard copy, for PDA, and on the CDHB intranet.

"The Pink Book" has three sections:

- **The PML section:**
  - Drugs should be selected from the Preferred Medicines List unless there is a compelling reason not to, e.g., patients admitted on a non-PML drug.
  - Drugs noted 'cons' should only be prescribed with consultant approval.
- **The Antibiotic Guidelines section:**
  - System based guidelines for specific infections and pathogens.
  - Antibiotic sensitivity tables.
  - Gentamicin/tobramycin and vancomycin dosing guidelines.
  - Surgical prophylaxis and postoperative antibiotic guidelines.
- **The Pharmacology Guidelines section:**
  - Cytochrome P450 drug metabolism and interactions.
  - Drug use in renal and liver impairment and the elderly.
  - Therapeutic drug monitoring (TDM).
  - Specific issues such as QT interval prolongation and serotonin syndrome.
  - Drugs in pregnancy and breastfeeding.
  - Drug and food interactions.

Drug Profiles:

- For commonly used drugs, key information is tabulated.

Obtaining the PML for PDA

To obtain the PDA version of the PML:

1. Go to http://www.fonz.org.nz/pml.
   - User name: pml
   - Password: captopril
2. Follow the instructions on this web page.

8.4 Drug Utilisation Review

Staff

- Jane Vella-Brincat, ☎ 89971

Function

- Regularly monitor drug usage throughout the CDHB hospitals and provide feedback to clinicians.
- Carry out drug utilisation reviews, clinical audits, and drug related campaigns.
- Produce regular clinical pharmacology bulletins.

8.5 Dose Individualisation and Therapeutic Drug Monitoring

Individualisation of patient treatment is the basis of good prescribing. Drug selection and choice of maintenance dose rates are important clinical decisions. How to adjust the dose rate in renal impairment is the example illustrated below.

Other patient characteristics such as age, weight, presence of liver impairment, diseases, interacting drugs, pregnancy, breast feeding etc. should be considered. See The Pink Book for more detailed information.

For some drugs with a narrow therapeutic index, therapeutic drug monitoring is recommended. See The Pink Book. Clinical Pharmacology reviews these laboratory results daily and provides advice.

For dose individualisation of aminoglycosides and vancomycin, contact the ward pharmacist or Drug Information ☎ 80900.

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.

How to adjust the dose:

1. Decide on an appropriate dose-rate (i.e. total dose in 24 hours) for a patient with normal renal function (D_{normal}).
2. Calculate the patient's creatinine clearance (CrCl) using the Cockroft and Gault equation:

$$\text{CrCl (mL/min)} = \frac{(140 - \text{age}) \times \text{lean body weight (kg)}}{\text{plasma creatinine (mmol/L)} \times 800} \quad (\times 0.85 \text{ if female})$$

   - 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.
3. Calculate the dose-rate for the patient ($D_{patient}$):
   - For drugs with fu ≥ 0.9:

$$D_{patient} = \left(\frac{\text{Calculated CrCl (mL/min)}}{100 \text{ (mL/min)}}\right) \times D_{normal}$$

   - For drugs with fu < 0.9:

$$D_{patient} = \left[(1\text{-fu}) + \text{fu}\left(\frac{\text{Calculated CrCl (mL/min)}}{100 \text{ (mL/min)}}\right)\right] \times D_{normal}$$

4. Decide whether to decrease the dose or increase the dose-interval (usually increase the dose-interval). Aim for once or twice daily dosing as this will maximise compliance.

Note: To calculate creatinine clearance, the creatinine must be stable. Calculated creatinine clearance is routinely reported by the laboratory (using the MDRD formula). Both methods give estimates that are valid for most 'normal' people. The further the patient is from normal (age, height, weight, etc.), the less valid the estimate. Normal creatinine clearance is > 90 mL/min.

9. Alcohol Related Problems

9.1 Alcohol Withdrawal

- During alcohol withdrawal the following sequence of events may be seen:
  - tremor
  - tachycardia
  - hallucinations
  - seizures
  - delirium tremens (autonomic hyperactivity, disorientation and hallucinations).
- Administer thiamine 100 mg IM **before** glucose given. Many patients with alcohol dependence 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 patients with alcohol dependence 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 cover the risk period.
- Alcoholic hallucinations - Haloperidol 2 mg IM then 1-3 mg BD maintenance. Oral therapy when appropriate.

  Note: *Haloperidol may provoke fits or hypotension. Give lower doses in the elderly.*
- 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/h infusion. If the Diazemuls preparation is prescribed, this is diluted in D5W. Oral therapy when appropriate.

  Note: *Phenothiazines are to be avoided because they are epileptogenic.*
- Seizure prevention is generally achieved with diazepam loading and withdrawal over 3-4 days. If there is a history of break-through seizures use carbamazepine 400 mg stat then 200 mg TDS for 5 days. Some authorities recommend phenytoin or sodium valproate as alternatives. To treat seizures use diazepam and see section on epilepsy.
- Wernicke's Encephalopathy should always be suspected. Look for the triad of confusion, ataxia, ophthalmoplegia. If the full syndrome is present give **IV** thiamine, 100 mg diluted in 100 ml normal saline over 30 minutes, three times daily, until signs resolve or plateau.

  For suspected Wernicke's, thiamine 100 mg IM twice daily may be used. All patients should be given **long term treatment** with thiamine 100 mg BD PO and Vit. B Complex twice daily at the end of the parenteral regime.
- Ethanol - Formula to convert units:

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

9.2 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 specialises in alcohol-related problems is employed at Christchurch Hospital in the Social Work Department (Ext: 80420 Beep 8986). 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 Zealand 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 ongoing patterns (ALAC):**

Males	Over 21 standard drinks per week.
Females	Over 14 standard drinks per week.
Male	Over 6 standard drinks per occasion.
Female	Over 4 standard drinks per occasion.

- **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 about 10 grams 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.
  - **Standard NZ drink** = one unit

    = 12 oz beer, or

    = 4 oz wine (small glass), or

    = 1oz spirit (1 pub nip)
  - 1 jug of beer = 3 standard drinks
  - 750ml bottle of wine = 8 standard drinks
  - 1125ml bottle of spirits = 40 standard drinks
- **Diagnostic Criteria for Alcohol Dependence** (adapted from Diagnostic and Statistical Manual of Mental Disorders. 4th Edition 1994)
  - 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.
- Continued use despite knowledge of significant medical or psychological consequences.
- 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 on those days?
  - 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 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 support groups to inpatient rehabilitation programmes. The Alcohol Social Worker (Beep 8986 or Ext. 80420) offers a discharge recovery programme which can include follow-up and referral to a community agency. Family members who express concern may also be referred.

9.2.1 Facilities available to help with alcohol related problems

- Christchurch City Mission

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

- Community & Alcohol Drug Service/ Methadone Programme

  Ph: 335 4350, Fax: 335 4351
  Sylvan Street, Hillmorton Site, Private Bag 4733

- Odyssey House

  Ph: 358 2690
  98 Greers Road (Residential - 26 beds for severe drug dependence. Long term therapeutic community and Youth Day Programme.)

- Kennedy Villa

  Ph: 339 1139
  Hillmorton Hospital (Detoxification)

- Salvation Army - Bridge Programme

  Ph: 338 4436
  35 Collins Street (Residential, day and women's programmes)

- Nova Lodge

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

- Te Rito Arahi

  Ph: 379 5709
  194 Madras Street (Maori Alcohol & Drug Resource)

- Wahine Whai Ora. Women's Alcohol and Drug Service

  Ph: 365 6601, Fax: 365 9936
  276 Hereford Street (Also open to client's children, and provides a day programme for outpatients)

- Thorpe House

  Ph: 379 1682, Fax: 371 0602
  228 Worcester Street
  (Non-medical detox)

- Vincentian Centre

  Ph 379 9338
  222 Wilsons Road (Inpatient programme for alcohol dependent males and women with children)

- Alcoholics Anonymous

  Ph 379 0860

- Home Detox Service

  Ph: 365 0635
  276 Hereford Street (for people over 18)

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.

10. Anaesthesia

If there are any concerns with regard to the preoperative preparation of a patient, then:

- Contact the Anaesthetist concerned.
- If Anaesthetist unknown, contact the Department of Anaesthesia, 364 0288, ☎ 80288 **or** contact the Duty Anaesthetist on Pager 8120 **or** contact the Operating Theatres ☎ 89385.
- For every non-elective patient requiring an anaesthetic, including out-of-theatre cases, e.g., cardioversions and DSA cases:
  - Fax a completed theatre booking form to the theatre co-ordinator, ☎ 81573
  - Inform the Duty Anaesthetist (Pager 8120) or, if out-of-hours, the registrar (Pager 8212).

10.1 Routine Preoperative Investigations

Pre-operative investigations serve two main purposes; to evaluate known or suspected medical conditions, and/or to confirm the apparent fitness of the patient for the procedure. The detection of abnormalities allows for corrections to be made, if possible, and thereby decrease the risk of complications with anaesthesia and surgery.

A history and clinical examination should be a guide to what investigations are required, if any are required at all.

- **Full Blood Count**

  Not indicated in healthy asymptomatic patients less than 60 years where blood loss is expected to be less than 10% of blood volume. (Blood volume 70ml/kg)

  Indications may include:

  - Major surgery
  - Anaemia
  - Rheumatoid arthritis
  - Cardiovascular disease
  - Chronic infection
  - Bleeding tendency
  - Chronic blood loss
  - Chronic renal failure
  - Malignancy
  - Respiratory disease
  - Acute inflammatory conditions
  - Malnutrition

- **Routine Biochemistry (Na, K, Creatinine, Glucose, LFTs)**

  Not indicated in healthy asymptomatic patients less than 60 years.

  Indications may include:

  - Major surgery
  - On cardiovascular drugs
  - Hypertension
  - Endocrine disease including diabetes
  - Malignancy
  - On steroids
  - Renal disease
  - Liver disease
  - Suspected sepsis

- **CXR**

  Not indicated in asymptomatic patients.

  Indications may include:

  - Acute respiratory symptoms or signs
  - Worsening existing cardiac or respiratory diseases
  - Possible metastases
  - Fractured NOF

- **ECG**

  Not indicated in asymptomatic males <50 years / females <60 years.

  Indications may include:

  - Clinical heart disease
  - Peripheral vascular disease
  - Haemodialysis
  - Rheumatic heart disease
  - Electrolyte abnormality
  - Severe chronic respiratory disease
  - Diseases associated with cardiac involvement
  - Hypertension
  - Diabetes mellitus
  - Collagen vascular disease
  - On Digoxin
  - Previous chemotherapy

Note: *There may be variations to these guidelines for certain surgical subspecialities, e.g., Aortic Aneurysm repair or specific anaesthetic request.*

10.2 Preoperative Fasting Instructions (Adults)

For patients undergoing elective surgery or anaesthesia. The latter includes patients who require general anaesthesia for procedures such as GI endoscopy, some X-ray investigations, bone marrow harvests, insertion of portacaths, etc.

- There is good evidence that maintaining oral intake with clear fluids up until 2 hours prior to surgery is both safe and advantageous. The evidence concerning the ideal Nil by Mouth period for solid foods and fluids containing particulate matter, protein or fat is lacking. However, many authorities recommend an overnight fast for morning procedures and a light early breakfast for afternoon procedures. The aim of these guidelines is to encourage the intake of clear fluids (water) up until 2 hours before the procedure but making allowances for possible changes to the timing of the procedure.
  - **Clear fluids** means water only
  - **Light breakfast** means a small quantity of toast or cereal with tea, coffee, or other drink.
- **Perioperative Fasting Instructions:**
  1. **ALL** patients should be instructed to drink water up until 2 hours before the scheduled start time of the list.
  2. **Patients on PM lists** may have a light breakfast 6 hours before the scheduled start time of the list.
  3. Other food or fluids may be consumed ONLY on the instructions of an anaesthetist. Verbal orders should be documented in the clinical notes.

10.3 Administration of Regular Medications

Unless otherwise instructed by an anaesthetist, all routine medications should be continued preoperatively on the day of surgery (given with water to enable comfortable swallowing).

Except for:

- Anticoagulants (warfarin and heparinoids).
- Diuretics/ACE inhibitors.
- Anti-inflammatory drugs, including aspirin.
- Oral diabetic drugs and insulin.

The anaesthetist concerned will advise on the management of patients taking the above medications.

For more information, refer to the CDHB intranet under Clinical information > Clinical pharmacology > Peri-operative drugs.

10.4 Perioperative Management of Diabetes

This will usually be supervised by the Anaesthetist; refer to QMR0330. If not, refer to the guidelines in Perioperative Management of Diabetes on page 103.

10.5 DVT Prophylaxis for Patients Undergoing Surgery

Refer to Surgical DVT Prophylaxis on page 258.

10.6 Guidelines for Perioperative Steroids in Patients already on Steroids

Refer to Guidelines for Perioperative Steroids in Patients already on Steroids on page 95.

10.7 Management of Patients on Warfarin Therapy Undergoing Surgery

Refer to Management of Patients on Warfarin Therapy Undergoing Surgery (see page 255).

11. Cardiology

11.1 Cardiology Department Information

Main Office

- 2nd Floor, Parkside, ☎ 81138, Fax 81415

Inpatient Services

- Five inpatient teams on Wards 12, 26, & CCU

Cardiologists:

- Dr P Bridgman, ☎ 81122, Beep 8574
- Dr I Crozier, ☎ 81138, Beep 8225
- Assoc. Prof. J Elliott, ☎ 81065, Beep 8222
- Dr J Lainchbury, ☎ 88070, Beep 8899
- Dr D McLean, ☎ 81416, Beep 8991
- Dr I Melton, ☎ 81121, Beep 8573
- Prof. M Richards, ☎ 81120, Beep 8227
- Dr D Smyth, ☎ 88073, Beep 8253
- Dr R Troughton, ☎ 80826, Beep 8949

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, page on-call Registrar and then fax the referral to 364 1137 (or 81137).

Outpatient Consultations

- Clerk, ☎ 81110
- Nurse, ☎ 81190

Consultation Guidelines

Chest pain, dyspnoea, documented angina, myocardial infarction, heart failure, hypertension, valvular heart disease, arrhythmias, syncope, congenital heart disease.

Other Services

- Cardiology Day Ward, ☎ 81071, Fax 81127

  Cardiac catheters, coronary angioplasty, stress echo, trans oesophageal echo, electrophysiology studies, radiofrequency ablation for tachyarrhythmia, pacemaker / defibrillator implantation
- Echocardiography Laboratory, Fax 81449
- Tilt Table Testing, Fax 81025
- ECG Department, Fax 80681

  12 lead ECG, exercise tests, Holter monitor tests
- Coronary Care Unit, ☎ 81099, Fax 81128
- Education & Rehabilitation Service, ☎ 81093, Beep 8262
- Cardioendocrine Research Group, ☎ 81116

  (clinical research, basic research)

- Cardiology Research Unit, ☎ 81096
  (clinical research studies)

11.2 Heart Failure

11.2.1 Definition

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

11.2.2 Management requires each of the following

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

11.2.3 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: dilated, hypertrophic, restrictive.
- Pericardial disease: constrictive pericarditis, tamponade.
- Congenital heart disease.
- High output states: cardiac beri-beri (alcoholics), Paget's Disease, thyrotoxicosis.

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:
  - Drugs with negative inotropic action such as beta-blockers, 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.

11.2.4 Investigations

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

- CXR (pulmonary venous congestion/oedema, cardiac size, pulmonary infiltrates).
- ECG (arrhythmia, ischaemia, past infarction)
- Myocardial injury markers: TNT, CK, Myoglobin.
- Na, K (urgently if ECG or rhythm abnormal), creatinine, Mg, Ca, PO_4.
- CBC + Diff.

- Echocardiography to assess LV function, valves, RV pressure estimate (urgent if tamponade or bacterial endocarditis suspected).
- Thyroid function tests.
- Plasma Pro-BNP (where there is doubt over cardiac vs non-cardiac cause of symptoms) - refer to Guidelines for Use of Pro-BNP Measurements in an Acute Medical Setting (see page 71).

11.2.5 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 Sat.O_2 >90%.
  - Glyceryl trinitrate spray under tongue. Repeat doses of nitrate every 5 minutes while the blood pressure remains high.
  - Morphine 2.5-5 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 73), 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 concentration 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 "refractory" 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 1-1.5 mg. Maintenance dose 0.25 mg per day initially, usually given at night. In renal failure and the elderly reduce the dose.

Table 7: Digoxin (see also digoxin poisoning on page 201)

- Therapeutic range (0.6 - 2 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 on page 58. 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. t½ in normal renal function = 36 hr. It is prolonged in impaired renal function.

- 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 refractory 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. Give **dopamine*** (2.5-5 mcg/kg/min) by IV infusion. Can be increased up to 7.5-10 mcg/kg/min if necessary (2 hourly steps of 2.5 mcg/kg/min).

***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.
  - Fluid restriction is indicated in hyponatraemia, ≤ 1000 ml/24hr.
- **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 possibly including Swan Ganz and arterial pressure (if using sodium nitroprusside) in CCU or ICU (see IV glyceryl trinitrate instructions (see page 73)).
  - ACE inhibitor dosing: The substantive evidence base for use of ACE inhibitors in heart failure pertains to captopril, enalapril, ramipril, lisinopril and trandolapril. Start with enalapril (Renitec), quinapril (Accupril) or cilazapril (Inhibace). The dose modification for quinapril and enalapril in renal failure is given in the table below. For the Cockroft and Gault creatinine clearance formula, see Dose Alteration in Renal Impairment on page 58. The starting dose of cilazapril for a patient with normal renal function is 0.5 mg to 1.25 mg/day.

Table 8: Quinapril and Enalapril Dosage in Renal Failure

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: Reduce dose if creatinine rises and reassess diuretic dose provided heart failure is adequately controlled. Consider renal artery stenosis if creatinine has risen quickly.
- 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 when added to ACE inhibitors and loop diurectics. Careful monitoring of potassium and creatinine is required. Similarly, amiloride or triamterene are sometimes necessary to counter

refractory congestion or loop diuretic induced potassium depletion. Potassium sparing diuretics should be used with caution if significant renal impairment and should not be combined.

- **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 enoxaparin 20-40 mg SC q24h. Start on admission. Consider full heparinization then warfarin in those with severe left ventricular impairment, or chronic atrial fibrillation.
  - 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.
  - Beta-blocker drugs do not have any role in the management of acute heart failure. However carefully titrated administration of beta-blockers reduces mortality in stable chronic heart failure associated with systolic dysfunction.

11.2.6 Guidelines for Use of Pro-BNP Measurements in an Acute Medical Setting

- Pro-BNP is useful in distinguishing between cardiac and non-cardiac dyspnoea in patients who are acutely unwell.
- In patients presenting acutely with new onset symptoms of breathlessness, where a diagnosis of heart failure is unclear (e.g., no clinical evidence, normal chest X-ray), a Pro-BNP of >220 pmol/L supports diagnosis of heart failure as the cause of breathlessness (80% positive predictive value, 95% negative predictive value).
- Repeat measurements during an acute admission are not useful.

Note:

- Normal range in healthy subjects is <40 pmol/L.
- Values greater than 220 pmol/L strongly suggest heart failure in a newly symptomatic (breathless) patient.
- In between these levels, heart failure is still possible, but all clinical information must be taken into account. Pro-BNP may be elevated by atrial fibrillation, LVH, valve disease, after myocardial infarction, in the elderly, and in severe renal impairment. Pro-BNP may be decreased by hypothyroidism, treatment with diuretics, vasodilators and ACE-inhibitors.
- Use of serial measurements to adjust therapy for heart failure rather than single tests for diagnosis is experimental.
- Pro-BNP does not add to the diagnosis in a patient with overt heart failure.

11.3 Myocardial Infarction

11.3.1 Definition

The diagnostic criteria for acute myocardial infarction are elevated biochemical markers of myocardial necrosis (Troponin T) associated with at least one of the following:

- Ischaemic symptoms
- New Q waves on ECG
- ST segment or T wave changes on serial ECGs.

11.3.2 Causes

Acute Coronary occlusion due to:

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

Notes:

- Other conditions such as myocarditis may mimic myocardial infarction.
- TNT may be elevated in pulmonary embolism.

11.3.3 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. **Patients with chest pain of low probability for coronary cause and other major pathology excluded, should be admitted to the Chest Pain Unit for exclusion of myocardial infarction.** 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. If ST segment depression is present, or ST-T wave changes are non-specific, but risk factors/symptoms suggest myocardial infarction, give beta-blockers, aspirin, and nitrates. See below.

11.3.4 Investigations

- ECG daily for 3 days and before discharge. Repeat ECG when pain resolved or if pain recurs. Check right sided leads for ST elevation, i.e., look actively for right ventricular infarction.
- Cardiac Enzymes: A Troponin T (see page 76) and Creatine Kinase (CK) should be done on admission and at 8 to 12 hours.
- 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, creatinine, glucose.
- Total fasting cholesterol, HDL cholesterol and triglycerides on admission and repeat at 3 months.
- Patients with suspected myocardial infarction require rhythm monitoring (CCU or telemetry).

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).

11.3.5 Management

- **"Time is muscle"** - expedite treatment and assess suitability for thrombolysis urgently.
- **Transfer to CCU** - any patient with definite acute myocardial infarction is at risk from an acute arrhythmia and should be admitted to CCU. Stable patients with suspected myocardial infarction require a telemetry bed or admission to CCU. For advice on admission contact the CCU 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, beta-blockers, calcium antagonists and morphine as required. Give morphine IV according to severity and repeat up to 4 hourly if necessary. Draw morphine 10 mg (1 ml) up with 9 ml of water for injection (1 mg/ml). Give 2-3 mg (2-3 ml) increments until pain is controlled observing the patient's BP and respiration. Metoclopramide (Maxolon) 10 mg IV may reduce nausea and vomiting.
- **Aspirin** 300 mg sublingual stat then 150 mg orally daily if no contraindications.

11.3.6 Current Indications for reperfusion therapy (thrombolysis, angioplasty)

- 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.
- Acute reperfusion therapy is beneficial if the duration from onset is <12 hours and occasionally up to 24 hours from onset of symptoms particularly if pain is ongoing or marked ST elevation present.
- **Thrombolytic Therapy (see page 74).** Streptokinase and tissue plasminogen activator (alteplase tPA) can both be used in this situation. Fibrin specific agents are preferred over streptokinase. Alteplase may be more effective and has a lower side effect profile. It is more expensive. Alteplase **must** be used **if**:
  - Streptokinase has been used before.
  - There has been a definite recent streptococcal infection.
  - Streptokinase provokes unacceptable side effects.
  - There has been a large anterior infarct.

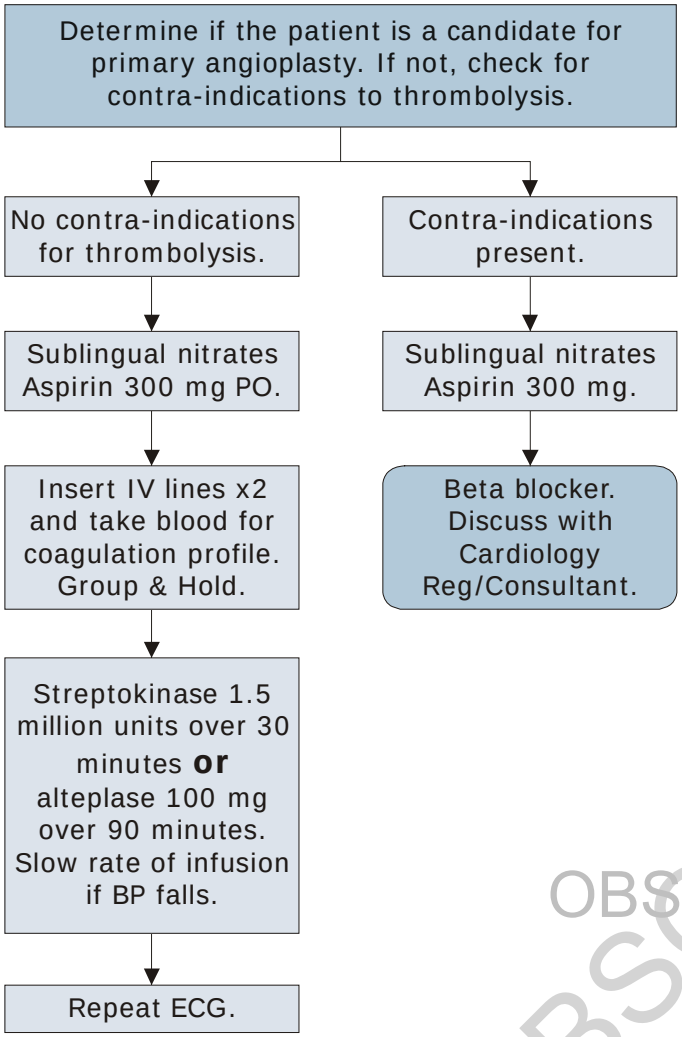
  Consultation with a cardiologist is essential if there is any doubt as to which agent to use.
- **Emergency coronary angiogram and primary angioplasty** is likely to be superior to thrombolysis and should be considered in all ST elevation myocardial infarction during normal working hours and in all patients who:
  - are in cardiogenic shock
  - have large anterior infarcts
  - have absolute contraindications to thrombolysis
  - have recurrent pain and ST elevation soon after thrombolysis
  - have had coronary stent implantation within the last ten days, or suspected acute saphenous vein graft occlusion early post CABG.

  In potential primary angioplasty candidates, 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 may be indicated. This will effectively treat angina and should permit early discharge. If the angiogram demonstrates multi-vessel disease, then bypass surgery may need to be considered.

  If a patient on the waiting list for angioplasty presents with unstable angina, please contact their Cardiologist to expedite inpatient 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 50 mg in 250 ml 0.9S. Start at 3 ml/h in average-sized adult and titrate up by 3 ml/h steps every 5 minutes until pain relieved or BP falls (can go as low as 90 mmHg systolic if otherwise well).

11.3.7 Thrombolytic Therapy

Table 9: Thrombolysis Therapy Flow Diagram



Contraindications to Thrombolysis

Absolute Contraindications:

- Any prior intracranial haemorrhage.
- Known structural cerebral vascular lesion.
- Known malignant intracranial or spinal neoplasm or arteriovenous malformation.
- Ischaemic stroke within 6 months.
- Neurosurgery within 6 months.
- Suspected aortic dissection.
- Active bleeding or bleeding diathesis (excluding menses).
- Significant closed-head or facial trauma within 3 months.
- Uncontrolled hypertension on presentation (SBP >180 mmHg or DBP >110 mmHg).
- Recent internal bleeding within 6 weeks.
- Major surgery or major trauma < 2 weeks.

Relative Contraindications (to be discussed with Cardiologist):

- Transient ischaemic attack < 6 months.
- Traumatic cardiopulmonary resuscitation < 2 weeks.
- Non compressible vascular puncture.

- Pregnancy.
- Active peptic ulcer.
- Current use of anticoagulants with an international ratio >2: the higher the INR, the higher the risk of bleeding.

Streptokinase versus Tissue Plasminogen Activator

Fibrin specific agents are preferred over streptokinase. Alteplase tPA is one of a number of preparations available. These agents are more effective than streptokinase and have fewer side effects, however they are more expensive. If used, streptokinase should not be readministered more than five days after a first dose. Recent streptococcal infection is a relative contraindication to first administration of streptokinase.

Administration of Alteplase

Give 15 mg over 2 minutes, 50 mg over 30 minutes, and then 35 mg over 60 minutes (max dose 100 mg).

Administration of Heparin in association with Thrombolysis

Unfractionated heparin should be given immediately **after** completion of the alteplase infusion. Give 4000 U IV bolus, and follow this with an infusion of 1000 U/hour. Check APTT at 3 hours. Ask your laboratory for their "therapeutic range" for APTT when monitoring unfractionated heparin infusions. Give heparin for 24 hours and then stop.

Note: The use of heparin in conjunction with streptokinase is controversial since the risk of bleeding may be greater. Seek advice from the Cardiologist.

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 in CCU.
  - 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.
  - Record times of:
    - Onset of pain.
    - First ECG/start Streptokinase/finish Streptokinase.
- **Complications**
  - Hypotension associated with Streptokinase infusions:
    - Slow or stop Streptokinase temporarily.
    - Head down tilt.
    - Consider giving IV 0.9S 250 ml boluses x 2-3 (contraindicated in LVF, particularly useful in right ventricular infarcts).
    - Consider Dopamine 2.5-10 mcg/kg/min or adrenaline **0.1 mg** IV (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.**
  - 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.
  - Haemorrhage - apply local pressure over all IV access and arterial puncture sites before starting thrombolysis. If significant bleeding occurs despite appropriate local pressure, 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.

- Cerebral haemorrhage occurs in 1% (higher in elderly). Suspect if headache, decreased level of consciousness or focal neurology. Stop thrombolysis - urgent CT head, if feasible. If confirmed, consider reversing thrombolysis - discuss with Haematology.

Note:

- Failed thrombolysis may be an indication for emergency angiography/ angioplasty.

11.3.8 Other treatments

- If thrombolytic therapy is not given, use low molecular weight heparin e.g., enoxaparin 1 mg/kg SC q12h with a maximum of 100 mg per dose. The usual duration of enoxaparin treatment is 2 - 3 days. Therapy of longer duration is associated with an increased risk of haemorrhagic complications and would normally only be considered in patients with ongoing unstable ischaemic symptoms, following discussion with the SMO, and with intensified monitoring for haemorrhagic complications. If there is moderate 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. The APTT "therapeutic range" will vary according to the test used in the laboratory. Contact the laboratory for their recommended therapeutic range.
- **Hypnotics** if sleep disturbed.
- **Beta-blockers** - continue if patient is already on them and no contraindication exists. Beta-blockers improve medium term prognosis and unless contraindications are present beta-blockers such as atenolol (25-50 mg PO daily) or metoprolol (23.75-47.5 mg), should be commenced on admission, and given IV if continuing pain/arrhythmias. Dosages should be increased as tolerated during admission. 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 beta-blocker therapy but remember that patients may benefit from early intervention. Nitrate infusion (see page 73) or full heparinization may also be helpful.

11.3.9 Troponin Testing

- **Cardiac troponins are highly cardiac specific. Troponin T is the troponin test routinely available at Canterbury Health Laboratories.**
  - No circulating troponin should be present in the serum of a healthy individual.
  - Troponins rise in the circulation 2-6 hours after myocardial injury, therefore troponins may be undetectable in blood taken from patients with acute myocardial infarction at the time of presentation to hospital. It is recommended that an initial negative sample be repeated once after 8 - 12 hours.
  - Troponins remain elevated for up to 14 days after acute myocardial infarction.
  - Other biochemical markers, such as myoglobin or CK must be used during this period if further acute myocardial infarction is suspected.
  - Once a biochemical diagnosis of myocardial infarction has been made there is little clinical utility in repeat testing.
  - If a patient presents with a suspected MI and renal failure, an elevated troponin level needs to be interpreted with caution. Troponin levels may be elevated in renal failure.
  - Not all myocardial injury is caused by coronary occlusion. Non acute myocardial infarction elevations in troponin may be seen in myocarditis, direct cardiac trauma, heart failure, and pulmonary embolism.
  - Small rises in troponin T are commonly seen following major surgery and major medical illness especially in the elderly. Whilst this almost certainly reflects myocardial necrosis, the clinical implications of troponin T elevations in these patients have not been defined. It is probable these patients will have underlying coronary disease and secondary prevention strategies for coronary disease should be considered (aspirin, statin, beta-blocker).

11.3.10 In-Hospital Management following Myocardial Infarction

- Mobilisation 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.
  - Coronary angiography should be considered in all patients, especially non ST elevation MI and post infarction angina.
- Medical therapy should be tailored to each individual patient, but should include aspirin unless it is contraindicated, beta blockers unless contraindicated and ACE inhibitors if there is evidence of left ventricular dysfunction. Statins should be considered in all patients unless contraindicated. 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 contraindications to beta blocker therapy and have good left ventricular function without clinical evidence of congestive failure.
- Action to reduce the effects of any risk factor present - smoking cessation, cholesterol lowering agents, control of hypertension, diet if overweight.
- Ask for Cardiac Rehabilitation Nurse (Beep 8262) to review prior to discharge.

11.3.11 Reference

ST-elevation myocardial infarction: New Zealand management guidelines. Cardiac Society of Australia and New Zealand. NZMJ 118 7 Oct 2005.

11.4 Cardiogenic Shock

11.4.1 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).

- If inferior MI consider RV infarction. Check RV leads on 12-lead ECG.
- Dopamine 2.5-10 mcg/kg/min to keep the BP above 80 mmHg and thus maintain coronary perfusion.
- 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 up to 2000 ml). These patients do not have pulmonary oedema. Swan-Ganz monitoring may be helpful.
- Consider early aortic balloon counter pulsation and coronary angioplasty with an acute myocardial infarction.
- Treat any arrhythmias.
- Consider other possible causes, e.g., sepsis, drugs.

11.5 Acute Coronary Syndrome

The following may be defined as acute coronary syndromes (ACS):

- 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.
- Non ST elevation myocardial infarction.

11.5.1 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 page 71).

11.5.2 Causes

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

11.5.3 Investigation and Management

This is similar to the treatment of acute myocardial infarction except that thrombolysis is not indicated.

- Daily ECG and cardiac enzymes on at least two occasions are mandatory, as is assessment of cardiac risk factors including lipids.
- Elevation of Troponin T indicates non ST elevation MI and high risk of subsequent events.
- ECG changes such as ST depression or T wave inversion or any serial change over the first 24 hours suggest a poorer prognosis.
- Enoxaparin at 1 mg/kg SC q12h (max 100 mg/dose) should be started in patients with ECG changes suggesting ischaemia, a positive Troponin T or a high index of suspicion of ACS. The usual duration of enoxaparin treatment is 2 - 3 days. Therapy of longer duration is associated with an increased risk of haemorrhagic complications and would normally only be considered in patients with ongoing unstable ischaemic symptoms, following discussion with the SMO, and with intensified monitoring for haemorrhagic complications.
- All patients with ACS should be given oxygen and require telemetric ECG monitoring for at least 24 hours; 48 hours if Troponin T positive.
- Start aspirin, nitrate and a beta-blocker (or calcium antagonist if beta-blocker is contraindicated). Clopidogrel may also be considered. Seek Cardiologist advice. If patient has presented with unstable angina on anti-anginal therapy, plan to discharge on increased doses or add another anti-anginal. Remember to investigate for anaemia, hyperthyroidism, heart failure and arrhythmias as precipitants for angina. Plasma lipids and body weight should be assessed and treated as appropriate. Statins should be considered in all patients unless contraindicated.
- Coronary angiography ± intervention should be considered prior to discharge.
- **Tirofiban,** a IIb/IIIa inhibitor should be considered if pain or ST changes recur despite above therapy in patients who are Troponin T positive or with dynamic ST changes. Consult Cardiologist.
- Review and treat risk factors as for myocardial infarction.

11.5.4 Reference

Non ST-elevation acute coronary syndromes: New Zealand management guidelines. Cardiac Society of Australia and New Zealand. NZMJ 118 7 Oct 2005.

11.6 Cardiac Arrhythmias

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

11.6.1 Classification

- Ectopic activity (atrial and ventricular).
- Heart block.

- Bradyarrhythmias.
- Supraventricular tachycardias.
- Ventricular tachycardias.

11.6.2 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.

11.6.3 Clinical features

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

11.6.4 Investigations

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

  Note: *Artefact may mimic some arrhythmias.*
- Check for abnormalities of K, Mg, Ca; acidosis and hypoxia. Metabolic factors may contribute to the initiation/perpetuation of the arrhythmia.
- Thyroid function tests.

11.6.5 Management

Ectopic Activity

- Atrial ectopics - often normal, benign. Look for atrial beat (may 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 reviewed.
  - 2nd degree block:
    - Type I, a 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. Indicates disease in or below the bundle of His. This may progress to complete heart block and a very slow ventricular escape rhythm; consider pacing.
- Bifascicular block (bundle branch block + hemi 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. If stable with regular ventricular escape rhythm and satisfactory blood pressure, may be observed overnight. Be prepared to use isoprenaline (isoprenaline 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) or cardiovascular compromise.

Bradyarrhythmias

- Sinus Bradycardia - check for excessive beta-blockade. Common after myocardial infarction. Treat with atropine 0.6 mg IV if symptomatic or hypotensive. Smaller additional doses of 0.3 mg may be required. Total dose of 2 mg before atropine side effects occur. Isoprenaline may also be used. Place 2 mg in 500 ml D5W (= 4 mcg/ml) and start at 1 ml (4 mcg) per minute but then run as slowly as possible (0.5-10 mcg/min) to keep heart rate >60.
- Sinus Arrest - common in inferior infarction and usually benign, as nodal escape rhythm maintains adequate heart rate. It may require treatment with atropine or isoprenaline but rarely needs pacing. When sinus arrest is not associated with infarction, it is due to the sick sinus syndrome and requires permanent pacing if symptomatic. Temporary pacing rarely required.

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.*

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 approximately 150/min (2:1 block) and may be misdiagnosed as another SVT. 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

  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 6 mg then 12 mg, then 18 mg, in step wise fashion at 2 minute intervals. Flush rapidly with 10 to 20 ml saline, effective for AV nodal re-entrant tachycardia but will not revert atrial flutter. **Contraindicated with severe asthma.**
- If unsuccessful and not on beta-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, hypotension.*
- If on beta-blockers and no structural cardiac disease present use flecainide 2 mg/kg (max 150 mg) over 10 minutes IV (telemetry required) or consider further beta-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 100 Joules, then 200 Joules, then 360 Joules. Do not shock more than twice with 360 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 as for paroxysmal tachycardia. If not compromised, control rate with digoxin (vide infra) or beta-blocker or calcium channel blocker using oral protocol given below. If spontaneous reversion to sinus rhythm does not occur within 24 hours, the patient should be considered for cardioversion.
- **Atrial Fibrillation - New onset atrial fibrillation with rapid ventricular rate:**
  - CBC + Diff, creatinine, Na, K, thyroid function tests
  - Control rate with either digoxin, calcium channel blocker, beta-blocker.
  - 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 1-1.5 mg. Do not digitalize if recently on digoxin.
  - Other options include:
    - Intravenous beta-blocker (not if already on a calcium antagonist).
    - Oral beta-blocker (e.g., Metoprolol; start with 47.5 mg daily, maximum dose 190 mg/day).
    - Oral calcium antagonist (diltiazem 60 mg TDS, verapamil 80 mg TDS).
    - Most patients with recent onset atrial fibrillation, will revert to sinus rhythm within 24 hours. Chemical cardioversion may be attempted in patients with structurally normal hearts (not in patients >70 years). Give flecainide with ECG monitoring: 200 mg PO stat, then 100 mg PO after 6 hours. If the patient fails to cardiovert within 12 hours, then consider electrical cardioversion.
  - Heparin:
    - All patients with atrial fibrillation or flutter should be treated with low molecular weight heparin, e.g., enoxaparin 1 mg/kg SC BD (max 100 mg per dose), unless there are contraindications. Warfarin may not be required if heparin started within 12 to 24 hours of onset of fibrillation and sinus rhythm achieved within 48 hours and there is no left atrial enlargement or major mitral valve abnormality.
  - Electrical cardioversion:
    - Indicated if there is cardiac compromise with hypotension, angina or impaired cerebral function or persistant atrial fibrillation. Consult cardiologist.
- **Chronic atrial fibrillation on digoxin with rapid ventricular rate:**
  - Exclude aggravating causes (ischaemia, heart failure, volume depletion, infection, alcohol).
  - Check digoxin concentration.
  - Add oral beta-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 >65 years, previous embolic event, heart failure, hypertension.

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 II, V1, or V2. 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. Unless an emergency, this should be undertaken in CCU or ICU. In an emergency situation proceed to 200-360 Joule shock. **If in doubt assume that all regular, broad complex tachycardias are VT.** Treatment of choice is cardioversion.

- **QT prolongation and torsades de pointes VT**
  - **Acquired long QT:** Generally, QT prolongation is acquired and is associated with bradycardias, myocardial ischaemia, metabolic disturbances or drugs. Causes of acquired QT prolongation include:

    Drugs:
    - Antihistamines.
    - Antiarrhythmics (Class 1: quinidine, flecainide, disopyramide and Class 3: amiodarone, sotalol).
    - Psychoactive drugs (lithium, tricyclics, haloperidol, phenothiazines).

    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 with syncope.
    - Withhold all QT lengthening drugs.
    - Check and correct any metabolic disturbances.
    - Beta-blockers may help suppress recurrent episodes.
    - Refer to Cardiologist for long term management.
- **Torsades 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 page 84).
- **Amiodarone** - Intravenous amiodarone is useful though slow acting in the treatment of atrial and ventricular arrhythmias. However, potentially important side effects may occur with long term therapy.

  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 over 24 hours as two successive 12 hour infusions as amiodarone is unstable in solution. Can give up to 1200 mg in 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.

11.7 Telemetry Guidelines

Placing patients on telemetry is a medical decision. However, as there are only a limited number of telemetry units available, all requests for telemetry should be discussed with Cardiology. Generally, it is inappropriate to have a "Not for Resuscitation (NFR)" patient on telemetry.

