Canterbury
District Health Board
Te Poari Hauora ō Waitaha

Management Guidelines for
Common Medical Conditions

13th Edition 2009

OBSOLETE

Internal Medicine Services

Issued: 1 December 2009 Expiry: 1 December 2011

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INTERNAL MEDICINE SERVICES

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

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Introduction - Thirteenth Edition 2009

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.

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 seizure, 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 the many Consultants, Registrars and other hospital staff not only in Medicine but from other disciplines. We are very grateful to Drs P. Chin, J. Geddes, T. Rahman, T. Reid, P. Tan, and C. Warren for their assistance with proof-reading, and to Helen Noble for her secretarial support. 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 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.*

Note: *These Guidelines must be used in conjunction with the Preferred Medicines List ("The Pink Book"). The following resources are also recommended:*

- UpToDate - on the CDHB intranet.
- Harrison's Online - on the CDHB intranet.
- Cochrane Database.
- Ovid Medline.
- PubMed.
- eMedicine.
- Current medical journals.

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
- **BiPAP:** bilevel positive airway pressure
- **Ca:** calcium
- **CBC + diff:** Hb, PCV, MCV, WBC and differential, platelets
- **CK:** creatine kinase
- **Cl:** chloride
- **CPAP:** continuous positive airway pressure
- **CPR:** cardiopulmonary resuscitation
- **CRP:** C-reactive protein
- **CTPA:** CT pulmonary angiogram
- **CrCl:** creatinine clearance
- **CXR:** chest x-ray
- **DIC:** disseminated intravascular coagulation
- **DKA:** diabetic ketoacidosis
- **ESR:** erythrocyte sedimentation rate
- **FEV_1:** forced expiratory volume/second
- **FVC:** forced vital capacity
- **GGT:** gamma glutamyltransferase
- **INR:** international normalised ratio
- **K:** potassium
- **KCl:** potassium chloride
- **LDH:** lactate dehydrogenase
- **LP:** lumbar puncture
- **MCV:** mean cell volume
- **Mg:** magnesium
- **MJ:** megajoule, a million joules
- **MSU:** mid-stream urine
- **Na:** sodium
- **NIV:** non-invasive ventilation
- **NSAID:** non-steroidal anti-inflammatory drug
- **PO_4**: phosphate
- **PTH:** parathyroid hormone
- **Sat.O_2**: haemoglobin oxygen saturation (pulse oximetry)
- **STI**: sexually transmitted infection
- **USS:** ultra-sound scan
- **VQ:** ventilation/perfusion scan

Drug Administration and Dosage

- **LBW:** lean body weight
  - LBW (kg) male = 50 kg + 0.9 kg for each cm >150 cm in height
  - LBW (kg) female = 45 kg + 0.9 kg for each cm >150 cm in height

  Note: Use actual body weight if it is less than LBW.
- **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.

Note: Do not use the abbreviation "OD"; write "once daily".

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:** <2000
- **Peritoneal:** <250
- **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. Alcohol Related Problems

2.1 Alcohol Withdrawal

- During alcohol withdrawal the following symptoms may be seen:
  - Sweating, tremor, anxiety, agitation, nausea and vomiting, hallucinations, disorientation, headaches, facial flushing, and seizures.
  - Record which of these are present and if so, the severity and/or frequency.

2.1.1 Immediate Treatment

- Administer thiamine 100 mg IM or IV **before** glucose is 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.
- Alcoholic hallucinations - haloperidol 2 mg IM then 1-3 mg BD maintenance. Oral therapy when appropriate. Note: haloperidol may provoke seizures or hypotension. Give lower doses in the elderly.

2.1.2 Early Withdrawal

- Limit all external stimuli, such as noise and visitors. Regularly re-orientate patient to the day, date, and their whereabouts, and explain the symptoms they may be experiencing. If the patient is confused (delirium) or agitated, they are at increased risk of personal injury and should be observed closely. Nurse the patient at floor level, if appropriate, to reduce the risk of falls and/or wandering off. If further treatment is required, give 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.

2.1.3 Moderate to Severe Withdrawal with Autonomic Hyperactivity and Disorientation

- Give diazepam 2.5-10 mg in 100 ml normal saline by IV infusion over 5-10 minutes then maintenance dose 1-2 mg/hour by IV infusion up to a maximum of 60 mg per 24 hours. Oral therapy when appropriate.

2.1.4 Alcohol Withdrawal and Seizures

- **Seizure prevention**:
  - This is generally achieved with diazepam loading and withdrawal over 3-4 days. If seizure activity is considered likely (e.g., history of previous alcohol withdrawal seizures), give diazepam 10 mg orally every 1-2 hours for 3-4 doses (30-40 mg) and reassess. Because of the long half life of diazepam, further sedation is usually unnecessary. If more diazepam is required, give 5-10 mg every 6 hours to a maximum of 60 mg diazepam per 24 hours. Taper off over 3-5 days. Consider also carbamazepine 400 mg stat then 200 mg TDS for 5 days. Some authorities recommend phenytoin or sodium valproate in this situation.
- **Seizure treatment**:
  - Diazepam or midazolam may be given. Refer to the Neurology section on page 160 for full treatment details.

2.1.5 Wernicke's Encephalopathy

- **Wernicke's Encephalopathy** should always be suspected. Look for the triad of confusion, ataxia, ophthalmoplegia. If the full syndrome is present give thiamine three times daily (100 mg IV over 10 minutes or IM). Give 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 Strong twice daily at the end of the parenteral regimen.

2.2 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.
- Social Work assistance with alcohol related problems is available to in-patients and their families/whanau. Please access this service through the ward Social Worker.
- **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 seizures.
  - 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
  = 360 ml (12 oz) beer, or
  = 120 ml (4 oz) wine (small glass), or
  = 30 ml (1 oz) spirit (1 pub nip)
- 1 jug of beer = 3 standard drinks
- 750 ml bottle of wine = 8 standard drinks
- 1125 ml 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.
  - Persistent 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. Referral to the Social Work Services is recommended (☎ 80420). They provide a social worker comprehensive assessment and intervention plan which can include referral on to other agencies such as the Community Alcohol and Drug service.

2.2.1 Facilities available to help with alcohol related problems

Community Alcohol and Drug Service/ Methadone Programme

Ph: 335 4350, Fax: 335 4351
Sylvan Street, Hillmorton Hospital
Private Bag 4733, Christchurch Mail Centre, Christchurch 8140

- Assessment, case management including relapse prevention, and referral to appropriate treatment agencies.

Christchurch City Mission

Ph: 365 0635
275 Hereford Street, Christchurch

- Assessment/overnight stay, youth alcohol and drug worker

Odyssey House

Ph: 358 2690
98 Greers Road, Christchurch

- Residential (male) - 26 beds for severe drug dependence. Long term therapeutic community and Youth Day Programme.

Kennedy Detoxification Unit

Ph: 339 1139
Hillmorton Hospital, Christchurch

- Medical detoxification for South Island by referral only from the Community Alcohol and Drug Service

Salvation Army - Bridge Programme

Ph: 338 4436
35 Collins Street, Christchurch

- Residential, women's programmes

Nova Lodge

Ph: 349 2053
Newtons Road, Templeton

- 61 bed residential/long term for chronic alcoholics

Care NZ

Ph: 365 8700
192 Cashel Street, Christchurch

- Intensive outpatient programme for men and women

Wahine Whai Ora. Women's Alcohol and Drug Service

Ph: 365 6601, Fax: 365 9936
276 Hereford Street, Christchurch

- Provides a day programme for outpatients

Thorpe House

Ph: 379 1682, Fax: 371 0602
228 Worcester Street, Christchurch

- Non-medical detox

Vincentian Centre

Ph 379 9338
222 Wilsons Road, Christchurch

- Inpatient programme for alcohol dependent males and women with children, and day programme for women

Alcoholics Anonymous

Ph 379 0860/0800 229 6757

Home Detox Service

Ph: 365 0635
276 Hereford Street, Christchurch

- For people over 18

Familial Trust

Ph: 981 1093; Fax: 942 1093
6 Wilsons Road, Christchurch

- Education/support for family members of people with addiction

Addiction Advocacy Service

Ph: 943 5584
PO Box 13496
Armagh, Christchurch 8141

Alcohol and Drug Helpline

Ph: 0800 787 797

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 8140.

3. 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).

3.1 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
  - Renal impairment or failure
  - 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.*

3.2 Preoperative Fasting Instructions (Adults)

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

Fasting before anaesthesia aims to reduce the volume and acidity of stomach contents thus decreasing the risk of regurgitation and aspiration. There is good evidence that maintaining oral intake with clear fluids up to 2 hours before surgery is both safe and advantageous.

Perioperative Fasting Instructions:

- **ALL** patients should be instructed to drink clear fluids up until 2 hours before the scheduled start time of the list.
- **Patients on AM lists** should be instructed not to take SOLID food for 6 hours before the scheduled start time of the list.
- **Patients on PM lists** may have a light breakfast 6 hours before the scheduled start time of the list.
- Other food or fluids may be consumed ONLY on the instructions of an Anaesthetist.

Clear fluids means water and clear non-particulate fruit juice only.

Light breakfast means a small quantity of toast or cereal with tea, coffee, or other drink.

Milk is considered as solids.

Reference: Soreide E, Ljungqvist O. Modern preoperative fasting guidelines: Best Practice and Research Clinical Anaesthesiology (2006); 20 (3): 483-491.

3.3 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
- Aspirin - if patient is taking aspirin for secondary prevention of cardiovascular events, the risks vs benefits of cessation should be discussed with the surgical team or Anaesthetist.
- 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 **View Departments** > **Clinical Pharmacology** > **Bulletins and Guidelines** > **Peri-operative Medications**.

3.4 Perioperative Management of Diabetes

This will usually be supervised by the Anaesthetist. If not, refer to the guidelines in Perioperative Management of Diabetes on page 102.

3.5 DVT Prophylaxis for Patients Undergoing Surgery

Refer to Surgical DVT Prophylaxis on page 258.

3.6 Guidelines for Perioperative Steroids in Patients already on Steroids

Refer to Guidelines for Perioperative Steroids in Patients already on Steroids on page 93.

3.7 Management of Patients on Warfarin Therapy Undergoing Surgery

Refer to Management of Patients on Warfarin Therapy Undergoing Surgery on page 267.

3.8 Spinal/Epidural Anaesthesia

Refer to Spinal/Epidural Anaesthesia on page 259.

4. Ancillary Services

4.1 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.

4.2 New Zealand Blood Service

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

Contact Telephone Number

Cross matching ☎ 80310

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 29) for further information.

4.3 Canterbury Health Laboratories

Service Provided

- The Core Laboratory functions 24 hours daily providing routine biochemistry, 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 - normal business hours.
- After hours, most laboratories provide an on-call service for urgent specialised tests not performed in the Core Biochemistry and Core Haematology Laboratories.

Contact Telephone Numbers

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

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 Information and Resources** > **CHLabs** > **Test Guide**.

Minimum labelling requirements for specimens:

- Two patient identifiers:
  - a hospital number **and**
  - 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., STI 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.

4.4 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 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.
- **The Princess Margaret Hospital** - radiology services are provided by the Christchurch Radiology Group (CRG). Consultant Radiologist on call at all times.

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.

Request forms with incomplete details will be sent back to the referrer for completion before the study can be actioned. Attention to detail will prevent unnecessary delays in getting an examination performed.

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. Please indicate at top of requisition form if your patient is for discharge. This helps the Radiologists when triaging the requisition forms and with patient flow through the hospital.

Radiology forms are available on the CDHB intranet under **View Department** > **Radiology** > **Forms and Docs**. Please note that the consent for contrast form is required for the majority of MRI and CT scans. The consent for contrast form must be filled in on the ward for patients who are unable to give consent, e.g., confused patients or patients with communication difficulties.

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. Radiologist office-related enquiries should be made to ☎ 80781 or 80782.

Radiology examinations during evenings, nights, and weekends at Christchurch Hospital (acute service only from 17:00 to 08:00)

Radiology examinations should only be specifically asked for during evenings, nights, and weekends if the examination is likely to significantly change the patient's management before the next working day. The Registrar on call must be consulted for acute examinations in specialised areas such as CT, MRI, ultrasound, and DSA. The Registrar pager is 8911. Registrar office in Radiology is ☎ 89369.

PACS

Christchurch Hospital, Christchurch Women's Hospital, and Burwood Hospital are all linked by PACS and are filmless, except for mammography at Christchurch Hospital. Radiology department staff can help with online access to the Christchurch Radiology Group's private imaging centres, and also to most other public hospitals in the South Island.

4.5 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. The information is also available in the CDHB intranet under **View Departments** > **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.

4.6 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.

5. Blood and Cancer Care - Haematology, Oncology, and Palliative Care

This service comprises the Departments of Oncology, Haematology, and Palliative Care.

Main Offices

Haematology, Ground Floor, Laboratories, ☎ 80300, fax ☎ 81432, referral fax ☎ 81432

Oncology, Oncology Building, ☎ 80020, fax ☎ 80759, referral fax ☎ 86233

Palliative Care, Oncology Building, ☎ 81473 (voice mail), fax ☎ 80759, referral fax ☎ 86233

Haematologists

Dr Andrew Butler, Dr Liam Fernyhough, Dr Peter Ganly, Dr Steve Gibbons, Dr Mark Smith, Dr Ruth Spearing.

Oncologists

Dr Brendon Anderson, Assoc Prof Chris Atkinson, Dr Scott Babington, Dr Bernie Fitzharris, Dr David Gibbs, Dr Dean Harris, Dr Melissa James, Dr Mark Jeffery, Dr Avtar Raina, Assoc Prof Bridget Robinson, Dr Iain Ward, Dr Chris Wynne, Dr Michelle Vaughan.

Palliative Care Physician

Dr Kate Grundy.

Inpatient Services

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

Oncology Ward, Ward 27, Riverside Block.

Consultation and On-call Services

- **Haematology** (Monday to Friday). Fax referrals to ☎ 81432. Please also put in internal mail to the Department of Haematology. The Registrar or Consultant taking referrals can be contacted on pager 7031. The Laboratory Registrar may be contacted on pager 8314. Please make it clear whether a full consultation is required or just a bone marrow examination. For urgent consults after hours, contact the on-call Haematologist.
- **Oncology** (Monday to Friday). For non urgent consults, fax referral to ☎ 86233 (internal) or ☎ 378 6233 (external). Some specialization exists and referral will be made to the appropriate Consultant. For urgent consults phone 80023 or 86271. For urgent consults after hours, contact the on-call Oncologist. It is departmental policy to re-admit under our care, patients who are undergoing active treatment.
- **Palliative Care** (Monday to Friday). This 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 Palliative Care Guidelines on the intranet (under **Clinical Information and Resources** > **Palliative Care Service and Guidelines**).

Consultation Guidelines

Diagnosis and management of malignant disorders, cytopenias, bleeding or thrombotic problems.

Other Services

- Haematology Laboratory Consultant or Registrar (pager 8314).
- Haemostasis Nurse (pager 8527 or ☎ 81246)
- Palliative Care Specialist Nurses ☎ 81473 (Willem Vink and Anne-Marie Evans) or ☎ 81885 (Anne Morgan). Palliative Care Registrar (pager 8970).
- 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).

Departmental Guidelines

- Haematology Department Protocols and Guidelines are available on the CDHB intranet (under **Clinical Information and Resources** > **Red Book**).
- Palliative Care Guidelines are available on the intranet (under **Clinical Information and Resources** > **Palliative Care Service and Guidelines**).

5.1 Haematology

5.1.1 Management of Haemorrhagic Disorders

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

Investigation of a patient presenting with a possible haemorrhagic disorder

- Family history, history of pattern of bleeding, recent drugs, dietary history, possibility of HIV.
- 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 (☎ 80374).*

Treatment

- This is entirely dependent on the results of the initial tests obtained. If a severe **thrombocytopaenia** (<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.
- **Refer to the Haematology Department Protocols and Guidelines (the Red Book)** under Inherited Bleeding Disorders for management of haemophilia, including local practice (these guidelines are based on the New Zealand National Guidelines for the Management of Haemophilia).
- 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 1: 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, desmopressin or CSL Factor VIII concentrate is used. Desmopressin may be given in a dose of 0.3 mcg/kg in 50 ml normal saline IV over 30 minutes (starting 60 minutes pre-op, if requiring surgery). Desmopressin 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.

5.1.2 Management of Severe Anaemia

The following investigations are suggested for anaemia in the absence of acute blood loss or shock. Some causes include: iron deficiency, B_{12} and folate deficiencies, leukaemias, myelodysplastic syndromes, aplasia, haemolysis, renal failure, and bone marrow infiltration.

Investigations

- CBC + diff, film, and reticulocyte count along with standard biochemistry and LDH. Get copies of previous CBC from Community Laboratory/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 (<250). Methylmalonic 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 are 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.*

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 IM 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).

5.1.3 Management of Severe Neutropaenia/Immunosuppression

- May present with patient feeling non-specifically unwell. Common signs include fever, tachycardia, and postural hypotension.
- 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.
- **Note:** Consider similar prompt management of infection in non-neutropaenic patients with malignant disease who may be immunosuppressed because of:
  - Chemotherapy (particularly corticosteroids, fludarabine).
  - Hypogammaglobulinaemia (particularly lymphoproliferative diseases such as chronic lymphocytic leukaemia, myeloma, and lymphoma).
  - Previous splenectomy.

Treatment

- If the neutropaenia 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 neutropaenia is chronic and the patient has been out in the community with neutropaenia, then there is no need for isolation.
- If the patient is febrile (fever >38.5 or history of fever >38 for one hour or any question of either of these) start empirical antibiotics after blood cultures (from peripheral vein and also central line if present) are taken, **before** doing other investigations, and seek Specialist advice.
- A detailed management plan for neutropaenic or otherwise immunosuppressed haematology and oncology patients is described in the Emergency Department's Clinical Pathway "Immunosuppressed Patients Clinical Pathway (Oncology, Haematology and Transplant Patients)". This is available in the Emergency Department.
- Other appropriate investigations include MSU, swab of any lesion or pustule, sputum for Gram stain and culture, faecal culture if diarrhoea is present, CXR.
- First line antibiotic therapy for the treatment of neutropaenic sepsis is:
  - Piperacillin/tazobactam (Tazocin) 4.5 g IV q8h plus gentamicin 5-7 mg/kg IV in 100 ml normal saline over 30 min q24h

  or, if there is a history of penicillin allergy,
  - Imipenem 500 mg IV q6h plus gentamicin 5-7 mg/kg IV in 100 ml normal saline over 30 min q24h.

5.1.4 Management of Nausea and Vomiting

Refer to Management of Nausea and Vomiting in the Oncology section (see page 26).

5.2 Oncology

5.2.1 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 for which early consultation is particularly inportant 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.

5.2.2 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 (☎ 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.

5.2.3 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 >5 cm 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 ☎ 80023, or contact the Oncologist on call after hours **before** biopsy to avoid prejudicing future surgery or radiation options.

5.2.4 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.
- 30% of cases of spinal cord compression involve multiple levels, so MRI of the whole spine should be requested.

5.2.5 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.

5.2.6 Management of Severe Neutropaenia/Immunosuppression

Refer to Management of Severe Neutropaenia (see page 24) in the Haematology section.

5.2.7 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 Hypercalcaemia on page 107) are appropriate, but where possible 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.

Treatment of hypercalcaemia of malignancy requires rehydration followed by 4 mg zoledronic acid over 15 mins by IV infusion.

5.2.8 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. See Causes of Vomiting (see page 118).

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.

Chemotherapy - Associated Nausea and Vomiting

- **Pre-chemotherapy:** anti-emetics are individually tailored to the emetogenicity of the regimen, and vary between metoclopramide 10 mg PO, or dexamethasone 4-8 mg PO for mildly emetogenic regimens, to a combination of ondansetron 16 mg PO/IV, aprepitant 125 mg PO, and dexamethasone 12 mg IV in 100 ml normal saline for highly emetogenic regimens.
- **Post-chemotherapy:** again treatment is tailored according to chemotherapy regimen.
  - Mildly emetogenic regimens - metoclopramide 10 mg PO prn QID or domperidone 10-20 mg PO prn QID.
  - Moderately emetogenic regimens - dexamethasone 4-8 mg mane for 3 days after chemotherapy.
  - Highly emetogenic regimens - dexamethasone 8 mg mane and aprepitant 80 mg on days 2 and 3 after chemotherapy.

For persisting emesis, more than 24 hours after chemotherapy:

- Give regular metoclopramide or domperidone, consider adding cyclizine 50 mg PO TDS.
- Within the first 3 or 4 days of chemotherapy increase dexamethasone to a maximum of 8 mg BD (8 mg mane only in patients on aprepitant), and adding ondansetron 8 mg BD may also be effective.

  Note: constipation may be severe with ondansetron, and may worsen nausea.

Other Antiemetics for Oncology, Haematology, 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)
- Domperidome 10-20 mg PO QID before food and nocte
- 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 and can be given subcutaneously.
- Combinations of 2 or 3 agents may be more successful than a single agent.
- Haloperidol is very effective 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 Guidelines on the intranet**. 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 is a broad-spectrum antiemetic and can be very effective in advanced disease. Refer to the **Christchurch Hospital Palliative Care Guidelines on the intranet**, 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 may incur a part-charge in the community but certain generic brands are exempt.
- If nausea and vomiting remain a problem, consult Oncology or the Palliative Care Service ☎ 81473 for further advice.

5.2.9 Radiotherapy

If a patient becomes unwell during a course of radiotherapy, even if the illness is unrelated to the oncological problem, the treating Radiation Oncologist should be contacted promptly.