Mandatory Monitoring

- Patients with ventricular arrhythmia that are life-threatening (these patients are often in CCU anyway).
- Patients with intravenous infusions that require cardiac monitoring (*Guidelines for Intravenous Administration of Drugs, Volume 5, Policy and Procedure Manual*). The list includes adenosine, amiodarone, dobutamine, dopamine, flecainide, phenytoin, beta-blockers, and verapamil.
- Patients with temporary pacemakers (usually nursed in CCU, but not compulsory).
- Malfunctioning pacemaker/AICD (actual or suspected).
- Patients post permanent pacemaker/AICD insertion (overnight).
- Patients post cardiac radiofrequency ablation (overnight).
- Patients post myocardial infarction (ECG changes evident or with positive troponin results) - for 48 hours or longer if documented arrhythmia.
- Patients strongly suspected to have had an acute myocardial infarction within the last 48 hours (refer criteria above), or until diagnosis confirmed or excluded.

Discretionary Monitoring

- Patients post PTCA (as ordered by the Interventionist Cardiologist).
- Syncope, if cardiac arrhythmia is suspected (suspected or confirmed heart block). Generally monitor for 24 hours or longer if required.
- Atrial fibrillation/flutter, especially with poor ventricular rate control.
- Drug overdoses at risk of cardiac arrhythmia.
- Unstable Acute Coronary Syndrome (ACS) patients with ongoing pain ± ischaemic ECG changes.

11.8 Cardiac Arrest

Commence basic life support - using the ABCs of CPR (see page 20). Call for the Cardiac Arrest Team and trolley. Precordial thump if witnessed.

REMEMBER:

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

11.8.1 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.
  - If unsuccessful give adrenaline **1 mg** IV. This may be repeated every 3 minutes. Perform CPR for 1 minute.
  - If still in VF defibrillate using 360 Joules x3, then 1 minute CPR, then further 360 Joules, x3 shocks.
  - If still in VF give lignocaine 50-100 mg IV bolus or amiodarone 150 mg IV bolus and repeat defibrillation using 360 Joules.
  - If further resistant VF give 1-2 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.
  - 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.
  - Consider an adrenaline infusion.
  - Definitive management is by pacemaker, transcutaneous (temporary) or transvenous.
- **Electromechanical dissociation (Pulseless Electrical Activity)**, i.e., Organised electrical activity on ECG but failure of effective myocardial contraction:
  - Consider and treat possible causes including hypovolaemia, major electrolyte imbalance, tension pneumothorax, cardiac tamponade, pulmonary embolism, overdose, anaphylaxis and may occur transiently following VF/asystole. Urgent echocardiography may be useful.
  - In absence of other specific therapy give adrenaline **1 mg** IV. This may be repeated every 3-5 minutes.

11.8.2 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.

11.9 Hypertension

11.9.1 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.

11.9.2 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 Mellitus:** Both Type I and II patients are commonly hypertensive.
- **Coarctation of the Aorta**.
- **Respiratory:** Obstructive sleep apnoea.
- **Drugs:** Presence or absence (e.g., clonidine withdrawal). NSAIDs, steroids, sympathomimetics (including non prescription drugs), alcohol, liquorice, cocaine, erythropoietin, cyclosporin.
- **Obesity**.

11.9.3 Investigation

- Blood pressure measurement - lying and standing.
- ECG and CXR.
- Urinalysis (diptest for proteinuria/haematuria, microscopy for cells and casts).
- Plasma, Na, K, Cl, creatinine, Ca. Haemolysis of samples may obscure hypokalaemia.
- Consider the following tests to look for secondary causes:
  - Collect blood for catecholamines before treatment if phaeochromocytoma possible as therapy will alter the blood levels (6 ml of blood into an EDTA tube. Requires rapid separation and storage at -20°C. Consult with Special Tests in Endocrinology any week day morning).
  - Renal ultrasound for renal size and calcification.
  - 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.
  - Morning plasma cortisol level.
  - Renal MRA for renal artery stenosis.

11.9.4 Management of acute hypertensive crisis

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 alpha-antagonist (e.g., doxazosin 1 mg) can be used. Alternatively captopril 6.25 mg PO may be used but should be avoided in the presence of hyponatraemia. Labetalol gives combined alpha- and beta-blockade and may be used if no contraindications to beta-blockade (200 mg PO stat then repeat as required up to 1200 mg daily). Avoid a beta-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 (20 mg in 100 ml or 100 mg in 500 ml, titrated against BP starting in the range of 0.3 -1 mcg/kg/min), only in the CCU or ICU.

Note:

- Do not treat cerebrovascular accidents with IV therapy - oral therapy is best as this 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 angiotensin II receptor antagonist (e.g., losartan).
- Phaeochromocytoma, if suspected, requires alpha-blockade (phenoxybenzamine) or the combination of alpha- plus beta-blockade (e.g., labetalol). Avoid beta-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 beta-blocker alone. Stopping clonidine may induce a phaeo-like state which is exacerbated by giving a beta-blocker. Labetalol is recommended as it provides alpha- and beta-blockade.

11.10 Thoracic Aortic Dissection

11.10.1 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, hypovolaemic shock and an abnormal mediastinum on chest X-ray suggest aortic dissection. Seek urgent advice from both the cardiologist on call and the cardiac surgical registrar on call. The immediate priority is to determine the presence and type of dissection urgently since this will influence management and prognosis.

1. Type A involves ascending aorta with dissection origin between the aortic leaflet and the innominate artery. It carries a mortality of 2% per hour therefore emergency surgery needs to be considered.
2. Type B does not involve the ascending aorta (i.e. origin distal to the innominate artery).

11.10.2 Aetiology

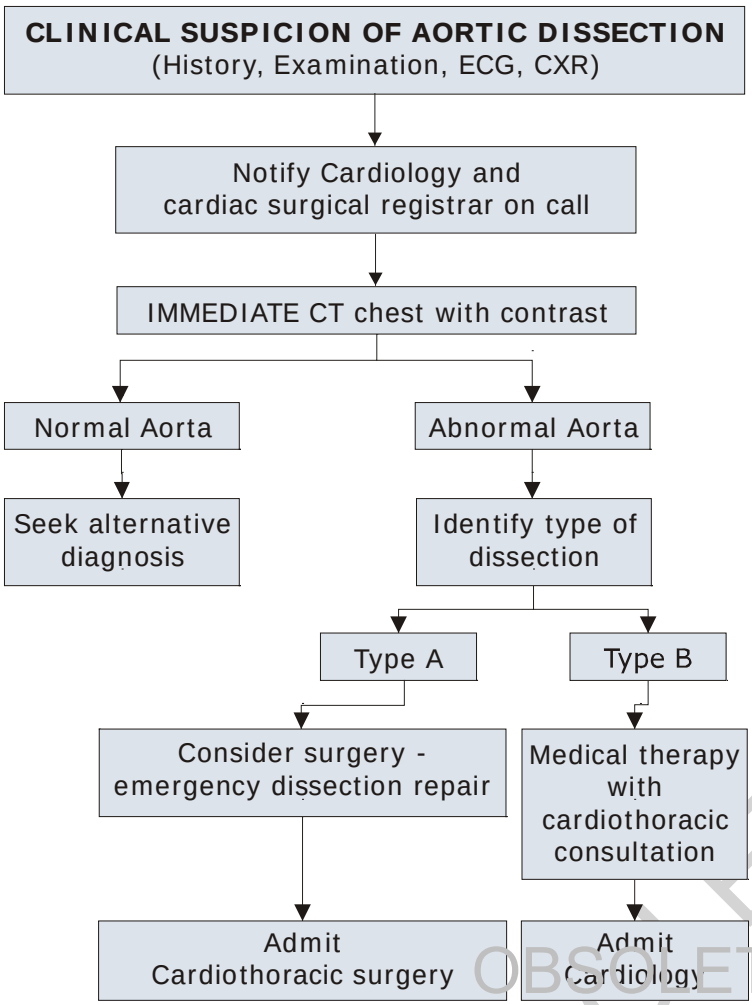
- Cystic medial necrosis.
- Marfan's Syndrome.
- Atherosclerosis.
- Hypertension.
- Trauma, post-cardiac surgery.

11.10.3 Investigations

See Table 10 - Diagnosis of Aortic Dissection.

- 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.
- **Immediate CT scan with contrast**.
- Crossmatch blood.

Table 10: Diagnosis of Aortic Dissection



11.10.4 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 beta-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 73) or a sodium nitroprusside infusion (see page 85).
- Analgesia, e.g., morphine 10-15 mg. Give prochlorperazine (Stemetil) 12.5 mg IM to prevent vomiting.
- Seek advice urgently from cardiologist and cardiothoracic surgeon.

11.11 Bacterial Endocarditis

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

11.11.1 Investigations

- Blood cultures. Three venepunctures inoculating 2 bottles each time, 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, ALT, AST.
- CBC + Diff.
- Echocardiogram.

11.11.2 Treatment

- Initial Therapy - benzylpenicillin 2.4 g q4h IV, plus gentamicin. Flucloxacillin should be added if staphylococcal sepsis suspected (e.g., IV drug user, acute presentation, early embolic lesions).
- 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.

11.12 Infective Endocarditis Prophylaxis

The following information is 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 http://www.nhf.org.nz/files/Research/tech_report76.pdf, or contact:

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

11.12.1 Cardiac conditions and endocarditis prophylaxis

Endocarditis prophylaxis recommended

- **High risk category**
  - All patients with a previous episode of endocarditis [1]
  - Prosthetic cardiac valves, including bioprosthetic and homograft valves [2]
  - 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 [3]

- **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. (4)
  - Major congenital right-sided lesions, e.g., Ebstein's anomaly of the tricuspid valve and significant pulmonary stenosis.

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). (5)
- 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. (6)
- Cardiac pacemakers (intravascular and epicardial) and implanted defibrillators.
- Physiological, functional or innocent murmurs. (7)

Notes: Cardiac conditions and endocarditis prophylaxis

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

(2) The risk of endocarditis remains high after replacement of the native valve with any prosthesis.

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

(4) 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.

(5) 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.

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

(7) 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.

11.12.2 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.

11.12.3 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.

11.12.4 Antibacterial Recommendations for Dental, Oral, Respiratory Tract or Oesophageal Procedures

Moderate Cardiac Risk

Antibacterial recommendations for dental, oral, respiratory tract or oesophageal procedures in cases involving **moderate** cardiac risk are as follows [1]:

- Standard [2]
  - Oral amoxycillin 2 g one hour before procedure and oral amoxycillin 1 g six hours later.
- Pencillin allergy [2, 3]
  - Oral cefuroxime axetil 1 g one hour before procedure and oral cefuroxime axetil 1 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.

High Cardiac Risk

Antibacterial recommendations for dental, oral, respiratory tract or oesophageal procedures in cases involving **high** cardiac risk are as follows [1]:

- Standard [2]
  - Oral/intravenous amoxycillin 2 g plus intravenous/ intramuscular gentamicin (2 mg/kg, not > 120 mg) within 30 minutes of procedure. No subsequent dose recommended.
- Pencillin allergy [2, 3]
  - Intravenous cefuroxime 750 mg plus intravenous or intramuscular gentamicin (2 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 g infused over 60-90 minutes ending within 30 minutes of procedure. No subsequent dose recommended.

Notes

(1) 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.

(2) 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.

(3) 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.

11.12.5 Antibacterial Recommendations for Genitourinary Tract and Gastrointestinal Tract (excluding oesophageal) procedures

Moderate Cardiac Risk

Antibacterial recommendations for genitourinary tract and gastrointestinal tract (excluding oesophageal) procedures in cases involving **moderate** cardiac risk are as follows [1]:

- Standard [1]:
  - Oral amoxycillin 2 g one hour before procedure and oral amoxycillin 1 g six hours later.
- Pencillin allergy [1,2]
  - Intravenous vancomycin 1 g infused over 60-90 minutes ending within 30 minutes of procedure. No subsequent dose recommended.

High Cardiac Risk

Antibacterial recommendations for genitourinary tract and gastrointestinal tract (excluding oesophageal) procedures in cases involving **high** cardiac risk are as follows [1]:

- Standard [1]:
  - Intravenous amoxycillin 2 g plus intravenous or intramuscular gentamicin 2 mg/kg within 30 minutes of procedure and oral or intravenous amoxycillin 1 g six hours later.
- Pencillin allergy [1,2]
  - Intravenous vancomycin 1 g infused over 60-90 minutes ending within 30 minutes of procedure plus intravenous or intramuscular gentamicin (2 mg/kg, not > 120 mg). No subsequent dose recommended.

Notes

(1) Prior or continuing penicillin treatment does not affect these regimens.

(2) There is no oral option for those with penicillin allergy.

12. Endocrinology / Diabetes / Metabolic Disorders

12.1 Endocrinology Department Information

Main Office

- 2nd Floor, Riverside, ☎ 80927, Fax 81159

Inpatient Services Ward 26

- Dr D Cole
- Dr P Hunt
- Dr S Soule

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, osteoporosis. 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 or Dr P. Hunt.

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 (July 2005) for details on request, logistics and interpretation of endocrine tests.

12.2 Diabetes Service Information

Main Office

- 245 Antigua Street, ☎ 80860, Fax 80171

Consultation and On-call Service

24 hours a day, seven days a week by mobile phone or through the Christchurch Hospital Operator. Referrals may be faxed to 80171. Urgent calls during working hours can be directed to the Diabetes Registrar (Beep 8688 or 027 268 9358). Out of hours, contact the on-call consultant.

- Dr D Cole, ☎ 027 229 0126
- Dr H Lunt, ☎ 0274 333 508
- Dr MP Moore, ☎ 0274 377 095
- Dr RS Scott, ☎ 0274 366 380
- Dr S Soule, ☎ 027 228 9537

Consultation Guidelines

Physician input. All new Type 1 diabetes mellitus. Consider physician input if metabolic decompensation is the primary cause of admission or if there are significant diabetes complications.

Other Services

- For a Diabetes Nurse specialist, contact the Diabetes Centre, ☎ 80860, Fax 80171
- Nurse Maude Diabetes District Nurse, ☎ 375 4156, Fax 355 0050
- Outpatient Appointments, Fax 80171
- Lipid and Diabetes Research Group (Tracey Hubert), ☎ 80449, Fax 80457

12.3 Adrenal Insufficiency

12.3.1 Causes

- Primary adrenal failure:
  - Autoimmune.
  - Tuberculosis.
  - Haemorrhage/ infarction (e.g., severe sepsis, antiphospholipid syndrome).
  - Metastases.
  - HIV infection.
- Secondary:
  - ACTH deficiency [pituitary failure]
  - Adrenal suppression, or glucocorticoids stopped or not increased at time of acute stress.

OBSOLETE

12.3.2 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.

12.3.3 Investigations

- Na, K, creatinine, urea, glucose - may all be normal (hyponatraemia common). In later phases of primary deficiency, low Na and high K, high urea, lowish glucose.
- CBC + 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 Synacthen - 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 falsely normal. Successive 0800 hr plasma cortisol levels may be indicated, and/or other tests (metyrapone etc.). Consult Endocrine Team.

- In the setting of severe acute illness, a random cortisol >950 nmol/L suggests adrenal insufficiency unlikely, <450nmol/L makes hypoadrenalism a likely possibility. For inbetween values (450-950) a cortisol increment of <200 nmol/L after Synacthen suggests adrenal insufficiency and the need for supplemental steroids.

12.3.4 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 [eg. IV cefuroxime and gentamicin].
- Hydrocortisone 50-100 mg IV then 50 mg 8 hourly for 24 hours, then reduce daily dose of hydrocortisone (eg. 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 fludrocortisone will usually be required once hydrocortisone dose is less than 50 mg/day.
- Diagnostic work-up and management should be completed in consultation with the Endocrine Department.
- **Steroid induced suppression of the hypothalamic-pituitary-adrenal (HPA) axis**. Patients receiving long term glucocorticoids [eg. 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.
- All patients with adrenal insufficiency should have a steroid card and medic alert.

12.3.5 Guidelines for perioperative steroids in patients already on steroids

Note: Prednisone 5 mg ≈ hydrocortisone 20 mg ≈ dexamethasone 0.75 mg ≈ methylprednisolone 4 mg.

- Patients with intrinsic lack of ACTH or with primary adrenal insufficiency are especially sensitive to acute stress illness.
- Patients taking supraphysiological doses of steroids (>5-7 mg prednisone or equivalent per day) for <3 weeks are unlikely to have significant HPA axis suppression, but if in doubt treat as steroid deficient. Patients on high doses of inhaled glucocorticoids (>1500 mcg beclomethasone or >750 mcg fluticasone daily) may have HPA axis suppression.

All patients should take their usual steroid doses on day of surgery (or IV equivalent) and supplementation (see Table 11). Monitor fluid status, electrolytes and glucose daily.

Table 11: Perioperative guidelines for patient taking steroids

Patients currently taking steroids:

- **≤5mg prednisone daily** (and not known to be steroid deficient):
  - Assume normal HPA response
    - Additional steroid cover not usually required.
- **>5mg prednisone daily and/or known steroid deficiency**:
  - **Minor surgery** e.g., hernia repair, tooth extraction, laparoscopic procedures
    - Double usual dose oral steroids on day of procedure or 25 mg hydrocortisone IV at induction
  - **Moderate surgery** e.g., hemicolectomy, open cholecystectomy, nephrectomy
    - 50 mg hydrocortisone IV at induction then 50mg q8h for 24 hours and reduce to maintenance over 1-2 days
  - **Major surgery** e.g., AAA repair, Whipples, major cardiothoracic surgery, liver resection
    - 50-100mg hydrocortisone IV at induction then 50-100mg q8h for 48-72 hours and reduce to maintenance over 2-4 days
  - **Critically ill** e.g., shock, sepsis induced hypotension
    - 50-100mg hydrocortisone IV q8h for 24-48 hours and taper to maintenance as condition improves, usually 2-4 days

Patients stopped taking steroids (>5mg prednisone/day):

- **<3 months:**
  - Check Synacthen test* pre-op, if normal do not give steroids; if urgent procedure, treat as if on steroids.
- **>3 months:**
  - No perioperative steroids necessary.

***to arrange test, phone Endocrine Special Tests on 80934**

12.4 Assessment of Thyroid Function

Abnormalities in thyroid function tests, not requiring treatment are often observed in patients with systemic non-thyroidal illness. These abnormalities are often referred to as the "sick euthyroid syndrome". Therefore thyroid function should not be assessed in seriously ill patients unless there is a strong suspicion of thyroid dysfunction.

In Christchurch Hospital, a Free T4 Index (calculated from Total serum T4) measurement is the routine thyroid function test. When Free T4 levels are low/normal or low, the laboratory will automatically measure a sensitive TSH on the same blood to confirm possible thyroid failure. If Free T4 is elevated or high/normal, the laboratory will measure T3 and TSH on the same sample. In acute non-thyroidal illness, conversion of T4 to T3 is reduced, and T3 measurements are usually unhelpful - particularly in ICU where the lowest T3 levels are seen.

- **High TSH with Free T4 normal -** These findings are consistent with sub-clinical hypothyroidism and may be associated with a small goitre and positive thyroid antibodies. Patients with TSH > 10 mU/L usually have primary hypothyroidism requiring treatment. In the recovery phase after acute non-thyroidal illness, TSH may transiently show a slight elevation, usually < 6 mU/L. TSH and Free T4 should be repeated after 6-8 weeks.

- **Low (or low/normal) Free T4 and normal TSH -** These results are often seen in serious non-thyroidal illness but also raise the question of secondary hypothyroidism. Repeating the thyroid function tests after 6-8 weeks is recommended unless there is a high suspicion of pituitary/ hypothalamic disease. In the latter case, screening for evidence of other pituitary dysfunction may be necessary, i.e., plasma prolactin, plasma cortisol at 0800 hours, LH/FSH and testosterone or oestradiol. Consult Thyroid physicians or Endocrinology for advice.
- **High Free T4 and suppressed TSH -** Thyrotoxicosis is likely, particularly if accompanied by a goitre and signs of hyperthyroidism. T3 levels give a guide to severity, but acute illness may lower a previously elevated T3. A radio isotope thyroid scan is helpful to distinguish toxic nodular disease from Graves' disease. Patients with suppressed TSH (< 0.2 mU/L) and normal Free T4/T3 have sub-clinical thyrotoxicosis. Consult Thyroid physicians.
- **Amiodarone -** This is a frequent cause of thyroid function abnormalities. Conversion of T4 to T3 is reduced and with long-term administration Free T4 may be modestly elevated with TSH and T3 normal. The high iodine content of amiodarone may also precipitate either thyrotoxicosis (suppressed TSH) or hypothyroidism (elevated TSH).

12.5 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 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.

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 30 60% in the morning and 40% at the evening meal, at a total dose of 0.3 u/kg per 24 hours. For example a 100 kg patient might be prescribed Penmix 30, 18 units before breakfast and 12 units before the evening meal time. Supplemental fast acting insulin such as lispro (Humalog) or aspart (Novorapid) 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.)
- Pen injectors (e.g., Novopen, Humapen) can be obtained from the Christchurch Hospital Pharmacy.

Description of Insulins currently available in New Zealand

Type of Insulin	Brand Names	Description of Action	Duration of Activity (1)		Common outpatient use (NB: all insulins listed here can be used with a pen injector)
			Peak (hours after injection)	Time to disappearance (hours after injection)	
Aspart	NovoRapid	Fast acting	1.5	6	t.d.s. with food - requires the addition of a once or twice a day intermediate or long-acting insulin.
Lispro	Humalog	Fast acting	1.5	6	Usage as for Aspart.
Neutral (soluble)	Actrapid Humulin R	Short Acting	2 - 4	10	t.d.s. half an hour before food in addition to a bedtime intermediate or long-acting insulin.
Premixed (2) insulin e.g., 30% neutral 70% isophane	Penmix30 (2) Humulin 70/30 (2)	Biphasic	As for component insulins	24	Half an hour before breakfast and the evening meal.
Isophane (NPH)	Protaphane Humulin N	Intermediate acting	3 - 8	24	Background (basal) insulin - often given at bedtime and used in conjunction with fast/short acting insulins or with oral anti-diabetic agents.
Glargine Detemir	Lantus Levemir	Long acting	4 - 24	>24	Reduced risk of nocturnal hypoglycaemia. Not subsidised - patient will need to self-fund.

Notes

(1) Insulin activity varies between injections (ie. within patient variability) and from patient to patient (i.e., between patient variability). This table of duration of action is an approximate guideline only.

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

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.
- Patients on fast acting insulins (lispro or aspart) may need to test 2 hours after mealtime injections.

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 dilutional hyponatraemia.
- For each 10 mmol rise in serum glucose, an approximately 3 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 >0.15 mmol/L), overt cardiac failure, acute myocardial infarction, severe hepatic impairment, hypoxia, severe dehydration and sepsis
- 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 may be reduced in patients with newly diagnosed as well as known diabetes, if an intravenous insulin 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 **48 hours before** patients on insulin leave hospital. Questions you should consider 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 of the correct length or 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 Registrar or Diabetes Nurse Specialist.

12.6 Diabetic Ketoacidosis (DKA)

12.6.1 General Principles and Precautions

- DKA is defined by hyperglycaemia with positive plasma ketones and an arterial pH ≤7.30 and/or a plasma bicarbonate ≤15mmol/l. Plasma or capillary beta hydroxybutyrate is typically > 1.2 mmol/L.
- 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 and adolescent DKA. A deterioration in the level of consciousness, despite improving biochemistry, suggests this complication. Monitor level of consciousness and undertake fluid replacement slowly.
- 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 page 102).

Discuss the management of these patients with the Diabetes Physician on call.

12.6.2 Common Causes of DKA

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

12.6.3 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 or capillary beta hydroxybutyrate.
- Arterial blood gases (venous pH and bicarbonate may be sufficient if patient has mild DKA only).
- CBC + Diff.
- Cultures of blood and urine and any other material as indicated.
- CXR.
- ECG.

12.6.4 Treatment

If the patient is severely ill (arterial pH <7.1 or obtunded or has DKA complicated by other medical conditions) consider admission 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 capillary ketones (beta hydroxybutyrate).
- A meter to measure capillary beta hydroxybutyrate is available for patients in the Special Nursing Unit. This provides a better measure of improvement than monitoring urine ketones.
- 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.
- Venous blood gases can be used to monitor progress once the patient is improving.
- 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. Use 0.45S in adolescents only after discussion with the Consultant (cerebral oedema risk).
- When the blood glucose approaches 15 mmol/L change to D5W.
- See below for K replacement. Many patients can receive K replacement using ready mixed 30 mmol KCl in 1L 0.9S. Some patients will however require K added to 0.9S. For safety reasons, K replacement must be given via an infusion pump.

Insulin

- Give a stat bolus dose of 10 units IV Actrapid.
- Add 50u Actrapid to 50 mL 0.9S in a 50 mL syringe.

- Administer IV using a pump such as an IVAC.
- The nursing staff will purge 10 mL through the plastic tubing, to saturate the insulin binding sites on the tubing.
- Start with an insulin infusion of around 5 mL (5 units) per hour, 'piggy backed' together with IV fluids such as 0.9S if glucose > 15 mmol/L or D5W if glucose is ≤ 15 mmol/L.
- Increase or decrease the insulin infusion rate according to the rate of fall of glucose.
- Aim to normalise glucose over 24 hours, no faster.
- When the glucose has fallen to around 20 mmol/L, slow down the rate of infusion of insulin (refer to the sliding scale on page 101).
- When glucose has fallen to around 15 mmol/L, change IV fluid replacement to D5W.
- If satisfactory progress is not occurring (particularly if the acidosis is not resolving) reassess volume status to ensure adequate repletion, check for hyperchloraemia and check the insulin mixture is correctly prepared and consider increasing the insulin infusion rate.

Suggested Starting Sliding Scale for IV Insulin Administration

Blood glucose (mmol/litre)	Insulin infusion rate (units/h)
>20	5
15 - 19.9	4
10 - 14.9	3
7 - 9.9	2
4 - 6.9	1
3 - 3.9	0.5 (1)

(1) Normally in Type I DKA continuous infusion of insulin is desirable but if blood sugar is less than 3, temporarily interrupt the infusion. Check glucose every 20 minutes and restart the insulin infusion as soon as possible.

Note: *Patients with increased insulin sensitivity (e.g., thin, elderly patients) or insulin resistance (e.g., patients with marked centralised adiposity) will probably require modification of this sliding scale.*

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.
- The key to adequate potassium replacement is regular monitoring.
- Most patients can have pre-mixed 30 mmol/L KCl in 0.9S. Occasionally, patients with severe total body potassium depletion will require greater concentrations.
- Discontinue potassium replacement once the patient is eating or K above 5 mmol/L.

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 aspart or 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 the evening meal, or aspart or lispro with main meals and Humulin N or Protaphane at bedtime.

- If the patient is converting to subcut aspart or lispro insulin, a small dose of long acting insulin such as Humulin N or Protaphane may also be required at the time the IV infusion is discontinued.

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.
- Abdominal pain and hyperamylasemia often occur in DKA. The hyperamalysemia may be the result of extra-pancreatic secretion and should be interpreted cautiously.
- Consider IV bicarbonate only if pH is very low (<7) and then give enough to raise the pH to 7.1 e.g., try giving 1 mmol $NaHCO_3$ per kg over 30-60 minutes with 10-20mmol of potassium and review pH in one hour.
- Always refer the patient to the Diabetes Service to assess overall diabetes management.

12.7 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%.

12.7.1 Investigations

As for DKA (see page 100) but include plasma osmolality.

12.7.2 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 less severe 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 taken 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 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 or Registrar.
- **Prevention of venous thrombosis**
  - These severely dehydrated comatose patients are at high risk of DVT. Consider prophylactic low molecular weight heparin.

12.8 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 1 L D5W, at 100 ml/hr, plus an insulin infusion at 1 unit/hr. Measure blood glucose 2-4 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 mmol/L. Decrease infusion rate to 0.5 units/hr if glucose <6.5 mmol/L.
- If there is any delay in the surgery or the patient does not resume normal intake promptly post operatively, Na, K, and creatinine will need to be closely monitored, at least 12 hourly. Seek advice under these circumstances from the Diabetes Service.

The above regimen is suitable for most patients but those on high daily doses of insulin may require more IV insulin than above.

12.9 Management of the Newly Diagnosed Patient with Diabetes

- Refer all patients to the Ward Dietitian.
- **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. Recent significant weight loss, age <40, and severe hyperglycaemia (>14-16 mmol/L) all suggest that insulin treatment is likely to be required 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 (creatinine ≥ 0.15 mmol/L), cardiac or liver disease and metformin should also be used with caution in the elderly.
- **Diabetes Nurse Specialists**: are available to help with education and practical management e.g., home blood glucose monitoring, insulin injection technique, use of pen injector devices, sick day and

"hypo" management. Referrals should be faxed to the Diabetes Centre. If the patient needs to be seen within 24 hours, a phone call to back up the faxed referral is helpful.

12.10 Hypoglycaemia

12.10.1 In Diabetic Patients

This is commonly seen in patients on insulin or sulphonylureas.

12.10.2 In Non-Diabetic Patients

If hypoglycaemia is suspected (bedside glucose low, <3 mmol/L) but the patient is not known to be on treatment for diabetes, i.e., possible insulinoma or inappropriate ingestion of a sulphonylurea: take **venous blood sample for glucose, insulin and C-peptide** (9 mL blood into EDTA tubes and contact Biochemistry for immediate 4°C centrifugation and freezing of plasma) **before** giving IV dextrose. If venous glucose confirms hypoglycaemia (< 3 mmol/L), consult the Endocrine team.

12.10.3 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.
- Take 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.

12.11 Hypernatraemia

Hypernatraemia (serum Na >145 mmol/L) is due to a deficiency of water relative to solute (Na) in the ECF and always represents a hyperosmolar state. Thirst and release of ADH are important defense mechanisms preventing hyperosmolar states. Therefore hypernatraemia is rarely found in alert patients with normal thirst and access to water. At risk groups include infants, the elderly, intubated patients, and those with altered mental status.

Symptoms - depend on time course (acute vs chronic) and level of Na

- Lethargy, weakness, irritability.
- Confusion, seizures, coma.

12.11.1 Causes

- **Pure water depletion**:
  - No water!
  - Hypodipsia (either 2° [e.g., severe illness, dementia, coma] or rarely 1° [hypothalamic injury]).
  - Diabetes insipidus (cranial or nephrogenic). (1)
- **Depletion of hypotonic fluid** (1) i.e. loss of relatively more water than Na.
  - Renal loss:
    - Loop diuretic, osmotic diuresis (glucose, urea, mannitol).
    - Postobstructive diuresis, polyuric phase ATN.
    - Intrinsic renal disease.
  - GI tract loss:
    - Vomiting, NG drainage, diarrhoea, laxatives.
  - Skin loss:
    - Burns, excessive sweating.
- **Solute excess** (uncommon)
  - Sodium: ingestion of NaCl, sea water, NaHCO3 infusion
  - Hyperalimentation: IV or parenteral nutrition

(1) Only sustained hypernatraemia if impaired thirst or access to water.

12.11.2 Approach to hypernatraemia

Evaluation includes history to determine likely cause, clinical assessment of volume status (usually depleted except in rare cases of sodium overload) and neurological function. Investigations should include:

- Plasma electrolytes, urea, creatinine, Ca and osmolality **[**(2 x Na) + urea + glucose**]** (all mmol/L).
- Urine osmolality, Na and glucose.
- Urine osmolality >700-800 mOsm/kg confirms normal ADH secretion and action and suggests non-renal fluid losses and/or a blunted thirst response.
- Low urine osmolality (<700 mOsm/kg) indicates a urine concentrating defect. Causes include solute diuresis (e.g., glucose), diabetes insipidus either cranial or nephrogenic (lithium, hypercalcaemia, severe hypokalaemia, congenital).
- Formal evaluation for diabetes insipidus requires a water deprivation test which should only be performed after initial fluid resuscitation and with close supervision. Please consult Endocrinology.

12.11.3 Management of hypernatraemia

Cerebral adaptation to hypernatraemia occurs within hours, involves accumulation of intracellular electrolytes and organic osmolytes, and minimizes the potential reduction in cerebral volume - therefore, as with hyponatraemia, acute hypernatraemia is more likely to be symptomatic and should be more aggressively managed than chronic hypernatraemia (>24hr). Treatment involves administering hypotonic fluid and addressing the cause. Principles include:

- In acutely hypernatraemic and symptomatic patients (e.g., accidental sodium loading) rapid correction is appropriate, reducing Na by 1 mmol/l/hr to approximately 145 mmol/l.
- In patients with hypernatraemia of longer or unknown duration, a maximal correction rate of 0.5 mmol/l/hr is appropriate - targeted fall 10mmol/l/day.
- The preferred route of administering fluids is **oral**, **nasogastric**, or **subcut**.
- Generally use pure water (orally) or 5% Dextrose - the lower the osmolality of the fluid, the lower the volume required for correction.
- Avoid normal saline unless frank circulatory collapse.
- Remember to allow for ongoing fluid losses both incidental and obligatory.

- As a guide to the rate of infusion use the following:

  Change in serum [Na] after infusion of 1 litre of fluid = [Na] in infusate - serum [Na] ÷ TBW + 1

  Total body water (TBW) is approx. 60% body wt males, 50% body wt females. The infusate [Na] is 0 mmol/l for 5% Dextrose, 30 mmol/l for D4S, 77 mmol/l for 0.45% Saline and 154 mmol/l for 0.9% Saline.

  e.g., For a 70 kg patient with Na 168 mmol/l due to pure water loss from insensible losses. Infusion of 1 litre of 5% Dextrose will reduce serum Na by [0-168] ÷ [(0.6 x 70) + 1] = -3.9 mmol/l. To reduce Na by 10 mmol/24h requires 2.5 litres [10 ÷ 3.9], plus insensible losses of 1.5 litres, giving total of 4 litres per 24h or 166 mls/h.
- Frequent clinical and biochemical reviews are essential in patients with severe and symptomatic hypernatraemia; repeat Na after 6-8hs initially.
- For acute cranial diabetes insipidus (CDI) Desmopressin (synthetic AVP) should be given parenterally in a dose of 1-4 mcg IM or IV with repeat doses as clinically required based on urine output and osmolality, usually 12-18 hourly. Consult Endocrinologists.
- Established CDI requires the use of Desmopressin in a dose adjusted according to clinical need, usually 10-20 mcg daily by intranasal spray.

12.12 Hyponatraemia

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

- Weakness, lassitude, headache, nausea.
- Confusion, convulsions, coma.
- Some patients may have no symptoms, especially if chronic ↓Na.

12.12.2 Causes

- These are many and varied (see page 106).
- Remember to consider factitious causes:
  - Laboratory error - check anion gap (see page 111) and calculate osmolarity **[**(2 x Na) + urea + glucose**]** (all mmol/L).
  - Drip arm specimen.
  - Pseudohyponatraemia (hyperlipidaemia or hyperproteinaemia). You may need to get a direct reading of Na (contact biochemistry).

12.12.3 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, recent use thiazide diuretic].

12.12.4 Assessment of Plasma Volume Status in Hyponatraemia

VOLUME DEFICIENT

- **History:**

  Renal or GI losses, burns, third space losses, diuretic use, aldosterone deficiency, cerebral salt wasting, history of heart failure, cirrhosis.
- **Examination:**

  Volume contraction with low JVP and postural hypotension, or signs of congestive heart failure or cirrhosis

- **Laboratory:**

  Hypo-osmolar plasma, hypo or hyperosmolar urine, urine Na <20 mmol/L (**not** if recent diuretics, tubular disorders, or cortisol deficient), normal or raised uric acid, urea, creatinine

NORMAL OR VOLUME EXPANDED

- **History:**

  Excess water ingestion, potomania (excess beer drinking), recent surgery / trauma / pain, thiazide diuretics, renal failure, SIADH (pulmonary; neurological; thyroid/adrenal insufficiency; drugs - DDAVP, oxytocin, SSRIs, tricyclics, vincristine, NSAIDs, carbamazepine).

- **Examination:**

  Normovolaemic clinically. No postural BP fall. JVP not low. Sometimes subcutaneous tissues feel "spongy".

- **Laboratory:**

  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.
- An estimate of plasma osmolarity may be derived from **[**(2 x Na) + urea + glucose**]** (all mmol/L).

12.12.5 Management

The brain gradually adapts to hypo-osmolality thus the presence or absence of symptoms gives some guide to chronicity and appropriate treatment. Thus rapid correction of chronic severe hyponatraemia in the "adapted" asymptomatic patient may result in osmotic demyelination (pontine myelinolysis). Conversely, the symptomatic patient with hyponatraemia warrants urgent correction of plasma Na (maximum increase 8-12 mmol/day) to 125-130 mmol/L.

- Withdraw inappropriate drugs.
- Exclude deficiencies of thyroid or adrenal function (FT4, TSH, synacthen test).
- Whatever the cause, treatment and 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 125-130 mmol/L. The speed of correction depends on presence of symptoms and careful monitoring of clinical state and sodium level is required. In the symptomatic patient, the initial rate of correction can be 1-2mmol/L per hour for several hours.
- Severe hyponatraemia may be life threatening (e.g., coma or convulsions) and may require hypertonic saline - consult before use.
- The appropriate infusion rate can be calculated according to:

  Total body water (60% body wt males; 50% body wt females)
  x desired correction rate = rate of sodium replacement (mmol/hr).

  e.g., desired correction rate of 1mmol/L/hr in 70kg man would require 42 mmol/hr of sodium (0.6 x 70 x 1). If using normal saline (154mmol/L) - infusion rate 273mL/hr. If using 3% saline (513mmol/L) - infusion rate 82mL/hr [i.e, 42 mmol/hr divided by 0.513 mmol/ml = 82 ml/hr].

- Investigate and treat underlying cause.

12.13 Hypercalcaemia

If marked (>3.5 mmol/L), this requires urgent attention - usually symptomatic if calcium is >3 mmol/L.

12.13.1 Causes

- Malignant disease - myeloma, carcinoma (eg. 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.

12.13.2 Symptoms

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

12.13.3 Investigations

Note: *The ionised calcium may be misleading and we do not advocate routine assay of ionised calcium. 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, calb, 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. Draw samples for PTH and calcium before giving hypocalcaemic drugs.
- 25OH Vitamin D.
- 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), consider radionuclide bone scan.
- Fasting morning urine calcium to creatinine ratio.
- 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.*

Calcium Correction Formula

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

12.13.4 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 1-2 litres 0.9S over 2 hours then 1 litre 0.9S 6-8 hourly and reassess at regular intervals. Potassium supplements 10-20 mmol KCl per 500 ml may be required.
- Bisphosphonates
  - Pamidronate 30 mg in 0.5 litre 0.9S IV over 2 hours or zoledronic acid 4 mg in 50 ml 0.9S IV over 15 minutes. 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. Frusemide may be useful by increasing urine calcium excretion, but give only when volume replete.
- 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.

12.14 Hypocalcaemia

Check albumin and if necessary adjust the calcium level (see page 108). If <2 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).

12.14.1 Causes

- Hypoparathyroidism or resistance to parathyroid hormone.
- Renal failure.
- Vitamin D deficiency.
- Low magnesium states.
- Pancreatitis, rhabdomyolysis.

12.14.2 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.

12.14.3 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 (magnesium chelate 50 mg tablets (2 mmol magnesium per tablet)).
- 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.

12.15 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.

12.15.1 Causes

- Familial syndromes/primary hyperlipidaemias.
- Alcohol.
- Diabetes Mellitus.
- Drugs (thiazides, steroids, oestrogens).
- Hypothyroidism.

12.15.2 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 acid and fibrates. Results can be disappointing. Diet is pivotal.
- Treat diabetes to normalise glucose.

OBSOLETE

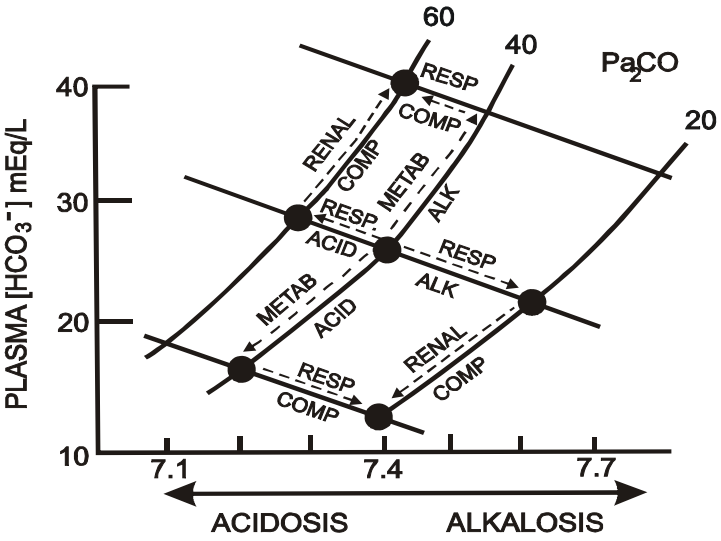
12.15.3 Follow Up

Specific dietary advice (dietitian) and specialist medical review is recommended.

12.16 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. The following table may be helpful:



Note: 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.

The causes of **respiratory acid/base disturbance** are usually obvious and reflect the underlying abnormal pulmonary function. This should be investigated and managed if necessary with the assistance of a Respiratory Physician.

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.

12.16.1 Causes of Metabolic Acidosis

Table 12: Calculate Anion Gap

Anion Gap = [K] + [Na] - [Cl] - [HCO_3]
Normal range = 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 (including metformin).
  - Poisoning: salicylate, ethylene glycol, methanol.
- Renal failure.

Normal anion gap (chloride 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.

12.16.2 Investigations

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

12.16.3 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.

12.16.4 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.

12.16.5 Investigations

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

12.16.6 Treatment

- Treat the underlying cause.

13. Gastroenterology

13.1 Gastroenterology Department Information

Main Office Investigative Unit

- 2nd Floor, Riverside, General enquiries ☎ 80920, Fax 80419
- Endoscopy unit ☎ 80965

Inpatient Services Ward 29, ☎ 89290

- Dr M Barclay
- Dr M Burt
- Dr B Chapman
- Dr J Collett
- Dr S Ding
- Dr A Ross
- Dr C Stedman

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, gastric, duodenal and colorectal stent insertion, motility investigations (oesophageal, ano-rectal, biliary), oesophageal pH studies, GI tract tumour ablation, GI tract food bolus and foreign body management, capsule endoscopy, enteral feeding tubes.

13.2 Haematemesis

13.2.1 Causes

- Mallory Weiss tear.
- Acute stress erosions (shock, sepsis, NSAID).
- Peptic ulceration (ask about NSAID + aspirin use).
- Varices including gastric (note: high mortality).
- Oesophagitis.
- Upper GI tract cancer.
- Abnormal haemostasis.
- Swallowed blood.

13.2.2 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, or banding of varices 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 Shock on page 23):
  - History often unreliable.

- Useful signs include:
  - Resting tachycardia.
  - Hypotension.
  - Postural BP 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 + Diff.
  - Coagulation profile.
  - Na, K, creatinine, LFTs.

Urgent surgical consultation if:

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

Gastroenterology consultation

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

13.2.3 Therapy

- **Varices:**
  - IV infusion of octreotide using a 50 mcg bolus followed by a continuous infusion for up to 72 hours. Consult Gastroenterologist for infusion details.
  - Urgent variceal ligation or occasionally sclerotherapy.
  - 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 (20 ml Mylanta 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. High dose omeprazole infusion beneficial in specific situations. This will be directed by the Gastroenterologist.
  - Eradication therapy for **Helicobacter pylori** when this has been identified - omeprazole 20 mg BD + Amoxycillin 1 g BD + Clarithromycin 500 mg BD for 7 days. If penicillin allergy, substitute metronidazole 400 mg BD for amoxycillin

Other regimens are available for treatment failures. Consult Gastroenterology.

13.3 Vomiting

13.3.1 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, theophylline, cytotoxics).
- Neurological:
  - Vestibular/middle ear.
  - Increased intracranial pressure.
  - Cerebrovascular accident (especially brain stem).
- Other:
  - Pregnancy.
  - Excess smoking, alcohol and other addictive drugs.

13.3.2 Complications

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

13.3.3 Treatment

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

- Dopamine antagonists:
  - Metoclopramide 10 mg q8h PO, IM, IV, but higher doses may be required.
  - Domperidone 10 mg QID PO.
- Phenothiazines:
  - Prochlorperazine 5 - 10 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, refer to Management of Nausea and Vomiting (see page 178).*

13.4 Acute Diarrhoea

(e.g., less than 2 weeks)

13.4.1 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.

13.4.2 Examination

- Look for signs of dehydration, sepsis, abdominal tenderness and rigidity.
- Digital rectal examination. If symptoms are prolonged, sigmoidoscopy and biopsy may be required.

13.4.3 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 and the specific requests should reflect the clinical setting:
  - **Microscopy**: Parasites (microsporidia, cryptosporidia in immunosuppressed).
  - **Bacteria**: Salmonella, Shigella, Yersinia, Aeromonas, Campylobacter and Plesiomonas are routinely cultured at Christchurch Hospital. (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.

13.4.4 Management

- 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 Na and 5-15 mmol 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 262).
  - Pseudomembranous colitis; always suspect when antibiotics have been taken within last few weeks. Sigmoidoscopy may sometimes be diagnostic but is usually unnecessary. If suspected, check for Clostridium difficile toxin and treat. Treatment of choice metronidazole 400 mg q8h PO 7-10 days. Is effective for relapse or recurrence. 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 HIV and AIDS on page 133).
  - Amoebic dysentery - metronidazole 800 mg PO, TDS for 10 days.
- **Acute inflammatory bowel disease is suspected**.
  - Gastroenterology consultation.
  - Toxic megacolon (diameter >5.5 cm) should be considered in any person with inflammatory bowel disease, systemic toxicity and increasing diarrhoea (can paradoxically be reduced). 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.
  - Sulphasalazine 1 g QID PO or mesalazine 1 g QID PO, 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.

13.5 Constipation

13.5.1 General Measures

- PR examination (a plain abdominal x-ray may be required).
- Look for possible 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.

13.5.2 Specific Measures

- Increase fluid intake.
- Bulking agents (e.g.,mucilax, metamucil, granocol). 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 Klean-Prep or oral Fleet Phospho-Soda or other agents may be needed. This procedure is relatively contraindicated in the elderly.
  - Glycerine suppositories/manual evacuation for faecal impaction.

13.6 Jaundice

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

13.6.1 Obstructive Jaundice (Cholestasis)

- Ultrasound is investigation of choice to exclude bile duct dilatation.
- Check coagulation and if necessary correct with parenteral vitamin K (absorption will be reduced).
- If extra hepatic cholestasis (dilated ducts), consider common bile duct stones, stricture and tumours. Appropriate investigations would include CT, MRCP, ERCP. Consult Gastroenterology.
- If no duct dilatation, consider hepatic jaundice.

13.6.2 Hepatic Jaundice

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

13.7 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, ZN stain and PCR if this infection is suspected.

The albumin serum/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 aiming for a weight loss of 0.5 - 1.0 kg / day. Remove ascitic fluid by peritoneal tap, if necessary combined with IV albumin infusion. Give 10 g albumin for every litre of ascitic fluid removed.

13.8 Liver Failure

Where this is suspected commence treatment early.

13.8.1 Clinical and Biochemical Features

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

13.8.2 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 ascitic fluid on page 117), consider cefotaxime 1g q6h IV until culture available.
  - Drugs (especially alcohol, benzodiazepines).
  - Electrolyte disturbance and volume depletion (diuretics, hypokalaemia).
  - Hepatocellular carcinoma. (Check alpha fetoprotein and/or ultrasound.)

13.8.3 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).
- Viral hepatitis testing (assume infectious until result available).
- Blood cultures.
- If cause not obvious consider smooth muscle and antinuclear antibodies.

13.8.4 Treatment

- Treat any underlying cause (e.g., bleeding varices, sepsis).
- Stop all offending drugs.
- Correct hypokalaemia, hypotension, hypoglycaemia.
- If ascites present (see page 117), aspirate for diagnostic purposes.
- Correct coagulation defects with vitamin K 10 mg IV slowly and fresh frozen plasma as indicated.

13.8.5 If encephalopathy suspected

- Give high carbohydrate/low protein diet.
- Gut sterilisation with neomycin 1 g q4h PO.
- Purge with lactulose 10-30 ml TDS adjusted to produce three loose stools per day. Enemas can also be used.
- Watch for alcohol withdrawal (see page 60).
- Consult gastroenterologist promptly.

13.9 Acute Pancreatitis

13.9.1 Clinical Features

- Epigastric 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.*

13.9.2 Diagnosis

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

13.9.3 Aetiology

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

13.9.4 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 types I and V hyperlipidaemia.
- CXR.

13.9.5 Management

- Treatment of shock (see page 23).
- 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 (severe) gallstone pancreatitis suspected. Features include - jaundice, abnormal LFTs and abnormal biliary tract on imaging.

The following are associated with a poor prognosis:

Table 13: 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

13.10 Acute Hepatocellular Dysfunction (Hepatitis)

13.10.1 History

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

13.10.2 Investigations

- U/S scan of the liver and biliary tract.
- INR, and Echis ratio if INR prolonged.
- Tissue auto antibodies, ANA, serum protein electrophoresis.
- Hepatitis A, B, and C serology.
- EBV & CMV serology.

13.11 Percutaneous Endoscopic Gastrostomy

- A percutaneous endoscopic gastrostomy (PEG) procedure may be required when oral food and fluid intake is impossible due to oesophageal obstruction or hazardous because swallowing mechanisms are impaired increasing the risk of aspiration. The gastrostomy tube is placed at gastroscopy, under conscious sedation and local anaesthesia.

  Note:

  - Informed consent is required. This requires consultation with Gastroenterology and often, review by the PEG Nurse Specialist. Ethical, procedural and overall medical issues need to be considered.
  - A PEG does not eliminate the risk of aspiration.
- **Complications**
  - **Skin Infection** - ensure that the tube is not too tight and can rotate freely in the subcutaneous tract. Antibiotics are likely to be required.

- **Peritonitis** - If leakage or early tube dislodgement, start antibiotics and seek advice from Gastroenterology (PEG Nurse ☎ 80965, Gastroenterology registrar or consultant).
- **Inadvertent Tube Removal**
  - Early - risk of peritonitis.
  - >2 weeks. By this time the tract has epithelialised. Place a Foley urinary catheter to maintain the tract which starts to close within 1-2 hours. Seek advice as above.

14. Haematology

14.1 Haematology Department Information

Main Office

Ground Floor, Laboratories, ☎ 80300, Fax 81432

Consultants

- Dr Peter Ganly, Pager 8163
- Dr Steve Gibbons, Pager 8324
- Dr Nigel Patton, Pager 8161
- Dr Mark Smith, Pager 8308
- Dr Ruth Spearing, Pager 8145

Inpatient Services

Clinical Haematology Unit, (BMTU) Lower Ground Floor, Riverside Block. Registrar - Beep 8191.

Consultation and On-call Service

24 hours per day, seven days per week. Fax referrals to 81432 (Monday to Friday). Please also put in internal mail to the Department of Haematology. To speak directly to the consultant, phone 80300 or switchboard and ask for the '**consult consultant**'. Registrars available on Pagers 8314 and 8507. 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 consultant or registrar (Pager 8314).
- Haemostasis Nurse (Beep 8527 or ☎ 81246)
- Haematology Notes are kept in Haematology and are available to the rest of the hospital by contacting the department - Reception during the day (80384) or the Laboratory staff after hours (80373).
- Haematology clinic letters are available electronically in ED on the consultants' computers.

14.2 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.

14.2.1 Investigation of a patient presenting with a possible haemorrhagic disorder

- Family history, history of pattern of bleeding, recent drugs, dietary history, possibility of HIV.
- CBC + Diff, ESR or CRP, 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).*

14.2.2 Treatment

- This is entirely dependent on the results of the initial tests obtained. If a severe **thrombocytopenia** (<10 x 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 (Haemostasis office) 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. Always contact a haematologist or the haemostasis nurse.
- In **haemophilia A** life threatening bleeding requires immediate Factor VIII infusions, with concentrated freeze-dried preparations or recombinant factor VIII. A rough guide is given by the following formula.

Table 14: Factor VIII Infusion

Units of Factor VIII required = (weight in kilograms x % rise desired) ÷ 2
• Currently each Biostate ampoule contains 250 IU. • Recombinant Factor VIII (Kogenate, Re Facto or Recombinate) is also available.

- You will need to know what level of Factor VIII it is desirable to achieve in any particular clinical situation (see above formula). Round to the nearest vial. Do not throw any product away. Every effort should be taken to ensure each patient receives the same specific concentrate that the patient has recently been using.
- In **Von Willebrand's disease** and mild haemophilia A, DDAVP or CSL Factor VIII concentrate is used. DDAVP may be given in a dose of 0.3 mcg/kg in 50 mL 0.9S IV over 30 minutes (starting 60 minutes pre-op, if requiring surgery). DDAVP can be given undiluted SC. Mild haemophilia A patients who infrequently use coagulation factor concentrates should receive recombinant products.
- In **Christmas disease** (Factor IX deficiency) Factor IX concentrate (Monofix) or the recombinant product (Benefix) is the treatment of choice. Consult Haematologist for this and less common coagulation disorders.

14.3 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.

14.3.1 Investigations

- CBC + Diff, film, and reticulocyte count along with standard biochemistry and LDH. Get copies of previous CBC from private lab/GP to ascertain duration of anaemia.
- MCV <80 fl - probable iron deficiency or an inflammatory anaemia. Consider thalassaemia. Request iron studies and ferritin and CRP.

- 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, myelodysplasia. In some patients, particularly the elderly, B_{12} deficiency may be present despite a B_{12} level in the lower range of normal (~<200). Methyl malonic acid measurement may be helpful but is falsely raised in renal impairment.
- MCV 80-100 fl - consider renal failure, hypothyroidism, acute blood loss, malignancy (e.g., do PSA, SPE), and chronic inflammation or infection.

Note: *Decide whether a bone marrow is required.*

Note: *Haemolytic anaemia may be suspected if the reticulocyte count and LDH is raised. A direct Coombs test and liver function tests should be done and if haemolysis is still suspected, the patient should be discussed with the Haematologist.*

14.3.2 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 325 mg 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 severe 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).

14.4 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.

14.4.1 Treatment

- If the neutropenia is a new feature, 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. If the neutropenia is chronic and the patient has been out in the community with neutropenia, then there is no need for isolation.
- If febrile take blood cultures from peripheral vein, and also 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, or a single reading of 38.5°C, parenteral broad spectrum antibiotics such as gentamicin and cefepime **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 page 130). Do not await the results of cultures in this situation. First line antibiotic therapy for the treatment of neutropenic sepsis is:
    - **Cefepime** 2 g IV q12h and **Gentamicin** 5 mg/kg IV q24h

    or, if the above is unsuitable for a particular patient,
    - **Imipenem** 500 mg IV q6h.

15. Infectious Diseases

15.1 Infectious Diseases Department Information

Main Office

3rd Floor, Riverside, ☎ 80951, Fax 80952

Inpatient Services Ward 30

- Professor Steve Chambers
- Dr Alan Pithie
- Registrar
- Kate Gallagher - Intravenous Antibiotic Service (IVAS) Specialist Nurse

Consultation and On-call Service

The on-call registrar and consultant can be contacted via the Christchurch Hospital Operator on 364 0640.

Consultation Guidelines

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

15.2 Meningitis

15.2.1 Clinical Features

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

15.2.2 Causes

- N. meningitidis; S. pneumoniae; H. influenzae (usually paediatric - rare since HIB vaccine). Listeria monocytogenes (immunosuppressed, elderly or pregnant); Mycobacterium tuberculosis.
- Syphilis, leptospirosis, Gram negative bacilli (rare but seen in neonates, post trauma, immunosuppressed).
- Viral - especially mumps, enteroviruses, and herpes simplex (type 2).
- Other - amoebae, fungi (rare).

15.2.3 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.

15.2.4 Investigations

- Blood cultures - 2 sets before antibiotics given.
- Lumbar puncture. **Caution - refer to Management (see page 126) and lumbar puncture technique (see page 52).**

  Collect 2 mL of CSF into each of three numbered sterile 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 x 10^6 cells/L of CSF. PCR testing for N.meningitidis, H.simplex, TB and enterovirus available, but not routine.
- CBC + Diff.
- Na, K, glucose, creatinine, AST, GGT, alk. phos., bili.
- Chest and sinus x-rays (not all cases).
- Coagulation profile.
- Special tests needed for cryptococcus, TB, amoeba, viruses - consult Microbiologists, if indicated.

15.2.5 Management

- **Lumbar puncture (see page 52). If there is any reason to suspect that the patient might have a space occupying lesion, obtain a CT/MRI head scan urgently before doing a lumbar puncture. Therefore, 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 - a CT/MRI head scan should be done before doing a 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, i.e.,** 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 AND DEXAMETHASONE 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, pneumococcal antigen or a pyogenic picture (see below) begin antibiotics. If in doubt, consult Microbiology or the Infectious Diseases Service.

Table 15: 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

15.2.6 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. Currently penicillin resistant pneumococci are rare <10%. The recommendations given here may need to change, depending on the local prevalence of these organisms.

- Proven or presumed pyogenic meningitis:
  - 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. **Add** amoxycillin (300mg/kg/day, up to 12 gm IV daily in 4-6 divided doses) **if** age >60, alcoholic, diabetic, immunosuppressed.
    - If Gram stain or antigen testing is suggestive of pneumococcal disease, then add vancomycin 1 gm IV over 90 minutes q12h 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 2.4g q4h is the preferred treatment if the organism e.g., pneumococci, is sensitive.
  - **Steroids and Meningitis:** A recent randomised controlled trial has demonstrated that dexamethasone [10mg 6 hrly] given immediately before or with the first dose of antibiotic in cases of acute bacterial meningitis in adults reduced unfavourable outcomes from 25% to 15% and mortality from 15% to 7%. The benefit was confined to patients with pneumococcal disease in whom mortality dropped from 34% to 14%. Patients with meningococcal disease were not significantly helped by dexamethasone therapy (although there was a trend towards improvement).
  - In light of these results (supported by early studies in adults and children) dexamethasone 10 mg should be given immediately before or with the first antibiotic dose in all adults with suspected bacterial meningitis (but not those with suspected septic [including meningococcal] shock). If pneumococcal meningitis is confirmed, dexamethasone (10 mg q6h) should be continued for 4 days. If the pneumococcal isolate is shown to have penicillin resistance, vancomycin should be continued with ceftriaxone and careful follow-up with consideration of repeated CSF examination is required. If a pneumococcal cause for the acute meningitis is not established the dexamethasone should be stopped after the first dose. (Ref: NEJM 2002:347:1549-1556)
- 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 600mg BD PO for 2 days 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 261).
- Under 15 years seek paediatric advice.
- **Tuberculous meningitis:**
  - PCR for M.tuberculosis is available. 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, herpes simplex, 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.

15.3 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 been defined in 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 granulocytes.

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 and fluid resuscitation 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.

15.3.1 Clinical Situations which may predispose to Septicaemia

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

15.3.2 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.

15.3.3 Investigations

- Blood cultures.
- 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.
- The diagnosis is based on culturing organisms from the blood so good technique is essential (see page 49).
- If endocarditis is suspected 3 venepunctures (6 bottles) should be taken, ideally spaced over 24 hours. If patients are acutely ill they may be taken stat from several sites. If antibiotics have been given during the past 2 weeks do 6 venepunctures (12 bottles).
- Other cultures:
  - Sputum if possible.
  - MSU, throat and nose swab.
  - Swab skin lesions and ears if local sepsis likely.
  - Consider LP if meningitis possible.
  - Aspirate fluid from joints or serous cavities and send aspirated material to laboratory.
  - If IV cannula sepsis is suspected then swab skin over entry site with alcohol. Remove and cut intravenous section into sterile container with sterile scissors. Consult Microbiology if these samples have to be stored for more than an hour.
  - CBC + Diff, coagulation profile for DIC screen.
  - Na, K, creatinine, glucose, AST, GGT, alk. phos., bili.
  - CXR.
  - Arterial blood gases.

15.3.4 Management of Septicaemia

Fluids:

- Resuscitate with 0.9S then alternate 0.9S with D5W. Blood or plasma may be needed. Large volumes may be needed, inadequate volume repletion is common.
- If the patient is in shock then a CVP line may be needed and larger volumes of fluid required (see page 23). If you are not competent to place a CVP line (see page 50) do not attempt it.
- If patient remains hypotensive (systolic <80 mmHg) despite adequate hydration then inotropic support will be needed. Transfer to ICU.
- Fluid management in septic shock can be difficult and CVP readings may be misleading. Swan Ganz catheterisation is frequently required.

Oxygen therapy (see page 223)

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 (see page 111) secondary to inadequate tissue perfusion may require partial correction.

- 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, and the Preferred Medicines List will give guidance as to which drugs to use initially.
- In hospitalised patients organisms may have been previously isolated and the sensitivities available.
- If infection is cryptogenic (no primary site identifiable) then cefuroxime plus gentamicin is a reasonable choice but this combination will not cover enterococci, anaerobes, Listeria and several other species.
- Reasonable choices include:
  - Cryptogenic sepsis:
    - Community acquired - cefuroxime + gentamicin.
    - Hospital acquired - ceftriaxone + metronidazole.
  - Intra-abdominal sepsis:
    - Amoxycillin + gentamicin + metronidazole.
    - Ceftriaxone + metronidazole.
  - Cellulitis (see page 131): Flucloxacillin.
  - Pseudomonas sepsis:
    - Piperacillin/tazobactam + tobramycin.
    - Cefepime + tobramycin.
  - Urinary tract:
    - Gentamicin.
    - Ceftriaxone.
  - Neutropenic fever/sepsis:
    - **Cefepime** 2 g IV q12h and **Gentamicin** 5 mg/kg IV q24h

    or, if the above is unsuitable for a particular patient,
    - **Imipenem** 500 mg IV q6h.

15.4 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.

15.5 Cellulitis and Erysipelas

- Definitions:
  - Cellulitis is an infection of the skin and soft tissues, most commonly caused by Group A Streptococci and/or Staphylococcus aureus.
  - Erysipelas is a form of cellulitis with rapid onset, clearly demarcated 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 or cerebrovascular disease have a high incidence of cellulitis and are prone to relapse.
- Investigations:
  - Blood cultures required if patient is very unwell (i.e., satisfies criteria for SIRS on page 128), has lymphatic obstruction, or is immunocompromised.
  - Aspirate any skin blisters or fluctuant areas and swab any skin lesions, ulcers or wounds in the area of the infection.
  - CBC + Diff, 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:
  - **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 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.

    Note: *Cellulitis commonly appears to worsen in the first few days but this should not automatically lead to a change in antibiotics unless the patient's 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 **or** flucloxacillin + gentamicin + metronidazole **or** cefuroxime + metronidazole.
  - **Erysipelas and cellulitis** thought to be caused by Group A Streptococci can be treated by penicillin alone. Give benzylpenicillin 1.2g q4h IV.

- **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 g/day) IV benzylpenicillin + IV clindamycin 600mg q6h + 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.

15.6 Infection with Antibiotic Resistant Organisms

- Infection with antibiotic resistance is of increasing clinical importance.
- In New Zealand, Methicillin resistant Staph aureus (MRSA), Penicillin Insensitive Pneumococci and multi-resistant gram negative organisms (e.g., Acinetobacter baumanii) are currently of great concern.
- Multi-drug resistant Mycobacterium tuberculosis, Vancomycin resistant enterococci and Vancomycin Insensitive Staphylococci are likely to become increasing problems.
- To prevent development and spread of resistant organisms - use antibiotics only when clinically indicated, avoid broad spectrum antibiotics, use as narrow spectrum as possible, keep courses short and isolate patients when such infection is likely or proven.
- **MRSA** This should be suspected in all patients who have been previously hospitalised within two years, either in New Zealand or overseas. Implement appropriate infection control measures and be guided by the Infection Control Nurses. Many patients are colonised and do not require specific therapy. For a true infection, consult an Infectious Diseases physician regarding antibiotic therapy.
- **Penicillin resistant/insensitive pneumococci** Consider in all serious pneumonias and especially in patients from overseas, the immuno-suppressed, those with chronic lung conditions, and patients who have had repeated courses of antibiotics. If confirmed, nurse in side room, high dose beta lactam antibiotics usually remain effective. If therapy fails or high level resistance found, consult the Infectious Diseases Service. For Pneumococcal meningitis, add vancomycin to ceftriaxone until sensitivities available. See Meningitis on page 127.

15.7 Herpes Simplex

15.7.1 Mucocutaneous and Oral Herpes Simplex

- **Primary attack**. This may occur in adulthood and can be severe causing fever, toxicity, oral ulceration and lymphadenopathy. Acute urinary retention may occur in pelvic disease. Healing occurs in 2 weeks.
- **Recurrent attacks**. Most attacks are mild and occur at site of initial infection.
  - Sun protection is useful in preventing recurrences.
  - Frequent recurrence may be prevented with prophylactic treatment.
  - Recurrences can be complicated by erythema multiforme which can be more troublesome than the infection itself.
- **Eczema herpeticum**. In the presence of dermatitis secondary attacks may disseminate causing a generalised eruption with groups of vesicles, weeping and skin tenderness.
- **Infectivity**. All lesions are infectious and may cause infection, particularly on the fingers (Whitlow) among staff. Use gloves.
- **Diagnosis**:
  - Usually clinical.
  - If in doubt take a sample of fluid and cells from the ulcer base for PCR or immunofluorecence with a cotton swab. Place in viral transport medium to send to the laboratory.

- **Treatment**:
  - Primary herpes infection:
    - Aciclovir 200 mg 5 times daily 7-10 days.
    - If cannot swallow or severely unwell aciclovir 5 mg/kg iv q8h.
  - **Recurrences**:
    - Normal host - minor attacks - povidone iodine cream 10% tds. Aciclovir cream of minimal benefit. Aciclovir orally 200 mg 5 times daily only of benefit if started early.
    - Immunosuppressed - aciclovir 400 mg 5 times daily 5-7 days.
    - Prophylaxis - aciclovir 400 mg BD.

15.7.2 Herpes Simplex Meningitis

- **Primary attack**:
  - Usually occurs with systemic illness and ulceration.
  - Aciclovir 5 mg/kg IV q8h for 7 days.
- **Recurrent attacks**:
  - Most commonly occurs with Herpes simplex type 2.
  - Natural history is benign and lasts 48 -72 hours in most cases.
  - Normal host - aciclovir therapy not recommended.
  - Immunocompromised - aciclovir 5 mg/kg IV q8h for 5 days.

15.8 Varicella Zoster (Shingles)

- This is caused by the chickenpox virus and may cause severe local infection complicated by secondary bacterial infection - usually S. aureus or S. pyogenes.
- It may occur in any dermatome.
- Aciclovir treatment improves the rate of recovery and reduces post herpetic neuralgia only if started within 72 hours of onset. Best results follow earliest possible treatment.
- All patients with ocular herpes or immune suppression should be treated with aciclovir.
- Steroids may reduce post herpetic neuralgia but proof is lacking.
- There are a number of strategies for reducing post herpetic neuralgia including low dose antidepressants.
- **Diagnosis**:
  - Clinical.
  - If in doubt, send samples of fluid and cells taken from ulcer base with a cotton swab in viral transport media for PCR.
- **Treatment**:
  - Normal host:
    - Aciclovir 800 mg 5 times daily orally for 7 days.
    - Prednisone 30 mg BD for 7 days, 15 mg daily for days 8-14 and 7.5 mg daily on days 15-21 **if >50 years and severe pain on presentation**.
  - Immunocompromised host:
    - Not severe - as for normal host.
    - Severe (more than one dermatome, dissemination) aciclovir 10 mg/kg iv q 8 hours 7-14 days.
  - **Ocular zoster**:
    - Aciclovir 800 mg 5 times daily orally and consult the ophthalmologist.
    - Sight-threatening disease Aciclovir 10 mg/kg iv q 8 hours.

15.9 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.

15.9.1 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 (i.e., Standard Precautions).
- The virus is present in body fluids and can 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 or has cognitive impairment, or for performing procedures.
- Goggles should be worn if splashes likely e.g., 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 Microbiologist or Infection Control **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 and the CDHB Intranet.

15.9.2 Antibody Testing

- Provide full explanation of test. 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.

15.9.3 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.

15.9.4 Clinical Presentation

- Acute infection:
  - "Mononucleosis-like" fever, lymphadenopathy, sore throat, truncal rash (maculopapular), diarrhoea.
  - Aseptic meningitis
    - These patients are **infectious** although the HIV antibody test is often 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 leukoplakia, herpes simplex, gingivitis
    - Skin - herpes zoster, fungal infections.
- Suggestive laboratory findings:
  - Anaemia.
  - Thrombocytopenia.
  - Leucopenia/lymphopenia.
  - Reduced CD4 T lymphocyte count.

15.9.5 Some Specific Complications of Late Stage HIV Disease

May be presenting feature.

Pneumonia - Pneumocystis jiroveci (previously known as P.carinii) is most 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 (e.g., blood and sputum cultures) and treat as community acquired pneumonia. Otherwise treat as pneumocystis jiroveci pneumonia.

- Pneumocystis jiroveci pneumonia: 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 and pulse oximetry - 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 pneumocystis.
  - Throat swab - viral immunofluoresence and culture.
  - Bronchoscopy may be indicated. Consult Infectious Diseases.
- Treatment:
  - Begin treatment for presumed pneumocystis pneumonia 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). Usually 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 glucose QID.

CNS Disease

- May be due to direct effects of HIV, opportunistic infection or neoplasm.
- 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. TB meningitis should be considered.

- Space occupying lesions - Lethargy and confusion progressing to fits and focal signs. Causes are lymphoma, toxoplasmosis and other infections.
- Investigations - please consult Infectious Diseases.

Note: *In all the above situations it is essential to obtain advice from an Infectious Diseases Consultant.*

Retinitis

- Most often due to cytomegalovirus. This infection may progress to **blindness** very rapidly.
- Consult Infectious Diseases urgently.

GI Disease

- Oesophagitis is generally due to candida or herpes simplex. Endoscopy or barium swallow may be needed for diagnosis.
- Consult Infectious Diseases.

Constitutional Disease

- Systemic symptoms - fever, weight loss >10%, sweats, fatigue
- If fever is documented but no localising symptoms, a systematic search is needed. Consult Infectious Diseases.

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

15.10 Malaria

15.10.1 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 important to assume Chloroquine resistance in all cases of P.falciparum until proven otherwise.**

15.10.2 Presentation

- Prophylaxis should be continued 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 prophylaxis 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.falciparum - cerebral malaria, focal signs uncommon, mortality 20%.
    - Renal failure and haemoglobinuria.
    - Pulmonary oedema.
    - Hypoglycaemia especially during pregnancy, in children and quinine therapy.
  - P.vivax - rupture of spleen, be careful on palpation (rare).

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

15.10.3 Investigations

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

15.10.4 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 chloroquine is unavailable, use atovaquone/proguanil 1000 mg/400 mg (4 tablets) once daily with food for 3 days.
  - 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 PO daily for 14-21 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 atovaquone/proguanil may be appropriate in some patients with mild/moderate disease. Give atovaquone/proguanil 1000 mg/400 mg (4 tablets) once daily with food for 3 days.
  - **IV therapy**: Quinine dihydrochloride 20 mg/kg in D5W by infusion over 4 hrs. Monitor ECG during infusion. Hypoglycaemia may occur. **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. Seek advice.

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

15.10.5 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.

15.11 Causes of Fever in the Returning Traveller

These include malaria as well as those listed below:

- Enteric fever.
- Dengue fever.
- Typhus.
- Legionnaires Disease.
- Tuberculosis.
- Amoebic liver abscess.
- Hepatitis (viral).
- Viral haemorrhagic fever.

15.11.1 Investigations

- Blood cultures - 3 sets.
- Urine culture.
- Stool culture.
- CBC + Diff, blood film and eosinophil count.
- Coagulation profile for DIC.
- Na, K, creatinine, AST, GGT, alk. phos., bili.
- Hepatitis markers.
- Specific serology and where possible, specific PCR testing.
- CXR.
- ECG.
- Abdominal ultrasound if diagnosis is unclear after the above tests have been done.

Blood cultures may 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 sensitivities are known. Aminoglycosides are ineffective.

16. Nephrology

16.1 Nephrology Department Information

Main Office

- 3rd Floor, Parkside West ,☎ 80655, Fax 80941

Inpatient Care Ward 14

- Prof Z Endre
- Dr K Lynn
- Dr D McGregor
- Dr R Robson
- Dr M Searle

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

16.2 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.
- Acute renal failure is common in hospital. The commonest causes are effective circulating volume depletion and nephrotoxins (drugs). It is preventable by avoiding these factors especially prior to surgery and radiological contrast procedures. Ask for advice!

16.2.1 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.

16.2.2 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 to assess kidney size and echo texture. A plain radiograph to screen for renal calculi.
- If you suspect Goodpasture's Syndrome or a systemic vasculitis such as Wegener's granulomatosis - rapid serological tests for anti-GBM, antiproteinase 3 and anti-myeloperoxidase antibodies are available from Immunology. These tests should not usually be ordered out of normal working hours and the clinical problem should be discussed with a physician.
- Urgent renal biopsy may be indicated, particularly when the urine evaluation suggests an aggressive glomerulonephritis.

16.2.3 Management

- Stop any potentially nephrotoxic drugs.
- Ensure optimal hydration with appropriate fluid - blood or 0.9S. 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.
- 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 16: 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.
>7.5 mmol/L	As above but also give 10-30 ml 10% calcium gluconate IV (to reduce risk of arrhythmias). 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.
  - pH <7.1 mmol/L

  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.*

  Note: *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 17: 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

16.3 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 58). This is particularly important for drugs such as the aminoglycosides, cephalosporins, cimetidine, ranitidine, digoxin, procainamide, the ACE inhibitors and some of the beta-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. The fu of commonly used drugs is available in the Preferred Medicines List.
- 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), fibrates and NSAIDs.

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 Table 8 on page 70 for dosage recommendations**.

16.4 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:

Creatinine Clearance Calculation

$$\text{CrCl (mL/min)} = \frac{(140 - \text{age}) \times \text{lean body weight (kg)}}{\text{plasma creatinine (mmol/L)} \times 800} \quad (\times 0.85 \text{ if female})$$

  - 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.
- **MDRD Formula** Christchurch Hospital Laboratories are now reporting estimated GFR using the MDRD formula. This complex formula has been shown in several trials to be as good or better than Cockroft and Gault. Estimated GFR is reported in ml/min (normal >90) and does not require weight to be entered.
- If in doubt concerning the prescribing of a drug in a patient with renal insufficiency seek advice.

16.5 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 with bacteriuria will also have pyuria (>10 x 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.

16.5.1 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.
- When interpreting the number of bacteria forming units/litre of urine (cfu/L) of any uropathogen, you will need to take into account the presence/absence of pyuria and symptoms, regardless of the patient's gender.

16.5.2 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.

16.5.3 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:

Table 18: Drug Guidelines for Cystitis

Single dose
▪ Trimethoprim 600 mg*
▪ Norfloxacin 800 mg
3 day course
▪ Trimethoprim 300 mg q24h
▪ 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.

Prophylactic Treatment for Patients with Recurrent Urinary Tract Infections

- Patients with recurrent UTIs (eg. >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 (eg. 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 or trimethoprim 150 mg (preferred agents), or norfloxacin 200 mg

  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.

16.6 Acute Pyelonephritis

- A syndrome of fever (>37.8°C) ± rigors, loin pain or tenderness together with infected urine. If no fever, reconsider the diagnosis.
- Lower urinary tract symptoms may be absent.
- Symptoms may be unilateral or bilateral.
- Patients with severe acute pyelonephritis (toxic, requiring IV fluids or parenteral analgesia) require hospitalisation.
- 10-15% will have a bacteraemia.

16.6.1 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**).

16.6.2 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.
- Rectal and vaginal examinations should be done only if clinically indicated.
- CBC + Diff.
- Na, K, and creatinine.
- Blood cultures. These are not indicated in uncomplicated acute pyelonephritis. Blood cultures should be taken if there is: doubt over the diagnosis; evidence of sepsis (i.e., satisfies criteria for SIRS on page 128); renal failure; or a prosthetic device is present.
- Patients with acute pyelonephritis who follow an atypical course, e.g., fever or severe loin pain > 48-72 hours, (?obstruction, kidney stone) should have an ultrasound examination of the urinary tract. A CT urogram is the best test if a urinary stone is suspected.
- A cystoscopy may very occasionally be indicated.

16.6.3 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 (e.g., Ciprofloxacin 250 mg BD) starting on the second day of treatment. Treatment should be continued for 5 days.

The choices of **parenteral** agents are:

- Gentamicin - initial dose 3 mg/kg.
- 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.

The urine should be recultured 10-14 days after completion of therapy.

17. Neurology

17.1 Neurology Department Information

Main Office

- 3rd Floor, Riverside, ☎ 80940, Fax 81226

Inpatient Services

These are provided by a single inpatient Neurology Team, headed by one of five Neurologists on a rotational basis:

- Dr S Avery
- Dr J Fink
- Dr D Mason
- Dr P Parkin
- Assoc. Prof. B Taylor

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, contact the Acute Neurology Registrar (Pager 8111), 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.

17.2 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 and at risk of brain herniation.

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.

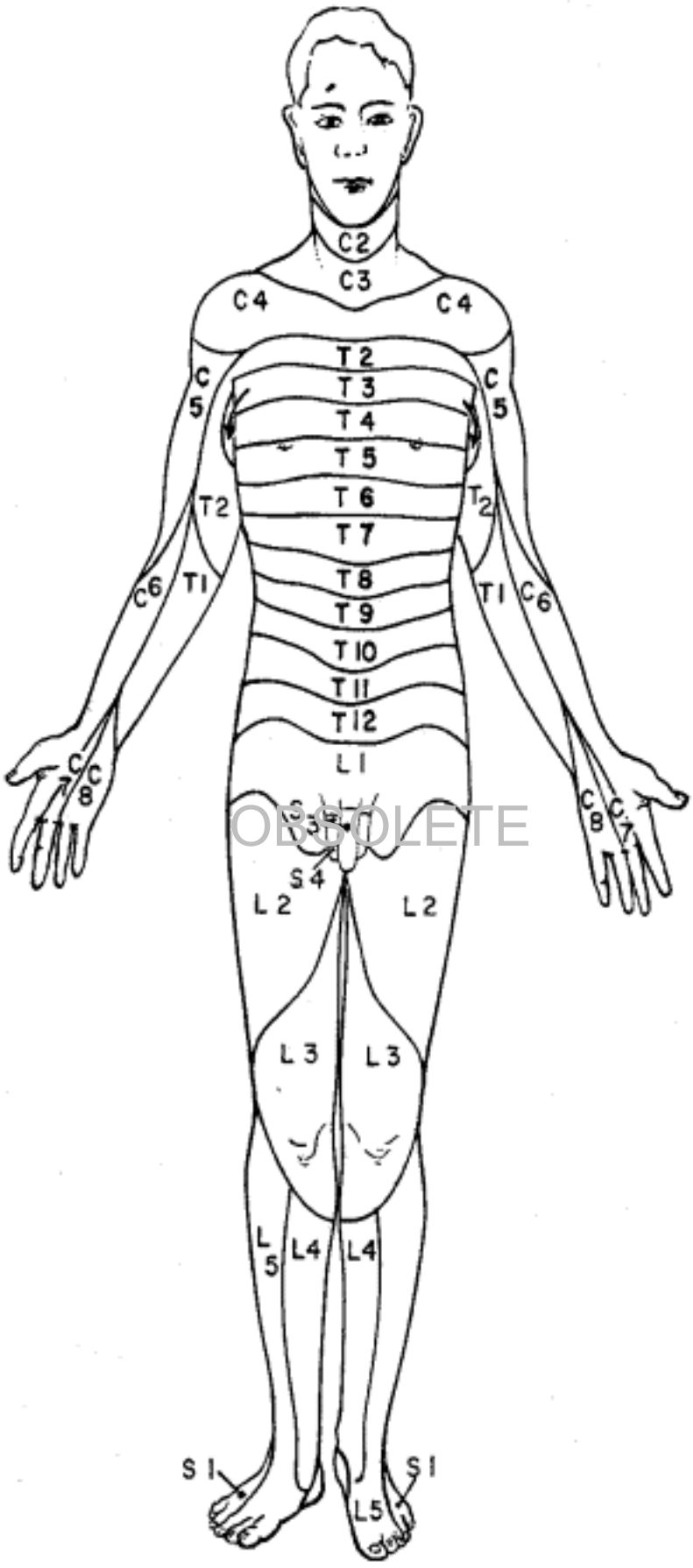
Reflexes

- 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)

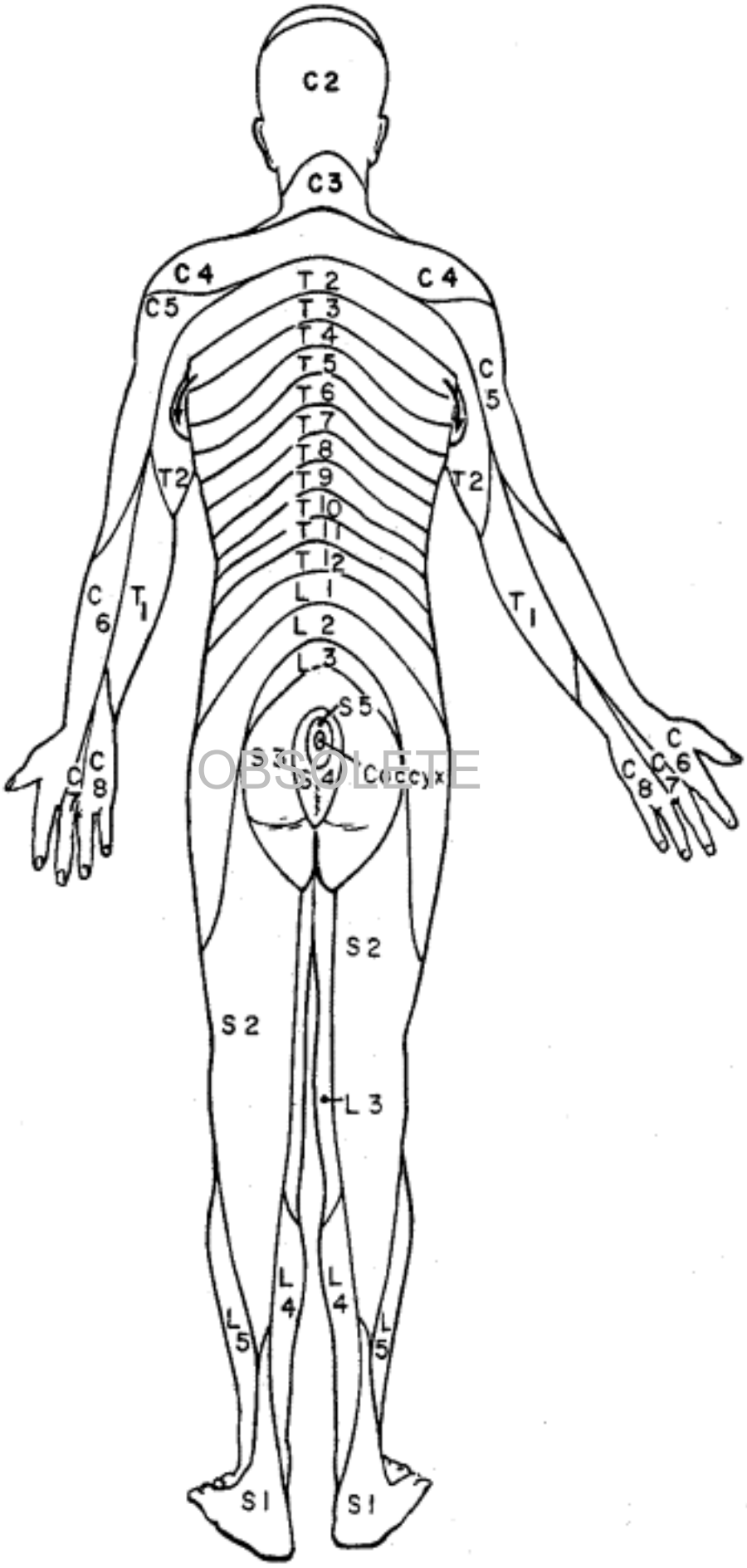
Segmental Innervation

- The segmental innervation of the skin is illustrated (see page 148). This can be more readily recalled by remembering certain "key" dermatomal levels e.g.:
  - C5: Skin over deltoid muscle
  - C6: Thumb
  - C7: Middle finger
  - C8: Little finger
  - T10: Umbilicus
  - L1: Groin
  - L3: Knee
  - L5: Anterolateral calf and dorsum of foot
  - S1: Lateral foot and little toe

17.2.1 Dermatome Distribution - Anterior



17.2.2 Dermatome Distribution - Posterior



17.3 Stroke

17.3.1 Acute Stroke Unit (ASU), Ward 31

Admission policies:

- All patients with stroke (or TIA) admitted to hospital should be admitted to the Acute Stroke Unit, either under Neurology or one of the GM teams responsible for stroke (teams 1, 4, 6, 8, 10, or 11).
- The establishment of the ASU is not intended to alter the previous admission policy regarding division of patients with stroke between general medicine (GM) and neurology. In general:
  - Admission under Neurology is indicated if specialised neurological assessment, monitoring, or management may be required. For example:
    - Patients considered for thrombolysis.
    - Patients with progressive, recurrent, or unstable stroke deficits.
    - Younger patients with large strokes who may be at risk of deterioration due to progressive brain swelling.
    - Patients where the diagnosis or aetiology of stroke is of uncertain or unusual kind, including younger patients (<65 years) without traditional vascular risk factors.
  - Patients who present with stroke as a manifestation of systemic cardiovascular disease without other acute neurological issues will be admitted to the Acute Stroke Unit under the care of the general physician of the day responsible for stroke.