Radiation Side Effects

When radiation is used to treat a cancer, normal cells can also be damaged and this may result in side effects. The side effects from radiation can be divided into acute and late side effects. Acute side effects are those which occur during the treatment and immediately after. Late side effects occur at least 6 months after the treatment. Tissues have differing tolerances to radiation and also differ in the types of radiation reactions and the timing of these.

Tissues which are rapidly proliferating (e.g., mucosal lining cells) tend to be the most sensitive to radiation treatment and express acute side effects. Patients being treated for a head and neck cancer for example, usually experience mucositis and have pain and difficulty swallowing about 2 weeks into the 6-7 week course of radiation treatment. Patients with rectal cancer often experience diarrhoea as a result of radiation damage to the gut lining cells. Patients being treated for a skin cancer may experience damage to the normal epithelial cells and the skin may develop erythema and in some cases sloughing and ulceration, known as desquamation.

These rapidly proliferating cells may also exhibit late side effects. The late side effects often involve fibrosis of tissues and scar formation. For example in the gut, fibrosis may develop leading to bowel obstruction or malabsorption. In the skin the late changes that may develop included atrophy of the skin, thinning of the skin, scar formation and telangiectasia.

More slowly proliferating tissues tend to develop late side effects so caution is taken to limit the dose of radiation treatment to these tissues. For example a late effect of treating above a certain dose to the spinal cord may be myelitis and subsequent development of neurological signs and symptoms. A late effect of treating the kidney may be hypertension and treating the optic nerve may be optic neuritis and possible visual loss.

Some tissues may exhibit subacute reactions which may not be evident during the treatment, but develop within a few weeks of it. For example a patient who has radiation treatment to the lung may present several weeks later with cough, dyspnoea and low grade fever and on imaging be found to have changes in the lungs consistent with radiation pneumonitis.

The challenge in radiation is to give the maximum dose to the tumour to optimise the chance of cure while sparing as much as possible the normal tissues to decrease the risk of acute and late side effects.

5.3 Palliative Care

For guidelines for referring a patient to the Palliative Care Service, please refer to the Palliative Care Guidelines on the intranet (under **Clinical Information and Resources** > **Palliative Care Service and Guidelines**).

For management of pain in the cancer/palliative setting, refer to Chronic/Persistent Pain in the Cancer/Palliative Setting (see page 193).

6. Blood Transfusion Practice

6.1 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 (Crossmatching) is on the Lower Ground Floor of Christchurch Hospital.

- Blood Bank ☎ 80310 - 24 hours.
- Transfusion Medicine Specialist - contact via the Blood Bank.
- NZBS on-call team - contact via the Blood Bank at any time.

Product information and clinical guidelines are available on the Transfusion Medicine Guidelines site on the CDHB intranet. See also the Transfusion Medicine Handbook 2008 available from the New Zealand Blood Service and the **Haematology Department Protocols and Guidelines (the Red Book)** on the CDHB intranet.

6.2 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 (pink top).
- 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. The sample must be signed. 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.

6.3 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 compatibility label (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 in cases of extreme urgency occurring outside laboratory hours when on the direct order of a senior medical officer, Group O Rhesus negative blood labelled specifically for emergency use, which has not expressly been labelled as suitable for the patient in question, may be issued. In such cases (which should be rare) the Blood Bank must be informed.**

6.4 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.

6.5 Transfusion Reactions

- Reactions occurring during transfusion of blood components or products 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 Guidelines for Management of Mild Adverse Transfusion Reactions (see page 31) and Management of Moderate and Severe Adverse Transfusion Reactions (see page 32).

- **Sepsis due to bacterial contamination** is a rare but serious transfusion reaction accounting for at least 10% of transfusion-related fatalities. It is especially common with platelet transfusions and in neutropaenic recipients. Overall, Gram negative organisms more commonly cause serious reactions but Gram positive organisms need to be considered if a platelet transfusion is implicated. 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 g IV q8h plus gentamicin 5 mg/kg IV, loading dose.
  - If, after testing (see page 32), no cause can be found for a moderately severe reaction, it may be presumed to have an allergic basis. Further transfusion can then be given 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 2: 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:
   - 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 - antipyretics are of unproven benefit
   - Urticarial reaction - antihistamine
2. Hydrocortisone - not usually needed.

Table 3: 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) (see page 30).
  - Anaphylaxis/anaphylactoid reaction: adrenaline intramuscular. See Anaphylaxis (see page 83). 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.

6.6 Blood Products Available

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

Table 4: Blood Components

Blood components available include:	
▪ Resuspended Red Cells ($253) ▪ Resuspended Red Cells Neonatal ▪ Whole Blood Autologous ▪ Whole Blood Plasma Reduced ▪ Cryoprecipitate ($365)	▪ Platelet Pool ($755) ▪ Platelets Apheresis ($755) ▪ Fresh Frozen Plasma ($195) ▪ Fresh Frozen Plasma Neonatal
• Cryoprecipitate contains on average 1.3 g of fibrinogen per bag. • The above prices (as at 2009/2010) are approximate.	

Table 5: Blood Products

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

6.7 Micro-Filters

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

6.8 Massive Blood Loss/Massive Transfusion

Seek advice from the Transfusion Medicine Specialist, the NZBS on-call team, or the Haematology Department as soon as possible.

6.9 Patients who Decline Blood Transfusion

Some patients, including Jehovah's Witnesses, do not wish to have blood products and should be treated according to their beliefs. Before treatment starts, an individual management plan should be agreed upon by the patient and the senior medical officer(s) responsible for providing care for the patient. For more information, refer to the Haematology Department Protocols and Guidelines on the intranet under **Clinical Information and Resources** > **Red Book**.

7. Cardiology

7.1 Cardiology Department Information

Main Office

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

Inpatient Services

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

Cardiologists

- Dr Paul Bridgman, Dr Ian Crozier, Assoc Prof John Elliott, Dr John Lainchbury, Dr Dougal McLean, Dr Iain Melton, Prof Mark Richards, Dr David Smyth, Assoc. Prof. Richard Troughton.

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, pager 8262
- Cardioendocrine Research Group, ☎ 81116 (clinical research, basic research)
- Cardiology Research Unit, ☎ 81096 (clinical research studies)

7.2 Heart Failure

7.2.1 Definition

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

7.2.2 Management requires each of the following

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

7.2.3 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.
- Anaemia.
- Thyrotoxicosis - particularly in the elderly.
- Infections (especially endocarditis and pulmonary infections).
- Pulmonary embolism.
- Fluid overload - e.g., transfusion, renal failure.

7.2.4 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: troponins, 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 BNP (where there is doubt over cardiac vs non-cardiac cause of symptoms) - refer to Guidelines for Use of BNP Measurements in an Acute Medical Setting (see page 38).

7.2.5 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 40), continuous positive airway pressure (CPAP) by face mask, and haemodynamic monitoring in ICU or CCU should all be considered.
- CPAP is useful if hypoxia persists after initial treatment and may avert the need for intubation and mechanical ventilation. It is best started before the patient becomes severely fatigued. If prolonged therapy with high O_2 concentrations is required, consider other ventilatory supports.

- Compromised myocardial function: Low output states can be managed by increasing myocardial contractility (inotropic support) or reducing the cardiac workload (pre load and after load reduction).
- Inotropic Support:
  - Digoxin - indicated for control of ventricular response in atrial fibrillation and atrial flutter and has value as third line agent in heart failure with sinus rhythm. The trials indicating benefit from digoxin are not confined to "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 6: Digoxin

See also digoxin poisoning on page 206

- 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, low magnesium 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 62. 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 5% dextrose (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 mm Hg systolic on dobutamine, a vasoconstrictor drug should probably be added (dopamine or adrenaline) to keep the BP above 80 mm Hg 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 such as glyceryl trinitrate (see page 40) or sodium nitroprusside with close monitoring possibly including Swan Ganz and arterial pressure (if using sodium nitroprusside) in CCU or ICU.
  - ACE inhibitor dosing: start with enalapril, quinapril or cilazapril. 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 62. The starting dose of cilazapril for a patient with normal renal function is 0.5 mg to 1.25 mg/day.

Table 7: Quinapril and Enalapril Dosage in Renal Failure

Creatinine Clearance (ml/min)	Dose	
	Starting	Maximum
>90	5-10 mg	30 mg q24h
48-90	5 mg	20 mg q24h
24-48	2.5-5 mg	10 mg q24h
12-24	2.5 mg	5 mg q24h
<12	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** 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 chronic atrial fibrillation. Also consider anticoagulation in those with severe left ventricular impairment. Discuss with Cardiologist.
- Potassium supplements will be needed with most diuretics. Requirements may be reduced or unnecessary in renal failure, with ACE inhibitor treatment or when using potassium sparing diuretic therapy.
- Re-evaluate the primary cause of the heart failure - attempt to confirm the primary disease process and exclude aggravating factors. This may include cardiac catheterization in selected cases.
- 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.

7.2.6 Guidelines for Use of Brain Natriuretic Peptide (BNP) Measurements in an Acute Medical Setting

- BNP is useful in distinguishing between cardiac failure and non-cardiac causes of 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 BNP of >80 pmol/l supports diagnosis of heart failure as the cause of breathlessness.

  Note:

  - Normal range in healthy subjects is <30 pmol/L. In this situation, heart failure is unlikely.
  - Values greater than 80 pmol/l 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. BNP may be elevated by atrial fibrillation, LVH, valve disease, after myocardial infarction, in the elderly, and in severe renal impairment. 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.
  - BNP does not add to the diagnosis in a patient with overt heart failure.

7.3 Myocardial Infarction

7.3.1 Definition

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

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

7.3.2 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.
  - Troponins may be elevated in other conditions. See Troponin Testing on page 42.

7.3.3 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.

7.3.4 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 (see page 42) and creatine kinase (CK) should be done on admission and at 8 to 12 hours.
- If suspicion of aortic dissection, arrange an urgent CT scan and inform the Cardio-Thoracic team - see Thoracic Aortic Dissection on page 54.
- CXR can usually wait until normal working hours or prior to discharge. Indications for urgent X-ray include 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).

7.3.5 Management

- **"Time is muscle"** - expedite treatment and assess suitability for reperfusion - thrombolysis or angioplasty - 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 normal saline.
- **Oxygen** - should be administered to all patients with MI or unstable angina for the first 12 hours aiming to keep Sat.O_2 at 96% unless there is a strong contraindication.
- **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 10 mg IV may reduce nausea and vomiting.

- **Aspirin** 300 mg chew and swallow stat, then 150 mg orally daily if no contraindications.
- **Clopidogrel**, 300 mg stat then 75 mg daily for patients under the age of 75.

Reference: Sabatine MS et al. for the CLARITY-TIMI 28 Investigators. Addition of clopidogrel to aspirin and fibrinolytic therapy for myocardial infarction with ST-segment elevation. NEJM 2005;352:1179-89.