Patients with stroke on other hospital wards:

- The Acute Stroke Clinical Nurse Specialist should be notified of all patients in the hospital with stroke and will visit them on their ward. Page 8978 (0730-1600 Mon-Fri) or leave a message with the Ward 31 ward clerk, ☎ 89310.

17.3.2 Stroke Classification/Causes

Infarction (85%):

- Oxfordshire clinical classification of stroke type:
  - TACI: Total anterior circulation infarction syndrome (hemiplegia+ hemianopia+ dysphasia/ neglect).
  - PACI: Partial anterior circulation infarction syndrome.
  - LACI: Lacunar infarction syndrome.
  - POCI: Posterior circulation infarction syndrome.

  All patients admitted to the ASU should have an Oxfordshire classification documented.
- TOAST* classification of stroke aetiology:
  - "Large artery" thrombosis or embolism: e.g., ICA stenosis or aortic arch atheroma (common), MCA stenosis (uncommon).
  - Cardioembolic: atrial fibrillation is the most common cause.
  - Lacunar: common in hypertension and diabetes.
  - Other, e.g.:
    - Carotid or vertebral artery dissection - consider in younger patients, especially if retro-orbital or neck pain.
    - Cerebral venous sinus thrombosis - see below.
  - Unknown

  *TOAST = **T**rial of **O**rg 10172 in **A**cute **S**troke **T**reatment. *Adams et al. Stroke. 1993; 24:35-41.*

Intracerebral Haemorrhage (ICH) (15%):

- Deep ICH: Usually caused by hypertension. Can be associated with vascular abnormalities in young (<45y) patients, or those with **no** history or evidence of hypertension.
- Lobar ICH: Hypertension, amyloid angiopathy (elderly), AVM, aneurysm, cerebral venous sinus thrombosis.
- Also consider:
  - Coagulation disorders.
  - Haemorrhagic infarction (e.g., cerebral venous sinus thrombosis).
  - Contusion (trauma).

Subarachnoid Haemorrhage (SAH) (see page 154)

Cerebral venous sinus thrombosis (CVT)

- Uncommon but important to recognise and treat. Diagnosis is often delayed and usually requires MRI with magnetic resonance venography (MRV) to detect.
- Clinical presentation is broad:
  - Headache and haemorrhagic infarction are usually present.
  - Onset may be abrupt, progressive, or step-wise.
  - Papilloedema may be present.
  - Seizures can occur.
- Consider CVT in the differential diagnosis of ICH, especially temporal lobe ICH (lateral sinus thrombosis) or "atypical" ICH.
- Treatment is anticoagulation with heparin, even when haemorrhage is present. **Neurological consultation is essential**.

17.3.3 Investigations

- Include:
  - CBC + Diff (polycythaemia, thrombocytosis).
  - ESR (arteritis).
  - Na, K, Ca, albumin, creatinine.
  - Glucose.
  - HbA1c.
  - Lipids.
  - ECG.
  - CT head scan.

  Note: *Cerebral haemorrhage is often not distinguishable from cerebral infarction on clinical grounds alone. CT distinguishes between haemorrhage and infarction, defines the location of the lesion and may define the nature of the underlying cause.*
- The following investigations may also be appropriate:
  - Coagulation profile (all patients with haemorrhage).
  - Duplex carotid ultrasonography (minor ischaemic stroke/TIA, ICA territory, possible surgical candidate).
  - MRI brain, including diffusion-weighted imaging.
    - Particularly helpful when diagnosis is uncertain, and for unusual stroke syndromes/younger patients e.g., carotid or vertebral artery dissection, cerebral venous sinus thrombosis.
    - Consider Neurological consultation.
  - Homocysteine.
  - Thrombophilia screen, lupus anticoagulant, anticardiolipin antibodies (generally younger patients only).
  - Echocardiogram (transthoracic or transoesophageal; particularly if recent MI, dilated cardiomyopathy, or if mitral valve disease or LV aneurysm suspected, or for younger patients with no other cause identified (?patent foramen ovale - TOE required).

- Syphilis serology.
- ANA.
- Angiography - MRA, CTA or DSA (particularly for patients with ICH, but may not be required for deep hypertensive haemorrhages). MRV or DSA for suspected cerebral venous sinus thrombosis.

- CXR is **not** a routine investigation for stroke. Request as clinically indicated.

17.3.4 Acute Management

- **Thrombolysis** with tissue plasminogen activator for acute ischaemic stroke.
  - May be considered for highly selected patients **within 3 hours of stroke onset**
  - May only be given in consultation with the Acute Neurology Team: call the Acute Neurology Registrar (working hours: pager 8111, other times via hospital operator) or on-call Neurologist. Dr Fink is also available to consult on possible thrombolysis cases during working hours by pager via the hospital operator.
  - A detailed thrombolysis protocol is available in the Acute Stroke Unit.
- **Aspirin 150-300 mg daily** should be initiated once ICH excluded by CT.
- **Maintain patient 'homeostasis':**
  - Avoid aspiration pneumonia. Document bedside swallowing assessment for all patients.
    - If in doubt, make nil by mouth (NBM) until formal speech language therapy (SLT) swallowing assessment.
  - Maintain hydration: subcutaneous, NG or IV fluids if NBM or inadequate intake.
    - Avoid glucose-containing **intravenous** fluids whenever possible (hyperglycaemia is associated with poor outcome after stroke).
  - Manage hyperglycaemia.
    - Detailed guidelines are available in the ASU. Particular care is needed for insulin-dependent diabetics who are made NBM following stroke.
  - Avoid hypotension.
  - Consider treating extreme hypertension:
    - Ischaemic stroke: >220/120.
    - Intracerebral haemorrhage: >180/105.
    - Labetalol is the preferred agent to control acute hypertension if this is necessary. Nifedipine should be avoided.
  - Treat pyrexia >37.5°C with paracetamol (pyrexia is associated with poor outcome after stroke).
  - Nutrition: NGT feeding should be considered if NBM/poor intake >24-48h.
  - DVT prophylaxis:
    - Full-length graduated compression stockings for all immobile or hemiparetic patients unless contraindications e.g., fragile skin, peripheral vascular disease.
    - Subcutaneous LMW heparin should only be considered after 48h for patients with contraindication to stockings, aspirin intolerance, or with past history of DVT.
  - Early mobilisation out of bed within 24h should be expected.
    - Even short periods are beneficial, e.g., up to commode for toilet.
- **Heparin:** intravenous heparin, subcutaneous heparin, LMW heparin or heparinoids are not routinely recommended for treatment of patients with acute ischaemic stroke.
  - IV heparin may be considered in carefully selected patients, (e.g., evolving basilar thrombosis, crescendo TIA, carotid or vertebral artery dissection, visible cardiac mural thrombus on ECHO). However, there is little evidence to support its use. Neurological consultation is recommended.
- **Cerebral Haemorrhage:**
  - Stop anticoagulants, including aspirin.
  - Manage hypertension as indicated above.
  - If life-threatening, surgical evacuation should be considered for superficial intracerebral or intracerebellar haematoma. Urgent neurosurgical consultation is required.
- **TIA**
  - By definition: **complete** resolution occurs within 24 hours. Most "true" TIAs resolve completely within **one hour.**

- In general, there is no difference in the management of TIA and completed stroke. Urgent investigation is required, including CT head.
- Hospital admission may not be required if **complete** resolution has occurred. However this decision needs to be taken by a physician with expertise in stroke management. If they feel it is appropriate, specialist assessment and investigations can be arranged as an outpatient within 7-14 days (contact Acute Neurology Registrar to arrange).
- If vertebrobasilar territory, consider subclavian steal syndrome. Check BP in both arms.
- Carotid territory - semi urgent carotid ultrasonography if patient potentially suitable for endarterectomy.

17.3.5 Secondary Prevention

- **Aspirin 75-150 mg daily** for all patients with ischaemic stroke not treated with warfarin, unless contraindicated.
- **Dipyridamole 150 mg bd** may be considered for additional antiplatelet therapy for patients with recurrent stroke or TIA taking aspirin or those who are intolerant of aspirin. Special authority application is required for funding.
- **Statin lipid-lowering therapy**. The results of one large randomised controlled trial support the use of simvastatin 40 mg daily to reduce risk of recurrent vascular events after stroke, even in patients with "normal" cholesterol levels (Lancet 2002;360:7-22). Other major trials of statin therapy after ischaemic stroke are ongoing. Some patients with low HDL, high triglycerides, and clinical features of a metabolic syndrome may be better treated with other therapies for correction of the lipid disorder and require further assessment.
- **Antihypertensive therapy** is recommended for all patients after stroke or TIA unless there is symptomatic hypotension.
  - Combination ACE inhibitor+diuretic is supported by PROGRESS trial (Lancet 2001;358:1033-41). This study demonstrated a 40% relative risk reduction of recurrent stroke with a mean 12 mmHg lowering of systolic BP, even for "non-hypertensive" patients. The blood-pressure lowering effect appeared to be most important in this trial, rather than a specific effect related to the particular medications used. Medications in this trial were **additional** to any previous antihypertensive therapy.
  - Treatment initiation generally delayed >7-14 days from stroke onset.
  - Cautious introduction low dose to avoid hypotension, titration subsequently.
  - This treatment is additional to any previous antihypertensive therapy.
- **Warfarin** is recommended for cardioembolic stroke. Target INR is generally 2.0-3.0 unless the patient has mechanical heart valves or antiphospholipid antibody syndrome, when higher ratios may be needed.
  - Optimal time for initiation of warfarin after stroke is not known. For patients with AF, the risk of early recurrent stroke is low and anticoagulation is usually delayed for 7-14 days, which may reduce the risk of haemorrhagic transformation of stroke. However, for **minor** stroke or **TIA**, initiation of anticoagulation after 48 hours is reasonable. It is preferable to commence warfarin treatment in hospital.
- For patients with atrial fibrillation (AF) **who have already had stroke or TIA** the benefit of warfarin usually **far outweighs** the risk of haemorrhage (including risk of subdural haematoma due to falls) due to warfarin.
- **Carotid Endarterectomy** is recommended for patients with minor ischaemic stroke or TIA in the internal carotid artery (ICA) territory when a severe (>70%) stenosis of the ipsilateral ICA is present.
  - Patients benefit most when endarterectomy is performed **early** after symptoms.
  - Some patients with ipsilateral 50-70% stenosis might benefit from endarterectomy, also. Some patients who are not surgical candidates could be considered for carotid angioplasty and stenting. Neurological consultation is recommended for these patients.
- **Smoking cessation advice** (see page 212) should be given to all current smokers.
  - QUITLINE 0800 778 778

17.3.6 Neurological Complications following Stroke

- **Brain oedema and raised intracranial pressure.**
  - Young patients with large infarcts are at risk of brain oedema causing raised intracranial pressure. This usually presents as progressive neurological deterioration 24-72 hours after stroke onset.

  Refer to management of raised intracranial pressure (see page 159). However:
  - Corticosteroids are **not** helpful for post-infarction brain oedema.
  - Surgical decompression can be life-saving, particularly for cerebrellar infarcts, but also may be considered for some young patients with large hemispheric strokes (hemicraniectomy). Neurological and neurosurgical consultations are recommended.
- **Seizures.**
  - Seizures occur in 6-8% of strokes. If a seizure has occurred, anticonvulsants should be prescribed. Neurological follow-up is also recommended to determine the length of anticonvulsant treatment required for the individual patient.
- **Other complications.**
  - E.g., DVT, pressure areas, shoulder pain, dehydration, aspiration, malnutrition.
  - Refer to "Maintain patient 'homeostasis'" under Acute Management (see page 152).

17.3.7 Rehabilitation

- Rehabilitation efforts should commence as soon as possible after stroke, e.g., mobilisation out of bed within the first 24 hours.
  - Refer to **Acute Management (see page 152).**
  - Identify patient goals.
  - Involve the multidisciplinary team.
    - Inform the Stroke Clinical Nurse Specialist, pager 8978.
  - Discharge planning:
    - Within 48-72 hours, consider whether discharge directly home may be feasible within a 7-10 day admission. If not, early referral to The Princess Margaret Hospital Stroke Area (>65 y), or Burwood Hospital (<65 y) should be made.

17.4 Subarachnoid Haemorrhage

17.4.1 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 %.

17.4.2 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.

17.4.3 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* ***contraindicated*** *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 (see page 52) could prove fatal.*

  Remember: *There are only three ways to diagnose a subarachnoid haemorrhage: lumbar puncture (see page 52), 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.

17.4.4 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. Start with the lower dose of 0.5 mg/hr if BP unstable and weight less than 70 kg.

  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.

17.4.5 Christchurch Hospital Admission Arrangements

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

17.5 Status Epilepticus

17.5.1 Definition and Implications

- Defined as continuous seizure activity lasting 30 minutes or more **or** intermittent seizure activity lasting 30 minutes or more and during this time the patient remains unconscious.
- Cerebral metabolic decompensation occurs after around 30 minutes or so of continuous, uncontrolled, convulsions.
- Status, and the conditions responsible for it, carries an overall mortality of 25% in adults. Urgent treatment is therefore imperative.

17.5.2 Causes

- Commonest cause is anticonvulsant withdrawal or non-compliance in a patient known to have epilepsy.
- If the patient is not known to be epileptic, consider whether the cause is due to an intracranial lesion or is secondary to a toxic-metabolic disorder. Remember, in particular, alcohol withdrawal, hypoglycaemia and infection.
- **Acute Disorders**
  - Electrolyte imbalance
  - Stroke
  - Cerebral trauma or surgery
  - Drug toxicity
  - Encephalitis, meningitis
  - Hypoxic brain damage
  - Sepsis/infection
  - Alcohol or benzodiazepine withdrawal
  - Renal failure
- **Chronic Disorders**
  - Pre-existing epilepsy
  - Poor drug compliance
  - Change in antiepilepsy drug therapy
  - Chronic alcoholism
  - Cerebral tumour or other structural lesion affecting the brain.

17.5.3 Investigations

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

17.5.4 Management

- Ensure adequate airway and oxygenation - monitor.
- Consult ICU team early if:
  - Inability to maintain adequate airway
  - Cardiovascular instability
  - Prolonged status with metabolic or systemic decompensation
  - Status unresponsive to IV diazepam
  - Underlying cause requiring intensive therapy, e.g., sepsis, encephalitis.
- Insert IV line and take bloods (above).
- Administer **diazepam** 5 mg IV bolus, then 2 mg IV/minute until seizures stop or 20 mg maximum. Seek advice from ICU if unsuccessful.
- If patient has not been on prescribed antiepileptic medication prior to developing status, start **IV phenytoin:**

Table 19: Phenytoin IV Infusion

- Usual IV dose in Status Epilepticus (Adults)
  - 15 mg/kg phenytoin in 100 ml 0.9S and given at a rate not exceeding 50 mg/minute (25 mg/minute in the elderly). Up to 20 mg/kg may be needed.
- IV phenytoin can cause hypotension, bradycardia and arrhythmia and thus all infusions loading or routine should be monitored at least an hour from the end of the infusion.
  - Pulse rate, respiration rate and blood pressure every five to ten minutes. Continuous ECG monitoring. If possible, a doctor should be present on the ward to interpret the cardiac monitoring.
- Maintenance Phenytoin 100 mg 6-8 hourly PO, NG tube or IV.

Note: *Phenytoin precipitates in all IV solution except 0.9S, because of pH incompatibility. Phenytoin also precipitates in tissues if given IM so that this route is contraindicated.*

- If status is not controlled following IV phenytoin, transfer to ICU will be necessary. IV thiopentone may be necessary.
- If the patient is already known to be on antiepileptic medication, obtain urgent blood concentration. If the level is subtherapeutic, continue to administer the same drug in modified dose. If the level is therapeutic, start a second antiepileptic drug. Choices include carbamazepine, sodium valproate or phenytoin.
- If there is no past history of epilepsy, or if there is a known history of chronic heavy alcohol use, consider thiamine 100 mg IM followed by an IV bolus of 50 mL 50% dextrose.

Summary of Drug Therapy:

Step 1: Diazepam 10-20 mg IV

Step 2: Phenytoin IV 15 mg/kg

Step 3: If no response, call ICU team for consideration of GA with thiopentone.

17.5.5 Further Management Points

- Be on the look out for systemic complications:
  - Dehydration
  - Hyponatraemia
  - Hyperkalaemia
  - Metabolic acidosis
  - Hypoglycaemia
  - Acute tubular necrosis
  - Acute pancreatitis
  - Acute hepatic necrosis
  - Vertebral fracture
  - Rhabdomyolysis
  - DIC
  - Multiple organ dysfunction syndrome
- Phenytoin has saturable kinetics. At the higher end of the therapeutic range, small dose increases may result in large plasma concentration increases.
- Phenytoin is highly protein bound. This makes interpretation of total plasma concentrations difficult in hypoalbuminaemia or severe renal impairment. Free plasma concentration measurements are advised if albumin is low or if there is known displacing drug (such as valproate or aspirin).

- An alternative to the use of IV phenytoin (such as the patient who is already prescribed phenytoin prior to admission, and in whom the plasma levels are therapeutic) is IV phenobarbitone. This is administered in a dose of 10 mg/kg at a rate not exceeding 100 mg/minute (max dose 1 g).
- Once status is controlled, it is essential to address maintenance antiepilepsy medication needs. If the patient is taking antiepilepsy medication at presentation, it is important to ensure that these are continued.
- Once status is controlled, it is essential to establish its cause. CT, MRI, CSF, toxicology screen, EEG may be required if the underlying cause is not obvious.
- Avoid the IM route for diazepam or phenytoin in the treatment of epilepsy - absorption is erratic and unpredictable.
- The most straightforward way of managing status is with one antiepileptic drug (in addition to diazepam) rather than using multiple drugs. Ensure full dosage with adequate blood concentration before discounting a drug as ineffective.

17.5.6 Four Principles of Treatment

Each should proceed simultaneously:

- Stop the seizures.
- Prevent recurrence of seizures.
- Identify the precipitating cause and treat it.
- Identify complications and treat them.

17.6 Epilepsy: Patients Presenting with their First Seizure

17.6.1 Diagnosis

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

17.6.2 Investigations

- CBC + Diff, ESR.
- Glucose, Na, Ca.
- Cranial imaging by MRI is required if there is a history suggestive of focal onset of the seizure. 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 and it may give a clue to the type of epilepsy (e.g., distinguishes the "absence" of temporal lobe epilepsy from that of petit mal).

17.6.3 Treatment

- Most patients who have recovered and are well following a single seizure, do not require hospital admission. All will require Neurology Outpatient assessment.
- If delay in return to normal mental status or if the patient is unwell, consider urgent neurological admission. If non-convulsive status is a possibility, undertake urgent EEG.
- If the seizure occurs as the result of a focal structural lesion in the cerebral hemisphere, commence anticonvulsants after the first seizure. Otherwise, generally do not commence anti-epileptic therapy following a single seizure.
- Sodium valproate, carbamazepine, phenytoin are the drugs of first choice for tonic-clonic or for partial (focal) seizures.

17.6.4 Driving

- **All** patients must be advised that the LTSA driving stand-down requirement is twelve months after any confirmed or **suspected** seizure. Document in the notes that this advice has been given. Patients should also be advised of potential risk of swimming alone, working at heights and other high risk activities.

17.7 Raised Intracranial Pressure

17.7.1 Clinical Features

These include lateralising (focal) features, focal seizures, drowsiness, or papilloedema.

17.7.2 Causes

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

17.7.3 Investigations

- CT or MRI head scan is mandatory to establish the cause.
- Remember that many patients with raised intracranial pressure sufficient to cause death **will not have or will never develop papilloedema**.
- Do not dilate pupils.

17.7.4 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. If necessary, intubation - 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 intracranial pressure include the following - lateralising (focal) features, focal seizures, drowsiness or papilloedema. When bacterial meningitis is strongly suspected, but features consistent with raised intracranial pressure are present, it may be appropriate to administer intravenous antibiotics immediately. Then arrange for urgent CT scan. If the CT shows no mass lesion or any evidence of raised intracranial pressure, perform a lumbar puncture for CSF examination. Refer to Meningitis - Management (see page 126) and Lumbar Puncture (see page 52).*

17.8 Encephalitis

17.8.1 Clinical Features

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

17.8.2 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, predominantly white matter changes.
- **Non-Viral:** Bacterial endocarditis, TB, syphilis, listeria, cat scratch, malaria, nocardia (with or without abscess), toxoplasmosis.

17.8.3 Investigations

Important differential diagnoses include meningitis, severe sepsis, cerebral neoplasia, SLE, toxic metabolic encephalopathy. (See Stupor and Coma on page 162.)

- MRI brain scan - to help establish a diagnosis of either post viral or HSV encephalitis and to exclude other diseases mimicking encephalitis.
- CSF exam (Provided no contraindication on MRI) - 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 of encephalitis. 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.

17.8.4 Treatment

- If HSV suspected treatment is urgent. Give aciclovir 10 mg/kg IV q8h. Consult Infectious Diseases regarding duration of aciclovir 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.

17.9 Spinal Cord Compression

17.9.1 Causes

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

17.9.2 Investigations and Management

- **Remember that quick action may prevent irreversible damage - tetraplegia, tetraparesis, paraplegia, paraparesis.**
- 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 in order to target investigations.
- If recent onset, rapid progression, and/or significant neurological deficit, obtain immediate **(i.e., at once)** neurological/neurosurgical consultation and MRI.
- Catheterise if urinary retention present and record residual volume.
- CBC + Diff, ESR, Na, glucose, K, Ca, creatinine, AST, GGT, alk. phos., bili, albumin, CXR. Serum protein electrophoresis and prostate specific antigen may be indicated. Search for underlying malignancy. Commonest primaries are lung, breast, melanoma, prostate, lymphoma and myeloma.
- 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.
- Corticosteroids e.g., methylprednisolone or dexamethasone should be considered once the diagnosis is confirmed. In particular, diagnosis of abscess or lymphoma must be considered before steroids are given.

17.10 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, with or without, localising signs.
  - Clinical picture of headache, intellectual change, alteration in alertness, and signs of bilateral hemisphere dysfunction.
- It is uncommon for marked 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 deficit e.g., hemiparesis rather than raised intracranial pressure.
- Although some small haematomas with only mild clinical signs can be treated 'medically', all patients must be referred for neurosurgical opinion.

17.10.1 Investigation and Management

- CT/MRI head scan.
- Commence neurologic recordings and consult Neurosurgeon for further advice on management.
- CBC + Diff and coagulation profile.
- Withhold anticoagulants. Administer Vit.K +/- FFP for patients taking warfarin.

17.11 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.

17.11.1 Emergency Management / Resuscitation

Refer to Emergency Management/Resuscitation. (see page 21)

17.11.2 Causes

The causes of coma are:

- Drug overdose: 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, cerebral venous sinus thrombosis.

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

17.11.3 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 22) 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, corneal reflex, 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 more likely to be metabolic rather than structural. 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 unilateral or bilateral III nerve lesions, or mydriatic eye drops, or anticholinergic drugs, or orbital trauma.
  - 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.

- **Corneal Reflex**

Gently touch the cornea with a wisp of cottonwool. Intact blink reflex confirms integrity of cranial nerves V (afferent) and VII (efferent) plus pontine connections. Brushing the eyelashes is an alternative but less potent stimulus if necessary.

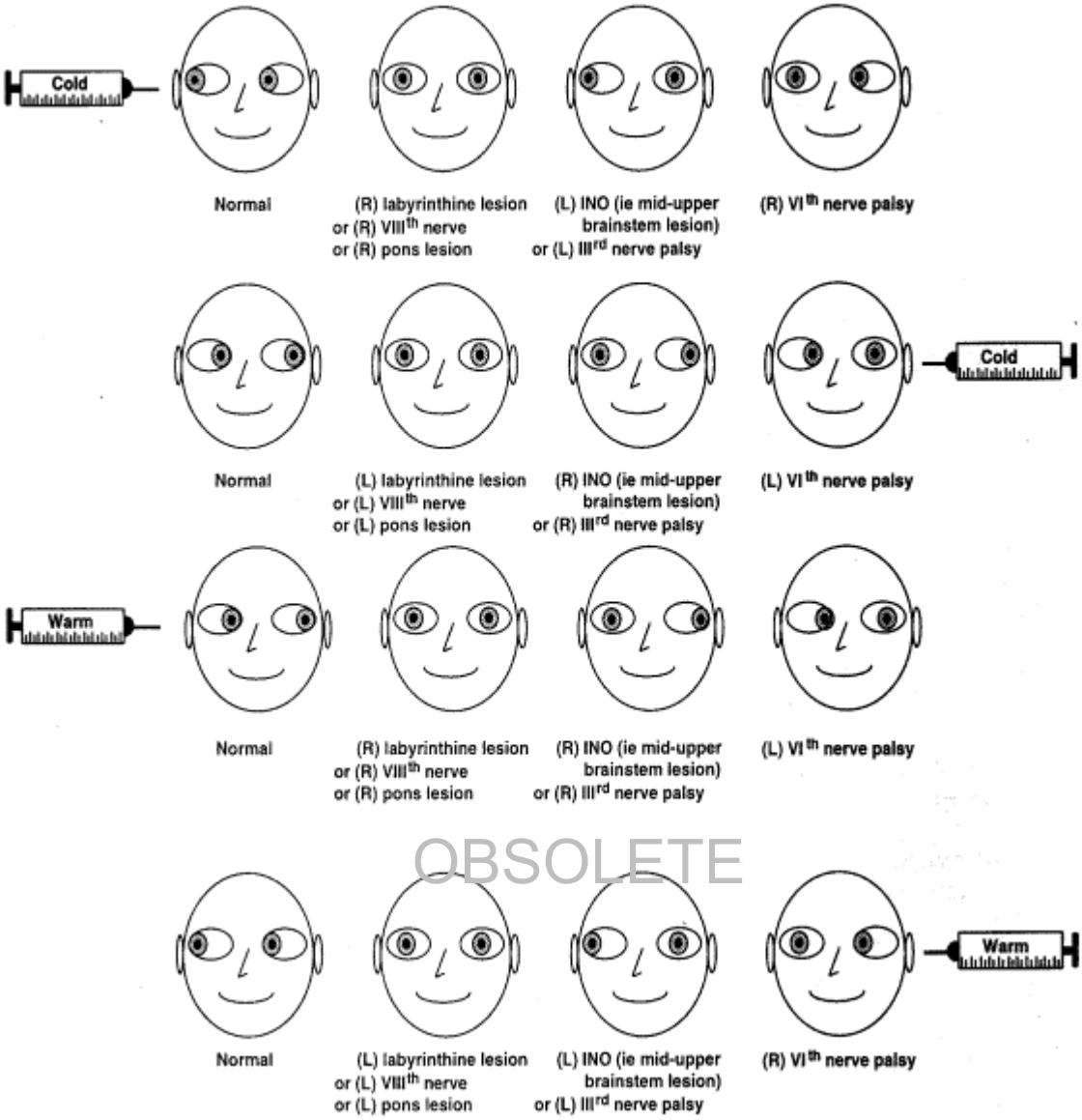
- **Eye Movements**

The oculomotor examination comprises observation of eye deviation, spontaneous eye movements, caloric testing, and oculocephalic reflex (Doll's eye response):

- **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 epileptic supratentorial focus.
- **Caloric Testing**.
  - Caloric testing is easily done by instilling at least 20 mL (usually 50-200 mL is required) of ice cold water 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 20 mL or 50 mL syringe is adequate.
  - The interpretation of caloric testing is depicted in the Caloric Responses in Coma Table (see page 165).
  - 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., labyrinthine or VIIIth nerve) lesion or if cold water testing is inconclusive.
  - If horizontal **nystagmus** is induced the implication is that the patient is **conscious**.
- **Oculocephalic reflex (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 to 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 page 165). 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.*

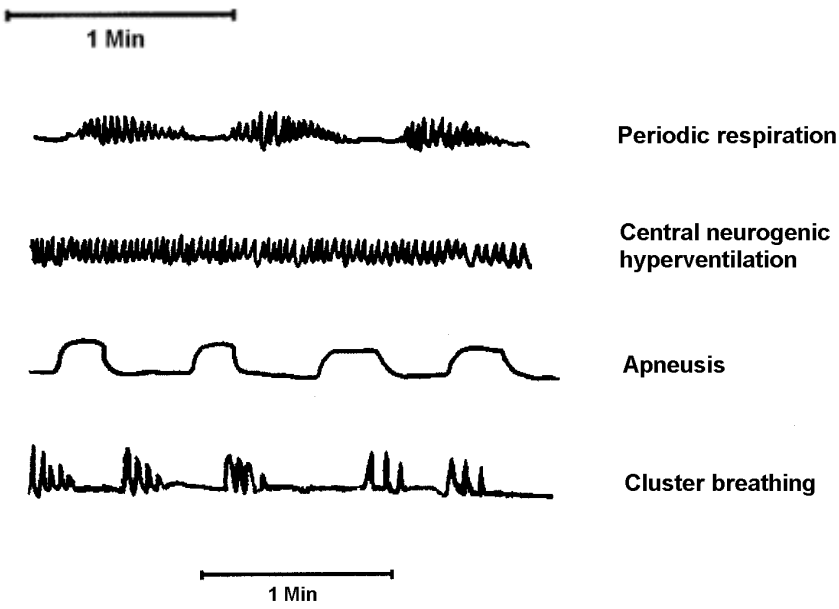
Table 20: Caloric Responses in Coma



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) suggests bilateral cerebral hemisphere dysfunction and if stable, usually implies a relatively good prognosis.
  - **Central neurogenic hyperventilation** (40-70 breaths 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.

Table 21: Respiratory Pattern in Coma



Motor Function

- Observe responses to noxious stimuli applied to nailbeds, sternum or supraorbital ridges. *Normal responses* include withdrawal of limb +/- grimace/groan, and implies intact sensory and motor pathways to and from cortex. Note that adduction/flexion of a limb can occur at spinal reflex level.
- **Abnormal 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 diencephalic 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 myoclonus or generalised seizures raises possibility of metabolic or ischaemic-hypoxic aetiology.

17.11.4 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 (see page 195)).
- CT or MRI scan if structural lesion suspected. MRI if brain stem abnormality suspected.
- If meningitis a possibility give antibiotics, do CT, then lumbar puncture (see page 52) if safe to do so.
- EEG may be considered to identify psychogenic unresponsiveness or partial complex status epilepticus.

17.12 Facial Nerve (VII) Palsy

17.12.1 Common Causes

- Bell's Palsy (idiopathic).
- Herpes zoster (Ramsay Hunt syndrome).
- Middle ear infection.
- Trauma.
- Tumour: **if there has been no recovery of facial nerve function after 3 months a tumour of the temporal bone / parotid must be excluded.**

17.12.2 Clinical Assessment

- Thorough clinical assessment is required.
- Neurological assessment:
  - Confirm that upper and lower facial muscles are involved. Lower facial weakness only is more suggestive of a central nervous system (upper motor neurone) disorder.
  - Complete cranial nerve examination to detect/exclude any other abnormality.
  - Confirm no neurological abnormality in the limbs.
- ENT assessment:
  - Examine for vesicles including pharynx, pinna, ear canal.
  - Otoscopy for middle-ear disorder.
  - Parotid gland examination to exclude clinical evidence of tumour.
  - Examination for cervical and cranial lymphadenopathy.

17.12.3 Bell's Palsy

- Idiopathic - but increasing evidence suggests an association with herpes virus infection.
- The prognosis is usually favourable, however aberrant reinnervation can result in synkinesis (e.g., movement of the mouth when the eye is closed) or 'crocodile tears'.
- A short course of prednisone is usually prescribed e.g., 40 mg daily for 5 days then 20 mg daily for 5 days then stop.
- If presenting within the first 3 days from symptom onset, there is some evidence that combination treatment with prednisone plus aciclovir reduces the incidence of poor outcome compared with prednisone alone: aciclovir 400 mg five times per day for 10 days. Check renal function.
- Bilateral VII palsy: this is **not** "Bell's palsy". Suspect: sarcoidosis, Guillain Barre syndrome, myasthenia, myopathy. Neurological opinion advised.

17.12.4 Ramsay Hunt syndrome

- Herpes zoster infection of the geniculate ganglion or VII nerve.
- Vesicles/scabs may be present on the face, pinna, ear canal, pharynx, upper neck
- Facial palsy is common with worse prognosis than idiopathic (Bell's) palsy.
  - Untreated with complete palsy: 10% complete recovery.
  - Untreated with partial palsy: 68% complete recovery.
- Maximum palsy usually occurs within 1 week but there is evidence that late denervation occurs up to 14 days after the onset of the palsy.
- Other cranial nerves may also be involved: e.g., VIII, IX, X.
  - Check for swallowing impairment - further management may be indicated.
- Treatment: aciclovir 800 mg 5x/day for 7-10 days. Check renal function.
- If facial palsy also treat with prednisone as for Bell's palsy (above).

17.12.5 Progression / failure to improve:

- Patients should be instructed to seek further medical attention and investigation if no improvement occurs within 6-12 weeks or if there is any evidence of involvement of **other** cranial nerves.

18. Obstetrics and Gynaecology

18.1 Obstetrics and Gynaecology Department Information

- Delivery Suite, third floor, ☎ 85711
- Acute Gynaecology Assessment, second floor, ☎ 85805
- Outpatient Department, ground floor, ☎ 85430

On-call Team

- One consultant covering both Obstetrics and Gynaecology, page via operator
- Obstetrics - registrar on pager 5059 at all times
- Obstetrics - house surgeon on pager 5068 at all times
- Gynaecology - Monday to Friday 0800-1600 pager number available via AGA ph 85805
- Gynaecology after hours covered by the Obstetrics house surgeon, registrar, and consultant.

Consultation and On-call Service

In life-threatening situations, contact the on-call consultant directly, via the telephone operator.

Non-urgent consultation requiring inpatient review can be with the acute team of the day as above. The registrar should be contacted in the first instance. If it is difficult to access the registrar, contact the on-call consultant.

Non-urgent consultation for outpatient review can be faxed directly to the outpatient department (fax 85423). It is recommended the case be discussed with the on-call team as well, to facilitate the timing of investigations.

Obstetric Medicine

If specialist medical input is required for pregnant patients, please contact an Obstetric physician via the Christchurch Hospital operator:

- Dr David Cole
- Dr Ruth Hughes
- Dr Peter Moore

18.2 Vaginal Bleeding or Abdominal/Pelvic Pain During Pregnancy

Vaginal bleeding or abdominal/pelvic pain in pregnancy should always be referred to the appropriate obstetric or gynaecological service as below:

- Gestation < 22 weeks: gynaecology
- Gestation > 22 weeks: obstetrics

18.3 Menorrhagia

Heavy bleeding per vaginum with negative beta-HCG.

Aetiology

- Dysfunctional uterine bleeding.
- Bleeding secondary to trauma (vaginal laceration/ blunt force trauma). Consider sexual abuse and ask about this. If confirmed, refer to DSAC (Doctors for Sexual Abuse Care) for advice before examination unless clinical condition renders this unsafe.

- AV malformation.
- Coagulopathy.
- Neoplasia - cervix, endometrial, vaginal.
- Post menopausal bleeding.

Investigations

- Beta-HCG - urine test sufficient.
- Speculum examination and check cervical smear status.
- Cervical and vaginal swabs (Chlamydia, Neisseria, high vaginal swab for vaginitis) if relevant.
- CBC + Diff and group and hold.
- Ultrasound scan.
- Screen for coagulopathy if history suggests this.

Management

- Local causes:
  - Polyp: remove and send histology.
  - Infection: treat with broad spectrum antibiotics.
  - Heavy bleeding: apply pressure with vaginal pack, foley catheter balloon.
- IV fluid replacement.
- If dysfunctional uterine bleeding:
  - Tranexamic acid - 1 g PO QID.
  - High dose progestogens (e.g., Norethisterone 10 mg PO hrly x 5).
- Call Gynaecology registrar.

18.4 Genital Tract Infection

	Diagnosis	Treatment	Contact trace
Candida	High vaginal swab Microscopy & culture ID on Cervical smear	Treat if symptomatic Intravaginal azole (e.g., Clotrimazole 500 mg stat)	If partner symptomatic
Bacterial vaginosis	High vaginal swab Gram stain clue cells PH > 4.5	Treat if symptomatic or before gynaecology surgery or pregnant	Nil
Trichomonas	Microscopy Culture Identify on cervical smear	Metronidazole 2 g stat Tinidazole 2 g stat	Treat partner Screen for other STI
Chlamydia	Positive EIA (enzyme linked immunoassay) from endocervical swab First void urine	Azithromycin 1 g stat Doxycycline 100 mg bd 7 days Erythromycin 500 mg QID 7 days in pregnancy	Screen and treat partners Screen for other STI

	Diagnosis	Treatment	Contact trace
Gonorrhoea	Endocervical swab Urethral/anal swab Throat swab	Ciprofloxacin 500 mg stat Ceftriaxone 250 mg IM stat if pregnant	Screen and treat partners Screen for other STI
Herpes simplex	Swab fluid from lesion in viral transport media	Aciclovir 200 mg 5x per day for 5 days Catheterise if needed	Screen for other STI
Pelvic Inflammatory Disease	Swabs for all of above Image for abscess - ultrasound or CT	Amoxil/clavulanic acid + doxycycline ± metronidazole or Gentamicin and clindamycin	

18.5 Gestational Proteinuric Hypertension (Pre-eclampsia)

Normally appears beyond 20 weeks gestation. Once present it will progress at a variable rate until the fetus is delivered. Resolution is not immediate after delivery, and severe hypertension or an eclamptic seizure post-partum may be the first presentation. The disorder is usually asymptomatic until at an advanced stage, at which time the patient may complain of headache, visual disturbance, or epigastric pain.

Clinical signs

- Hypertension.
- Proteinuria.
- Oedema.
- Hyperreflexia/clonus.
- Epigastric tenderness.
- Seizures.
- Placental abruption.

Investigations

- CBC + Diff, Na, K, creatinine, urea, urate, albumin, bili, alk. Phos., AST, GGT, ALT, LDH, coagulation profile, and urinary protein/ creatinine ratio.
- Group and hold if delivery imminent.
- Ultrasound scan of fetus including doppler studies.
- Cardiotocograph (CTG) if >24 weeks gestation.

Management

- **Call obstetric registrar**.
- If BP ≥ 170 mmHg systolic or 110 mmHg diastolic, institute antihypertensive therapy:
  - Loading dose of methyldopa 1 gm and then 250-500 mg 6 hourly
- **If urgent reduction in BP necessary, the options are**:
  - Labetolol 20 mg IV bolus repeated as required or followed by a continuous infusion (avoid in patients with a history of asthma), **or**
  - Nifedipine 10 mg orally and repeated as required, **or**
  - Hydrallazine 5 mg IV repeated as required (beware of precipitating hypotension).

Whichever option is used, the fetus needs to be monitored by CTG if the blood pressure is being acutely lowered.

- If at high risk of eclampsia (indicators include severe hypertension, headache, epigastric pain, and hyperreflexia with clonus) start seizure prophylaxis with magnesium sulphate (protocol available on delivery suite - **seek specialist advice**).

18.6 Ovarian Hyperstimulation Syndrome

Aetiology

Follows IVF or ovulation induction.

Assessment

Examine woman for signs of:

- Hypovolaemia.
- Third space fluid redistribution (ascites, pleural effusion).
- Thromboembolism.

Investigations

- CBC + Diff, Na, K, urea, creatinine, alb, bili, alk. phos., AST, ALT, GGT, coagulation profile, blood gases if tachypnoeic.
- Ultrasound scan.

Management

- Rehydration with crystalloid and colloid.
- Strict fluid balance.
- Analgesia.
- Further management should be dictated by the oncall Gynaecologist.

OBSOLETE

19. Older Persons Health

19.1 Older Persons Health Department Information

Older Persons Health Service is based at the Princess Margaret Hospital. It is responsible primarily for the assessment, treatment, and rehabilitation of elderly people with physical and mental health problems and associated disabilities. There is a strong emphasis on a patient-focussed multi-disciplinary approach that is provided in the location most appropriate to the patient, be it as an inpatient, outpatient, or in the person's normal residence.

Consultant Physicians

- Dr J Elliot, Dr V Fletcher, Dr B Franks, Dr N Gilchrist, Dr C Hanger, Dr J Kidd, Dr S Lynn, Dr N Millar, Prof R Sainsbury, Dr A Sidwell, Dr J Thwaites, Dr T Wilkinson

Consultant Psychiatrists

- Dr C Collins, Dr M Croucher, Dr B Deavoll, Dr K Fox, Dr J Kirwan, Dr C Peebles

Consultation and On-call Service

- There is a duty physician on call at all times for the Older Persons Health department, contactable through the Princess Margaret Hospital switchboard. Every clinical area in the CDHB has a nominated Older Persons Health physician. Do not hesitate to use this person's expertise. Please phone the consultant physician directly or fax referrals to the Admissions Coordinator on 66914.
- There is a psychiatrist available Monday - Friday via the PSE (Psychiatric Services for the Elderly) Community Team. At the weekends, a psychiatric nurse is available. After-hours assistance is available through the Psychiatric Emergency Service. PSE referrals, including Delirium Team and Consultation-Liaison referrals, should be made as follows:
  - 0830-1630 weekdays: ☎ 66997, fax 66998 (PSE Community Team).
  - 0830-1630 weekends: ☎ 337 7899 and ask for the PSE Duty Psychiatric Nurse).
  - All other times: ☎ 364 0640 and ask for the Psychiatric Emergency Service.

Consultation Guidelines

Refer to an Older Persons Health physician or psychiatrist for:

- Specialist medical or psychiatric opinion about an elderly patient.
- Assessment for ongoing management by Older Persons Health Service.
- Assessment for hospital-level care or dementia-care facility.

Points to Remember

- The best value is obtained by referring for a consultative service rather than a "takeaway" service, and by referring early.
- Rehabilitation and discharge planning can occur in any hospital setting, and should never be put on hold pending review by an Older Persons Health representative.

Philosophy

As a person ages, there is often a decline in the resources that keep them healthy and independent. These may be internal (e.g., physical health and cognitive functioning) and external (e.g., dwindling social networks and negative attitudes towards ageing). Older Persons Health Service specialises in the recognition and management of these issues, both before and as they arise, to maximise the health and independence of elderly people.

19.1.1 Attitudes

Elderly patients make up 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 competent and confident when dealing with the elderly.

Poor staff attitudes to older people can adversely influence the standard of care they receive. It is important that older people are not considered an imposition or an inappropriate admission. In particular, labels such as “social admissions” 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. Terms such as "acopia" must never be used; the term "threatened independence" is much more useful for describing when an older person is having difficulties maintaining their normal level of functioning in the community.

Try not to be over-familiar with elderly patients. For example, avoid calling them by their first names unless invited to do so. Treat them respectfully and handle them gently when performing the physical examination.

19.2 History and Examination

- The case history should follow the normal format. In cases where the patient is unable to give the required information, **collateral history** from family, friends, carers, GP, neighbours, etc. can be invaluable.
- **Social History** should not be limited to smoking and alcohol use. Of equal or greater importance in the elderly patient is to know their circumstances prior to admission. Ask about:
  - Place of domicile.
  - Usual (premorbid) level of functioning, including ability to perform Activities of Daily Living (ADLs) such as personal care and mobility.
  - Use of aids such as walking aids and hearing aids.
  - What support is provided and by whom.
  - How carers (usually family members) are coping. Remember that "carer stress" is becoming increasingly recognised.
- **Medications**: for many reasons, older persons are at greater risk of being harmed by medication than any other group. It is therefore crucial to ensure that you have an accurate record of your patient's drug regime; this may necessitate checking with their GP or pharmacist. Review and rationalisation of an elderly person's medications should take place at each admission, particularly with regard to dose adjustment, potential interactions, and side effects. Always ask if they are using over-the-counter (OTC) medications, particularly eye drops, laxatives, and hypnotics.
- **Systems review**: as well as the standard systems examination, the following checklist should be completed:
  - Bladder and bowels: Ask about urgency, incontinence, use of continence aids, prostatic symptoms, altered bowel habit, constipation.
  - Eyes and ears: Ask about problems with vision and hearing. Does the person wear spectacles or use a hearing aid? Does the hearing aid work (suspect battery failure if not) and can the patient use it? Have spectacles and hearing aid been brought into hospital?
  - Mouth and nutrition: enquire about dentures and whether they fit. Has there been recent weight loss? Are there obstacles to good nutritional intake, e.g., swallowing problems, availability of food, excessive alcohol intake?
  - Postural stability: have there been any recent falls?
  - Cognition: are there memory problems? Ask about unpaid bills, leaving the oven or fire on, burned cooking and other accidents in the home, and getting lost outside.
- **Examination**: in addition to the standard examination, pay particular attention to the following:
  - Visual acuity.
  - Impacted wax in ears.

- Evidence of poor nutrition, e.g., evidence of weight loss, angular stomatitis.
- Evidence of poorly fitting dentures.
- Cognitive function. Perform MMSE if cognitive impairment is suspected, and consult CAM (see page 207) to help decide whether or not delirium is a likely cause.
- Is the bladder palpable?
- Rectal examination for prostatic disease and constipation.
- Is vaginal examination required?
- Rectal temperature if peripheral temperature is low.
- Joints. Look for arthritis and changes of gout.
- Feet. Look for lesions such as corns, uncut nails etc., requiring attention. Is there evidence of impaired vascular supply or peripheral neuropathy? Is footwear safe?
- Gait. Is there instability? Look for signs of pain and neurological or joint disease. Are walking aids appropriate?

- **Summary**
  - List the problems, starting with those that are in most urgent need of attention.
  - Remember that multiple morbidities often co-exist in elderly people, and that the interaction between these may be contributing to the patient's presentation.

19.3 Altered Presentation

Altered or abnormal presentation is the rule rather than the exception in the elderly. Falls, delirium (acute confusional state), lost or threatened independence, and reduced mobility ("gone off legs") are common non-specific presentations. These patients need meticulous examination and work up as there is almost always an underlying medical condition that has contributed to their decompensation.

Beware painless myocardial infarction and sepsis with normal temperature.

Always consider medication as a cause or contributor of the acute presentation.

19.4 Management of the Confused Elderly Patient

Accurate diagnosis is the key to management. It is essential to find out the duration of the patient's confusion and distinguish between acute confusional state (delirium) (see page 206) and chronic cognitive impairment (dementia). Collateral history from a family member or carer is invaluable.

*Note: these patients are at **very high risk**. Delirium affects 25% of elderly patients admitted to hospital and is associated with a 1 month mortality of 33%. Remember, too, that cognitive impairment is often missed by medical staff.*

- Remember the predisposing factors for delirium, including advanced age, pre-existing dementia, sensory impairment, and Parkinson's disease.
- Use the MSQ (see page 146) routinely, the MMSE if cognitive impairment is suspected, and the CAM Screen (see page 207) to help identify delirium.
- Try to make an accurate diagnosis.
- Treat any underlying cause (infection, dehydration, faecal impaction, etc).
- Consider stopping or reducing medication that may be contributing.
- Consider benzodiazepine and alcohol withdrawal as potential precipitants.
- Use small regular doses of oral medication such as haloperidol 0.5 - 1 mg on a regular basis. Titrate up or down according to the response and withdraw as soon as possible.
- Avoid using intramuscular injections.
- Avoid using sedation unless absolutely required.
- Avoid using “cocktails” of several drugs.

- Aid orientation by providing visual and verbal cues - attend to spectacles and hearing aid if necessary, helpful communication and reassurance from staff and friends/carers, adapt surroundings, e.g., clock, familiar photographs, and other objects from home.
- Avoid using bed rails. Consider nursing on a mattress near to or on the floor if there is a risk of falling.
- Use a soft night-light.
- Minimise changes of staff members. Using relatives to stay with the patient may be helpful.

19.4.1 Continence Problems

Urinary Incontinence

- Attempt to ascertain whether the patient has stress or urge incontinence.
- Consider medication as possible contributors.
- Check MSU.
- Perform abdominal, rectal, and vaginal examinations to exclude faecal impaction, prostatism, urinary retention, atrophic vaginitis, etc.
- Consider measuring bladder residual volume by ultrasound scan.
- Use an incontinence chart to identify any problem times or pattern of the incontinence.
- A trial of an anticholinergic (e.g., oxybutynin) may be worthwhile if uninhibited neuropathic bladder is suspected and no contraindications exist. Remember that cognitive impairment may be worsened by anticholinergics.
- Referral for urodynamic studies may be required in a small number of cases.

Faecal Incontinence

- Faecal impaction with overflow is the leading cause in older people.
- Perform abdominal and rectal examinations (± abdominal x-ray) to exclude faecal impaction, painful rectal and anal conditions.
- Check the medication list for contributors, e.g., opioids, aperients.
- Consider the use of bulking agents.
- Do not use constipating agents until you are certain that high faecal impaction is excluded.
- Use commonsense measures such as encouraging the patient to use the toilet after a meal.

19.4.2 Loss of Functional Abilities/Deconditioning

Older people can lose function/abilities as a result of their acute illness or the treatment they are given or simply by being in hospital. It is important to optimise their recovery by combining medical treatment with measures to maintain independence as much as possible. Specific rehabilitation may also be required. Ensure that the older person has their normal aids (e.g., spectacles, hearing aid, walking aid, comfortable shoes) and gets dressed in day clothes (where appropriate) to facilitate recovery of function.

20. Oncology

20.1 Oncology Department Information

Main Office

- Oncology Building, ☎ 80020, Fax 80759
- Assoc. Prof. Chris Atkinson, ☎ 80366
- Dr Al Abdelaal, ☎ 80179
- Dr Scott Babington, ☎ 80363
- Dr Bernie Fitzharris, ☎ 80207
- Dr David Gibbs, ☎ 81291
- Dr Mark Jeffery, ☎ 80417
- Assoc. Prof. Bridget Robinson, ☎ 80361
- Dr Iain Ward, ☎ 80020
- Dr Chris Wynne, ☎ 89733

Consultation and On-call Service

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

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.

The Christchurch Hospital Palliative Care Service is located within Oncology.

- Main Office (voice mail), ☎ 81473
- Fax for referrals, 80759
- Dr Kate Grundy, Clinical Director, ☎ 89611
- Anne Morgan, Specialist Nurse, ☎ 81885
- Willem Vink, Specialist Nurse, ☎ 81473

Palliative Care is a **consultation** service, and patients are **not** admitted under Palliative Care unless by arrangement with the Clinical Director. For guidelines for referring patients to Palliative Care, please refer to the Christchurch Hospital Palliative Care Service Clinical Guidelines (Ed. 2003), Chapter 17. A copy can be obtained by calling ☎ 81473.

20.2 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. All require specialist consultation for staging and treatment.
- Please do not wait until all investigations or histology reports are complete. Potentially curable cancers include:
  - Testicular cancer.
  - Germ cell tumours - ovary, extragonadal, retroperitoneal and mediastinal.
  - Gestational trophoblastic disease.
  - Undifferentiated cancers, especially in younger patients.
  - Any cancer in children or teenagers.

- Osteosarcoma, Ewing's sarcoma and other sarcomas.
- Leukaemias.
- Lymphomas - Hodgkins and non-Hodgkins.
- Early stage head and neck cancer, cervical cancer and prostate cancer.

20.3 Lymph Node or Other Tissue Biopsies

Before biopsies are organised, consider whether any additional tests will be needed on the material other than routine diagnostic histology. Contact on-call Oncologist or Haematologist before biopsies are performed. 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.

20.4 Sarcoma Biopsies

Sarcomas are rare but should be considered in patients who present with a mass, particularly in:

- Children and young adults
- Any patient with a mass >5cm in diameter
- Any patient where the mass appears to be fixed to other structures
- Any mass that has grown rapidly or is painful

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 essential to discuss the patient with the Sarcoma Clinic (Ext. 80023, or contact the Oncologist on-call after hours) before biopsy to avoid prejudicing future surgery or radiation options.

20.5 Spinal Cord Compression

- Consider in all patients with cancer and back pain, especially if accompanied by resistance to analgesia, sensory loss, alteration in bladder or bowel function, limb weakness or lack of co-ordination.
- If cord compression is suspected, give dexamethasone 16 mg stat PO or IV and cover with omeprazole (prior to investigation) and refer **immediately** to on-call oncologist.

20.6 Superior Vena Caval Obstruction

- Consider Superior Vena Caval Obstruction (SVCO) in patients presenting with dyspnoea or "heart failure" (raised JVP).
- If SVCO is suspected, give dexamethasone 16 mg stat PO or IV and cover with omeprazole and refer **immediately** to on-call oncologist.

20.7 Neutropenic Sepsis

- Consider in patients who have had radiation or chemotherapy within the past two months.
- May present with patient feeling non-specifically unwell. Common signs include fever, tachycardia, and postural hypotension.
- If suspected, start empirical antibiotics after cultures are taken, **before** doing other investigations.
- First line antibiotic therapy for the treatment of neutropenic sepsis is:
  - **Cefepime** 2 g IV q12h and **Gentamicin** 5 mg/kg IV q24h

  or, if the above is unsuitable for a particular patient,
  - **Imipenem** 500 mg IV q6h.

20.8 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 Endocrinology (see page 108) 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.

20.9 Management of Pain in Cancer Patients and Palliative Care Patients

Refer to Cancer Associated Chronic Pain (see page 191) and the Christchurch Hospital Palliative Care Service Clinical Guidelines, Chapter 1 and Chapter 2.

20.10 Management of Nausea and Vomiting

Hypercalcaemia, electrolyte imbalance, opioid 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. Recent onset of renal impairment will cause or exacerbate morphine-related nausea due to retention of toxic morphine metabolites - dose reduction may be needed. Give intravenous fluids if dehydrated. Use specific treatment if cause identified e.g. dexamethasone for cerebral metastases, hydration for hypercalcaemia.

20.10.1 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 8 hourly for 24 hours;
    ± Dexamethasone 8-16 mg IV [in 100 ml normal saline];
- For persisting emesis, more than 24 hours after chemotherapy:
  - Dexamethasone PO 8 mg BD for 2 days, then 4 mg BD for 2 days

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

20.10.2 Other Antiemetics for Oncology and Palliative Care Patients

- Metoclopramide

  10 mg PO, IV, or SC 4-6 hourly (commonly given QID before food)
- Haloperidol

  0.5-3 mg PO, SC or IV 6-12 hourly, or single nocte dose (max 5 mg/day)
- Cyclizine

  25-50 mg PO or slow IV BD or TDS (max 150 mg/day)

- Prochlorperazine

  5-10 mg PO 6 hourly or

  25 mg PR 8 hourly
- Dexamethasone

  2-4 mg PO daily
- Lorazepam

  0.5-1 mg PO 6-8 hourly

Note:

- Avoid metoclopramide in bowel obstruction; haloperidol or cyclizine are preferred as they can be given subcutaneously.
- Combinations of 2 or 3 agents may be more successful than a single agent.
- Haloperidol is very helpful for opioid-related nausea.
- Dexamethasone can be useful for liver metastases.
- Subcutaneous injections of metoclopramide and haloperidol can be effective given regularly or in varying combinations in a continuous infusion. Refer to Christchurch Hospital Palliative Care Service Clinical Guidelines, Chapter 16. Appendix A of these Guidelines details drug compatibilities for continuous subcutaneous infusions (note - a maximum of 3 drugs can be mixed together in an infusion).
- Methotrimeprazine (Nozinan) is a broad-spectrum antiemetic and can be very effective in advanced disease. Refer to the Christchurch Hospital Palliative Care Service Clinical Guidelines, Chapter 3, or refer to the Palliative Care service.
- Domperidone is an alternative prokinetic agent (oral only) that can be used if dystonic reactions or other side-effects are encountered with metoclopramide.
- Cyclizine has a significant part-charge in the community - a special authority number is usually applicable. Forms are available from Pharmacy.
- If nausea and vomiting remain a problem, consult Oncology or the Palliative Care Service ☎ 81473 for further advice.

21. Ophthalmology

21.1 Ophthalmology Department Information

Main Office

- Ground Floor, Pathology Building, 19 St Asaph Street

Inpatient Services

Inpatient teams are by consultant and are associated with Ward 32. The consultants are:

- Dr Jim Borthwick
- Associate Professor Mark Elder
- Dr Sean Every
- Dr Ainsley Morris
- Dr Allan Simpson
- Dr K Tarr
- Dr R Weatherhead

Consultation and On-call Service

24 hours a day, 7 days a week. Contact the Ophthalmology Registrar or Consultant on call through the Operator on 364 0640. There is a specific on-call pager which is handed from registrar to registrar.

Outpatient Consultations

Outpatient consultations are achieved either by ringing the on-call registrar or by faxing a referral to 364 1479.

Other Services

Ophthalmology offers a comprehensive diabetic screening programme and this can be accessed by faxing the clinical details to the above numbers. The Department also undertakes visual field tests including Humphrey and Goldmann visual fields, fluorescein angiography, and retinal photography. The Department has a counselling service for patients having ophthalmic-related issues.

Referral Guidelines

All referrals require a visual acuity. If the visual acuity is not normal, then the test must also be repeated with a pinhole. This is mandatory for all referrals with the rare exception of the unconscious patient and the pre-verbal child. Please specify the preferred time-frame for the consultation.

21.2 Clinical Conditions

Many conditions may require ophthalmology assessment. These include - acute red eye, acute visual loss, chronic visual loss, ophthalmic pain, diplopia, problems of eyelid position, eyelid lesions, trauma (including chemical burns), infection including intraocular pre-septal and orbital cellulitis and conjunctivitis.

Common Causes for Decreased Vision

- **Sudden painless:**
  - Retinal artery occlusion (pale retina), retinal vein occlusion, ischaemic optic neuropathy (inc. giant cell arteritis), vitreous haemorrhage, retinal detachment.
- **Painful:**
  - Acute angle closure glaucoma, uveitis, optic neuritis (pain on eye movement).
- **Gradual painless:**
  - Cataract, age-related macular degeneration, diabetic retinopathy, open angle glaucoma, refractive error.
- **Transient:**
  - Amaurosis fugax, vertebrobasilar artery insufficiency, migraine, impending central retinal vein occlusion, giant cell arteritis.

Common Causes for Red Eye

Differential diagnoses:

- **Eyelids:**
  - Blepharitis, trichiasis, foreign bodies.
- **Conjunctiva:**
  - Conjunctivitis, subconjunctival haemorrhage, inflamed pterygium.
- **Sclera/episclera:**
  - Episcleritis, scleritis.
- **Cornea:**
  - Corneal ulcer, foreign bodies.
- **Anterior chamber:**
  - Iritis.

21.3 Management

- **Penetrating eye injuries** should not have any topical medication applied to them, and they should not have an eye pad applied, but simply have a shield installed over the eye. The patient must be kept nil by mouth and the on-call registrar notified immediately.
- **Acute red eyes**, where there is unexplainable loss of vision or severe pain, need referring acutely by phone to the on-call registrar.
- Eye pain unresolved by paracetamol and in particular associated with nausea or vomiting requires urgent referral.
- Any post-operative ophthalmic patient whose pain is not relieved by paracetamol requires that the on-call registrar see the patient.
- If the visual loss is less than 12 hours we would consider treating with hyperbaric oxygen so please phone the on-call registrar for advice.
- Always consider temporal arteritis as a common cause of acute visual loss, especially as the other eye is at risk. The Ophthalmology Department offers a biopsy service. Starting steroids does not alter the biopsy findings in the first week. Refer to the section on giant cell arteritis (see page 246) for more details.

22. Orthopaedic Medicine

22.1 Orthopaedic Medicine Department Information

Main Office

- Older Persons Health, The Princess Margaret Hospital.

Inpatient Services

- Ward 18 and 19, Christchurch Hospital.
- Orthopaedic Rehabilitation Unit

Staff

- Dr B Franks
- Dr N Gilchrist
- Dr S Lynn
- Professor R Sainsbury
- Dr A Sidwell
- Dr J Thwaites

Consultation and On-Call Service

- Consultants can be contacted through the switchboard at TPMH or CPH. The Ortho Medical Registrar can be contacted through TPMH (☎ 66899).

Consultation Guidelines

- Background: Shared care between Orthopaedic Surgeons and Orthopaedic Medicine Specialists has been highly successful in decreasing mortality, morbidity, and length of stay in elderly patients with fractures. An Orthopaedic Medicine Specialist or Registrar does daily ward rounds in Wards 18 and 19 and will also see specific patients upon request from Monday to Friday. Out of hours consultations are normally handled by the appropriate acute medical or surgical specialty. This also applies over the weekend.

22.2 Identification, Treatment, and Management

At risk patients

The patients who are most at risk of complications are elderly males, patients with dementia, institutionalised patients, insulin and non-insulin dependent diabetics, and patients who are underweight. All of these patients are at increased risk of cardio respiratory complications as well as infection.

Fall aetiology

A detailed history must be obtained to ascertain whether the fall was mechanical in nature or whether there was a secondary cause such as arrhythmia, silent MI, postural hypotension, drug effect, neurological event.

Pre-morbid function

It is important to record pre-morbid level of functioning as this will provide important information for rehabilitation and discharge planning.

Drugs and Hip Fractures

Many drugs are recognised as having an association with hip fractures including benzodiazepines, tricyclic antidepressants, SSRIs, antipsychotics, and polypharmacy. Please discuss with the Ortho medical registrar or consultant or seek advice from Psychiatric Services for the Elderly (PSE) as to how these drug regimens might be optimised.

Thromboprophylaxis

Deep vein thrombosis occurs commonly in patients with fractures of the lower limbs:

- All patients should receive low molecular weight heparin unless contraindicated.
- Aspirin may be an alternative.
- All patients should have TED stockings unless contraindicated.

Antibiotic Prophylaxis for Fracture Surgery

- Reduces deep and superficial wound infections.
- All patients undergoing surgery for fracture fixation should receive antibiotic prophylaxis perioperatively.

Cardio-respiratory Problems

Where there is concern or if a patient is unstable from cardio-respiratory problems, early consultation should be made to the cardiology or respiratory services. Early anaesthetic consultation must be made if the patient is awaiting surgery.

Nutritional Management

Oral protein supplementation is beneficial in reducing minor post-operative complications, preserving body protein stores, and decreasing length of stay. All patients should receive protein supplementation. For further information, refer to the guidelines for Nutrition Support (see page 46).

Analgesia

Refer to the guidelines on analgesia on page 187. Please note regular low dose analgesia should be used rather than PRN analgesia. Avoid Tramadol as first line analgesia due to its side effect profile and its non-funded status.

Constipation

Constipation is very common in these patients. Please ensure early and optimal use of laxatives as outlined in Constipation (see page 117). Bowel washout is relatively contraindicated in elderly patients. It should be used with caution and only after other measures have been taken.

Management of Patients on Anticoagulants

Refer to the Thrombosis section for management of patients on warfarin therapy undergoing surgery (see page 255).

Delirium

Delirium occurs in up to 2/3 of older patients with hip fracture and can last up to several months. It carries an adverse prognosis with increased length of stay, mortality, and institutionalisation. It is more common in patients with pre-existing dementia and memory loss. Secondary causes of delirium must be excluded i.e. alcohol and drug withdrawal, infection, analgesia, hypoxia.

Refer to the guidelines for management of delirium (see page 206). The delirium service is available (contact through the operator at TPMH) for consultation and advice.

Rehabilitation / Discharge Planning

If the patient is very independent, discharge home directly from Christchurch Hospital may be possible. However most elderly people will require a period of rehabilitation following an orthopaedic injury. If they are medically and surgically stable, then transfer to the Orthopaedic Rehabilitation Unit is recommended. Those with ongoing medical problems are best rehabilitated on the medical wards at TPMH.

Osteoporosis Treatment

Please use the orthopaedic osteoporosis protocol for all patients with osteoporotic fractures that enter the Orthopaedic Wards. For further information, refer to the guidelines for management of osteoporosis (see page 185).

23. Osteoporosis

23.1 Bone Clinic

Main Office

- PMH outpatients, ☎ 66949, fax 66842.

Consultation service, outpatient clinic service, arranging bone density scans

- Dr John Elliot, Dr Anna Fenton, Dr Nigel Gilchrist, Dr Penny Hunt, Dr Steven Soule

Consider clinic referral: very low bone mineral density (BMD), intolerance/poor response to therapy for osteoporosis, unusual conditions associated with low BMD, Paget's disease, metabolic bone disease, multiple fractures.

23.2 Osteoporosis

A condition of reduced bone mass and strength resulting in fractures. The most important consideration is an individual's absolute risk of fracture.

- **Major risk factors for osteoporosis**:
  - Prior fracture. Vertebral fracture is associated with 5-10 times the risk of future fracture. 10-20% will refracture over the next year. Peripheral fractures double the risk of future fracture.
  - Age - 4% of 50 yr olds have osteoporosis compared to 33% of 70 yr olds.
  - Steroid usage - the higher the cumulative dose, the greater the risk of fracture. Over 7.5 mg prednisone /day is associated with 5 times the risk of fracture).
- **Other risk factors**:
  - Maternal hip fracture, weight less than 57 kg, smoking, proximal muscle weakness.
- **Conditions commonly associated with osteoporosis**:
  - Hypogonadism (e.g., premature menopause, anorexia, prostate cancer survivors, prolonged depoprovera), coeliac or thyroid disease, anticonvulsant use, COPD, alcoholism, hyperparathyroidism.

Consider BMD scan, to assess risk of fracture and need for treatment. Results reported as T score (standard deviation score (sds) compared to normal young adult) and Z score (sds score compared to age matched normal control). WHO defines osteoporosis as T score < -2.5, osteopenia T score -1 to -2.5. Degenerative bone disease can give falsely reassuring bone mineral density scores at hip and spine.

Note: *Patients over 75 with a significant osteoporotic fracture demonstrated radiologically do not necessarily require a BMD before treatment.*

Guidelines for treatment based on BMD results:

- **T score < -2.5**: treat.
- **T score -1 to -2.5**: treat if fracture, otherwise correct risk factors.
- **T score > -1**: don't treat.
- **If on supraphysiological steroid therapy, treat if T score < -1.5**.

23.2.1 Investigations

All patients should have Ca, $P0_4$, albumin, ALT, creatinine, and CBC + Diff. If BMD for age is low (i.e., Z < -2), consider secondary causes of osteoporosis. Possible tests include: Vit D, PTH, Testosterone & SHBG (in males), LH, FSH, coeliac antibodies, TFTs, SPE.

23.2.2 Treatment

Treatment, as well as addressing any underlying cause, involves:

- **Calcium**: by diet or supplement to at least 1000 mg per day.

  Calcium carbonate (Osteo 500) one bd with food or Ca Sandoz one nocte.

- **Vitamin D**: consider loading dose (i.e., calciferol forte 50,000 IU, one daily for 10 days) if likely to be low, for example over 65 yrs, institutional care, anticonvulsant medication. Maintenance dose (recommended for most patients on bisphosphonates): calciferol forte 50,000 IU monthly.

  Calcium and vitamin D probably reduce fractures in frail older persons but usually osteoporosis requires additional treatment.

- **Bisphosphonates**:
  - **Etidronate**: taken cyclically for 2 weeks every 3 months as 400 mg daily (2x 200 mg tablets) with water only on an empty stomach (no food or drink other than water for 2 hours before or after tablets). After 2 week cycle completed, give calcium 500 mg daily for 76 days. Then repeat the 90 day cycle. Associated with 50% reduction in spinal fractures, not peripheral fractures.
  - **Alendronate**: 70 mg once a week 30 mins before breakfast with water, remain upright after taking tablet. More effective than etidronate, 50% reduction in all fractures but **special authority required** (a specialist or vocationally registered GP).
  - **IV bisphosphonates - pamidronate and zoledronate**: These should only be considered for patients who have:
    - Intolerance to oral bisphosphonates with a T score of < -3 and a fracture, or
    - Multiple fractures.

    IV bisphosphonates are used "off label, non experimental", and patients should be informed of this, but a signed consent is not required. Information packs for patients are available through the Bone Clinic. Consult Bone Clinic physicians if necessary.
    - Zoledronate 4 mg in 100 ml normal saline over 15 minutes, once yearly
    - Pamidronate 30 mg in 250 ml normal saline over 1-2 hours, 3 monthly

    Side effects of oral bisphosphonates include nausea, indigestion, abdominal pain, diarrhoea. All bisphosphonates may cause transient mild bone pain, also hypocalcaemia if vitamin D deficient. IV bisphosphonates may also cause transient fever and flu-like symptoms but usually GI tract side effects are not seen.

- **Hormone Replacement Therapy (HRT)**:

  It is important in young hypogonadal females, for example premature menopause, to preserve bone mass. Seek advice re management. In postmenopausal women, risks of vascular disease and breast cancer need to be considered and therefore not recommended. Testosterone replacement should be considered in all hypogonadal males.

- **Consider falls risk:**

  Review medications (anti-hypertensives, hypnotics), safe environment, physiotherapy, hip protectors. Consider referral to "Stay on your feet Canterbury" through GP.

24. Pain Management

24.1 Pain Management Contact Information

Pain Management Centre (Burwood)

- Prof. E. Shipton, Clinical Director - for chronic pain problems, ☎ 99831

Acute Pain Management Service (Christchurch Hospital)

- Richard Craig, Beep 8114

Palliative Care

The Christchurch Hospital Palliative Care Service is located within Oncology.

- Main Office (voice mail), ☎ 81473
- Fax for referrals, 80759
- Dr Kate Grundy, Clinical Director, ☎ 89611
- Anne Morgan, Specialist Nurse, ☎ 81885
- Willem Vink, Specialist Nurse, ☎ 81473

Palliative Care is a **consultation** service, and patients are **not** admitted under Palliative Care unless by arrangement with the Clinical Director. For guidelines for referring patients to Palliative Care, please refer to the Christchurch Hospital Palliative Care Service Clinical Guidelines (Ed. 2003), Chapter 17. A copy can be obtained by calling ☎ 81473.

24.2 Principles

Pain is a symptom which requires thorough evaluation and appropriate management. The aim is to control pain adequately while diagnosis and 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 opioids should be given cautiously to patients with respiratory disease, although this is less of an issue in chronic/persistent pain compared to acute pain. Morphine may cause bronchospasm in addition to respiratory depression.

Note: If the patient is already enrolled on the Methadone programme, follow the guidelines on page 213 since the treatment of these patients is covered by legislation.

24.3 Severe Acute Pain

Opioids are the most potent analgesics and should be used only where there is a diagnosis of severe pain.

- Morphine remains as the gold standard and is generally well tolerated, although nausea can be a problem along with constipation.
- Pethidine is still used occasionally, mainly where there is an intolerance to morphine. It does not have any specific benefit in smooth muscle spasm. It does have a shorter half-life. Pethidine is almost never used in chronic/persistent pain, as there are more effective and less toxic alternatives such as transdermal fentanyl and methadone.
- Tramadol is now used as the next line of management after morphine in acute pain. It causes less constipation and has a quick onset of action. The parenteral dose is the same as the oral dose.
- Fentanyl, a synthetic opioid, can also be used, especially where severe pain is anticipated in certain procedures. Discuss with consultant.
- Local anaesthetic agents can be useful for providing sensory block of specific dermatomes, e.g., femoral nerve block for fractured femur.

A wide range of modalities are 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 Pager 8114 or via the on-call anaesthetist, can advise on the appropriate technique.

Acute episodes of pain also occur in patients receiving opioids for chronic/persistent pain. In these situations, higher doses of breakthrough analgesia may be needed to gain effect compared to opioid-naive patients. During working hours, advice is available from the Palliative Care Service ☎ 81473 (or page via the Operator).

24.3.1 Doctor's Responsibilities

- Initial assessment of patient:
  - Magnitude and cause of pain.
  - Existence of factors that might affect the patient's handling of opioids 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 Table 22 for general guidelines) and chart according to hospital protocols.
- Reassess at regular intervals and adjust prescription accordingly.

24.3.2 Nurse's Responsibilities

- Assess opioid dosing levels at regular intervals.
- Administer opioid 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 opioid. See Table 23: Frequency of Observation during Acute Pain Medication on page 189 and Table 24: Pain and Sedation Scores on page 190.
- Request an urgent medical review if the pain protocol/prescription is not fully effective.

24.3.3 Management of Complications

Manage appropriately any untoward effects (see page 190).

Table 22: Dosage Guidelines for Systemic Opioids in Acute Pain

Drug	Route	Dose For Adults	Notes & Dose Intervals
Morphine	IM SC	0.15 mg/kg/dose ie. 10 mg/dose/70kg	3-6 hourly
	IV over 1 minute	0.03 mg/kg/dose ie. 2 mg/dose/70kg	Can be repeated at 5 min intervals until desired effect achieved and respiration and sedation are satisfactory
	IV infusion	0.02 - 0.04 mg/kg/hr ie. 2-3 mg/hr/70kg	Use 1 mg/mL solution **with** infusion or syringe pump
Tramadol	IM	1 mg/kg/dose	3-6 hourly
Pethidine (when intolerant of morphine)	IM	1.5 mg/kg/dose ie. 100 mg/dose/70 kg	2-3 hourly
	IV over 1 minute	0.3 mg/kg/dose ie. 20 mg/70kg	Can be repeated at 4 min intervals until desired effect achieved and repeated as required
Fentanyl	TD Durogesic patch	Commence with smallest patch as instructed by specialist - size 25, 50, 75, 100 mcg/hr	

Notes:

- The oral and rectal routes are not usually recommended for acute pain.
- Doses for neonates and children vary. Refer to paediatric guidelines.
- Morphine can be given subcutaneously rather than IM or IV, particularly if the patient is already on maintenance morphine for chronic/persistent pain. Morphine is approximately **twice** as potent SC as oral. Doses of **up to** 60 mg morphine sulphate (2 mls of 30 mg/ml) OR 120 mg morphine tartrate (120 mg/1.5 mls) can be given as a SC bolus. This may be appropriate for patients already taking high dose morphine, either orally or via continuous SC infusion (e.g., via a Graseby™ syringe driver). Refer to the Christchurch Hospital Palliative Care Service Clinical Guidelines, Chapter 1 and Chapter 16.

Table 23: Frequency of Observation during Acute Pain Medication

Route	At least
IM	1 hour after each dose
IV	Every 5 minutes for 15 minutes, then hourly for 2 hours
IV 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 24: 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 attempt must be made to rouse the patient)
5 = Worst imaginable pain	

Table 25: Management of Severe Complications

Complication	Management
RESPIRATORY DEPRESSION ▪ Life threatening	▪ stimulate patient ▪ support ventilation and airway - bag and mask ▪ oxygen by mask ▪ stop opioid administration ▪ give naloxone (1)
▪ Non life threatening	▪ stop opioid administration ▪ give oxygen by mask
EXCESSIVE SEDATION (not rousable by verbal stimuli)	▪ oxygen by mask ▪ stop opioid administration ▪ nurse in recovery position ▪ consider other causes

(1) Naloxone 0.2-0.4 mg IV injection repeated every 2-3 minutes until desired effect. May need up to 10 mg (maximum dose). Monitoring essential as the effect of naloxone can wear off before that of the opioid. (The t½ of naloxone is ~1 hour which is shorter than most opioids.)

24.4 Adjuncts to Opioids for Severe Acute Pain

NSAIDS remain the standard approach, but are relatively contraindicated where there is a bleeding disorder or upper GI dysfunction. Orthopaedic surgeons have a reluctance to use these agents in prosthetic work.

The Cox 2 inhibitors are generally well tolerated, although not entirely free of upper GI side effects. Their efficacy is no better than standard NSAIDs and they are expensive. Recent controversy over their long term cardiovascular side effects does limit their use. They should not be administered before there has been an adequate trial of standard NSAIDS. They are preferred, however, where there is a concern about the possibility of bleeding or dyspepsia, but caution is advised.

Note: Rofecoxib is no longer available.

24.5 Chronic or Persistent Pain

This is broadly classified into two categories - cancer and non-cancer pain. Refer to the Christchurch Hospital Palliative Care Service Clinical Guidelines, Chapter 1 and Chapter 2.

24.5.1 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 in combination with analgesics.
- **For moderate to severe pain, use morphine as first-line**.
- **Guidelines for starting morphine for palliative therapy:**
  - Commence using **either:**
    - morphine elixir (1 mg/mL, 2 mg/mL, 5 mg/mL, or 10 mg/mL), **or**
    - Sevredol™ 10 mg or 20 mg tablets.
  - Starting dose: 10 mg 4 hourly, **regularly** throughout 24 hours. Extra doses must also be available for severe pain (maximum of hourly prn).
  - Gradually titrate dose to effect before converting to sustained release morphine.
  - Reduce dose and/or frequency in elderly (e.g., 6 hourly rather than 4 hourly).
  - Increase starting dose if already on regular codeine. 60 mg codeine is equivalent to at least 5 mg oral morphine.
  - Give dose 6-8 hourly if impaired renal function (no need to change dosing interval for mild to moderate hepatic failure).
  - Patients commenced on morphine may develop some tolerance. Therefore, there can be a need to titrate the dose up, to maintain efficacy.

Starting sustained release morphine (morphine sulphate SR):

- **Currently available preparations:**
  - **m-Eslon™ (10 mg, 30 mg, 60 mg, 100 mg, and 200 mg)**
  - **LA Morph™ (10 mg, 30 mg, 60 mg, and 100 mg).**
- Add up morphine doses over 24 hours during which pain was controlled, and divide by 2 to get the 12 hourly m-Eslon™ or LA Morph™ dose given twice daily.
- When stabilised on morphine sulphate SR, an appropriate dose of breakthrough morphine should be charted (15 - 20% of the **total** daily dose q3-4h as elixir or Sevredol™ tablets).
- Continue to prescribe oral (or PR) paracetamol and/or NSAID even when taking regular morphine.
- Morphine is approximately **twice** as potent SC as orally, and can be given both as a bolus injection and as a continuous infusion. Refer to the Palliative Care Service Clinical Guidelines, Chapter 1 and Chapter 16, or refer to the Palliative Care Service ☎ 81473 (or page via the Operator).

Note: If commencing directly on morphine sulphate SR without prior dose titration with elixir or Sevredol™, caution is needed as a safe and effective starting dose is difficult to predict.

Breakthrough Pain:

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

  If several breakthrough doses needed per day, increase morphine sulphate SR dose.