7.3.6 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 41). Tissue plasminogen activator (e.g., tenecteplase tPA) is recommended in this situation. Fibrin specific agents are preferred over streptokinase. They may be more effective and have a lower side effect profile.

- **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".

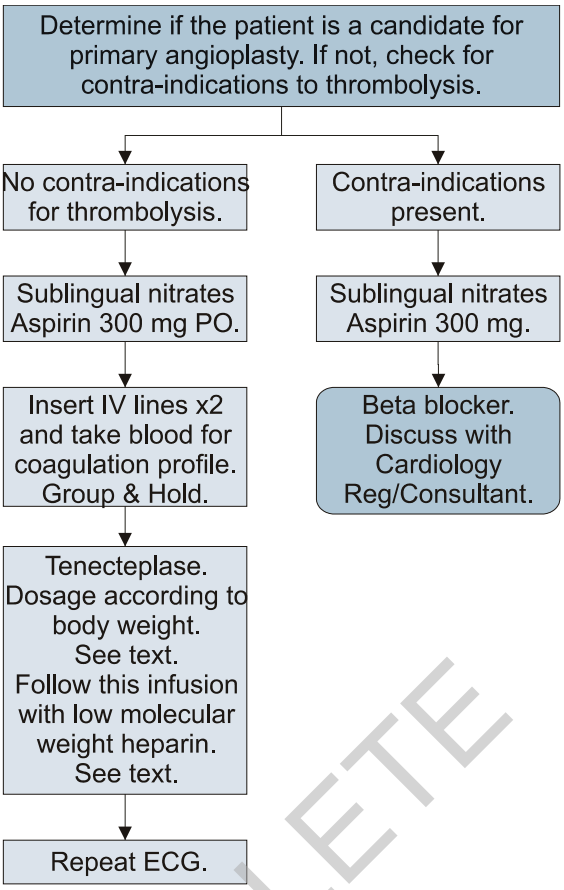
- **The role of balloon angioplasty/stenting post infarction** - if in-hospital cardiac catheterization demonstrates a critical stenosis and/or slow flow in one coronary artery, then balloon angioplasty and/or stenting may be indicated. This will effectively treat angina and should permit early discharge. 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 or isosorbide 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. Nitrates should be given in ICU/CCU. 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 5% dextrose in a non-PVC bag (200 mcg/ml). Start infusion at 10 mcg per minute (3 ml/h). Increase infusion rate by 10 mcg/minute every 3 to 5 minutes. Boluses of 10 - 20 mcg can be given until pain relieved or BP falls (can go as low as 90 mm Hg systolic if otherwise well).

7.3.7 Thrombolytic Therapy

Table 8: 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 mm Hg or DBP >110 mm Hg).
- 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 INR >2: the higher the INR, the higher the risk of bleeding.

Fibrin-specific Thrombolytic Agents

Fibrin specific agents are preferred over streptokinase. A number of preparations are available, such as tenecteplase and alteplase tPA. These agents are more effective than streptokinase and have fewer side effects. We currently recommend the tissue plasminogen activator **tenecteplase**.

Administration of Tenecteplase

- Give single bolus IV, dosage according to weight:

Weight	Dose of Tenecteplase
<60 kg	30 mg
60 - 70 kg	35 mg
70 - 80 kg	40 mg
80 - 90 kg	45 mg
>90 kg	50 mg

Administration of Heparin in association with Tenecteplase

Enoxoparin 1 mg/kg SC BD for 48 hours post thrombolysis (maximum of 100 mg per dose). Give first dose after completion of tenecteplase **if no abnormal bleeding has occurred**.

Note: *Failed thrombolysis may be an indication for emergency angiography/angioplasty.*

7.3.8 Other treatments

- Unless the patient has had an angioplasty, or thrombolysis is contraindicated, give a 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 48 hours. 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 Consultant, 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 40) may also be helpful.

7.3.9 Troponin Testing

- **Cardiac troponins are highly cardiac specific. Troponin I is the troponin test currently 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. Elevations in troponin may also be seen in myocarditis, direct cardiac trauma, heart failure, and pulmonary embolism.
- Small rises in troponin 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 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).

7.3.10 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:
  - 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, hypotension, or new or changing murmurs.
  - 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 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.
- Aim 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 (pager 8262) to review prior to discharge.

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

7.4 Cardiogenic Shock

7.4.1 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 mm Hg 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 normal saline, 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, pulmonary embolism.

7.5 Acute Coronary Syndrome

7.5.1 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 38).

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.

7.5.2 Causes

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

7.5.3 Investigation and Management

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

- Daily ECG and cardiac injury markers on at least two occasions are mandatory, as is assessment of cardiac risk factors including lipids.
- Elevation of Troponins 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 or a high index of suspicion of ACS. The usual duration of enoxaparin treatment is 48 hours. 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 Consultant, and with intensified monitoring for haemorrhagic complications.
- Oxygen should be given if there is ongoing angina at rest. Telemetry monitoring if Troponins are raised or the patient's condition is unstable; monitor for 48 hours.
- 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 positive or with dynamic ST changes. Consult Cardiologist.
- Review and treat risk factors as for myocardial infarction.

References:

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

Eikelboom et al. Unfractionated heparin and low-molecular-weight heparin in acute coronary syndrome without ST elevation: a meta-analysis. Lancet. 2000 Jun 3;355(9219):1936-42.

7.6 Cardiac Arrhythmias

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

7.6.1 Classification

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

7.6.2 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.

7.6.3 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.

7.6.4 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.

7.6.5 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 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 5% dextrose (= 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 usually due to the sick sinus syndrome and requires permanent pacing if symptomatic. Temporary pacing rarely required.

Note: *Sinus arrest is common in vasovagal syncope. These patients only have bradycardia at the time of symptoms. They can usually be managed medically and pacing is only infrequently 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 IV 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 (see below) 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, Mg.
  - 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). Discuss with Cardiologist.
  - 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. Heparin may be stopped at 48 hours under these circumstances.
  - Electrical cardioversion:
    - Indicated if there is cardiac compromise with hypotension, angina or impaired cerebral function or persistent 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 may be tried, but consult Cardiologist. Temporary pacing at 90 beats/minute may suppress this arrhythmia.
- **Ventricular Fibrillation** (VF) - D.C. shock (see page 49).
- **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 ml of 5% dextrose 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 PICC central venous line if planning to give more than 24 hours intravenous infusion.

Patients receiving intravenous amiodarone should be on continuous ECG monitoring.

- **Recurrent Ventricular Tachycardia/Fibrillation in a patient with implantable defibrillator ("arrhythmic storm").**
  - This is a medical emergency.
  - The patients are often distressed by multiple shocks and will benefit from sedation.
  - If the shocks are inappropriate, the implantable defibrillator can have the shocks disabled by placing a magnet over the device.
  - Consult Cardiology immediately and arrange early transfer to CCU.
  - Look for exacerbating causes for arrhythmias, and correct if present.

7.7 Cardiac Arrest

Commence basic life support - using the ABCs of CPR (see page 74). Call for the **Clinical Emergency Team** and resuscitation trolley. Consider precordial thump if witnessed.

REMEMBER:

- External chest compression at 100/min.
- **30 compressions to 2 ventilations for both 1 and 2 person CPR. Minimise interruptions to chest compressions.**
- 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. Give uninterrupted chest compressions at 100/min once intubated.
- When the defibrillator arrives identify the rhythm utilizing the electrode pads (Lifepak 20) or paddles (older models) and/or by attaching ECG limb leads.
- Position pads or paddles at right of upper sternum below the clavicle, and left of the left nipple in the anterior axillary line.
- Do not use dilated pupils as an indication to stop resuscitation.

7.7.1 Identify the Cardiac Rhythm

- **Ventricular fibrillation (VF):**
  1. Defibrillate immediately using maximum setting (360 joules on Lifepak 20). Give three shocks rapidly, briefly checking rhythm in between each shock.
  2. If unsuccessful give adrenaline **1 mg** IV. Perform CPR for 2 minutes.
  3. If still in VF give one shock at maximum setting (360 joules), then 2 minutes CPR.
  4. If still in VF give amiodarone 300 mg IV bolus. Continue one shock / 2 minutes CPR sequence.
  5. Repeat adrenaline every 3-5 minutes.
  6. If still in VF consult Cardiologist regarding the use of other agents. Consider metoprolol 1-2 mg IV.
- **Pulseless Electrical Activity (Electromechanical dissociation)**, i.e., Organised electrical activity on ECG but failure of effective myocardial contraction:
  1. 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.

2. In absence of other specific therapy give adrenaline **1 mg** IV. Repeat every 3-5 minutes.

- **Ventricular asystole:**

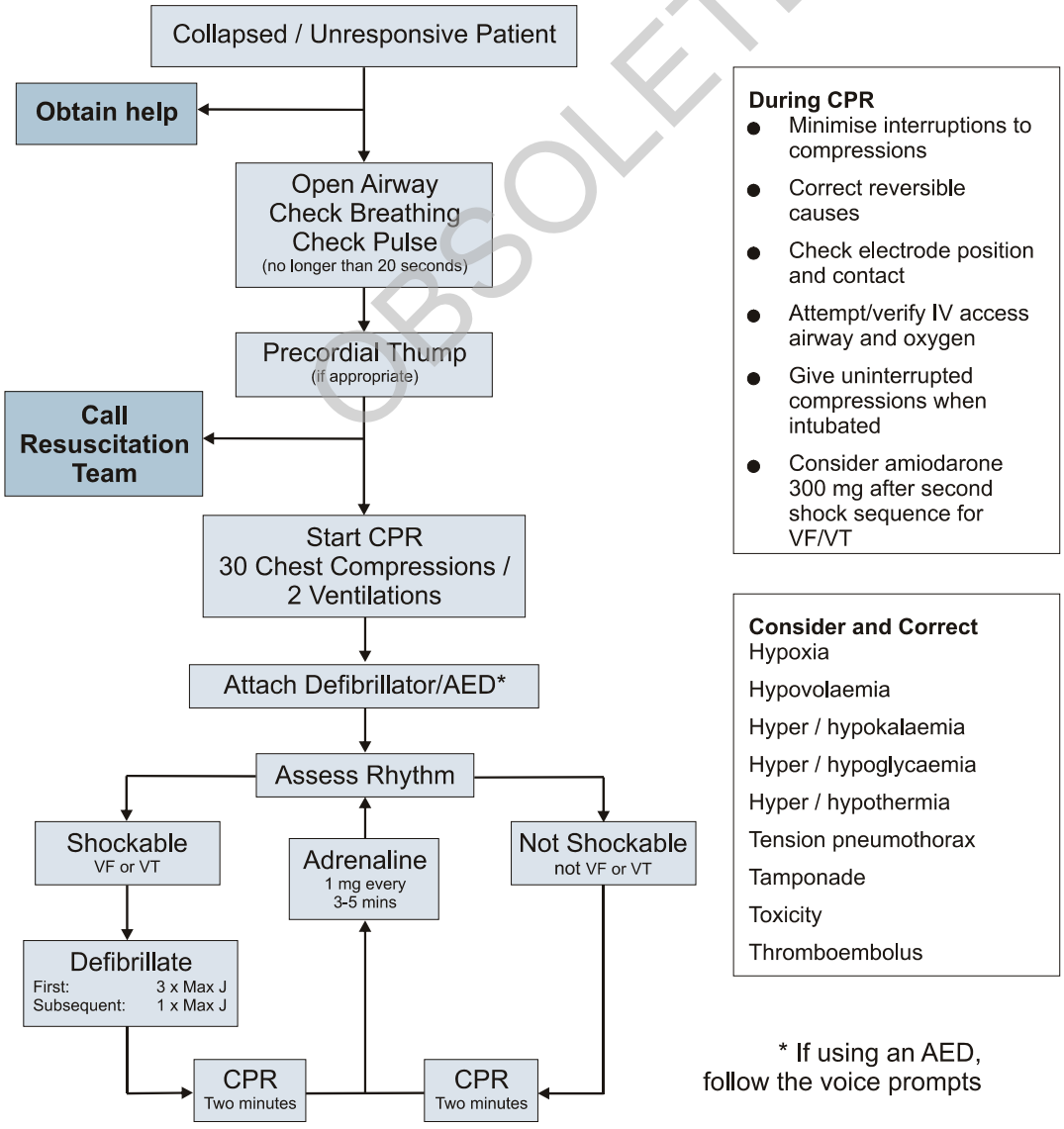
  Note: *Exclude the possibility of monitor failure resulting in apparent asystole. Consider thump pacing and check for evolved QRS and pulse for effectiveness.*