Incident Pain:

- Use morphine elixir, 20% of total daily morphine dose, before activity which causes pain. Adjustment of the morphine sulphate SR dose is generally not recommended.

Alternative Opioids to Morphine:

- Transdermal fentanyl
- Methadone
- Oxycodone

Occasionally these drugs may be required. For advice regarding indications and prescribing, refer to the Palliative Care Service Clinical Guidelines. A referral to the Palliative Care Service is strongly recommended - ☎ 81473 (or page via the Operator).

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

Co-analgesics:

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 (TENS), antidepressants (e.g., nortriptyline or amitriptyline), anticonvulsants (gabapentin has the greatest efficacy and the lowest side effect profile) for neuropathic pain. Consultation with Palliative Care ☎ 81473 or with Pain Management Centre, Burwood Hospital is recommended.

Management of Opioid Side Effects

Nausea and vomiting

- Metoclopramide 10 mg PO/SC q4-8h (or QID before food)
- Haloperidol 0.5-1.5 mg PO q8h (can be given as a single nocte dose)
- Cyclizine 50 mg PO q6-12h (maximum dose of 150 mg/24 hrs)
- Prochlorperazine 5-10 mg PO q6h, 3 mg buccal q6h, 25 mg PR q8h

Nausea and vomiting due to opioids tends to subside over the first week. Therefore reassess need for antiemetics.

Metoclopramide has theoretical advantages in the presence of constipation as it stimulates peristalsis. However it is contraindicated if obstruction is likely. An oral alternative is domperidome (has fewer side effects, including no extrapyramidal reactions).

Methotrimeprazine (Nozinan) is a broad-spectrum antiemetic and can be very effective in advanced disease states. Referral to the Palliative Care Service ☎ 81473 (or page via the Operator) is recommended for persistent or intractable nausea.

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.

Dexamethasone 2 - 8 mg PO or via a continuous SC infusion can also be effective for intractable nausea.

Constipation

Regular stool softeners (e.g., Coloxyl) with stimulants (e.g., Bisacodyl, Senna) or a combination laxative such as Coloxyl and Senna™ or Danthron and Poloxamer (Conthram™, Codalax™) should be used routinely when taking opioids. Refer to Christchurch Hospital Palliative Care Service Clinical Guidelines, Chapter 4.

24.5.2 Non Cancer Chronic Persistent Pain

NSAIDs continue to be widely used as basal analgesics. Dextropropoxyphene, a component of Di-gesic, is not favoured because of the long duration of effect and potential for abuse. DHC and oral tramadol

provide slightly better efficacy, but do have a higher adverse event profile. Tramadol is expensive, but is available as an oral and parenteral preparation. Tricyclic antidepressants or anticonvulsant agents are useful in neuropathic pain (burning, shooting). Gabapentin is gaining pre-eminence in this field as an effective agent with limited adverse side-effects (an application from a specialist or vocationally trained general practitioner is required but a referral to Palliative Care or Pain Management is strongly recommended before prescribing). The use of oral opioids, particularly the slow release preparations, should only be prescribed where a comprehensive evaluation has been conducted.

It is suggested that advice be sought either from the Christchurch Hospital Palliative Care Service ☎ 81473 (Palliative Care is **not** just for patients with a cancer diagnosis), or the Pain Management Centre, Burwood Hospital.

OBSOLETE

25. Poisoning / Drug Overdose

25.1 Management Priorities

1. **The Airway, Breathing and Circulation**.

   The first priority in all unwell patients is attention to:

   - Airway, Breathing, Circulation: See The ABCs (see page 20)

   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.

   The majority of poisoned patients can be managed supportively and expectantly. A few require gastrointestinal decontamination. A small number of drugs have specific antidotes.

2. **"Toxidromes"**

   A number of toxins/poisons produce recognisable toxic syndromes which may be rapidly identified allowing prompt evaluation and management.

   - **Anticholinergic, e.g., tricyclics, antihistamines**
     - Dry, warm skin
     - Thirst and tachycardia
     - Hyperthermia
     - Confusion and hallucinations
     - Urinary retention
     - Visual disturbances
   - **Sympathomimetics, e.g., amphetamines, cocaine, caffeine, theophylline**
     - CNS excitation and convulsions
     - Hypertension
     - Tachycardia
     - Sweating
     - Mydriasis
   - **Muscarinic, e.g., organophosphates, some mushrooms**
     - Defecation, urination, miosis, bradycardia, emesis, lacrimation, salivation (DUMBELS).
   - **Nicotinic e.g., insectides**
     - Tachycardia, hypertension, paralysis, muscle fasciculations.
   - **Narcotic**
     - CNS depression
     - Hypotension
     - Hypoventilation
     - Miosis
   - **Withdrawal e.g., from opioids, alcohol, benzodiazepines**
     - Diarrhoea, mydriasis, tachycardia, lacrimation, abdominal pain, hallucinations.

3. **Gastrointestinal Tract Decontamination**

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

   The options include:

   1. Activated charcoal
   2. Gastric lavage
   3. Whole bowel irrigation

   Note: *Usually gut decontamination is not required.*

25.1.1 Activated Charcoal

- First choice for gastrointestinal decontamination, if any is indicated.
- Provided as 50 g in 250 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 it may 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.

25.1.2 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 drowsy or likely to become drowsy.

25.1.3 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 Klean-Prep or oral Fleet at approximately 2 L/hr orally until the rectal effluent is clear (usually 4-6 hours).
- Contraindications: ileus, perforation, obstruction.
- Cautions:
  - Klean-Prep or oral Fleet 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, sodium bicarbonate for tricyclic antidepressants, or ways of enhancing the elimination of the drug e.g., haemodialysis, haemoperfusion, alkaline diuresis, etc.

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

- Poisons Internet database (TOXINZ) - a computerized reference available in the Emergency Department and Clinical Pharmacology Drug Information Service.
- Clinical Pharmacology Drug Information Service (Ext: 80900) or via the Intranet.
- The Emergency Medicine Physician, Pharmacologist or General Physician.
- National Poisons and Hazardous Chemical Information Centre

  Telephone:
  URGENT 0800 POISON / 0800 764 766
  NON URGENT (Monday to Friday 0900-1700 hours) (03) 479 1200 and ask for Poisons Centre.

25.1.4 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.
- In Christchurch, toxic screens are only performed on urine.
- 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.

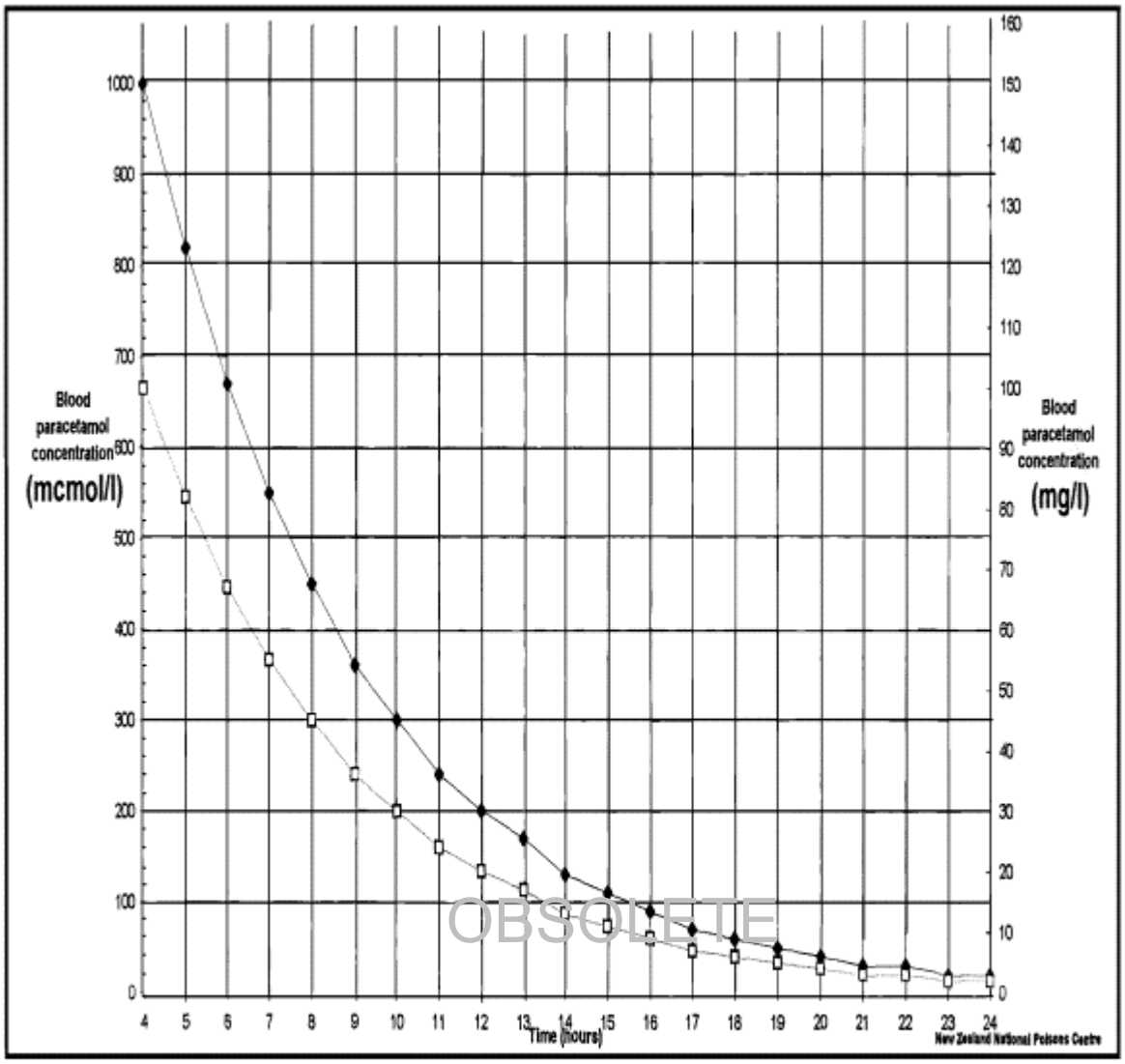
25.2 Paracetamol

- 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. Use the green lithium heparin tube.
- 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 8 hours, especially if symptomatic, should have N-acetylcysteine without waiting for the blood level result.
- Clinical toxicity follows four approximate stages:
  - 1/2 - 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 (see page 197).

Table 26: Modified Rumack-Matthew Paracetamol Nomogram (mcmol/L and mg/L)



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

Treat all patients with levels above the upper line.

Table 27: N-acetylcysteine Dosage in Paracetamol Poisoning

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

25.3 Tricyclic Antidepressants

- Numerous receptor effects (sodium channel, calcium channel, muscarinic and alpha-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. Greater than 10 mg/kg can be 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.
- 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 benzodiazepines if required.
- 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.
- 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 199).*

25.4 Monoamine Oxidase Inhibitors

- Examples of MAO-I drugs currently available include phenelzine, selegiline, moclobemide and tranylcypromine.
- More than 2-3 mg/kg is potentially life threatening.
- Effects of an overdose include CNS stimulation, tachycardia, hypertension, hyperpyrexia and tachypnoea. In severe overdoses there is CNS and cardiovascular depression.
- Diagnosis and assessment of severity is clinical. Blood levels for MAO-I are not done routinely.
- Treat hyperthermia with aggressive external cooling and sedation. May need neuromuscular paralysis. Dantrolene may also be useful.
- Control BP and tachycardia with labetalol (combined alpha- and beta-blockade may be better than beta 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 199).

25.5 Fluoxetine/Paroxetine/Citalopram

(Selective Serotonin Re-uptake Inhibitors, SSRIs)

- To date overdoses with these drugs alone 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 (see Serotonin Syndrome on page 199).
- In most cases CNS depression will predominate.
- Treatment is supportive.

25.6 Serotonin Syndrome

- May occur with combination of drugs such as MAOIs with SSRIs, clomipramine, other tricyclic antidepressants, lithium or pethidine, tramadol.
- Diagnosis requires such a combination, or the increase in dosage of serotonergic 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 and may need to be aggressive. Diazepam for agitation.

25.7 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.

25.8 Neuroleptic Malignant Syndrome

- An idiosyncratic reaction to neuroleptic drugs, e.g., haloperidol, chlorpromazine, prochlorperazine, metoclopramide.
- May occur at any time during patient's treatment with these drugs.
- Develops over hours to days.
- Features include:
  - High fever
  - Muscle rigidity
  - Altered level of consciousness
  - Autonomic instability (tachycardia, sweating, labile blood pressure)
- Treatment
  - Stop the drug. If on lithium and other anticholinergics, consider stopping these drugs as well. Cooling and fluids and may need cardiovascular and respiratory support.

25.9 Designer Drugs

e.g., Ecstasy, GHB.

- **Gamma Hydroxybutyrate (GHB)**
  - Formed from precursor gamma butyrolactone and metabolised to gamma aminobutyric acid.
  - Usually supplied in liquid form.
  - Acts as a CNS depressant with euphoria inducing capabilities therefore commonly present in profound coma. Other symptoms include:
    - Neurological - euphoria, seizures, headache, miosis, nystagmus
    - Respiratory - apnoea, respiratory arrest
    - Cardiovascular - bradycardia, hypotension
    - Gastrointestinal - nausea, emesis
  - May be accompanied by myoclonic jerks, confusion, and combativeness.
  - Rapidly absorbed, short half life.
  - Individuals should be observed for 6 hours after symptom resolution.
  - Treatment is supportive with particular attention to airway management. Seizures should be treated with benzodiazepines.
- **Ecstasy (MDMA)**
  - Hallucinogenic amphetamine derivative.
  - Rapid absorption and onset of effects.
  - Symptoms include:
    - Neurological - euphoria, agitation, hallucinations, seizures
    - Cardiovascular - hypertension, tachycardia, ventricular arrhythmias (usual cause of death)
    - Respiratory - hyperventilation, pulmonary oedema.
    - Renal - acute renal failure secondary to rhabdomyolysis.
    - Ocular - mydriasis
    - Gastrointestinal - nausea, vomiting and anorexia.
    - Other - hyperthermia, hyperkalaemia, trismus, diaphoresis.
  - Toxic dose not established as the strength of the ingested dose varies, however deaths have occurred after ingestion of a single tablet.

Treatment - mainly supportive care

- Consider activated charcoal
- Benzodiazepine for agitation and/or seizures.
- ECG monitoring - treat tachyarrhythmias with beta blockers.
- Hypertension may be treated with short acting agents, e.g., nitroprusside 2-10 mcg/kg/min titrated to response.
- Hyperthermia (see page 35) - aggressive cooling measures.
- IV fluids to ensure adequate hydration.
- Observe for at least 6 hours after resolution of symptoms.

25.10 Benzodiazepines

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

Note:

- T½ flumazenil is one hour, i.e., sedation can recur.
- It may be helpful if trying to establish the cause of 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) is contraindicated as it may induce seizures.

25.11 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 2-3 minutes. May need up to 10 mg (maximum dose). If no response after 10 mg then question diagnosis.
  - Monitoring essential as the effect of Naloxone can wear off before that of the opioid (the t½ of Naloxone is ~1 hour which is shorter than most opioids). 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.

25.12 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 30 mg/kg.
- Serum levels important. 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.

25.13 Digoxin

- 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.
    - 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 140), but avoid calcium chloride/calcium gluconate as calcium may potentiate digoxin cardiotoxicity.

- Fab fragments (Digibind - kept in ICU):
  - 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.
    - If still no response in adults, see TOXINZ internet database.
  - 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.*

25.14 Carbon Monoxide Poisoning

- 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 but useful diagnostically. There is also no correlation between severity at presentation and development or severity of delayed sequelae.
- There remains controversy over the role of hyperbaric oxygen treatment (HBOT) but a recent randomised double-blind clinical trial suggests that where it is readily available, as in Christchurch, it should be used in some patients (see page 202).
- Consider the diagnosis in all burns, smoke inhalation, coma or attempted suicide cases.

Treatment

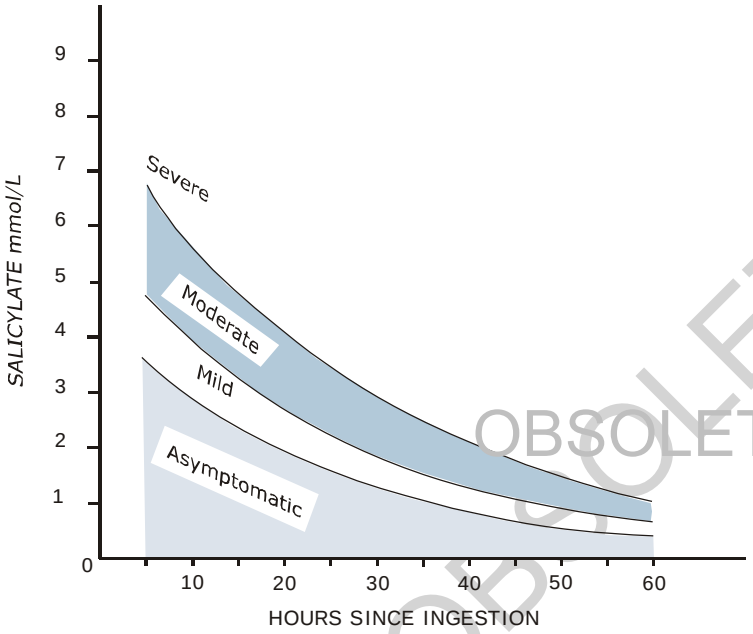
- 100% oxygen via a breathing system with a tight-fitting mask, reservoir bag and high fresh gas flow for at least 6 hours. This also hastens the elimination of CO.
- HBOT indications are:
  - Loss of consciousness (clear history of, or on arrival in ED)
  - Any neurological symptoms or signs (including cognitive, behavioural or psychological) except headache and/or nausea alone **plus** any one of:
    - Age >50 years
    - Metabolic acidosis
    - COHb >25%
  - Pregnancy
  - Delayed deterioration after normobaric oxygen therapy. This may occur in 8-30% of patients. Therefore, daily follow-up for several days is strongly advised.
- To arrange HBOT or for advice, see Referral of Patients to Hyperbaric Unit (see page 264).

25.15 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 only. 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.

Table 28: 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 metabolic acidosis is present 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 and the rate adjusted according to regular measurements of pH, K, Na and hydration to maintain an alkaline urinary pH.

  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.

25.16 Methanol

25.16.1 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.
  - 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 q2h in orange juice orally or via NG tube, **or** 0.15 ml/kg 100% ethanol/hour IV. 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.

25.16.2 Management of the chronic alcoholic / meths drinker

- **In the community**
  - 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 the Emergency Department, Christchurch Hospital.
  - 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.
- **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, urate, Ca, LFTs, 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 100 mg/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, e.g., 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, require urgent management (see page 204).

26. Psychiatry

26.1 Psychiatric Services Contact Information

- **Psychiatric Emergency Service (PES)**: ☎ 83960 between 0830 - 1700 (after-hours, page via the Christchurch Hospital operator).
- **Psychiatric Consultation Service**: ☎ 83100 between 0830 - 1700 (after-hours, contact PES via the Christchurch Hospital operator).
- **Delirium Service**: contact via the PMH switchboard ☎ 66000 (external number 337 7899).
- **Psychiatric Services for the Elderly (PSE)**: refer to Older Persons Health Department Information on page 172 for contact details.

26.2 Introduction

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, bipolar disorder).
- 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.

26.3 Delirium

Refer also to 'Guidelines for Care of Patients with Delirium', Canterbury DHB 2002, Ref. 0020, which is available on the wards.

26.3.1 Clinical features

- **Acute confusion** - an abrupt change in mental state and ADL functioning.
- **Fluctuation** during the course of the day (often worse at night).
- **Difficulty focusing, sustaining, or shifting attention** is the most striking cognitive deficit; also forgetfulness and disorientation.
- **Change in level of alertness** - either reduced level of consciousness or increased (hypervigilant), sleep/wake cycle often disturbed.
- **Disorganised thinking** (rambling, illogical, or incoherent), suspiciousness.
- **Psychomotor changes** - either agitation or retardation.
- **Misperceptions** - vivid 'dreams', recognition errors, illusions, hallucinations.
- **Emotional changes**, anxiety, tearfulness, anger, blunting.

The presence of any of these features should trigger a diagnostic evaluation for delirium using the Confusion Assessment Method (CAM) (see page 207) and a cognitive screening test, MSQ (see page 146) or MMSE.

Delirium can be missed when superimposed upon pre-existing dementia. It is therefore vital to obtain collateral history regarding pre-morbid cognitive function from relatives, friends or GP.

Table 29: The Confusion Assessment Method (CAM)

Feature 1: Acute onset and fluctuating source
This feature is usually obtained from a family member or nurse and is shown by positive responses to the following questions: ▪ Is there evidence of an acute change in mental status from the patient's baseline? ▪ Did the (abnormal) behaviour fluctuate during the day, that is, tend to come and go, or increase and decrease in severity?
Feature 2: Inattention
This feature is shown by a positive response to the following question: ▪ Did the patient have difficulty focusing attention, for example, being easily distracted, or having difficulty keeping track of what was being said?
Feature 3: Disorganised thinking
This feature is shown by a positive response to the following question: ▪ Was the patient's thinking disorganised or incoherent, such as rambling or irrelevant conversation, unclear or illogical flow of ideas, or unpredictable switching from subject to subject?
Feature 4: Altered levels of consciousness
This feature is shown by any answer other than 'alert' to the following question: ▪ Overall, how would you rate this patient's level of consciousness? (Alert [normal], vigilant [hyperalert], lethargic [drowsy, easily aroused], stupor [difficult to arouse], or coma [unrousable]).
The diagnosis of delirium by CAM requires the presence of features 1 and 2 and either 3 or 4.

26.3.2 Risk factors for developing delirium

- The very young or elderly.
- Pre-existing cognitive impairment.
- Structural brain disease (e.g., previous CVA, Parkinson's disease, brain-damage).
- Impaired functional status (especially poor mobility).
- Chronic comorbidities, with multiple medications.
- Severe acute illness or major surgery.
- Nutritional deficiencies.
- History of alcohol abuse.
- Visual and/or hearing impairment.
- Use of physical restraints.
- Use of a bladder catheter.

26.3.3 Common Causes

Systemic Disease

- Toxic:
  - Drugs:
    - Medication toxicity/ withdrawal (see Table 30 on page 208)
    - Alcohol - intoxication/ withdrawal
    - Street drugs of abuse
  - Heavy metals
- Infections
- Metabolic: electrolyte imbalance, acid base disorders, renal failure, liver failure.
- Hypoxia: cardiovascular disease, respiratory disease, anaemia.
- Endocrine: thyroid disorders, parathyroid disorders, hypoglycaemia, hyperglycaemia.
- Vitamin deficiency: thiamine (Wernicke's), B12 and folic acid.
- Hypothermia.
- Recent surgery/anaesthesia.
- Pain
- Faecal impaction / urinary retention

CNS Disease

- Head injury.
- Space-occupying lesion.
- Encephalitis, meningitis.
- Acute stroke.
- Subdural haematoma.
- Epilepsy: post-ictal, absence seizures.

Table 30: Some Drugs that may Cause or Worsen Confusion

- **Sedatives/hypnotics:** benzodiazepines, zopiclone.
- **Analgesics:** opioids, nefopam, non-steroidal anti-inflammatories.
- **Drugs with strong anticholinergic properties:** antihistamines, some antiparkinsonians, antispasmodics, tricyclic antidepressants, neuroleptics.
- **Cardiac:** antiarrhythmics, some antihypertensives, digoxin.
- **Gastrointestinal:** H_2 - antagonists, proton-pump inhibitors (occasionally), prochlorperazine, metoclopramide.
- **Miscellaneous:** anticonvulsants, corticosteroids, antiparkinsonian drugs, lithium, antibiotics (occasionally), pro-serotonergic drugs ('serotonergic syndrome').

26.3.4 Management

- Prevention: vigilance in high risk patient, accurate medication/drug/alcohol history, optimise hydration, nutrition, oxygenation, mobility, avoid unnecessary medications.
- Seek, identify and treat underlying cause(s).
- Educate and support patient and their family (explanatory leaflet available from Delirium Service).
- Ensure a safe and secure environment for patient and staff (refer to Restraint Policy). A nurse-aide sitter may be required. Occasionally, Mental Health Act certification may need to be sought if the patient is persistently unwilling to consent to vital treatment or is endangering others.

- General supportive management:
  - Re-orientation and reassurance (utilise support of friends/family) - includes provision of clock, calendar, familiar objects, view to outside.
  - Quiet, single room whenever possible; minimise room changes.
  - Make sure glasses and hearing aids are worn.
  - Minimise physical restraints and tubes, avoid unnecessary bed rest.
  - Encourage oral fluids and good nutrition; vitamin supplements for malnourished/alcoholic patients.
  - Close, sympathetic surveillance - ideally by consistent nursing personnel.
  - At night keep the room quiet with low-level lighting, relaxation strategies to help sleep and reduce anxiety.
- Psychotropic medication (in parallel with general measures, not as a substitute):
  - Indicated if the patient is distressed from psychotic symptoms/anxiety or is posing a risk to themselves or others.
  - Haloperidol is generally the tranquillizer of choice. Exceptions include patients with:
    - Parkinsonism and dementia with Lewy bodies (see below).
    - Alcohol or benzodiazepine withdrawal delirium (see below).
  - For elderly patients haloperidol 0.5 mg orally or SC, once or twice daily. In urgent situations, higher doses (0.5-2.5 mg) of haloperidol may be necessary (best given SC, IM, or slow IV) with additional doses every 30-60 minutes as required. IV treatment seldom causes extrapyramidal side effects. Discuss with consultant before giving IV haloperidol (see below).
  - Younger patients may need higher doses (1 mg SC, IM, or slow IV initially, for milder symptoms; up to 5 mg for severe), repeated every 30-60 minutes as required.
  - **Discuss with the Consultant before giving IV haloperidol** as there is a risk of prolongation of QT interval and torsades de pointes tachycardia with higher doses.
  - Haloperidol should be tapered gradually as target symptoms resolve - usually over 1-2 weeks. Try to have patient free of psychotropic drugs by discharge.
  - **Parkinsonism**: atypical neuroleptic treatment is a safer alternative for patients with parkinsonism, especially if more prolonged treatment proves necessary (first choice is quetiapine 25-100 mg daily; watch for inital sedation).
  - **Dementia with Lewy bodies**: typical neuroleptics can cause serious neurotoxic reactions. Use atypical neuroleptics with caution, in very low dosage.
  - **Alcohol withdrawal** (see page 60) **or benzodiazepine withdrawal delirium** (see page 212): Diazepam is the treatment of choice (generally by oral administration).
  - Apart from the above situation, benzodiazepines should be avoided if possible, especially in the elderly. However, do not stop habitual benzodiazepines abruptly (especially short-acting). Short-term use of lorazepam or clonazepam (PO, SC, IM, or IV) as an adjunct to haloperidol may be appropriate for severe agitation, anxiety, or sleep disruption.
  - Resolution of delirium may take up to several weeks.

26.4 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.
- 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.

A combination of antipsychotic and benzodiazepine medications 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.

26.5 Preferred Neuroleptic/Antipsychotic Drugs

Range of dosage suggested:

Table 31: Oral/parenteral doses of anti psychotic drugs

Drug	Acute (mg)		Daily (mg)
	IM	Oral	
Haloperidol	1 - 5	0.5 - 5	2 - 10
Risperidone	N/A	0.5 - 2	3 - 6
Lorazepam	1 - 2	1 - 2	8
Chlorpromazine	Do not give IM	50 - 100	50 - 600

Common side effects:

- **Low potency** (chlorpromazine).
  - Sedation
  - Hypotension.
  - Marked anticholinergic effects; dry mouth; constipation; urinary retention.
- **High potency** (haloperidol).
  - Extrapyramidal effects (especially oral dosage in range 5-20 mg/day and the elderly).
  - Dystonia (torticollis, opisthotonus, oculogyric crisis - commoner in the young patient).
  - Dyskinesia (acute, tardive).
  - Akathisia
- **Atypical neuroleptics** (risperidone, olanzapine)
  - Weight gain.
  - Agitation (initially).
  - Fewer extrapyramidal effects.
- **Treatment of 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)

Some practical management guidelines:

- Review early and frequently.
- Once symptoms show some modification, reduce dosage frequency.
- Write clear instructions to nursing staff about indications for 'repeat' dosage.

26.6 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.

26.6.1 Management

- Beware of sensitivity to the side effects of antidepressant medications in the medically ill, particularly the tricyclics.
- Selective serotonin re-uptake inhibitors (SSRI) e.g., citalopram, fluoxetine, paroxetine - 20 mg mane (may use 10 mg initially in the elderly).
- Citalopram has least drug interactions, thus generally preferred in the medically ill.
- Nortriptyline (not amitriptyline) is the most tolerable of the tricyclics. Dosage range of nortriptyline 50 - 150 mg nocte, but start low and titrate upwards. Check serum therapeutic level.
- ECT should not be forgotten as an option (if fit for GA).
- Tricyclic antidepressants may have co-analgesic activity. SSRI's do not.
- The physically ill should not be allowed to suffer an untreated depressive disorder.
- SSRIs may render codeine ineffective.

26.7 Suicidal Ideation

This should be regarded as a very serious situation that requires both further evaluation and a response. Patients may be suicidal for a number of reasons and it should not be assumed that the patient is depressed (though this may be the case). Patients in pain or attempting to obtain drugs of abuse may express suicidal ideation as part of their general distress. Further evaluation of why the patient is suicidal at this time is vital to establishing an effective management strategy. This includes finding out whether the patient has a plan and whether they have previously attempted suicide, and evaluating the level of intent and means to carry out the plan.

Management

- Discuss all such situations with a consultant or registrar as soon as possible.
- Be cautious and err on the side of safety until you are sure of the level of immediate risk.
- Utilise constant watches or one-on-one nursing to ensure safety as required.

Note: *A psychiatric consultation is mandatory. Patients expressing suicidal ideation and attempting to leave can be detained under a number of legal provisions. You are able to detain a person against their will in this situation and will not be criticised or encounter legal difficulties for doing so.*

26.8 Nicotine Dependent Patients

Inform all patients of the CDHB Smokefree Policy. Ask if they have considered quitting. Offer Nicotine Replacement Therapy (NRT) while in hospital, whether or not they wish to quit.

Nicotine Withdrawal Symptoms

- Depressed mood, irritability or anger, insomnia, increased appetite, anxiety, decreased heart rate, difficulty concentrating, restlessness.

Nicotine Replacement Therapy (NRT)

- If you have nicotine dependent patients who want to quit smoking, discuss NRT and prescribe if appropriate. There is a subsidised Smoking Cessation Programme available through General Practitioners, or through QUITLINE on 0800 778 778.

Recommended NRT Dosage		
<10 cigs/day	10 - 20 cigs/day	>20 cigs/day
None usually necessary, but 7 mg Habitrol[1] patch per 24 hrs (transdermal route) may be prescribed if deemed useful	14 mg Habitrol[1] patch per 24 hrs (transdermal route)	21 mg Habitrol[1] patch per 24 hrs (transdermal route)
Duration of treatment should be for 4 weeks and then the dose should be reduced.		
1. Habitrol is the transdermal (patch) NRT currently funded in NZ.		

- Gum: 2 mg and 4 mg gum is available in the community.
- Useful facts about NRT medications:
  - They do not contain the toxic substances found in cigarette smoke, such as carbon monoxide, cyanide, ammonia, vinyl chloride, and tar.
  - They do not produce dramatic surges in blood nicotine levels.
  - They do not produce strong dependence.
  - Nicotine passes through the placenta to the foetus, and via breast milk to the baby. This occurs whether the mother smokes or uses NRT, however NRT is preferable, for the reasons explained above.
- **Contraindications** are the same as for smoking, i.e., acute MI, unstable angina pectoris, severe arrhythmias, recent CVA. However, if the options are NRT or smoking, NRT is preferable.
- Symptoms of NRT **overdose** include abdominal pain, nausea and vomiting, diarrhoea, dizziness, tachycardia, headache, hypotension, and confusion.
- Symptoms of NRT **underdose** are the same as for nicotine withdrawal.

Note: *Aromatic hydrocarbons in cigarette smoke induce hepatic drug metabolising enzymes, notably CYP1A2. Smoking cessation may result in elevated concentrations of drugs that are metabolised by this pathway such as theophylline, caffeine, and clozapine.*

26.9 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 5-10 mg PO hourly until control then reduce dose by 5-10% daily.

- Opioid intoxication.
  - Naloxone 0.2 mg IV and repeat in 2-3 minutes. May need up to 10 mg (maximum dose) in this situation. Monitoring essential as the effect of naloxone can wear off before that of the opioid. (The t½ of naloxone is ~1 hour which is shorter than most opioids.)
- 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. For the management of alcohol-related problems, including withdrawal, refer to Alcohol Related Problems (see page 60).*

26.10 Management of Patients on the Methadone Programme

- **Methadone prescribing and supply**.
- The following extract is taken from the CDHB Manual Volume 12, Fluid and Medication Management. It represents a summary of CDHB policy in this area and should be particularly helpful if such patients require admission. This is covered by legislation, and it is an offence for a medical practitioner to prescribe controlled drugs for the treatment of dependence unless the practitioner is approved or authorised under the Misuse of Drugs Act 1975.
- The prescription and administration of methadone to patients on the Christchurch Methadone Programme (CMP) is strictly regulated.
- SMOs, RMOs, and pharmacists working in CDHB hospitals need to be aware of the relevant regulations contained in the Ministry of Health publication, *Opioid Substitution Therapy, New Zealand Practice Guidelines, 2003*. The following aspects of these guidelines are drawn to your attention.

  Exception: *The restrictions do not apply to those using methadone for chronic pain, such as those in palliative care, and who are not enrolled with CMP.*
- Patients who are enrolled with CMP are not to receive methadone in a CDHB hospital until their daily dose (in mg) has been confirmed with their CMP case manager. This can be done by the doctor, pharmacist, or nurse, and should be documented in the notes.
  - Weekdays between 0800 and 1700 hours, ☎ 335 4350
  - After-hours, phone the Kennedy Detox Centre, ☎ 339 1139
- Written authorisation must be obtained from the CMP before hospital doctors can prescribe methadone to in-patients. The CMP have provided a form that can be faxed to obtain appropriate authorisation. Each authority to prescribe lasts one week. The CMP can extend or cancel authorities on request.
- The CMP or Kennedy Detox will inform the community pharmacy that prepares the daily dose of methadone that the patient is an in-patient in a CDHB hospital. This is so that extra supplies of methadone cannot be collected by a third party while the patient is admitted.
- Prescribers of methadone to patients in the CMP need to ensure that:
  - The potential for overdose is minimised,
  - The patient is not unsafely intoxicated with other drugs, and
  - The potential for methadone diversion is limited.
- Once CMP has confirmed the dose, methadone can be sent from a CDHB pharmacy to be used while the patient is admitted. After discharge, the methadone will be removed from the ward. The only wards to hold a methadone supply in their controlled drug safes are those who require it for chronic pain management.
- Patients receiving opioid substitution therapy should be prescribed analgesia for pain as for other patients.

- At discharge, the CMP case manager or (if after hours) the Kennedy Detox Centre must be called to arrange reinstatement of methadone supply in the community.
- Do **not** discharge any patient with a methadone supply or prescription.

27. Respiratory Medicine

27.1 Respiratory Medicine Department Information

Main Office

2nd Floor, Riverside, ☎ 80280, Fax 80914

Inpatient Services

Three inpatient teams on Ward 25:

- Resp 1 - Dr P Thornley / Dr M Epton / Dr R Laing
- Resp 2 - Dr C Drennan / Dr J Gillies
- Resp 3 - Dr R Meyer / Dr L Beckert

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, ☎ 88303, Fax 80849
  - For home-based service and education
  - Domiciliary oxygen service
  - Respiratory rehabilitation programme
  - Maori Respiratory Educator
- 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
  - Sue McLeod, Research Manager, ☎ 81157, Fax 81184
- Sleep Unit
  - Judy Jones, Technical Director, ☎ 81089, Fax 81089

27.2 Respiratory Failure

27.2.1 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.

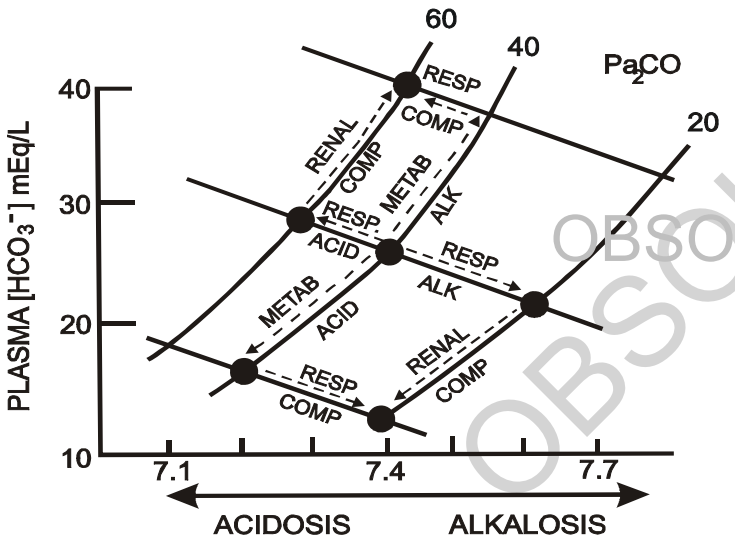
27.2.2 Classification

- 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.**

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

Blood gas interpretation may be assisted by the following diagram (which is also discussed under Acidosis / Alkalosis on page 111):



27.2.3 A-a gradient in the assessment of hypoxaemia

Calculation of the A-a gradient assists in differentiating between hypoventilation and V/Q mismatching as the source of hypoxaemia. It predicts the degree of shunt by comparing the partial pressure of O_2 in the (**A**) alveoli to that in the (**a**) artery. The difference between them gives us an idea how well the oxygen is moving from the alveoli to the arterial blood.

Normal A-a gradient for a young adult is < 20. The A-a gradient increases with age. For elderly patients (>75 yrs) a normal A-a gradient is < 25.

Calculation of the A-a Gradient

A-a Gradient = PAO_2 - PaO_2

- The **PaO_2** is obtained from the arterial blood gas.
- The **PAO_2** is obtained from the Alveolar Gas equation.

Estimation of PAO_2

$PAO_2 = PiO_2 - PaCO_2/R$

- PiO_2 = inspired partial pressure of oxygen = (barometric pressure minus water vapour pressure) X FiO_2
- R = respiratory quotient = ratio of CO_2 production to O_2 consumption = V_{CO2}/V_{O2} = 0.8 (usual).

  Therefore: $PAO_2 = (760 - 47) \times FiO_2 - PaCO_2/0.8$

Note: A-a Gradient is best calculated for blood gas taken on room air (FiO_2 = 0.21). Whilst on supplemental oxygen it is usually difficult to obtain an accurate assessment of FiO_2 due to variability in actual % of oxygen delivery.

27.2.4 Management of Respiratory Failure

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. Naloxone 0.2 mg IV and repeat in 2-3 minutes. May need up to 10 mg (maximum dose) in this situation. Monitoring essential as the effect of naloxone can wear off before that of the opioid. (The t½ of naloxone is ~1 hour which is shorter than most opioids.)
- 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 and some other pulmonary conditions. Patients who need CPAP should be managed in ICU. Discuss with ICU/Respiratory Physician.
- BiPAP should be considered in acute acidotic exacerbation of COPD (see page 221).

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 223)

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 urgent 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.
  - 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.

27.3 Obstructive Sleep Apnoea

Obstructive Sleep Apnoea (OSA) is a common medical problem occurring in at least (but not confined to) 4% of the middle-aged population. OSA is part of a spectrum of sleep-disordered breathing characterised by disturbed sleep arising from increased upper airway resistance. Risk factors for OSA include obesity, increased neck circumference, craniofacial abnormalities, hypothyroidism and acromegaly.

Patients who present with a history of loud snoring and excessive daytime sleepiness should be investigated for OSA. Snoring and excessive daytime sleepiness are both markers of adverse outcome, but are very prevalent (approximately 40% of the adult population report snoring and/or excessive daytime sleepiness) and are non-specific.

- If a patient presents with a history of snoring but without excessive daytime sleepiness they should be referred to an Ear Nose and Throat specialist service or Oral Health specialist service for assessment.
- If a patient presents without a history of snoring but with a history of daytime sleepiness they should be referred to a Sleep Disorders specialist service for assessment.
- All patients who present with both a history of snoring and excessive daytime sleepiness should have two initial assessments to facilitate timely and appropriate management:
  - Epworth Sleepiness Score (8 item questionnaire)
  - Overnight oximetry to estimate a desaturation index (DI)

  Subsequent management decisions can be made by applying the following 'Rule of 10':
    - Snoring patients with an ESS ≥ 10 and a DI ≥ 10 almost certainly suffer obstructive sleep apnoea and should be referred to a Sleep Disorders specialist service for consideration of CPAP treatment.
    - Snoring patients with an ESS ≥ 10 and a DI < 10 should be referred to a Sleep Disorders specialist service for further clinical assessment. These patients might suffer mild obstructive sleep apnoea, upper airway resistance syndrome, Disorders of Initiating and Maintaining Sleep or a variety of other less prevalent sleep disorders.
    - Snoring patients with an ESS < 10 and a DI ≥ 10 should be referred to a specialist Respiratory Physician for further clinical assessment. These patients almost certainly suffer some form of sleep-disordered breathing which might include a variety of cardio-respiratory disease.
    - Snoring patients with an ESS < 10 and a DI < 10 probably don't suffer significant OSA. Some of these patients need reassurance, others might want to pursue management of problematic snoring and should be referred to an Ear Nose and Throat specialist service or Oral Health specialist service for further assessment.

Table 32: Epworth Sleepiness Score

Epworth Sleepiness Score	
How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently, try to work out how they would have affected you. Use the following scale to choose *the most appropriate number* for each situation: **0** = would *never* doze **1** = *slight* chance of dozing **2** = *moderate* chance of dozing **3** = *high* chance of dozing	
	Chance of Dozing
Sitting and reading	
Watching television	
Sitting inactive in a public place (e.g., theatre, meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	
Thank you for your co-operation	

27.4 Chronic Obstructive Pulmonary Disease

27.4.1 Summary

- Smoking is the most important risk factor for COPD.
- Consider COPD in patients with other smoking-related diseases.
- Consider COPD in all smokers and ex-smokers older than 35 years.
- The diagnosis of COPD rests on the demonstration of airflow limitation which is not fully reversible.
- If airflow limitation is fully or substantially reversible, the patient should be treated as for asthma.

27.4.2 Definition

Chronic Obstructive Pulmonary Disease (COPD) is characterised by airflow limitation that is **not fully reversible**. The airflow limitation is in most cases both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases. It is a progressive, disabling disease with serious complications and exacerbations that are major burdens for healthcare systems.

Small-airway narrowing (with or without chronic bronchitis) and emphysema caused by smoking are the common conditions resulting in COPD. Chronic bronchitis is daily sputum production for at least three months of two or more consecutive years. Emphysema is a pathological diagnosis, and consists of alveolar dilatation and destruction. Breathlessness with exertion, chest tightness and wheeze are the results of airway narrowing and impaired gas exchange. The loss of lung elastic tissue in emphysema may result in airway wall collapse during expiration, leading to dynamic hyperinflation and consequent increased work of breathing.

The clinical features and pathophysiology of COPD can overlap with asthma, as most COPD patients have some reversibility of airflow limitation with bronchodilators. By contrast, some non-smokers with chronic asthma develop irreversible airway narrowing. However patients with complete reversibility of airflow limitation should be treated as asthma.

Differentiation of COPD from asthma is often difficult, and is best undertaken by a detailed history. COPD often presents later in life, with insidious and gradual onset of breathlessness, with less diurnal variation, associated with a history of exposure to noxious gas (usually cigarette smoking).

27.4.3 Causes of acute deterioration

- Acute bronchitis (viral or bacterial).
- Pneumonia.
- Pneumothorax.
- Increased bronchial irritability.
- Pulmonary embolism.
- Left ventricular failure.
- Sepsis.
- Drugs (beta-blockers, NSAIDS, sedatives).
- Acute abdomen.
- Chest pain (trauma, rib fracture, osteoporosis).
- Post-operative sedation / retention of secretions.

27.4.4 Investigations

- Arterial Blood Gases (pulse oximetry alone is **not** adequate).
- CXR.
- Sputum Culture and microscopy.
- CBC + Diff, U+E.
- ECG.
- Consider Pro-BNP to assess contribution of LV dysfunction.

27.4.5 Severity Assessment in COPD

Make an immediate assessment of severity (see page 220) and initiate treatment accordingly. Confirm the diagnosis, identifying precipitating factor(s) and estimate the degree of functional impairment, **referring to old notes** for information about previous functional status, spirometry, and **blood gas analysis**. Old notes may also contain previous discussions with patients about ceiling of care, and wishes about resuscitation and ventilation.

Table 33: 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 HCO3 level is raised and pH normal this suggests chronic CO2 retention.			

27.4.6 Management

General Principles

- Inhaled bronchodilators are effective treatments for acute exacerbations.
- Systemic glucocorticoids reduce the severity of and shorten recovery from acute exacerbations.
- Non-invasive positive pressure ventilation is effective for acute hypercapnic ventilatory failure.
- Exacerbations with clinical signs of infection (increased volume and change in colour of sputum and/or fever, leukocytosis) benefit from antibiotic therapy.
- Controlled oxygen delivery (24-28% or 0.5-2 L/min) is indicated for hypoxaemia.

Emergency Treatment

- **If respiratory arrest, unconscious patient, upper airway compromise, call ICU immediately.**
- Prepare for emergency intubation and assisted ventilation.
- Consider tension pneumothorax.
- Notify Respiratory Physician or General Physician on call.
- Initiate action for severe exacerbation (see below).

Management of Severe Exacerbation

- **Immediately obtain ABG.**
- Commence controlled oxygen therapy to maintain a PaO_2 >60 mmHg or Sat.O_2 >90% (0.5-2 L/min by nasal prongs or 24-28% by venturi mask). For more information, see Oxygen Therapy on page 223. Monitor for rising $PaCO_2$.
- Nebulised salbutamol 5 mg and ipratropium 0.5 mg stat and 2-6 hourly according to clinical response. Nebulise using compressed air if $PaCO_2$ elevated.
- Give oral prednisone 40 mg (IV hydrocortisone 200 mg if unable to swallow).
- Review ABG:
  - If pH <7.35 and $PaCO_2$ > 45 mmHg, all patients should be considered for ventilatory support and must be discussed with the Respiratory Physician on call. See Non-Invasive Ventilation on page 221.
- Consider oral antibiotics (IV if unable to swallow) if patient has two out of three of the following:
  - Purulent sputum.
  - Increased sputum production.
  - Increasing dyspnoea.
- If consolidation on CXR, treat as community-acquired pneumonia (see page 232).
- If concern about sputum retention, consider chest physiotherapy.
- There is no evidence of benefit for intravenous bronchodilators, either IV salbutamol or aminophylline, over inhaled treatments. There is evidence of greater adverse events with IV aminophylline. If inhaled treatments do not improve the situation consider ICU, Non-Invasive Ventilation, or discuss ceiling of care.

Non-Invasive Ventilation (NIV)

All patients should be assessed for the need for NIV through the use of Bilevel Positive Airway Pressure (BiPAP) ventilation using a face mask. NIV has been shown to be an effective treatment for acute hypercapnic respiratory failure, particularly in COPD. In this patient group, NIV has been shown to reduce mortality, hospital stay and costs.

Patients will be eligible for NIV in the Ward 25 NIV Unit if they fulfil the following entry criteria:

- The patient must have a clearly established diagnosis of COPD, **and** be acidotic (pH <7.35) **and** hypercapnic ($PaCO_2$ > 45 mmHg) on admission ABG.
- If the patient's pH is <7.25 then NIV should be delivered in ICU, unless the patient has been assessed by the medical team as **not** for endotracheal intubation or ICU referral. If this is the case, referral to Ward 25 NIV unit should still be considered
- **Every patient fulfilling the above criteria must be discussed with and agreed to by the acute Respiratory Physician before NIV is started. Thereafter the care of the patient will continue under Respiratory Services.**
- Before NIV is commenced, a ceiling of treatment must be established. A decision must be made whether the patient is for endotracheal intubation and transfer to ICU if NIV fails. This decision must be clearly documented in the patient's clinical notes along with their resuscitation status.
- If the patient is for endotracheal intubation in the event of clinical deterioration, then the cardio-respiratory registrar must notify the ICU team that the patient is being admitted to the Ward 25 NIV unit.
- All patients admitted for NIV on Ward 25 must be reviewed by the cardio-respiratory registrar within 30 mins of being notified of the arterial blood gas result obtained after 1 hour of NIV treatment, or sooner if requested by nursing staff. Their status is to be reported to the acute Respiratory Physician.

Patients **other** than those with hypercapnic respiratory failure secondary to COPD may be considered for NIV at the discretion of the acute Respiratory Physician and in consultation with the ward 25 nurse in charge.

- NIV nurse - beep 8750 or ☎ 89250.

Exclusion Criteria: NIV is generally excluded if the patient has any of the following:

- Facial trauma/ burns/ surgery.
- Recent upper airway surgery.
- Fixed upper airway obstruction.
- Persistent vomiting.
- Life threatening hypoxaemia.
- Haemodynamic instability.
- Severe co-morbidity.
- Impaired consciousness/ confusion/ agitation.
- Copious respiratory secretions.
- Focal consolidation on CXR.
- Undrained pneumothorax.

Treatment of Mild or Moderate Exacerbation

- Oxygen (see Oxygen Therapy on page 223).
- 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. There is no evidence of benefit for oral steroids beyond 14 days. Regular oral steroid treatment has not been shown to alter outcomes, and is associated with a poor side effect profile, including muscle weakness and osteoporosis.
- Oral antibiotics may be appropriate.
  - Consider chest physiotherapy.

Monitor Progress

- Oxygen therapy:
  - Monitor Sat.O_2 and aim to maintain >90%.
  - Monitor for hypercapnia (symptoms of drowsiness and / or confusion).
  - Perform ABG if evidence of falling Sat.O_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, fluid and electrolyte requirements and bronchial secretions (chest physiotherapy for retained bronchial secretions). Commence oral therapy as soon as condition stabilizes. Bronchodilators should be given by Metered Dose Inhaler (MDI) and spacer.

Discharge Planning/Rehabilitation

- Involving the patient's general practitioner in a case conference and developing a care plan may facilitate early discharge. Discharge planning should start on admission and be documented within 24-48 hours.
- It is helpful to obtain spirometry and arterial blood gases at discharge.
- Most patients will benefit from enrolment into an out-patient rehabilitation programme, including COPD education and a self-management plan. Contact Respiratory Outreach to arrange this.
- Arrange smoking cessation advice for current smokers (each ward has nurses trained in smoking cessation - ask the Charge Nurse for details).
- Consider nutritional supplements and advice for underweight patients.
- Advise influenza vaccination each autumn.
- Suggested criteria for a patient's readiness for discharge include:
  - The patient should be in a clinically stable condition and have had no parenteral therapy for 24 hours.
  - Inhaled bronchodilators are required less than four-hourly.
  - Oxygen delivery has ceased for 24 hours (unless home oxygen is indicated).
  - If previously able, the patient is ambulating safely and independently, and performing activities of daily living.
  - The patient is able to eat and sleep without significant episodes of dyspnoea.
  - The patient or caregiver understands and is able to administer medications.
  - Follow-up and home care arrangements (e.g., home oxygen, home-care, Meals on Wheels, community nurse, allied health, GP, specialist) have been completed.

Reference: *The COPDX Plan: Australian and New Zealand Guidelines for the management of Chronic Obstructive Pulmonary Disease 2003* by David K McKenzie, Peter A Frith, Jonathan G W Burdon and G Ian Town MJA 2003 178(6 Suppl 17 Mar): S1-S40.

27.5 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.

27.5.1 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.

27.5.2 Indications

- PaO_2 less than 60 mmHg or Sat.O2 <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.

27.5.3 Pulse Oximetry

- This is very useful for determining haemoglobin oxygen saturation ($Sat.O_2$) 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.

27.5.4 Administration

- 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.

27.5.5 Adjusting the Dose

- Do the arterial blood gases 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 making CO_2 retention worse.
- 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 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.

27.5.6 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.

27.6 Long Term Oxygen Therapy

27.6.1 Long Term Oxygen Therapy (LTOT) (16-24 hours daily)

The aims of LTOT are to:

- Correct hypoxaemia without introducing dangerous hypercapnia.
- Improve survival.
- Reduce polycythemia.
- Improve neuropsychological status.
- Improve sleep quality and prevent nocturnal hypoxaemia.
- Prevent right heart failure.
- Improve quality of life.
- Reduce health cost.

Indications for LTOT:

- COPD PaO_2 < 55 mmHg.
- COPD PaO_2 55-60 mmHg with evidence of polycythemia, clinical cor pulmonale, or pulmonary hypertension.
- Restrictive lung disease with PaO_2 < 55 mmHg.

To initiate LTOT, fax a referral to Respiratory Outreach, fax 80849.

27.6.2 Short Term Oxygen Therapy (STOT)

Short term oxygen therapy is required for patients with COPD, restrictive lung disease, and other respiratory disorders in which there is significant hypoxaemia (PaO_2 < 50 mmHg), who need supplemental oxygen while recovering from acute illness. The primary purpose is to enable hospital discharge. Patients must be followed up within six weeks and reassessed. Patients will need to be informed that this is for short term only.

27.6.3 Portable O2

Portable O_2 must be approved by a Respiratory Consultant.

27.7 Spirometry

Spirometry should be considered for patients with symptoms or signs of lung disease such as cough, dyspnea, wheezing, hyperinflation. It can be useful to assess the severity of disease, progression of respiratory disease, or response to treatment.

The first step in interpreting spirometry should be to assess the FEV1/FVC ratio. If this ratio is less than the lower limit of the reference range (included with the test report) an obstructive defect is present. If the ratio is greater than the lower limit of normal then the spirometry is normal or a restrictive defect may be present. In this situation the next step is to look at the FVC. A normal FVC effectively excludes restrictive lung disease, a reduced FVC can be spurious, but further testing should be considered as 50% of these patients may have a restrictive process.

Once you diagnose obstructive lung disease on the FEV1/FVC pattern, the FEV1 can be used to assess the severity of obstructive lung disease. The Thoracic Society of Australia and New Zealand suggests the following pattern:

- FEV1 > 60% predicted = mild obstructive lung disease
- FEV1 40 - 60% predicted = moderately severe obstructive lung disease
- FEV1 < 40% predicted = severe obstructive lung disease.

Occasionally it might be useful to test for reversibility after bronchodilator administration. This test has a 90% specificity for the diagnosis of asthma, but only a 50% sensitivity (i.e., asthma can be missed by this test). The Thoracic Society of Australia and New Zealand suggests that an improvement in the FEV1 of 12% and/or 200 ml suggests significant reversibility and asthma should be considered.

27.8 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.

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.

27.8.1 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 34: 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

DANGER SIGNS: *Exhaustion, confusion, cyanosis, bradycardia, unconsciousness, silent chest on auscultation, signs of respiratory muscle fatigue (indrawing of lower costal margin, abdominal paradox).*

27.8.2 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 35 (see page 228) gives guidelines for the management of patients with asthma according to severity.

Table 35: Management of Asthma

MILD Asthma: Management

- Nebulized salbutamol 5 mg q4h + prn [1]. Prednisone 40 mg orally stat then daily.

Monitoring:

- PEFR after initial treatment then QID. Pulse, respiratory rate QID.

MODERATE Asthma: Management

- Nebulized salbutamol 5 mg q4h + prn [1]. Prednisone 40 mg orally stat then daily.
- **Add** Oxygen to maintain O_2 sat > 95% (usually 2 L/min by nasal cannulae).
- **Contact** Medical Registrar if not improving.
- **Perform** CXR [2] if condition deteriorates or evidence of a complication.

Monitoring:

- PEFR 2-4 hourly. Pulse oximetry [3]. Pulse, respiratory rate, BP QID. Monitor for hypokalaemia which may be exacerbated by beta-agonist therapy.

SEVERE Asthma: Management

- **Increase** Nebulized salbutamol 5 mg up to 2 hourly. Nebulized ipratropium 0.5 mg q4h. Oxygen 8 L/min by Hudson mask. Adjust to maintain O_2 sat >95%.
- **Add** Intravenous access. IV hydrocortisone 200 mg stat then q6h (for 24 hours). Fluids 0.9S 1L 6 hourly initially. IV bronchodilator if not responding to nebulized bronchodilator.
- **Contact** Respiratory Physician.
- **Perform** CXR [2] in all cases.

Monitoring:

- ICU or high dependency unit. Oximetry / arterial blood gases
- Continuous ECG. Pulse, respiratory rate, BP 2 hourly. Special nurse.
  Serum potassium 12 hourly

Asthma With DANGER SIGNS PRESENT: Management

- **Increase** Oxygen to high flow system.
- **Contact** ICU team and Respiratory Physician.
- **Add** IV salbutamol 250 mcg loading dose then salbutamol infusion. See Table 36 (see page 229).
- **Monitoring:**
- Resuscitation or Intensive Care Unit. Nurse and doctor to stay with patient at all times.

Notes

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

(2) 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.

(3) 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.

Table 36: Life Threatening Asthma

Clinical

- FEV_1 or PEFR <33% predicted (or of usual best).
- Silent chest, cyanosis, or feeble respiratory effort.
- Bradycardia or hypotension.
- Exhaustion, confusion or coma.

Management

- High Flow Oxygen (40 - 60%).
- Salbutamol + ipratropium 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.

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.

27.8.3 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.
    - $Beta_2$ 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.
- **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 $beta_2$ agonist.
    - Add ipratropium bromide 0.5 mg q6h via a nebuliser.
    - Consider using an intravenous bronchodilator.
  - Consider the possibility of a complication or an alternative diagnosis:
    - Pneumothorax.
    - Cardiac arrhythmia.
    - Left ventricular failure.
    - Laryngeal or tracheal obstruction.
    - ARDS.
    - Pulmonary embolism.
    - Post transfusion acute lung injury.

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 contraindicated.
  - 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.
- **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.**

27.8.4 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.

27.8.5 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 Self-Management 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 nurse educator or respiratory physiotherapist.
  - Check inhaler technique and instruction on the use and interpretation of readings from a peak flow meter.
  - Introduction to the Asthma Self-Management Plan and basic self-management skills.
  - An arrangement for ongoing follow-up and education as an outpatient.

Options for Ongoing Education as an Outpatient

- Respiratory Physician.
- Respiratory Nurse Educator - Respiratory Outpatient Unit.
- General Practitioner/Practice Nurse.
- Community Asthma Educator - Canterbury Asthma Society.

27.8.6 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 $beta_2$ 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.
  - $Beta_2$ agonist inhaler to use as required (**NOT** regularly).
  - Advice regarding common side effects of these medications:
    - $Beta_2$ agonists: palpitations, anxiety, cramps.
    - Inhaled steroids: dysphonia, thrush - use mouth rinsing and a spacer.
    - Prednisone (short courses): euphoria or dysphoria, hypertension, hyperglycaemia, mild indigestion, insomnia.

Note: *Patients prescribed inhaled corticosteroids in higher doses (>800 mcg) should be encouraged to use a large volume spacer device.*

27.9 Community Acquired Pneumonia (CAP)

27.9.1 General Points

- Over 300 patients a year are admitted to Christchurch Hospital with CAP. It can be a severe disease with mortality of around 5%.
- Clinical features and initial investigations seldom identify a causative agent so empiric therapy based on local epidemiological data and disease severity is typically required.
- Streptococcus pneumonia is the most common causative pathogen in CAP (approx. 50% of cases).
- Early delivery of antibiotics is one of the few factors shown to favourably influence patient outcome - **SO DON'T DELAY (delays increase mortality)**.

27.9.2 Diagnosis

- CAP typically presents with a variable complex of symptoms including fever, pleuritic chest pain, shortness of breath, cough, and sputum. Elderly patients with CAP more frequently present with non-specific symptoms and are less likely to have fever than younger patients. Chest signs on clinical examination are variable, ranging from clear signs of consolidation with focal bronchial breath sounds, to just a few crackles, to no focal signs at all. Chest x-ray should be used to confirm the clinical suspicion of CAP.
- Differential Diagnosis - Consider LVF, PE, aspiration pneumonitis, lung carcinoma, and chronic interstitial lung disease.

  Note: acute intrabdominal pathologies such as pyelonephritis and acute cholecystitis can mimic CAP.

27.9.3 Investigations

- Refer to the description of the CURB-age score (see page 233).
- All patients with severe CAP (CURB-age score 3-5) should have blood drawn for blood culture - 2 sets before antibiotics (10 mls in each bottle).

  Immunocompetent patients with mild to moderate CAP (CURB-age score 0-2) and no complications do **not** require blood cultures to be taken. However, blood cultures should still be taken from this group of patients if they have a prosthetic device, evidence of sepsis (fever >38.5°C, hypotension etc), history of possible bacteraemia (e.g., rigors), or suspicion of Staphylococcal pneumonia.
- 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 hours of warming to room temperature.
- Consider whether specific tests are indicated:
  - Urinary pneumococcal antigen.
  - Legionella immunofluorescence, PCR, urinary legionella antigen.
  - ZN stain and culture for TB.
  - Stains for Pneumocystis carinii (induced sputum).
- Oximetry (or ABGs for severe cases or where there is chronic respiratory or cardiac disease.)
- 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].

27.9.4 Additional Investigations

- Pleurocentesis (see page 50):
  - 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 bottle as soon as possible after taking.*
  - Contact Respiratory service **early** if empyema or complicated para-pneumonic effusion suspected.
- Bronchoscopy. Indications include:
  - Immunosuppressed patient.
  - Life threatening pneumonia.
  - Multiple CXR changes.
  - Deterioration despite appropriate initial treatment.
  - Contact the Respiratory Physician on-call.

27.9.5 Management of CAP

Resuscitate

- **A**irway
- **B**reathing
- **C**irculation

Severity assessment, site of care, and antibiotic selection

This is an essential aspect of the initial management of patients presenting with CAP. It should be used to guide admission decision, antibiotic selection, and site of inpatient care (Ward vs ICU).

In general, clinicians are poor at identifying both high risk and low risk CAP patients. In turn they tend to under-treat severe CAP with high mortality risk and over-treat mild CAP with low mortality risk. In response there are now 2 well-validated disease severity assessment tools for CAP, based largely around prediction of mortality to aid clinician decision making. The Pneumonia Severity Index (PSI) involves a 2 step process involving over 30 clinical variables. The **CURB**-age score is used in Christchurch largely due to ease of use.

CURB-age score stratifies mortality risk with 5 variables (1 point each):

- **C** = Confusion (MSQ 8 or less, or new disorientation).
- **U** = Urea > 7 mmol/L.
- **R** = Respiratory rate > 30/min.
- **B** = Systolic BP < 90 mmHg or diastolic BP < 60 mmHg.
- Age = ≥ 65 years.

If **CURB**-age score 0-1 (Mild CAP):

- Low mortality (< 2%).
- Consider outpatient management so long as no significant co-morbidity and adequate social supports.
- Use single agent oral antibiotic e.g., macrolide, beta-lactam or doxycycline.

If **CURB**-age score 2 (Moderate CAP):

- Intermediate mortality (5-10%).
- Inpatient management.
- **Use single agent beta-lactam IV antibiotic.**
  - Young patients (< 50yrs), non smoking, and no underlying lung disease: **IV Amoxycillin 1 g q8hr** (alternatives - IV benzylpenicillin or clarithromycin).
  - Older patients (> 50yrs) COPD, smokers: **IV Augmentin 1.2 g q8hr** (alternative IV cefuroxime).
- Consider early switch to oral antibiotic - see below.

If **CURB**-age score 3-4 (Severe CAP):

- High mortality (10-50%).
- Inpatient management.
- **Dual antibiotic therapy:**
  - **IV Augmentin 1.2 g q8h + IV Clarithromycin 500 mg q12h**.

For **CURB**-age score 4-5, consider ICU referral and wider spectrum antibiotic cover:

- **IV Clarithromycin 500 mg q12h + IV Ceftriaxone 2 g q12h + IV Gentamicin 5 mg/kg q24h initial dose**.

Note:

- IV Clarithromycin causes phlebitis and should be diluted in 250 mls 0.9S and given via a large vein over 30 minutes.
- If staphylococcal pneumonia suspected (multifocal pneumonia ± cavities) IV flucloxacillin 2 g q4h should be added to usual empiric therapy.
- If pneumonia due to Mycoplasma or Legionella species is suspected in mild/moderate CAP a macrolide should be added.
- If Legionella pneumonia is suspected and a patients condition is deteriorating then add oral rifampicin 600mg q12h or oral ciprofloxacin q12h and contact Respiratory or Infectious Diseases Physician.

Failure to respond to initial antibiotic and supportive therapy

- Consider the presence of penicillin resistant Streptococcus pneumoniae (PRSP) - consider the role of high dose penicillin.
- Alternate diagnoses.
- Resistant organism (always consider TB).
- Development of complication (e.g., complicated parapneumonic effusion/empyema).
- Alternate source of fever (e.g., drug or phlebitis).

Switch from IV to oral antibiotic therapy

Duration of IV antibiotic therapy has been shown to be the major determinant of length of hospital stay in Christchurch Hospital. Indications for switch from IV to oral:

- Clinical improvement.
- Afebrile >24 hr.
- Decreasing WCC.
- Able to tolerate oral therapy.

Discharge planning

- **Duration of therapy**
  - There is little scientific evidence for optimal duration of antibiotic therapy for CAP.

- Recommendations:
  - 7-10 days for uncomplicated pneumonia.
  - 14-21 days for complicated disease (e.g., Legionella pneumonia, COPD, severe CAP).

At discharge:

- Appropriate oral antibiotic as above.
- 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.

Notes:

- 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.

27.9.6 Common Complications

- Parapneumonic effusion - seen in up to 40% of cases. Should always be aspirated to exclude empyema and complicated parapneumonic effusions (see page 239).
- Large simple parapneumonic effusions (>1/3 of hemi thorax), all complicated parapneumonic effusions and all empyemas should be immediately referred to the Respiratory Service.

27.9.7 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 / bronchiectasis.
- Consider referral to a Respiratory Physician.

27.10 Hospital Acquired Pneumonia

- The incidence of Hospital Acquired Pneumonia is around 0.7% in 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.

27.10.1 Investigations

- Sputum sample - involve a physiotherapist if necessary.
- Blood cultures - 2 sets. 10 mL in each bottle.
- WBC + diff.
- CXR.

27.10.2 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, age >65):
    - ceftriaxone 2 g IV q12h **and**
    - gentamicin 5 mg/kg IV initial dose **and**
    - clarithromycin 500 mg q12h IV.

Note: *If on a ventilator or pseudomonas suspected substitute piperacillin/ tazobactam or ceftazidime or imipenem for ceftriaxone.*

27.10.3 References

British Thoracic Society Guidelines. Thorax 2001; 56 (Suppl IV)
American Thoracic Society Guidelines
AMJ Respiratory Critical Care Medicine 2001; 163:1730-54.

27.11 Aspiration Pneumonitis

- Chronic occult microaspiration of gastric contents 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.

27.11.1 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.

27.11.2 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.

27.12 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 37: Acute Pulmonary - Renal Syndrome

Suspected diagnosis	Screening tests
A Idiopathic Vasculitis	
Wegener's Granulomatosis	PR3 ANCA (1)
Microscopic Polyangiitis	MPO ANCA (1)
Anti GBM Disease	Anti-GBM (1)
SLE	ANA
Mixed cryoglobulinaemia	Cryoglobulins
B Non Vasculitic	
Renal vein thrombosis/PE	Radiological
TTP	Blood film
C Infective (a rare cause)	
Mycoplasma	IgM antibody
Legionella	PCR
Mycobacterium	Sputum ZN stain
S. pneumoniae	Sputum Gram stain

(1) Request "urgent vasculitis screen".

27.13 Severe Acute Respiratory Syndrome (SARS)

See Canterbury District Health Board website for the lastest information with regard to this condition and its management.

27.14 Pleural Effusion

See *BTS Guidelines published in Thorax 2003;58 suppl II*

- *For the investigation of a unilateral pleural effusion in adults (pp ii8-ii17).*
- *For the management of pleural infection (pp ii18-ii28).*
- *For the management of malignant pleural effusions (pp ii29-ii38).*

27.14.1 Classification

The differentiation between **exudates and transudates** is the essential first step in the diagnostic evaluation.

Aspiration should not be performed for bilateral effusions in a clinical setting strongly suggestive of a pleural transudate unless there are atypical features or they fail to respond to therapy. At Christchurch Hospital it is suggested to refer patients with large unilateral effusions to the Respiratory Service early (before attempting therapeutic thoracocentesis).

27.14.2 Investigations

- Diagnostic pleurocentesis (see page 50). May be undertaken by medical staff with appropriate experience. Ultrasound guided aspiration should be considered if the effusion is small or loculated. 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.

27.14.3 Contraindications for pleural aspiration

- Unwilling or uncooperative patient.
- Abnormal bleeding tendency. Check history, examination, PT, APTT, and platelets. If in doubt, discuss with Consultant before proceeding.
- Insufficient pleural fluid.
- Chest pyoderma or herpes zoster.

27.14.4 Tests that should routinely be performed on pleural fluid

- Note and document the appearance and any odour of the 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 (if malignancy is a possibility).

27.14.5 Exudate/Transudate

- 99% of exudates meet one or more of the following criteria (Light's 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.
  - Pleural fluid LDH > two-thirds of upper limit of normal serum LDH.
- If transudate - further tests are usually not needed - seek cause, e.g., heart failure, cirrhosis, nephrotic syndrome, acute glomerulonephritis, peritoneal dialysis, myxoedema (but 5% of malignant effusions are transudates).
- Pseudoexudate = treated transudate - a fluid/serum cholesterol ratio of < 0.3 may be seen in patients with LVF treated with diuretics.
- 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.
  - If a diagnosis cannot be obtained on pleural aspiration or if a tuberculous aetiology is suspected, consider a closed pleural biopsy before drainage of the fluid (refer Respiratory Services).
- Tests which may help elucidate the cause of an exudate:
  - Cytology - carcinoma, lymphoma. Repeat specimen if the initial aspirate is negative but malignancy is still suspected.
  - Cell surface markers - to distinguish between reactive and malignant lymphoid proliferation.
  - ZN stain and TB culture if tuberculosis is suspected (consider pleural biopsy and PCR testing).

- Haematocrit - if >50% of peripheral blood haematocrit = haemothorax.
- Rheumatoid factor - rheumatoid effusion.
- Triglyceride - chylothorax.
- Amylase - pancreatitis, ruptured oesophagus, malignancy.

- Other investigations and treatment options include:
  - CT scan (consider contrast enhancement).
  - Closed or image guided pleural biopsy.
  - Intercostal drain.
  - Thoracic surgical referral with regards to a thoracoscopy or thoracotomy and biopsy.

Notes:

- Do not drain the pleural fluid until the diagnosis is established, unless the patient is very dyspnoeic. The presence of some pleural fluid is necessary to perform a closed pleural biopsy.
- If a complicated parapneumonic effusion or empyema is suspected, **do not delay specialist input**. Early treatment with drainage and possibly adhesiolysis (Streptokinase) may prevent a thoracotomy.

27.14.6 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.

The management of anything but a simple parapneumonic effusion should lead to referral to the Respiratory Services. If a complicated parapneumonic effusion or empyema is suspected, **do not delay specialist input**. Early treatment with drainage and possibly adhesiolysis (Streptokinase) may prevent a thoracotomy.

27.15 Spontaneous Pneumothorax

See BTS guidelines for the management of spontaneous pneumothorax. *Thorax 2003;58 (suppl II):ii39-ii52.*

27.15.1 Causes

- Primary: No (known) underlying lung disease.
- Secondary: Underlying lung disease such as COPD, acute severe asthma, Cystic fibrosis, Lymphangioleiomyomatosis.
- Other causes of pneumothorax in general include:
  - Traumatic pneumothorax (these patients should be referred to/discussed with the Cardiothoracic Surgical Service), and
  - Iatrogenic pneumothorax (usually after attempted cannulation of a central vein or after lung or pleural biopsy procedures).

27.15.2 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.

27.15.3 Investigations

- CXR - at the bedside if patient unwell.

  Note: *The CXR tends to underestimate the size of the pneumothorax*

- If the CXR is normal, consider a lateral or lateral decubitus film.
- CT chest is recommended when differentiating a pneumothorax from a complex bullous lung disease, when the plain CXR is obscured by surgical emphysema or when aberrant chest tube placement is suspected. A high resolution CT chest may show evidence of underlying parenchymal lung disease.

27.15.4 Treatment

- Discuss case with Consultant.
- Treatment is not always required. A small closed pneumothorax in the absence of breathlessness should be managed with observation alone.
- Simple aspiration is recommended for a larger spontaneous pneumothorax without underlying lung disease. See protocol in the Respiratory Ward or Emergency Department Guidelines (the Black Book).
- Simple aspiration is less likely to succeed in secondary pneumothoraces and in this situation is only recommended as an initial treatment in small (= less than 2 cm) pneumothoraces in minimally breathless patients under the age of 50 years. These patients should then be admitted under the Respiratory Service for observation.
- **Intercostal tube drainage is recommended in any of 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.
  - Haemopneumothorax.
  - Failed simple aspiration >2500 ml.
- As a general rule tubes are placed in the 'safe triangle', i.e., 5th or 6th intercostal space in the anterior axillary line. These can also be placed in the posterior axillary line. Drains for a pneumothorax are directed upwards, to the apical area. Drains for a pleural effusion or haemothorax are directed downwards to the basal area.

  There is no evidence that large chest tubes (>20 French) are better than smaller tubes (10-14 French). Smaller tubes may be more comfortable for the patient, however there is a greater risk of kinking or blockages and the airflow rate may prove to be insufficient. If a small drain is chosen for a patient with a pneumothorax, the patient must be closely monitored. Signs of an insufficient or dysfunctional drain include worsening respiratory compromise or surgical emphysema.
- Inpatient cases with pneumothorax should be managed by either the Respiratory Service on the Respiratory Ward or by the Cardiothoracic Surgical team if traumatic.
- Technical competence for pleural aspiration or chest drain insertion is essential.

27.15.5 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 [contraindicated].

27.16 Intercostal Tubes

The insertion and management of intercostal tubes is a complex and specialised area. Internal medicine patients requiring chest tube management should be cared for by the specialist respiratory or cardiothoracic surgical team in their respective wards.

The choice of the particular drain and drainage collection system should be discussed with the consultant in charge before the procedure.

Unless it is an emergency, intercostal tubes are inserted or supervised by trained staff only.

27.16.1 Indications

- Pneumothorax.
- Pleural effusion.
- Parapneumonic effusion/empyema.
- Haemopneumothorax.

27.16.2 Contraindications

- Coagulopathy.
- Possibility of bullous lung disease creating the impression of a pneumothorax - consider CT chest.
- Bronchial obstruction on the affected side.
- Chest wall infection.
- Loculated pleural effusion. CT scan advised in this setting. US scan advised prior to the chest drain insertion.
- Known thickening of the visceral pleura seen on CT chest (discuss with Respiratory physician).
- Previous pneumonectomy (discuss with thoracic surgeon or respiratory physician).

27.16.3 Care of chest tubes

Duty medical staff are often asked to assess patients with chest tubes for potential or actual problems. At Christchurch Hospital, the nursing staff from the Respiratory Ward have information and knowledge which may be helpful. If unsure, contact the Respiratory Physician on call.

A worsening pneumothorax or surgical emphysema in a patient with a chest tube in situ means that this is not performing adequately; it may be blocked, kinked, outside the pleural space, or simply too small - with an insufficient flow rate!

- Assessment should include the following:
  - Check the insertion site, all tubes and connections for patency.
  - 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 (= bronchopleural fistula).
  - Obtain a CXR if there is any concern about the patient. Ensure the chest tube is within the pleural space and not in the subcutaneous tissues. Consider a CT chest if still in doubt after the CXR.
  - Consider flushing the intercostal tube with 20-50 ml of sterile Normal Saline under aseptic conditions.
  - Do not clamp chest tubes unless the patient can be closely monitored. Do not clamp tubes during patient transfers.
  - Do not tape over the connections.
  - Never advance a chest tube after the insertion procedure itself (tubes may be withdrawn).

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 surgical emphysema = subcutaneous air:
  - This suggests an insufficiently treated bronchopleural fistula.
  - After checking that the tube is not blocked or kinked and is in the right place, consider the use of low-pressure suction and urgent insertion of a larger tube (24 French or larger).
  - Administer oxygen.
  - Urgent bedside CXR.
  - Call the Respiratory Physician if unsure.

27.16.4 Removal of chest drains

- See special protocol in respiratory services and cardiothoracic surgical services.
- A CXR should be performed after removal of the drain and must be reviewed by the RMO. If the patient deteriorates after the drain removal, an urgent medical assessment is required and, if indicated, a further urgent bedside CXR.

27.17 Haemothorax

- If a heavily blood stained effusion is noted, use a (purple top) EDTA blood container to measure the haematocrit of the fluid; if >50% of peripheral haemotocrit, haemothorax is diagnosed.
- In most cases this is probably due to tearing of pleural adhesions.
- It may be due to malignancy, arterio-venous malformation, but also due to a leaking aortic aneurysm.
- It may be a complication of a pleural aspiration or drain insertion.
- Cases should be discussed with the Cardiothoracic Surgical Service in the first instance.
- In most instance a large bore chest drain is required.

28. Rheumatology and Immunology

28.1 Rheumatology and Immunology Department Information

Main Office

3nd Floor, Riverside, ☎ 80953, Fax 80201

Inpatient Services Ward 24

- Dr P Chapman, ☎ 80953
- Dr J O'Donnell, ☎ 80950
- Dr L Stamp, ☎ 80953

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, ☎ 80492, Fax 80491
- Immunodeficiency and allergic disorders - Chris Martin, ☎ 80950, Fax 81241

The department is closely associated with the Immunology Laboratory, Canterbury Health Laboratories.

28.2 Acute Swelling of a Single Joint

The cause of the acute swelling must be established before any rational form of treatment can be given.

28.2.1 Possible Causes

- Trauma ± haemorrhage.
- Infection (septic arthritis signs may be modified if on steroids or in the presence of chronic arthritis e.g., RA).
- 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.

28.2.2 Investigations

- CBC + Diff, platelets and ESR or CRP.
- Aspirate joint fluid and send to Microbiology for:
  - Gram stain and culture (Send aspirate in sterile tube, capped syringe or inoculate into blood culture bottle).
  - Cell counts and differential (put fluid into EDTA tube and mix).
  - Compensated polarised light examination for crystals (capped syringe).
- Blood culture - 2 sets. Aim for 10 mls per bottle of each set. 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.

28.2.3 Treatment

- **Septic Arthritis**:
  - Splint joint and give analgesia.
  - Use appropriate antibiotic.
    If Gram positive cocci seen or Staphylococci suspected give penicillin 2.4 g and flucloxacillin 2 g both IV q6h.
    If allergic to penicillin give cephazolin 1 g IV q8h if allergy mild or vancomycin if allergy severe (see page 130).
  - Repeat aspiration of synovial fluid daily when effusion is recurrent.
  - Consult orthopaedic service. Most non prosthetic infected large joints will be considered for arthroscopic washout. All suspected prosthetic joint infections should be referred to/discussed with the orthopaedic service.
- **Acute gout or pseudogout**:
  - Initial therapy - Indomethacin 75 mg PO stat then 50 mg q6h or naproxen 750 mg stat then 500 mg BD. 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 20-40 mg PO daily until the acute attack has settled followed by a gradual taper over 7-14 days to avoid rebound attacks **or** intra-articular steroid if 1-2 joints involved and they are accessible to injection) **or** colchicine 1.2 mg stat, followed by 0.6 mg six hourly, up to a maximum dose of 2.4 mg per 24 hours as tolerated. Dose of colchicine should be reduced in the elderly and those with renal impairment and colchicine is relatively contraindicated in patients with a creatinine clearance <50 mls/min.
  - 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 or prednisone for 6 weeks or longer.

  Note: *If renal impairment is a concern, consider oral prednisone or intra-articular steroids.*
- **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.

28.3 Polymyalgic Syndrome/Systemic Inflammatory Disease

- A physician is often faced with the challenge of investigating a patient with the effects of chronic systemic inflammation without obvious cause. There are a multitude of potential causes however many will be associated with specific symptoms and signs that will dictate a sequence of investigations which lead to a diagnosis.
- The following outline is intended as a guide to investigation in those patients without specific symptoms and signs. It should be emphasised that in up to 25% of patients demonstrating chronic systemic inflammation no diagnosis is made.
- The major pro-inflammatory cytokines (IL-1, TNF, IL-6) can per se produce polymyalgia so the diagnosis of polymyalgia rheumatica should be made with caution and only after careful consideration of other potential causes.
- The following list of investigations is not to be used by an uncritical clinician. The list is to be used as a prompt to thoughtful consideration of possible diagnoses and how they might be expeditiously investigated.

28.3.1 Definition

Symptoms of diffuse, often ill-defined muscle and joint pain and stiffness or non-specific malaise associated with raised acute phase proteins and the anaemia of chronic inflammation (usually normochromic normocytic but may be microcytic).

28.3.2 Clinical

A careful (and often repeated) clinical assessment should be the main guide to investigation.

28.3.3 Differential

- Infections (e.g. bronchiectasis, bacterial endocarditis, abdominal abscess).
- Malignancy (especially renal cell carcinoma and lymphoma).
- Connective Tissue Disease/Primary necrotising Vasculitis (consider systemic onset rheumatoid arthritis, polymyalgia rheumatica, Giant cell arteritis, Wegener's Granulomatosis and other small vessel vasculitides).
- Metabolic Disorder (thyroid disease, hypopituitarism, adrenal insufficiency).
- Toxin/Drug (consider all drugs the patient is on and minimise their use as far as possible).