  1. Perform CPR. Briefly check rhythm every 2 minutes. Minimise interruptions to CPR.
  2. Give adrenaline **1 mg** IV. Repeat adrenaline every 3-5 minutes.
  3. Consider an adrenaline infusion.
  4. Consider transcutaneous or transvenous pacing.
  5. Consider and treat possible causes including hypovolaemia, major electrolyte imbalance, tension pneumothorax, cardiac tamponade, pulmonary embolism, and overdose.

- **Bradycardia and Heart Block:**

  1. Thump pacing may be effective in inducing ventricular depolarization and an adequate cardiac output.
  2. Atropine 0.6 mg IV and repeat if necessary.
  3. Consider an adrenaline infusion.
  4. Definitive management is by pacemaker, transcutaneous (temporary) or transvenous.

Table 9: Adult In-Hospital Resuscitation



Authorised by the CDHB Resuscitation Coordinator 2006.

7.7.2 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.

7.8 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 a ventricular arrhythmia that is life-threatening:**
  - Monitoring should be continued until the arrhythmia is controlled.
- **Patients with cardiac instability receiving intravenous infusions that require cardiac monitoring:** (The list includes adenosine, amiodarone, dobutamine, dopamine, flecainide, phenytoin, beta-blockers, and verapamil. See Guidelines for Intravenous Administration of Drugs, Policy and Procedure Manual, Christchurch Hospital.)
  - Monitoring should continue for up to six hours post drug administration.
- **Patients with symptomatic bradycardia or documented heart block:**
  - Monitoring must continue while patient remains symptomatic and/or in heart block.
- **Patients with temporary pacemakers (usually nursed in CCU, but not compulsory):**
  - Monitoring must continue while a temporary pacemaker is in situ.
- **Malfunctioning pacemaker/Automatic Implantable Cardioverter Defibrillator (AICD):**
  - Monitoring must continue until satisfactory pacing check/appropriate corrective action taken.
- **Post permanent pacemaker/AICD insertion:**
  - Monitor overnight or until pacing check done.
- **Patients post cardiac radiofrequency ablation:**
  - Monitor overnight.
- **Patients post myocardial infarction (ECG changes evident or with positive troponin results):**
  - Monitor for 48 hours. May require longer if documented adverse arrhythmia.
- **Patients suspected of having an Acute Myocardial Infarction:**
  - Monitor until diagnosis is excluded.
  - Monitor for 48 hours if diagnosis confirmed.

Discretionary Monitoring (discuss with Cardiology Registrar on call)

- **Patients post PTCA (or as ordered by the Interventionist Cardiologist):**
  - Monitor for 12 hours.
- **Syncope, if a cardiac arrhythmia is suspected:**
  - Monitor for 24 hours. Remove after 24 hours if no evidence of arrhythmia.
- **Atrial fibrillation/atrial flutter if:**
  - Poor ventricular rate control (rapid ventricular response).
  - Concern that the rhythm may be associated with syncope or underlying ACS.

Note: *Elderly patients with a ventricular rate of less than 150/min may be able to be observed on AMAU using a bedside monitor.*

- Monitor during treatment, e.g., electrical or chemical cardioversion.
- Monitoring can be discontinued once the patient has been in sinus rhythm for one hour post cardioversion.

- **Drug overdoses at risk of cardiac arrhythmia:**
  - Patients who have taken tricyclic antidepressants and who have syncope, seizures, or an abnormal ECG on presentation.
  - Monitor for 24 hours.
  - Monitor asymptomatic patients until the QRS is less than 100 milliseconds.
- **Potassium and/or magnesium electrolyte abnormalities:**
  - Potassium level of less than 2.5 mmol/l.
  - Potassium level of greater than 6 mmol/l.
  - Magnesium level of less than 0.6 mmol/l.
  - Monitor for at least 6 hours after normalisation of serum potassium.
  - If no arrhythmias have occurred, patients can be observed on AMAU using a bedside monitor.
- **Patients without cardiac instability receiving intravenous infusions that require cardiac monitoring:** (The list includes adenosine, amiodarone, dobutamine, dopamine, flecainide, phenytoin, beta-blockers, and verapamil. See Guidelines for Intravenous Administration of Drugs, Policy and Procedure Manual, Christchurch Hospital.)
  - Monitor for at least 6 hours post drug administration.
  - These patients can be observed on AMAU using a bedside monitor.

7.9 Hypertension

7.9.1 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.

7.9.2 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**.

7.9.3 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.
- Consider the following tests to look for secondary causes (e.g., if patient <40 years, or has resistant hypertension or has clinical features that suggest a secondary cause):
  - If phaeochromocytoma suspected, get a 24 hour urine for catecholamines and metanephrines (into an acid bottle) **or** blood for plasma metanephrines (4 ml blood into green lithium heparin tube). This should be done before treatment is given, as treatment may modify these results.
  - Renal ultrasound for renal size and calcification.
  - 24 hour urine for creatinine clearance, Na, K, VMA.
  - Renin and aldosterone plasma levels if Conn's syndrome possible.
  - Morning plasma cortisol level.
  - Renal MRA for renal artery stenosis.

7.9.4 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 mm Hg and diastolic >130 mm Hg but can occur at lower levels if there has been a rapid rise in pressure. Aim to reduce diastolic to around 100 mm Hg 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 a labetalol infusion. Add 500 mg (100 ml) of labetalol to 400 ml normal saline giving a concentration of 1 mg/ml. Start infusion at 2 mg/min (120 mg/hour). The usual dose needed to control BP is from 50 to 200 mg/hour. An 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 in this situation.
  - 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.

7.10 Thoracic Aortic Dissection

7.10.1 Clinical Features

This diagnosis should be considered in all patients presenting with chest pain. There are no specific clinical features, and therefore a high index of suspicion is necessary as this diagnosis is often missed. Patients nearly always present with severe knife-like pain often described as stabbing.

Once the index of suspicion is raised, it is to be treated as though it is a type A dissection until proven otherwise. This is because the mortality for a type A dissection is approximately 2%/hour.

Type A dissection involves the ascending aorta and in type B dissection the ascending aorta is not involved.

7.10.2 Aetiology

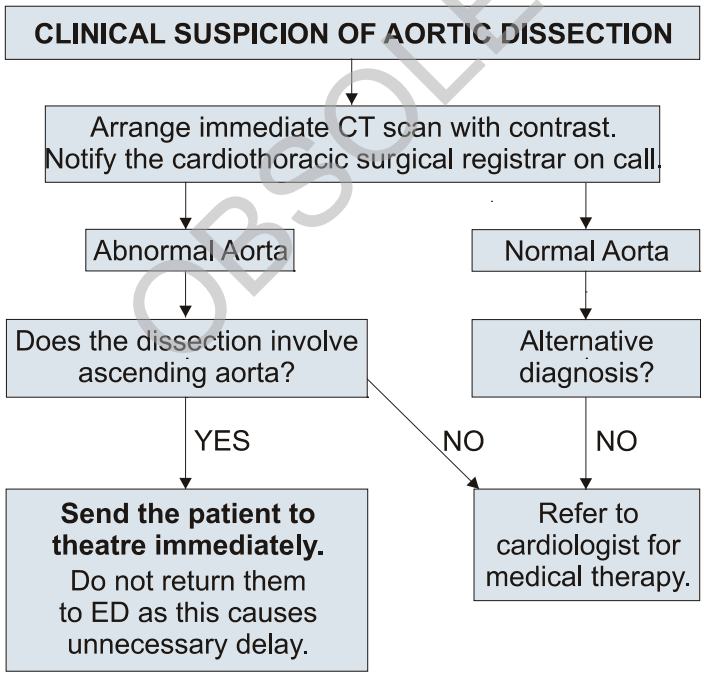
Cystic medial necrosis; Marfan's syndrome; atherosclerosis; hypertension; trauma; post-cardiac surgery; pregnancy.

7.10.3 Investigations

See Diagnosis of Aortic Dissection.

There are only two useful investigations on which decisions should be made. They are CT scan with contrast or a trans-oesophageal echo. The preference at Christchurch Hospital is to perform a CT scan. Delaying a diagnosis to obtain a chest x-ray or ECG etc. is dangerous and inappropriate.

Table 10: Diagnosis of Aortic Dissection



Note: The CT suite needs to be contacted and advised to stop scanning other patients with this patient taking priority.

7.10.4 Treatment - Type A

- Utilise time while waiting for transport to CT scanner. **Contact the cardiothoracic surgical Registrar on call.** Place urinary catheter, give adequate analgesia, e.g., IV morphine. Initiate infusion of hypotensive drugs. See below. Draw bloods for cross-match, CBC and biochemistry. Cross-match six units. An ECG should also be done provided this does not cause delay.
- Medical management: Until the patient is placed on by-pass, aggressive medical management should be pursued immediately. **This will be guided by the Cardiothoracic Surgical Registrar**, who will also ensure that the operating theatre is kept fully informed. The principle is to lower the

absolute blood pressure and the force of contraction. This is best done with vasodilators and accompanying negative inotropes. Do not use vasodilatation on its own as this will only increase cardiac output and stress the false lumen.