28.3.4 Investigations

- Urinalysis including microscopy.
- CBC + Diff, Na, K, creatinine, urate, Ca, PO_4, alb, bili, alk. phos, AST, ALT.
- Blood cultures (x3 sets with 10mls of blood per bottle).
- Urine culture including TB.
- Chest x-ray (if evidence of chronic lung disease consider chronic pulmonary sepsis and further imaging).
- CT abdomen and pelvis (renal cell Ca, lymphoma, abscess, signs of infection).
- Serology - ANA, ANCA, rheumatoid factor, anti-CCP antibodies, serum and urine protein electrophoresis for paraproteinaemia, total immunoglobulins.
- Mantoux test.
- Temporal artery biopsy.
- Bone marrow examination for leukaemia, myelodysplasia, and TB culture.

Note: *Older persons are often affected by what appears to be an age related "low grade pro-inflammatory state". It is unclear whether this "pro-inflammatory state" is primarily an age related process or secondary to accumulated morbidities e.g. cardiovascular disease. Not infrequently acute phase proteins such as CRP will be 4-5 times the upper limit of the reference range compared to a younger age group.*

28.4 Polymyalgia Rheumatica (PMR)

- PMR is a diagnosis of exclusion. As many diseases associated with a systemic inflammatory response can produce muscle and joint pains, careful clinical assessment of a patient presenting with such symptoms is required.
- Typically however a patient with PMR presents with either the acute or subacute onset of upper and lower limb girdle pain and stiffness. The pain and stiffness may start asymmetrically or just involve the upper or lower limb girdle.
- The patient is generally over the age of 50 and symptoms are almost invariably associated with an elevation in either ESR or CRP. CRP is arguably more sensitive and certainly more specific than the ESR in demonstrating serological evidence of inflammation.

28.4.1 Investigations

- Urinalysis, microscopy, protein culture.
- CBC + Diff, Na, K, creatinine, urate, Ca, PO_4, alb, bili, alk. phos, AST, ALT.
- Other investigations as determined by clinical assessment.

28.4.2 Treatment

- Generally PMR responds to low dose prednisone 10-20 mg per day and this response is seen by many as supporting the diagnosis. In contrast response to high dose prednisone 40-60 mg per day or greater has no clinically discriminatory value. Typically a steroid dose that results in symptom resolution is maintained for 2-4 weeks before gradual reduction. There is no agreed steroid reduction regimen but if a patient starts on 20 mg per day it would be continued for 2 weeks, reduced to 15 mg per day for a further 2-4 weeks, then to 10 mg per day with subsequent reduction by approximately 1 mg per month.
- It is often necessary to maintain a patient on low dose prednisone (8 mg per day for males and 6 mg per day for females) for a period of 6 months or more before complete steroid withdrawal at 1 mg per month.
- In those patients suffering relapse the steroid dose should be increased to that required to control symptoms and further attempts made at steroid reduction and withdrawal.
- If there are continued relapses and the steroid dose cannot be reduced to below 10 mg per day consideration should be given to the introduction of methotrexate. Referral to a Rheumatologist is recommended in this circumstance.
- On average, steroid therapy is maintained for 2-2½ years and therefore there is a risk of steroid induced side effects, in particular osteoporosis. Care should be given to ensuring the patient receives an adequate calcium and Vitamin D intake and if necessary serum Vitamin D levels may need to be measured to ensure no deficiency exists. Patients requiring higher or prolonged doses of prednisone should be considered for a baseline bone density study particularly if they are elderly and relatively immobile.

28.5 Giant Cell Arteritis (GCA)

Giant Cell Arteritis is an infrequent association of polymyalgia rheumatica. Biopsy evidence of GCA may occur in up to 1:20 patients. However patients may present with sudden blindness without prodrome in which case the acute phase markers may not be elevated. Immediate treatment with high dose prednisone is important in reducing the high incidence of blindness developing in the other eye. Temporal artery biopsy is strongly recommended but can be delayed for up to 5-7 days without substantially hindering histological interpretation. Steroid regimen is empirical; a common regimen is prednisone 40-60 mg/day for 1 month, 30-40 mg 1 month, 20-30 mg 1 month, 15-20 mg 1 month, 12.5-15 mg 1 month, then as for PMR (or slower). Most patients require treatment for 2yrs minimum hence importance of osteoporosis prophylaxis.

28.6 Biological agents in rheumatic diseases

- The use of immunosuppressive agents is a major risk factor for infections in patients with rheumatic diseases.
- Signs of infection may be masked in these patients by the underlying disease and its treatment.
- Biological agents targeted against TNF-Alpha (Etanercept (Enbrel), Infliximab (Remicade), adalimumab (Humira)) and IL-1 (Anakinra) are associated with risk of infection.
- Specific infections associated with TNF blockade:
  - Mycobacterium tuberculosis (especially with infliximab).
  - Strep pneumoniae.
  - Listeria monocytogenes.
  - Aspergillus fumigatus.
  - Histoplasma capsulatum.
  - Cryptococcus.
  - Pneumocystis pneumonia.
- Assessment of patients on biological agents:
  - Have a high index of suspicion for sepsis in the unwell patient on biological agents.
  - Patients should be investigated thoroughly for underlying organism; initial investigations should include CBC + Diff, Na, K, creatinine, bili, alk. phos., AST, ALT, blood cultures, urinalysis, sputum culture, CXR.
  - Consider culture/stains for AFB.
  - Other diagnostic imaging may be required to determine the source of infection.
- Empiric treatment:
  - Immunosuppressive therapy should be minimised and no further biological agent should be given until the sepsis has been adequately treated.
  - Broad spectrum antibiotics may be required until the organism is isolated.

OBSOLETE

29. Thrombosis, Embolism, and Anticoagulation

29.1 Venous Thromboembolism DVT and PE

29.1.1 Clinical Features

DVT and PE are difficult to diagnose clinically.

- Suspect DVT if there is swelling, bluish discolouration, pain, tenderness, and dilated superficial veins.
- Suspect PE if central chest pain, dyspnoea, hypoxia, collapse/shock, raised JVP, tachycardia, or arrhythmia are present. If PE has progressed to infarction, haemoptysis, pleuritic chest pain, and pleural effusion may occur.

29.1.2 Precipitating Causes of DVT and PE

- Surgery. Immobilization including travel. Oestrogen therapy (OCP/HRT). Malignancy. Pregnancy and puerperium. Polycythaemia. Thrombocytosis.
- If the patient is under 45, has had recurrent thromboses or there is a family history of thrombosis, **consider** activated protein C (APC) resistance (Factor V Leiden), anti-thrombin III deficiency, protein C or protein S deficiencies, prothrombin gene mutation, hyperhomocysteinaemia, or antiphospholipid antibody syndrome.

29.1.3 Risk Assessment for DVT

Clinical Model to Determine Pre-Test Probability for Deep Vein Thrombosis	Points
Active cancer (treatment within previous six months or palliative)	1
Recent immobilisation >3 days or major surgery within 4 weeks.	1
Local tenderness along the distribution of the deep venous system.	1
Entire leg swollen.	1
Calf swelling 3 cm > asymptomatic side (measured 10 cm below tibial tuberosity).	1
Pitting oedema (greater in the symptomatic leg).	1
Collateral superficial veins (non-varicose).	1
Alternative diagnosis as likely or greater than that of DVT.	-2
Clinical Probability calculated as follows:	
Total points	**Risk**
3 or greater:	**HIGH**
1 or 2:	**MODERATE**
0 or fewer:	**LOW**

Reference: *Lancet* 1997;350:1795-1798

29.1.4 Investigations for Suspected DVT

Assess the clinical probability of DVT (refer to Risk Assessment for DVT on page 248), and get D-dimer result. Proceed as guided below.

- **A negative D-dimer** in a patient with a low/moderate pre-test probability score **excludes a clinically significant DVT.** If the pre-test probability is high, consider an ultrasound.
- **A positive D-dimer** in a patient with suspected DVT needs to be correlated with the pre-test probability score **and** an ultrasound of the affected leg(s). Then proceed as follows:
  - **If the clinical probability is low**:
    - and the U/S is **normal**, DVT is unlikely.
    - and the U/S is **positive**, treat with anticoagulant therapy.
  - **If the clinical probability is moderate**:
    - and the U/S is **normal**, repeat U/S in 48-72 hours(1). If **normal** at 72 hours, this rules out DVT. If **positive** at 72 hours, treat with anticoagulant therapy.
    - and the U/S is **positive**, treat with anticoagulant therapy.
  - **If the clinical probability is high**:
    - and the U/S is **normal** or **equivocal**, consider CT venography (± CTPA). If there is any delay, consider anticoagulant therapy.
    - and the U/S is **positive**, treat with anticoagulant therapy.

(1) There is controversy here. Inpatients should be rescanned within 72 hours. For outpatients, 3 - 7 days may be adequate.

Notes on Investigations for DVT

- The D-dimer assay has a sensitivity for DVT of 89-100% and negative predictive value of 95-100%.
- **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. Hence the need to repeat the ultrasound under these circumstances.
- If there has been a previous clot, it may be difficult to distinguish post phlebitic changes from a fresh clot. Comparison with earlier ultrasounds and assay of D-dimer may help.
- If the ultrasound is negative for DVT, remember that a ruptured Baker's cyst may produce a similar picture and may also be visualised by ultrasound.

29.1.5 Risk Assessment for PE

Geneva Score for Pre-test Probability		
Criteria:		**Points**
Age 60-79 years		1
Age >79 years		2
Prior DVT/PE		2
Recent surgery		3
Heart rate >100 beats/minute		1
$PaCO_2$ mmHg:		
• <36		2
• 36-39		1
PaO_2 mmHg:		
• <49		4
• 49-60		3
• >60-71		2
• >71-82		1
Chest radiograph:		
• Platelike atelectasis		1
• Elevation of hemidiaphragm		1
Interpretation of the score		
Score range	**Mean prob. of PE (%)**	**Risk**
0-4	10	**LOW**
5-8	38	**MODERATE**
9-12	81	**HIGH**

Reference: Wicki J et al *Arch Intern Med* 2001;161:92-97

29.1.6 Investigations for Suspected PE

- All patients with suspected PE should have their pre-test probability assessed using the Geneva Scoring system (see page 250).
- All patients with suspected PE should have bloods taken for a D-dimer assessment. At Canterbury Health Labs an IL D-dimer test is used, and a level of less than 250 ng/ml is considered to be negative. Please check with your laboratory as they may use a different commercial kit.
- Patients with signs of right ventricular dysfunction should have urgent investigations with a CTPA and/or ECHO, as they may benefit from thrombolysis therapy using the standard tPA protocol on page 254.
- Out of normal working hours, and if it is clinically safe to delay establishing the diagnosis, consider anticoagulation overnight and investigate the following morning.

29.1.7 Action to Take When Results are Available

If the D-dimer is negative and:

- the patient has a low or moderate pre-test probability, a clinically significant PE can be excluded.
- the patient has a high pre-test probability, consider CTPA or VQ scans.

If the D-dimer is positive:

- If the CXR is normal, VQ scanning or CTPA scanning can be considered. VQ scanning has fewer potential side effects than CTPA scanning, and may be preferred if renal function is impaired, in iodine allergy, and for young women. **In pregnancy**, a perfusion-only scan may be appropriate rather than a CTPA. Discuss with an obstetric physician. A VQ scan could also be considered if CTPA is equivocal or indeterminate for technical reasons.
- **Patients with a low/moderate pre-test probability** and a negative CTPA or normal VQ scan: excludes clinically significant PE.
- **Patients with a low/moderate pre-test probability** and an intermediate probability VQ: investigate further with CTPA scanning.
- **Patients with a low/moderate pre-test probability** and a positive CTPA and/or high probability VQ: treat with anticoagulation.
- **Patients with a high pre-test probability** and a negative CTPA may require further investigations with U/S of leg veins or VQ scanning. The higher the D-dimer is, the more likely it is that patients have a PE.
- **Patients with a high pre-test probability** and a positive CTPA and/or high or intermediate probability VQ: treat with anticoagulation.

Reference: *BTS Guidelines Thorax* 2003;58:470-484.

29.2 Management of DVT and PE

29.2.1 Management of DVT

The standard treatment consists of LMW heparin and warfarin (refer to Drug Therapy for DVT or PE on page 252).

29.2.2 Management of PE

Pulmonary embolism:

- Anticoagulate (refer to Drug Therapy for DVT or PE on page 252).
- Oxygen.
- Analgesia as required.
- Patients with signs of right ventricular dysfunction should be considered for urgent investigations with a CTPA and/or ECHO, as these may benefit from thrombolysis therapy. Refer to Thrombolytic Therapy for PE on page 254.

29.2.3 Drug Therapy for DVT or PE

- **Low molecular weight heparin**, e.g., Enoxaparin (Clexane) 1 mg/kg q12h SC, or Dalteparin (Fragmin) 200 IU/kg q24h SC.
  - **Duration:** until INR >2 for 2 consecutive days.
  - **Monitoring:** Not usually required for LMWH.

AND

- Commence warfarin 5 mg or 10 mg PO on Day 1 - see below and the nomogram on page 253 for essential information concerning both this initial dose and subsequent doses.
  - **Duration:**
    - **DVT -** 3 months if reversible risk factors. 6 months if idiopathic.
    - **PE** - 6 months if reversible risk factors. 12 months if idiopathic.

    Consider indefinite treatment if rethrombosis risk greater than risk of bleeding from therapy.
  - If recurrent DVT or PE, consider lifelong anticoagulation.

Monitoring: Check INR daily for 5 days. Then if stable twice weekly for 2 weeks then PRN (maximal interval 1 month). Check the desired INR range (refer to Recommended INR Levels for Warfarin Treatment on page 253).

Note: *The following information needs to be considered before starting this therapy.*

- CBC+ Diff.
- Na, K, creatinine, alb, bili, alk. phos., GGT, AST.
- **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 prothrombin gene mutation and fasting homocysteine assay.
- **Heparin**
  - **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 fewer bleeding complications.
  - Low molecular weight heparins that are currently available for the treatment of DVT include dalteparin (Fragmin) and enoxaparin (Clexane).
  - Studies have shown that **outpatient treatment of DVT and less extensive PE** with LMW heparin is safe and effective. If you wish to treat as an outpatient, use the referral form provided and contact the haemostasis nurse (☎ 81246).
- **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.
  - Aim to start warfarin 5 days before it is planned to stop heparin. During this time check PT daily.
  - Patients may be more sensitive to warfarin if they are over 65, and/or have low body weight, altered liver function tests, or are on drugs known to increase sensitivity to warfarin.
  - **Patients who are generally well, relatively young, and with no co-morbidities could be started on warfarin 10 mg on Day 1 and 10 mg on Day 2 and then follow the nomogram on page 253 . For all other patients, we recommend 5 mg warfarin on Days 1 and 2.**

Nomogram for the first 5 days of warfarin treatment

Day:	INR:	Warfarin Dose
1	Within normal range	5 mg or 10 mg, see text
2	<1.5	5 mg or 10 mg, see text
	1.5-1.9	3 mg
	2.0-2.5	1 mg
	>2.5	seek advice
3	<1.5	5 mg
	1.5-1.9	3 mg
	2.0-2.5	2 mg
	2.5-3.0	1 mg
	>3.0	seek advice
4	<1.5	10 mg
	1.5-1.9	6 mg
	2.0-3.0	2 mg
	>3.0	seek advice
5	<1.5	10 mg
	1.5-1.9	8 mg
	2.0-3.0	3 mg
	>3.0	seek advice

Notes:

- Two commercial preparations of warfarin are available in New Zealand - Marevan®, 1, 3, and 5 mg tablets and Coumadin® 1, 2, and 5 mg tablets. They are not pharmacologically interchangeable! i.e., 1 mg of one may not equate to 1 mg of the other.
- We suggest for inpatients and at discharge only 1 mg tablets of warfarin are prescribed, to minimise confusion over dosage and tablet size.

29.2.4 Recommended INR Levels for Warfarin Treatment

Recommended INR Levels		
	Prothrombin Ratio (INR)	**Duration**
Pre and perioperative anticoagulation	1.5-2	Days
Treatment of DVT	2-3	12-26 weeks
Treatment of PE or massive DVT	2-3	26-52 weeks
Treatment of recurrent DVT or PE(1)	3-4	? life long
Atrial Fibrillation	2-3	life long
Prosthetic heart valves	2-4	life long
Arterial disease	3-4	life long
(1) Recurrence despite prothrombin ratio between 2 and 3.		

29.2.5 Drug Interactions with Coumarin-type Oral Anticoagulants

Some Drugs Expected to Potentiate Warfarin Effect(1)

- **Antibacterials**: cephalosporins, cotrimoxazole, isoniazid, macrolides, metronidazole, penicillins, quinolones, tetracycline.
- **Antifungals**: fluconazole, itraconazole, ketoconazole, miconazole.
- **Cardiovascular**: amiodarone, antilipid drugs, propranolol, quinidine, verapamil,
- **Central Nervous**: antidepressants (tricyclics, selective serotonin reuptake inhibitors (SSRI)).
- **Gastrointestinal**: cimetidine, omeprazole.

Some Drugs Expected to Decrease Warfarin Effect(1)

- **Antibacterials**: rifampicin.
- **Cardiovascular**: colestipol.
- **Central Nervous**: barbiturates, carbamazepine, phenytoin.
- **Others**: cholestyramine, Vitamin K, Vitamin K rich foods, e.g., avocados, broccoli.

Note: Herbal medicines may interact with oral anticoagulants (e.g., gingko, fish oils - increased warfarin effect; e.g., St. John's wort, ginseng - reduced warfarin effect).

(1) Care may be required for drugs or herbal medicines not included in this list. Check with Ward Pharmacist or Drug Information (Ext. 80900) if unsure.

29.2.6 Thrombolytic Therapy for PE

- Thrombolytic agents should be considered in **life-threatening PE**.
- Thrombolysis should not be used as first line treatment in non-massive PE.
- If there has been a massive PE - that is, one so severe as to cause circulatory collapse and/or right ventricular dysfunction - recommended practice is to use thrombolysis, the earlier the better. In patients with right heart thrombus, mortality with thrombolysis is a third of that with heparin.
- **If** the investigations have confirmed PE and the above criteria are satisfied, give tPA (alteplase) 10 mg IV over 1-2 mins and then 90 mg by IV infusion over 2 hours. The total dose for patients less than 65 kg should not exceed 1.5 mg/kg.
- **If no abnormal bleeding occurs, follow the alteplase infusion with low molecular weight heparin and warfarin as recommended in Drug Therapy for DVT or PE** on page 252.

Contraindications to Thrombolysis

Absolute Contraindications:

- Any prior intracranial haemorrhage.
- Known structural cerebral vascular lesion.
- Known malignant intracranial or spinal neoplasm or arteriovenous malformation.
- Ischaemic stroke within 6 months.
- Neurosurgery within 6 months.
- Suspected aortic dissection.
- Active bleeding or bleeding diathesis (excluding menses).
- Significant closed-head or facial trauma within 3 months.
- Uncontrolled hypertension on presentation (SBP >180 mmHg or DBP >110 mmHg).
- Recent internal bleeding within 6 weeks.
- Major surgery or major trauma < 2 weeks.

Relative Contraindications (to be discussed with Physician):

- Transient ischaemic attack < 6 months.
- Traumatic cardiopulmonary resuscitation < 2 weeks.
- Non compressible vascular puncture.
- Pregnancy.
- Active peptic ulcer.
- Current use of anticoagulants with an international ratio >2: the higher the INR, the higher the risk of bleeding.

Note: *Long term benefits of fibrinolysis for life-threatening PE are not yet clearly defined. The risk of bleeding is higher with thrombolysis than heparin and it is less easily reversed.*

Reference: *BTS Guidelines Thorax* 2003;58:470-484.

29.3 Management of Patients on Warfarin Therapy Undergoing Surgery

Long term oral anticoagulants may be given for atrial fibrillation, prosthetic heart valves, history of venous thromboembolism or arterial emboli. In each patient the risk of surgical bleeding must be balanced against the risk of recurrent (or new) thrombosis or emboli. The following is a suggested management plan for patients having elective surgery. However the final decision on what prophylaxis to use (if any) is taken by the surgeon caring for that patient.

29.3.1 Management of Patients on Warfarin undergoing Surgery [1]

If DVT or PE <1 month ago (defer surgery if possible) or Acute Arterial emboli[2] <1 month ago

- **Before Surgery:**
  1. **Withold warfarin for 4 days prior to operation day**. The aim is to allow INR to drop to <1.5 on day of surgery.
  2. Commence LMWH (e.g., enoxaparin 1 mg/kg BD) **at treatment dose when INR <2**. Last dose prior to surgery given in morning, the day BEFORE surgery, i.e., no LMWH for 12-24 hours prior to surgery.

     OR
  3. Commence IV unfractionated heparin when INR <2. Stop 6 hours prior to surgery.
  4. **Test INR on day of surgery**. If still ≥ 1.5 discuss with surgeon and anaesthetist.
- **After Surgery:**
  1. Restart warfarin (patient's usual daily dosing) AND either IV unfractionated heparin or LMWH at treatment dose, commencing 12-24 hours after surgery. Discuss with surgeon/ anaesthetist prior to recommencing therapy.
  2. Continue with heparin until INR >2.

If DVT or PE >1 month ago or Acute Arterial emboli[2] >1 month ago

- **Before Surgery:**
  1. **Withhold warfarin for 4 days prior to operation day**. The aim is to allow INR to drop to <1.5 on day of surgery.
  2. Commence on LMWH at **prophylactic dose** eg. Enoxaparin 40 mg SC daily. Last dose given on the day BEFORE surgery.
  3. Test INR on day of surgery. If INR ≥1.5 discuss with surgeon and anaesthetist.
- **After Surgery:**
  1. Continue with LMWH at prophylactic dose after procedure, preferably on day of surgery.
  2. Restart warfarin (patient's usual daily dosing) 12-24 hours after the surgery. Ensure therapy commenced only after discussion with surgeon and/or anaesthetist.
  3. Continue with heparin until INR >2.

If in Atrial Fibrillation

- **Before Surgery:**
  1. **Withhold warfarin for 4 days prior to operation day**. The aim is to allow INR to drop to <1.5 on day of surgery.
  2. Test INR on day of surgery. If INR ≥1.5 discuss with surgeon and anaesthetist.
- **After Surgery:**
  1. Restart warfarin (patient's usual daily dose) preferably on evening of day of surgery. Ensure therapy is recommenced only after discussion with surgeon and/or anaesthetist.

Note: *The INR is likely to be subtherapeutic for 5-7 days.*

If Prosthetic Heart Valves

If uncertain about management before or after surgery, discuss with cardiac surgeon.

1. **Mechanical aortic valve** [3] **only** inserted >6 months ago and no other additional risk factors:
   - **Before Surgery:**
     Thromboembolic risk is low, follow regimen as for atrial fibrillation.
   - **After Surgery:**
     Regimen as for atrial fibrillation
2. **Other valves, multiple valves, valve replacement <6 months ago or additional risk factors** [4]**:**
   - **Before Surgery:**
     Thromboembolic risk is **high,** follow regimen as for DVT/PE <1 month ago.
   - **After Surgery:**
     Regimen as for DVT/PE <1 month ago.

Notes

(1) For **emergency surgery** in patients on warfarin therapy, an INR of <1.5 can usually be achieved by infusion of fresh frozen plasma and IV vitamin K 1mg. **However, do not give vitamin K to a patient with a prosthetic valve without prior discussion with cardiac surgeon.**

(2) With a history of arterial emboli, concurrent aspirin therapy is also an important part of the prevention of further episodes - cessation of aspirin therapy as well as warfarin needs to be considered in light of the risk of bleeding versus emboli.

(3) St Jude Medical Bileaflet aortic valve, CarboMedics Bileaflet Aortic Valve, Medtronic-Hall tilting disk aortic valve.

(4) **Risk factors:** History of TIA's, CVA, systemic emboli, atrial fibrillation, severe LV systolic dysfunction, recurrent CHF, previous thromboembolism, hypercoagulable conditions.

Haemostasis Service, Haematology*: Phone 81246. This service comprises a haematologist (Dr Mark Smith) and two nurse specialists (Alison Inder and Carolyn Lauren) and is a specialist coagulation resource for the CDHB. It provides for outpatient anticoagulation including administering and/or teaching self administration of LMWH and is a consultancy service for difficult cases of coagulopathy.*

29.4 Treatment of Anticoagulant Overdosage

29.4.1 Heparin

- Protamine sulphate has been used to reverse overdosage with unfractionated heparin. Reversal is only necessary if there is serious bleeding. The dose of protamine is 1 mg IV per 100 IU heparin estimated to be remaining in the circulation. Protamine must be given slowly, and may cause serious allergic reactions (increased risk in those with either previous protamine exposure or with fish allergy). There is less experience with its effect in neutralising LMWHs. Studies in healthy volunteers indicate that 65-80% of the anti-Xa activity is neutralised by protamine sulphate 1 mg IV per 100 anti-Xa units of LMWH. A return of anti-Xa effect is seen 3 hours after LMWH reversal, probably due to continuous absorption of LMWH from the subcutaneous depot. It may be necessary to give protamine intermittently or as continuous infusion to achieve and maintain neutralisation of subcutaneous LMWH for 12-24 hours. The patient must be carefully monitored.

  Caution: *Excess protamine sulphate may act as an anticoagulant itself.*

29.4.2 Warfarin

The Australasian Society of Thrombosis and Haemostasis have prepared the following consensus guidelines (Medical Journal of Australia 2000. 172:600-605):

Managing Bleeding or an Overdose during Oral Anticoagulant (Warfarin) Therapy

▪ **Clinical Setting:** INR greater than 5 but less than 9
  ▪ **Action:** Stop warfarin, give 1-2.5 mg vitamin K_1 IV, measure the INR in 6-12 hours, restart warfarin at a reduced dose once the INR is less than 5.
▪ **Clinical Setting:** INR greater than 9 with no bleeding
  ▪ **Action:** Stop warfarin, give 5 mg vitamin K_1 IV, measure the INR in 6-12 hours, restart warfarin at a reduced dose once the INR is less than 5. Coagulation factor replacement to be given if there is a high risk of bleeding.
▪ **Clinical Setting:** Major bleeding at any level of INR
  ▪ **Action:** Stop warfarin, give 5 mg vitamin K_1 IV, transfuse coagulation factor replacement, measure the INR as required, assess need to restart warfarin.

Give Vitamin K_1 by slow IV infusion and Prothrombinex (Factors II, IX, and X) together with Factor VII (if available). The dose should be based on 50 IU of Factor IX and/or Factor VII per kg body weight. If no concentrate is available, fresh frozen plasma should be transfused but this is less effective. The dose for an adult is 12-15 mls/kg.

29.4.3 Streptokinase or tPA

Abnormal haemorrhage may be very difficult to correct at least for some hours. If fibrinogen level is low, cryoprecipitate may help.

29.5 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.

- **Recommended Prophylaxis Schedule**

  Enoxaparin (Clexane) 40 mg SC daily.

- **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**
  - Early mobilisation and optimal hydration should be maintained as far as possible from the outset.
  - All patients will have the adequacy of the lower limb arterial circulation, sensation and fragility of the skin assessed by the admitting medical staff to determine whether graduated compression stockings are contraindicated.
  - Graduated compression stockings should be applied in all stroke patients who are immobile and/or with a paresis of their legs, providing such stockings are not contraindicated as above.
  - Graduated compression stockings should be full length in patients with hemiparesis.
  - Patients who are fully anticoagulated with warfarin do **not** need graduated compression stockings as above.

  For patients with ischaemic stroke only:
  - Aspirin 100-150 mg per day should be given unless contraindicated.
  - Prophylactic anticoagulation with standard or low molecular weight heparin should **not** be used routinely because of risk of haemorrhagic transformation within an 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). A recent study showed a significant reduction in DVT rate using enoxaparin 40 mg daily.

29.6 Surgical DVT Prophylaxis

Surgery is a significant cause of deep vein thrombosis and post operative death from pulmonary embolism may occur from an unrecognised DVT. The risk of venous thromboembolism depends on the type of surgery, patient characteristics and the underlying disease.

Risk Factors For DVT

- **Patient Factors**: Age (>40); obesity; varicose veins; immobility; pregnancy; oestrogen therapy; previous venous thromboembolism; thrombophilia, e.g., deficiency of AT III, proteins C or S, factor V Leiden, prothrombin mutation, or presence of antiphospholipid antibodies.
- **Disease Factors**: Pelvic and leg trauma; malignancy; heart failure; paralysis of leg(s); infection; inflammatory bowel disease; nephrotic syndrome; polycythaemia.
- **Surgery/Anaesthesia**: Duration of surgery; leg surgery.

Risk Prediction

By combining the above factors, surgical patients can be stratified into low, moderate or high risk groups:

- **Low Risk:**
  - Minor surgery (<30 minutes): any age and no other risk factors other than age.
  - Major surgery (>30 minutes): no risk factors, age less than 40 years.
- **Moderate Risk:**
  - Minor surgery and thrombophilia or previous venous thromboembolism.
  - Major surgery, age >40 but no other risk factors.
- **High Risk:**
  - Major surgery and thrombophilia or previous venous thromboembolism.
  - Pelvic or abdominal surgery for cancer.
  - Orthopaedic surgery of pelvis or leg.

Guidelines for Prophylaxis

Confirmation of any pre-operative heparin dose and timing must be made with the surgeon and/or anaesthetist who will make the final decision with regard to what prophylaxis is used (if any). The use of spinal/epidural anaesthesia introduces the potential risk of spinal haematoma if heparin is administered close to the time of the procedure or the removal of the epidural catheter (see below).

- **Low Risk:**
  - Early mobilisation
- **Moderate Risk:**
  - Dalteparin (Fragmin) 2,500IU subcut two hours pre-op then daily.
  - **or** Enoxaparin (Clexane) 20mg subcut two hours pre-op, then daily.

  See guidelines below for use of LMWH if spinal/epidural anaesthesia likely.
- **High Risk:**
  - Dalteparin 5,000IU subcut evening before surgery then daily.
  - **or** Enoxaparin 40mg subcut two hours pre-op then daily.

  See guidelines below for use of LMWH if spinal/epidural anaesthesia likely.

If heparin is contraindicated use compression techniques.

Spinal/Epidural Anaesthesia

- If spinal/epidural is a possible mode of anaesthesia, then LMWH is required to be given at least **12 hours or more BEFORE** procedure is to take place (eg. 5.00 pm administration day before surgery would be suitable). Subsequent dose is given at least **two hours or more AFTER** the procedure.
- If unable to administer LMWH 12 hours or more before the procedure, eg. day of surgery admission, then omit pre-op LMWH unless it is certain that spinal/epidural will not be administered. The anaesthetist will administer the LMWH or give instructions as to when LMWH may be given.
- Removal of the epidural catheter also requires that the LMWH was given at least 12 hours earlier. Once catheter is removed, any subsequent dose of LMWH should not be given for at least two hours. If any concerns contact the Duty Anaesthetist (Pager 8120).

29.7 Acute Limb Ischaemia

29.7.1 Common causes

- Arterial embolism
  - Usually no previous history of peripheral occlusive arterial disease (POAD).
  - Sudden onset usually in a patient in atrial fibrillation but sometimes following myocardial infarction.
- Arterial thrombosis in situ
  - Often associated with previous history of POAD.
  - Examination findings may suggest widespread vascular disease (e.g., absent contralateral pulses).

29.7.2 Clinical findings

- Acute limb ischaemia is a clinical diagnosis. Hand-held Doppler analysis of peripheral pulses is a valuable adjunct, but is not a substitute for clinical examination. This may or may not be supplemented by radiological imaging.
- Symptoms and signs: Ischaemic rest pain with/without paraesthesia or loss of function (paralysis) in the context of absent pulses, ± pallor, ± reduced temperature of the affected limb. (Remember the 6 Ps: Painful, Pale, Pulseless, Paraesthesia, Paralysis, and Perishing cold.) This is usually obvious with emboli but can be more subtle with acute-on-chronic ischaemia.

29.7.3 Actions

1. Assess severity:
   - **Rutherford I**: Pain at rest resolved and no paraesthesia or paralysis.
     Consider admission for heparin anticoagulation.
   - **Rutherford II**: Ischaemic rest pain ± paraesthesia.
     Consider angiography ± intra-arterial thrombolysis.
   - **Rutherford III**: Ischaemic rest pain, paraesthesia, and paralysis.
     Consider immediate surgical exploration.

   Rutherford II and III should be managed as a medical emergency. The window for therapeutic intervention is approximately 6 hours from onset before muscular necrosis will occur. Urgent surgical consultation is mandatory.
2. Investigations: CBC + Diff, Na, K, creatinine, and urea.
3. Contact vascular registrar/consultant on call.
4. Analgesia.
5. Give oxygen.
6. If no major contraindications, heparinise (5000 IU bolus of unfractionated heparin IV) to minimise secondary thrombosis.
7. Leave limb alone. Protect from trauma, take pressure off heel, and do not heat or cool.

30. Diseases Notifiable in New Zealand

(Includes suspect cases)

30.1 Information for the Medical Practitioner

Section 74 of the Health Act (1956) places the following obligations upon medical practitioners:

"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.
2. Repealed.
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."

30.2 Venereal Diseases

Section 89 of the Health Act (1956) places the following obligations upon medical practitioners:

"Duty of medical practitioner as to patient suffering from venereal disease:

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 [having a sexual relationship] until he has been cured of that disease or is free from that disease in a communicable form; and

(c) Give to the patient such printed information relating to the treatment of venereal disease, and to the duties of persons suffering from such disease, as may be issued by the directions of the Minister.

Venereal Diseases Regulations 1982

"Regulation 7. Duties of medical practitioner

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)."

30.3 Diseases Notifiable in NZ (including suspect cases)

Note: *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.*

Notifiable Infectious Diseases Under the Health Act 1956

Section A - Infectious Diseases Notifiable to Medical Officer of Health and Local Authority

- Acute gastroenteritis[1]
- Campylobacteriosis
- Cholera
- Cryptosporidosis
- Giardiasis
- Hepatitis A
- Legionellosis
- Listeriosis
- Meningoencephalitis - primary amoebic
- Salmonellosis
- Shigellosis
- Yersiniosis
- Typhoid and paratyphoid fever

Section B - Infectious Diseases Notifiable to Medical Officer of Health

- Acquired Immunodeficiency Syndrome (AIDS)
- Anthrax
- Arboviral diseases
- Brucellosis
- Diphtheria
- *Haemophilus Influenzae b*
- Hepatitis B
- Hepatitis C
- Hepatitis (viral) - not otherwise specified
- Highly Pathogenic Avian Influenza

- Hydatid disease
- Leprosy
- Leptospirosis
- Malaria
- Measles
- Mumps
- *Neisseria meningitidis* invasive disease
- Pertussis
- Plague
- Poliomyelitis
- Rabies
- Rheumatic Fever
- Rickettsial diseases
- Rubella
- Severe Acute Respiratory Syndrome (SARS)
- Spongiform encephalopathies including *Creutzfeldt-Jakob Disease*
- Tetanus
- Viral haemorrhagic fevers
- Yellow Fever

(1) **Note:** 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*.

Diseases Notifiable to Medical Officer of Health (other than Notifiable Infectious Diseases)

Notifiable to the Medical Officer of Health

- Cysticercosis
- Decompression sickness
- Taeniasis
- Trichinosis
- Lead absorption equal to or in excess of 15mcg/dl (0.72mcmol/L)(1)
- Poisoning arising from chemical contamination of the environment

(1) Note: Blood lead levels to be reported to the Medical Officer of Health (15mcg/dl or 0.72 mcmol/L) are for environmental exposure. Where occupational exposure is suspected, please notify OSH through the NODS network.

Notifiable Diseases Under Tuberculosis Act 1948

Notifiable to the Medical Officer of Health

- Tuberculosis (all forms)

30.4 Immunisation Information For International Travel

Information on immunisation for overseas travel is available from the following internet web page: http://www.fit-for-travel.de/en/default.asp.

31. Referral of Patients to Hyperbaric Medicine Unit (HMU)

A double-compartment, four-patient recompression chamber for treatment with **Hyperbaric Oxygen (HBOT**) is operational at Christchurch Hospital. The chamber is administered by the Hyperbaric Medicine Unit.

The CDHB Intranet provides general information on the Hyperbaric Medicine Unit and HBOT under Our Organisation > Christchurch Hospital > Christchurch Hospital Departments > Hyperbaric Unit.

31.1 Emergency Referrals

- Ring Christchurch Hospital Operator (Internal '0' or external 03 364 0640) and request the Hyperbaric Unit Duty Doctor.
- Give the Operator your name, contact phone number, and location.

  Note: *Trying to contact individual clinicians may result in delay.*

31.2 Acute Emergency Indications for HBOT

(Refer to the HMU section of the CDHB Intranet.)

- Decompression Illness (the 'Bends' and cerebral arterial gas embolism; refer to the Emergency Department Guidelines).
- Arterial gas embolism (from any cause, including iatrogenic, e.g., cardiopulmonary bypass).
- Anaerobic necrotising soft tissue infections, irrespective of the suspected causative organism(s) (e.g., clostridial myonecrosis, streptococcal necrotising fasciitis); combined in a planned way with surgery and antibiotics.
- Carbon monoxide poisoning (possibly cyanide and H_2S also); smoke inhalation (see page 202).
- Crush injury with acute traumatic ischaemia.
- Intracerebral abscess.
- Compromised skin grafts and flaps.
- Thermal burns (referral from Regional Burns Unit only).

Referral for these conditions is **URGENT.** The Hyperbaric Medicine Unit has the capability to care for critically ill patients. Other indications may be considered on a one-off basis.

31.3 Non-Emergency Referrals

Patient referrals and any further information or non-urgent enquiries should be addressed to:

- **Fax:** 03 364 0187 ☎ 80187
- **Phone:** 03 364 0045 ☎ 80045
- **Email:** hyperbaric.medicine@cdhb.govt.nz
- **Mail:** Duty Medical Officer, Hyperbaric Medicine Unit, Christchurch Hospital, Private Bag 4710, CHRISTCHURCH.

31.4 Non-Acute Indications for HBOT

(Refer to the HMU section of the CDHB Intranet.)

- Osteo- and soft-tissue radionecrosis, including planned surgery in a previously irradiated area.
- Diabetic ulcer and other selected "problem" wounds (including preparation for grafting).

  Note: *Post-traumatic non-healing wounds in both diabetic and non-diabetic patients are covered by a prior-approval, non-acute ACC contract for HBOT.*

  "Problem" wounds are those that "fail to respond in a reasonable timeframe to established medical and surgical management". Limb-threatening wounds may be referred at an early stage.
- Refractory osteomyelitis.

These conditions require extended treatment courses (4-8 weeks, 2 hours daily). Referrals will be considered from both family practitioners and hospital specialists, except for patients under the ACC Contract who must be referred by a specialist. Some other conditions may be considered on an individual patient basis.

32. Christchurch Hospital External Emergency Plan/Mass Casualty Incident Plan

These plans are contained in the **Christchurch Hospital Emergency Procedures Manual**, which is available on all wards and departments. Details of how and when the Mass Casualty Incident Response is activated are provided, and unit-specific responses are explained. These plans may be activated at any time. It is your responsibility to know your role in the plan.

When you are advised that the Emergency Response has been activated, you should immediately report to the senior member of your clinical team, usually on your home ward.

In general, your first responsibility is to your clinical team. If this team has an active responsibility for the acute management of casualties from the incident, you will work under the general jurisdiction of the Emergency Department Controller.

If your team does not have this direct responsibility, there will be several ways in which you may be asked to assist:

- Clearing your team's patients from EOA to create space for the Mass Casualty Response.
- Identifying inpatients who could be transferred or discharged, to accommodate the influx of patients from the incident. These could be Surgical/Orthopaedic and/or Medical, depending on the incident.

  Note: The Gridlock tasks are based on the same principles and the cue cards are a useful prompt.

If you are unclear about any aspect of the Emergency Plans, Christchurch Hospital has an Emergency Planner who can be contacted on ☎ 81686 or email john.coleman@cdhb.govt.nz.

33. Document Management

The purpose of Management Guidelines for Common Medical Conditions ("The Blue Book") is to provide guidelines for medical staff working for the Canterbury District Health Board. It is an addition to the formal documentation of the various departments.

Content

The Blue Book contains guidelines for the management of medical conditions in the CDHB.

Scope

The Blue Book is for the use of all medical staff.

Document Control

The Blue Book Issue and Expiry dates are documented on the first page.

The printed Blue Book has a blue cover and a coloured stripe which is different for each issue. For the current period of December 2005 - November 2007 the stripe is

BROWN

The Blue Book will be updated every second year in November at which time owners of the previous year's document must discard the expired copy.

Distribution

Copies of the Blue Book are available free to all medical staff, and further copies can be purchased from Gerard Thomas (☎ 03 364 0640 ext 80034).

The Department of Medicine/RMO Unit maintains a distribution list of the paper version.

The Blue Book is available on the CDHB Intranet, and can be downloaded to PDA.

Updates and Amendments

The Blue Book is updated and reprinted in November of each second year.

There will be no amendments to the printed version during the year.

34. Index