- Start a labetalol infusion (see page 53). Give intravenous and later oral beta blockers, e.g., labetalol, unless contraindicated (systolic BP <120, cardiac failure, bradycardia <60/minute, heart block, obstructive airways disease) and start a glyceryl trinitrate infusion (see page 40) or a sodium nitroprusside infusion (see page 53). Aim to reduce the systolic blood pressure to between 100 and 120 mm/Hg to reduce the contractility of the left ventricle. Monitor blood pressure and urine output. Consider stemetil to reduce the risk of vomiting.
- The patient must be accompanied to the CT suite by both doctor and nurse so that medical management, i.e., blood pressure control is applied aggressively and continued until the patient is in theatre and placed on by-pass.
- **The aim is to have a patient in the operating theatre within half an hour of a clinician raising the question "is this an aortic dissection?".**

7.10.5 Treatment - Type B

- Refer to Cardiologist. These patients are generally managed in CCU. Aim to keep systolic BP 100-120 mm Hg. The first line drug to use is labetalol by IV infusion, then oral with other anti-hypertensive drugs as required.
- Morphine for pain with antiemetics.
- Patients should be monitored for complications of dissection. These include mesenteric, renal, or lower limb ischaemia. Consult the Vascular Surgeon if any of these complications occur.

7.11 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.

7.11.1 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 for urinary deposit.
- Na, K, Ca, glucose, creatinine, bili, ALT, AST.
- CBC + diff.
- Echocardiogram.

7.11.2 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.

7.12 Infective Endocarditis Prophylaxis

The following information is taken from the National Heart Foundation "Guideline for the Prevention of Infective Endocarditis associated with Dental and other Medical Interventions" (December 2008). The Heart Foundation has given permission for sections of these guidelines to be reproduced here. The full guidelines, including references, are available from the National Heart Foundation website **(www.nhf.org.nz) under Heart Health > Guidelines > Downloads**, or The National Heart Foundation of New Zealand, PO Box 17160, Greenlane, Auckland 1130.

7.12.1 Cardiac Conditions

The number of cardiac conditions for which prophylaxis is recommended has been reduced significantly (see below). These conditions have been selected because of a high lifetime risk of endocarditis and a high risk of mortality or major morbidity resulting from bacterial endocarditis, should it occur. In line with other recent recommendations we no longer recommend differentiation into high and moderate-risk groups.

The main difference from other recent national recommendations is the retention of rheumatic heart disease in the list of conditions requiring prophylaxis. This reflects the known high lifetime risk of endocarditis in this population and the potential for significant adverse outcomes after endocarditis. Rheumatic heart disease remains a major cause of morbidity and mortality in New Zealand and our recommendations take into account this difference from other developed countries. Although it is possible that the risk of endocarditis may differ with the severity of rheumatic valvular involvement, there is no clear evidence to this effect and prophylaxis is therefore recommended regardless of severity. Prophylaxis is not recommended for those who have had previous rheumatic fever without cardiac involvement. We hope that this pragmatic approach will allow for straightforward interpretation.

Table 11: Cardiac conditions for which endocarditis prophylaxis is recommended

- Prosthetic heart valves (bio or mechanical).
- Rheumatic valvular heart disease.
- Previous endocarditis.
- Unrepaired cyanotic congenital heart disease (includes palliative shunts and conduits).
- Surgical or catheter repair of congenital heart disease within 6 months of repair procedure.

7.12.2 Dental Care

This new NHF guideline highlights the imperative that at-risk patients should remain free of dental disease. This requires emphasis on improved access to dental care and improved oral health in patients with underlying cardiac risk factors for infective endocarditis, rather than a sole focus on dental procedures and antibacterial prophylaxis.

Optimal oral health is maintained through regular professional care and the use of appropriate products such as manual and powered toothbrushes, floss and other plaque-control devices such as antibacterial mouthwashes. Patients need to be strongly advised to comply with a continuing oral and dental care regimen.

Treatments to achieve this goal include:

- Removal of impacted teeth and unerupted teeth.
- Treatment of all teeth with periapical disease by endodontic debridement and root filling or apical surgery or extraction.
- Removal of all carious teeth that cannot be restored.
- Treatment of other abnormalities such as cysts or intra-bony lesions associated with the dentition and related structures.
- Treatment of oral ulcers including those caused by ill-fitting or irritating dental appliances.

- Treatment of inflammatory periodontal disease.
- Oral hygiene instructions for the patient to ensure maintenance of ideal oral health.

Table 12: Dental procedures (plus tonsillectomy/adenoidectomy) for which endocarditis prophylaxis is recommended

All dental procedures that involve manipulation of gingival tissue or the periapical region of teeth or perforation of the oral mucosa*

* The following procedures and events do **not** need prophylaxis:

- Routine anaesthetic injections through non-infected tissue.
- Taking dental radiographs.
- Placement of removable prosthodontic or orthodontic appliances.
- Adjustment of orthodontic appliances.
- Placement of orthodontic brackets.
- Shedding of deciduous teeth.
- Bleeding from trauma to the lips or oral mucosa.

7.12.3 Non-Dental Procedures

Endocarditis prophylaxis is no longer recommended for non-dental procedures (including respiratory, gastrointestinal and genitourinary procedures), unless the procedure is at a site of established infection (see Antibacterial regimen for surgery/procedures at sites of established infection on page 59). Antibacterial prophylaxis to prevent non-endocarditis infections after these procedures may be indicated but recommendations for this are not within the scope of this guideline.

Table 13: Non-dental procedures for which endocarditis prophylaxis is NOT recommended (1), (2)

The following procedures do **not** need endocarditis prophylaxis:

- Surgery involving respiratory mucosa (other than tonsillectomy/ adenoidectomy).
- Bronchoscopy.
- Oesophageal, gastrointestinal or hepatobiliary procedures (including oesophageal stricture dilatation, ERCP).
- Gastrointestinal endoscopy.
- Genitourinary or gynaecologic procedures (including TURP, cystoscopy, urethral dilatation, lithotripsy and hysterectomy).
- Vaginal or caesarean delivery.
- Cardiac procedures (including percutaneous catheterisation).

(1) *Endocarditis prophylaxis may be recommended if the procedure is at a site of established infection*

(2) *Antibacterial prophylaxis to prevent non-endocarditis infection after these procedures may be indicated*

7.12.4 Education and Identification of At-Risk Patients

District Health Boards and other organisations where at-risk patients may be identified are responsible for educating patients and staff about the need for good dental care and appropriate antibacterial prophylaxis. Patient education cards and resources for dentists and healthcare professionals are available from the Heart Foundation.

Electronic alerts should be placed for these patients in appropriate public and private medical information systems. From a dental practitioner's perspective, the Heart Foundation wishes to re-

emphasize the need for improved access to dental care and improved oral health in patients with underlying cardiac risk factors for infective endocarditis, rather than a sole focus on dental procedures and antibacterial prophylaxis.

7.12.5 Antibacterial Prophylaxis

Prophylaxis for dental procedures and tonsillectomy is directed against viridans streptococci. While they are not the only organisms that cause bacteraemia following these procedures, they are the organisms most likely to cause endocarditis.

There have been many reports of viridans streptococci with reduced susceptibility to penicillins, both in New Zealand and internationally. These strains are typically also less susceptible to cephalosporins, especially the oral first-generation cephalosporins. This has contributed to our decision to no longer recommend cephalosporins as oral alternatives. Viridans streptococci have shown a similar increase in resistance to macrolides while their resistance to clindamycin has also increased, but to a lesser extent.

The principles of prophylaxis for prevention of endocarditis from viridans streptococci have been well established in animal models. Successful prophylaxis depends more on prolonged antibacterial activity than prevention of bacteraemia. Indeed, failure of a regimen to suppress post-procedure bacteraemia is not a surrogate marker for failure of prophylaxis. Because of this, both bactericidal (e.g., amoxycillin) and bacteriostatic or non-killing regimens (e.g., clindamycin or clarithromycin) are very effective so long as the antibacterial agent is present in the blood stream for long enough. This can be achieved with a single dose of these agents, provided the correct dosage is given.

Table 14: Antibacterial regimen for dental procedures (plus tonsillectomy/adenoidectomy)

Amoxycillin 2 g (child: 50 mg/kg up to 2 g), administered

- Orally, 1 hour before the procedure, or
- IV, just before the procedure, or
- IM, 30 minutes before the procedure.

Administer parenterally if unable to take medication orally; administer IV if IV access is readily available.

For penicillin allergy or if a penicillin or cephalosporin-group antibiotic is taken more than once in the previous month (including those on long-term penicillin prophylaxis for rheumatic fever):

Clindamycin 600 mg (child: 15 mg/kg up to 600 mg), administered

- Orally, 1 hour before the procedure, or
- IV, over at least 20 minutes, just before the procedure, or
- IM, 30 minutes before the procedure.

Or

Clarithromycin 500 mg (child: 15 mg/kg up to 500 mg) orally, 1 hour before the procedure.

Clindamycin is not available in syrup form in New Zealand.

Beware potential interactions between clarithromycin and other medications.

If the antibacterial agent is inadvertently not administered before the procedure, it may be administered up to 2 hours after the procedure.

Prophylaxis is optimal when antibacterial treatment is begun just before the procedure, to ensure adequate levels are present in the blood stream at the time of the procedure. If it is begun hours or days beforehand, it may select strains with decreased susceptibility so that if endocarditis occurs it is more difficult to treat.

Bacteraemia may complicate established focal infection and its surgical management at any site, such as drainage of an abscess (dental, skin and soft tissues, lung etc) or of peritonitis. It may also complicate procedures (including urinary catheterisation) through infected fluids, such as urine, bile or peritoneal fluid. At all of these sites bacteria commonly associated with infective endocarditis may be present. Patients with established infections at these sites will necessarily receive antibacterial treatment and those at cardiac risk are advised to have appropriate antibacterial agents included (see below) in their overall antibacterial regimen before their procedure.

Table 15: Antibacterial regimen for surgery/procedures at sites of established infection

Treat promptly with antibacterial agents expected to cover the majority of causative organisms. For the purposes of endocarditis prevention, this should include:

- Dental or upper respiratory tract infections - amoxycillin (clindamycin or clarithromycin if penicillin allergy).
- Gastrointestinal, hepatobiliary, genitourinary or obstetric/gynaecological infections – amoxycillin (vancomycin if penicillin allergy).
- Skin, skin structure or musculoskeletal infections - flucloxacillin (a cephalosporin if mild penicillin allergy; clindamycin if severe penicillin allergy or suspect MRSA).

8. Clinical Pharmacology

8.1 Clinical Pharmacology Department Information

- For specific pharmacology information, refer to The Preferred Medicines List ("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, Assoc Prof Murray Barclay.

Consultation Service

- For consultations, Fax 81003.
- For urgent consultations, contact the Registrar ☎ 88354, or Drug Information ☎ 80900.

Consultation Guidelines

Any pharmacology issues, such as:

- Interpretation of drug concentrations, advice on therapeutic drug monitoring and toxicology.
- Complex polypharmacy.
- Guideline writing.
- Drug utilisation/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 Preferred Medicines List (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.
- Drug Concentration Monitoring: detailed drug profiles.
- PHARMAC link: to the Pharmaceutical Schedule.
- MEDSAFE link: includes drug manufacturers' datasheets.
- Drug utilisation review: drug expenditure, campaigns.
- Bulletins/Guidelines: clinical pharmacology bulletins/guidelines (including Peri-Operative Medication).

To access this site, go to the CDHB intranet home page and select **View Departments** > **Clinical Pharmacology**.

Another useful drug information site is UpToDate (under **Clinical Information and Resources** > **UpToDate**).

8.2 Drug Information Service

Staff

- Tracey Borrie, Pam Buffery, Judy Dalrymple, Kathryn Henshaw
- ☎ 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 within the working day.
- Written, referenced answers are provided for more complex questions.
- The service also issues regular bulletins, available via the Clinical Pharmacology intranet site.
- The service has an external website (www.druginformation.co.nz) that contains bulletins, information on drugs in pregnancy and lactation, and other useful drug information.

The service is not set up to:

- Undertake literature searches to assist with research or assignments.
- Provide information directly to patients under any circumstances.

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 contains "preferred" drugs and is not restrictive. It is updated annually, and is written in conjunction with local Specialists. It is available in hard copy from the Department of Clinical Pharmacology, and on the CDHB intranet.

The Pink Book has three main 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.
- Surgical prophylaxis and postoperative antibiotic guidelines.

The Pharmacology Guidelines section:

- Gentamicin/tobramycin and vancomycin dosing guidelines.
- Drug use in renal and liver impairment, the elderly, and the obese.
- Pharmacogenetics.
- Drug and food interactions.
- Drugs in pregnancy and breastfeeding.
- Drug concentration monitoring.
- Adverse drug reactions.
- Drug metabolism (including Cytochrome P450) and interactions.
- Drug profiles, for commonly used drugs.
- Antibiotic sensitivity tables.

8.4 Drug Utilisation Review

Staff

- Jane Vella-Brincat, ☎ 89971

Functions

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

8.5 Dose Individualisation and Drug Concentration Monitoring

Individualisation of patient treatment is the basis of good prescribing. Drug selection and choice of maintenance dose rates are important clinical decisions. Patient characteristics, such as age, weight, presence of renal or liver impairment, diseases, interacting drugs, pregnancy, breast feeding etc. should be considered. See the Pink Book for more detail.

For some drugs with a narrow therapeutic index, drug concentration monitoring is recommended. Laboratory results are reviewed daily and advice provided.

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. Choose the dose-rate (DR) that you would use in this patient if renal function were normal (DR(normal)).
2. Calculate renal function. For estimated renal function, use either the estimated GFR (eGFR) as supplied by Canterbury Health Laboratories, or calculate CrCl using the Cockcroft and Gault equation as follows:

$$CrCl\ (\text{ml/min}) = \frac{(140 - \text{age}) \times \text{lean body weight (kg)}}{\text{plasma creatinine (mcmol/l)} \times 0.8} \quad (\times 0.85 \text{ if female})$$

   - 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. For drugs with fu ≥0.9, calculate the dose-rate for the patient (DR(patient)) as follows:

$$\text{DR(patient)} = \frac{\text{Calculated CrCl (ml/min)}}{100\ (\text{ml/min})} \times \text{DR(normal)}$$

   For drugs with fu <0.9, calculate the dose-rate for the patient (DR(patient)) as follows:

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

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: The creatinine must be stable for the calculated CrCl to be valid (whether using the eGFR result or the Cockcroft and Gault result). In addition, the further the patient is from normal (age, height, weight, etc.), the less valid the estimate. For the purpose of the above calculation, normal creatinine clearance is assumed to be 100 ml per minute.

9. Clinical Procedures

9.1 Introduction

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

A Clinical Skills Unit 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 CDHB intranet under **View Departments** > **Clinical Skills Unit**.

9.2 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 (e.g., 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 may 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 mm Hg.
  - 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 may cause ongoing 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. See Tranfusion Reactions on page 30.

9.3 Central Venous and PICC Lines

Central Venous Access Device (CVAD) require special expertise in their insertion and ongoing care. In general, we recommend that CVADs are placed in Radiology, ICU, Anaesthesia. Seek advice from your Consultant.

Remember that peripherally placed long lines may obviate the need for a CVAD 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 (☎ 81410). These should also be considered for patients with difficult peripheral venous access who require ongoing IV therapy.

9.4 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.

9.5 Blood Culture Collection

- Blood cultures should ideally be taken via the **vacutainer compatible (safety) butterfly method** (see page 65), rather than using a needle and syringe, which may result in poorer quality samples and risk to the staff member.

  *Note: If **safety** butterfly is **not** used, extreme caution is required to ensure a needlestick injury does not occur during disposal.*

- **It is important to avoid air being injected into the anaerobic bottle** - with a butterfly there is often air in the tube, and with the syringe method there is often air in the plunger end. Therefore:
  - If using the butterfly method (see page 65), aseptically inoculate the **aerobic** bottle first.
  - If using the syringe method (see page 66), aseptically inoculate the **anaerobic** bottle first.
- 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.

9.5.1 Vacutainer Compatible (Safety) Butterfly Blood Culture Technique

Assemble the equipment:

- Vacutainer compatible safety butterfly
- 3 alcohol swabs
- Tourniquet
- Blood culture bottles
- Routine blood tubes, if required
- Large blue vacutainer hub, sharps container, tape, and bandaid/plaster.

Ensure that you have the **vacutainer compatible safety butterfly**, then:

- Thread the vacutainer compatible safety butterfly onto the blue blood culture bottle adapter.
- Remove caps from blood culture bottles. Swab each bottle with approved alcohol based skin wipe and leave swab on top of bottle until ready for use.
- Apply tourniquet and vigorously swab skin using alcohol based skin wipe. Use a circular motion to at least 3 cm around the vein to be accessed. **Allow to dry** and do not touch the area with fingers before inserting the butterfly needle.
- Access the vein with the butterfly and secure the wing in place with tape (optional).

  Important: Ensure that the blood culture bottles remain upright.

- Push and hold the blue adapter firmly onto the blood culture bottle and fill each bottle with 10 ml of blood (incremental markings are on the side of the culture bottles).
- Aseptically inoculate the **aerobic** bottle first and then the **anaerobic** bottle (as there may be air in the butterfly tubing that will get into the first bottle).
- If routine blood tubes are also required, fill subsequent tubes in correct order of draw following the cultures. Invert gently to mix.
- Undo the tourniquet.
- Remove the tape and withdraw the butterfly from the vein. Apply plaster. Immediately activate the safety lock device till securely in place over the needle. **Immediately discard entire unit into sharps container**.

9.5.2 Peripheral Blood Culture Technique (Syringe Method)

- Wash hands before and after the procedure. Examination gloves should be worn to protect yourself.
- Remove caps from blood culture bottles. Swab each bottle with approved alcohol based skin wipe and leave swab on top of bottle until ready for use.
- Apply tourniquet and vigorously swab skin using alcohol based skin wipe. Use a circular motion to at least 3 cm around the vein to be accessed. **Allow to dry** and do not touch the area with fingers before inserting the needle and 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 needle 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.
- Fill each bottle with 10 ml blood (i.e., 20 ml per set). Aseptically inoculate the **anaerobic** bottle first when using a syringe, then the **aerobic** bottle. Inoculate blood culture bottles before other blood tubes. Send to lab as soon as possible. Do not refrigerate.

9.6 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 10 mm width free fluid is identified on a lateral decubitus CXR or if loculated fluid is identified and can be reached with ultrasound guidance. Use a 20 ml syringe with a 22G 38 mm 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 (USS) 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 USS.
- Using aseptic technique:
  - Infiltrate with local anaesthetic, then using a 20 ml syringe with a 22G, 38 mm needle, enter the pleural space by progressively advancing while aspirating. This needle is usually of sufficient length to reach the pleural space.
  - Aspirate 20 ml of pleural fluid. Stop the procedure if you aspirate air or the patient develops pain or coughing. If this occurs, 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. Put 2 ml into an ABG syringe and cap it with a bung. 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 240) for advice on which tests to do on the pleural fluid obtained.

Note - when to use Ultrasound:

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

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 66). A local anaesthetic should be used.
- Insert a 14 - 16 G 50 mm 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 50 ml syringe with 20G needle should be put into the rubber giving port in the giving set, and 50 ml aspirated. This will allow flow to start, in a siphoning fashion.
- Aspiration should be stopped when:
  - 1000 - 2000 ml 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.

9.7 Insertion of Intercostal Tubes

The insertion and management of intercostal tubes is a complex and specialised area. Internal medicine patients requiring an intercostal tube should be referred to the Specialist Respiratory or Cardiothoracic surgical team for care 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 Spontaneous Pneumothorax - Treatment on page 243 for further advice.

See also: Intercostal Tubes on page 244.

9.8 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 only by those who have been trained to do so.

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 248.

9.9 Lumbar Puncture

See also: Meningitis on page 128, and Subarachnoid Haemorrhage on page 158.

- 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 1 mm. 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 1-2 mm 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. If possible, put 2 ml CSF into each.

  The following minimum approximate volumes are required for:

Microbiology	Culture, Gram stain, cell count and antigens	1 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 is 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.**

9.10 Urethral Catheterisation

Refer to the guidelines in the Urology section (see page 275).

10. Common Emergency Presentations

10.1 Emergency Department

Main Office

Ground Floor, Parkside, ☎ 80270, Fax 80286

Staff

- Dr Angela Pitchford (Director), Dr Michael Ardagh (Professor of Emergency Medicine).
- Doctors Stuart Barrington-Onslow, Jan Bone, Sarah Carr, Claire Dillon, Dominic Fleischer, Paul Gee, Amanda Holgate, Sandy Inglis, Rob Ojala, Scott Pearson, David Richards, Martin Than.
- Secretary, ☎ 89614
- Triage Nurse, ☎ 80274

Patient Handling

- Patients requiring resuscitation or stabilization in the Emergency Department will be managed in a 'shared', co-operative manner by staff of both the Emergency Department and the relevant inpatient team.
- Patients for whom admission is warranted, in the opinion of the Emergency Department staff, will be admitted. Alternatively they can be assessed in the Emergency Department by the inpatient team and discharged at their discretion.
- Inpatient teams who are expecting a patient referred by a GP or Outpatient Clinic, should advise the Triage Nurse.
- Inpatient teams are expected to respond in a timely fashion in accordance with the triage waiting times:
  - Triage 1: immediately
  - Triage 2: 10 minutes
  - Triage 3: 30 minutes
  - Triage 4: 60 minutes
  - Triage 5: 120 minutes
- 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.

10.2 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.

10.3 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.

10.3.1 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 decompression or drain where appropriate.
3. Administer high flow oxygen.

Circulation

1. Assess circulation.
2. Arrest external haemorrhage by local pressure.
3. Insert 2 large bore IV cannulae. If no IV access, consider alternatives - intraosseous, 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 29).
5. Monitor the patient with an ECG and BP monitor and a pulse oximeter.

Disability

1. Determine the level of consciousness (AVPU).

   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.

10.3.2 Resuscitation and Monitoring

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

Monitoring the progress of 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. A urethral catheter should be inserted if there are no contraindications (see page 275).

10.3.3 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.

Note: Nearly all multisystem trauma patients require CT imaging. Many centres perform "trauma CT scan" imaging from head to pelvis inclusive. If this CT is done, lateral cervical spine x-rays can be omitted, as CT is more sensitive for detecting injury than plain films.

10.3.4 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.

10.3.5 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, and look at the tympanic membranes.

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.
- A urinary catheter should only be inserted if there is no blood at the urethral meatus, no perineal bruising, and the rectal examination is normal.
- 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 76).
- Look for any localising signs.
- Re-evaluate the pupils.

10.4 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.
  - Laryngeal mask 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).
  - Vasodilatation (sepsis, drugs, anaphylaxis).
  - Obstruction (tension pneumothorax, massive pulmonary embolism, cardiac tamponade).

10.5 Altered Level of Consciousness

10.5.1 Initial assessment and resuscitation

See also: Stupor and Coma on page 165.

- 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, 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.

10.5.2 Definitive Management

According to the cause (see page 166).

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

- Trauma
  - Consider CT head 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 head scan if suspected.

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

Table 16: Glasgow Coma Scale

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

10.6 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 in this context.
- 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.
- Haemorrhagic shock with hypotension suggests 1500-2000 ml of blood loss and demands rapid infusion of 2000 ml of crystalloid (see table below).
- 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). See Collection of Blood from Blood Bank on page 29. 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 foetus' expense); therefore shock manifests later in the mother (but earlier in the foetus).*

Table 17: 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%)

10.7 Syncope

Definition - a transient loss of consciousness.

10.7.1 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 51.

10.7.2 Complete assessment

History

The most important part of the assessment is a detailed history, which often requires talking to a witness. The ambulance report is extremely helpful here.

Medications

Current medications, particularly hypotensive drugs, e.g., alpha-blockers, ACE inhibitors, diuretics.

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 (a doctor should be close by).
- 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 - look for acute ischaemia and conduction abnormalities.
- Random blood sugar.
- Na, K and creatinine.
- CBC + diff.

10.7.3 Possible causes

Possible causes, in order of prevalence:

- Vasovagal syncope (90%)

  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 (5 - 10%)

  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 (e.g., tongue bite). There may be a preceding aura. There is often post-ictal confusion and drowsiness for one to two hours. More common during sleep.

- 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.

10.8 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 biconvex lenses) enhances observation and removes optic fixation. Any vertiginous patient without nystagmus in the sitting position **must** have a provocative positional test.

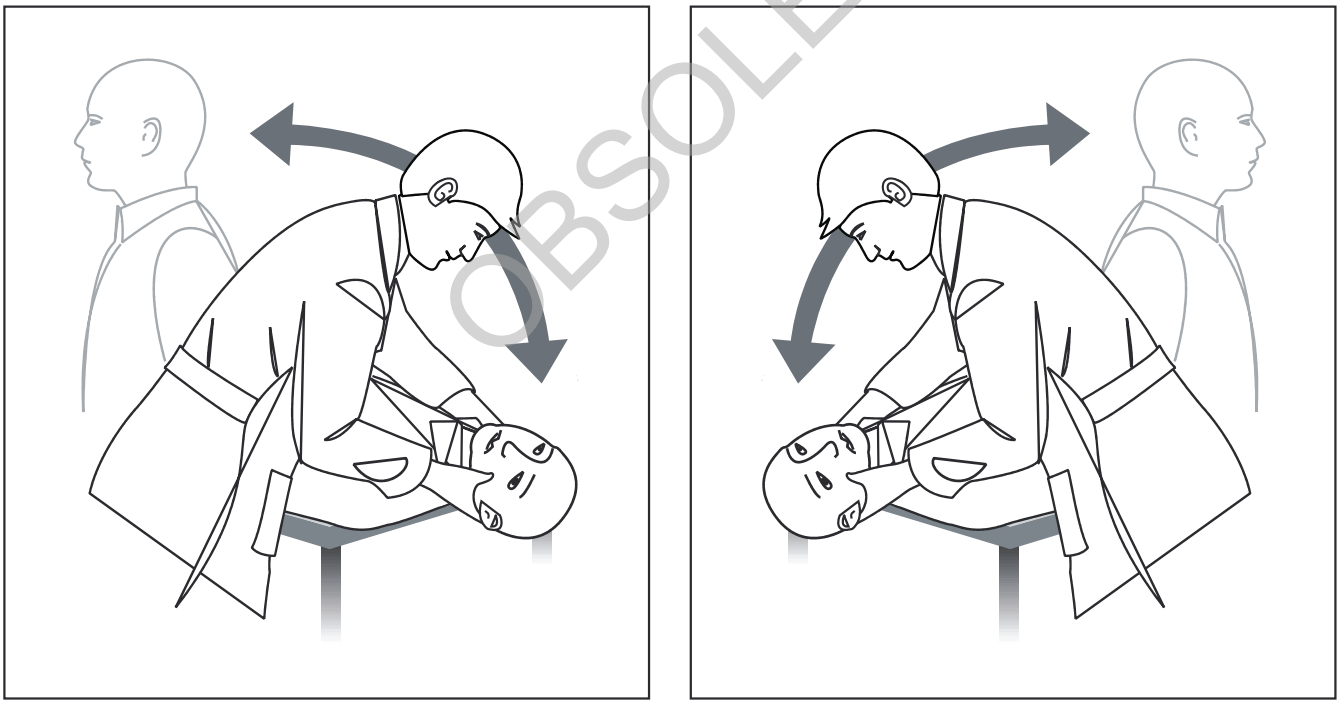
10.8.1 Peripheral Causes of Vertigo

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 semicircular canal. There is no nystagmus when the patient is upright.
- Diagnosis is by the Dix-Hallpike positional test (see below).
- 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 is treated by "repositioning" of the otoconia by the Epley Repositioning Procedure (see page 80).
- Most common in middle aged and elderly. In younger adults it may follow head trauma or vestibular neuritis.

Diagnosis of BPV

This is established by the Dix Hallpike test.

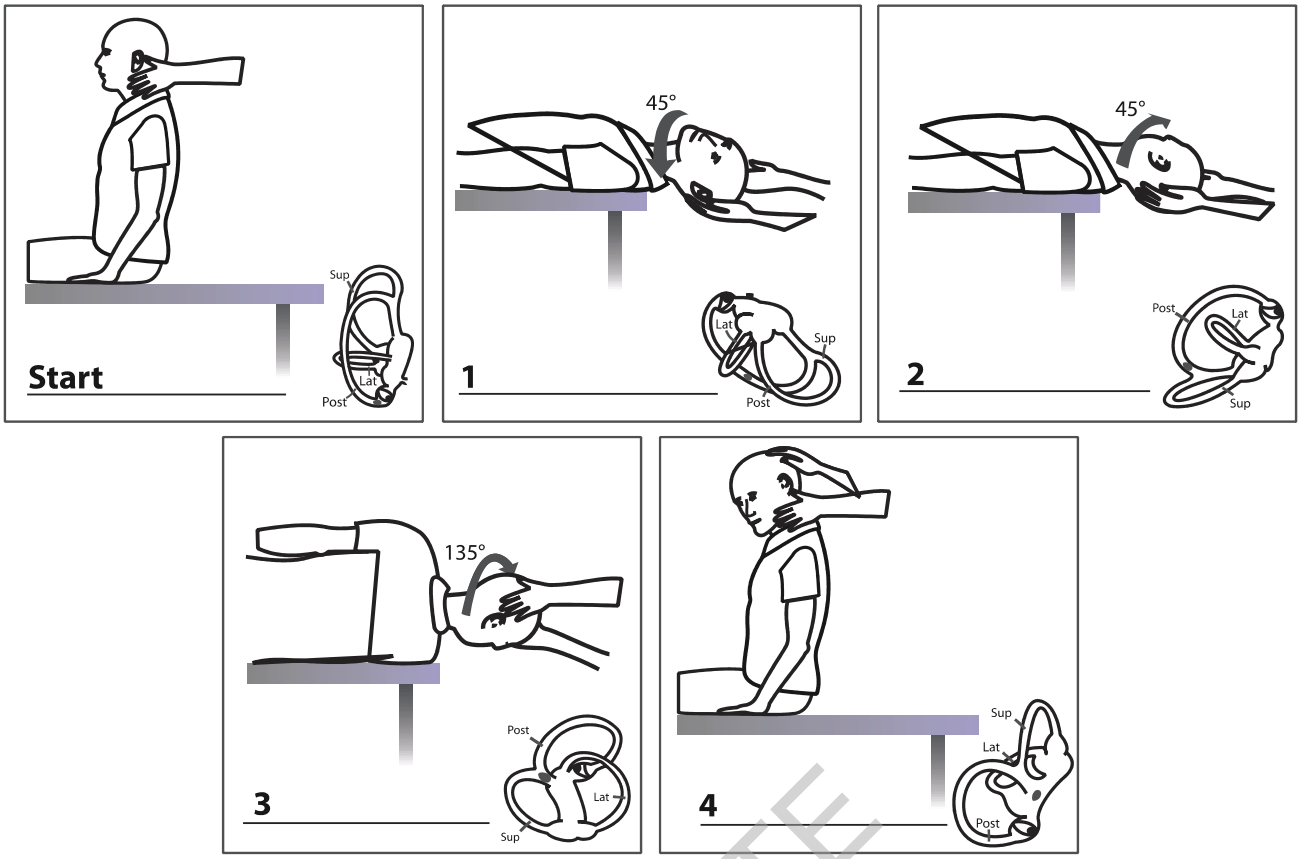


The patient sits with legs extended near the end of the examination table. The examiner turns the head 45 degrees to one side and lies the patient down so the head is below the table. For a positive test the patient must experience acute vertigo and have brisk torsional nystagmus which is anticlockwise to the right ear or clockwise to the left ear.

The test requires some experience to perform well and safely. Seek advice from a more senior colleague if you are unsure.

Treatment of BPV

The Epley Canalith Repositioning Procedure (CRP) can be performed once a certain diagnosis of BPV has been made. Once again, this procedure requires experience. Seek advice.



The repositioning procedure is designed to remove the particle(s) from the posterior semicircular canal. The drawings illustrate treatment for the left ear. The physician stands or sits behind the patient at the head of the table. From the start position the head is turned 45 degrees and the patient tipped back (1) (identical to the Dix-Hallpike test). Wait for the nystagmus to cease, then turn the head 45 degrees to the other side (2). Wait. Ask the patient to turn the body and head to 135 degrees (looking down at the floor) (3). Wait. Ask the patient to straighten up and sit up with the head tilted to the treated side (4). Repeat the Dix-Hallpike test (see page 80). If no response, cease. If positive, repeat the Epley CRP once.

Special care is required 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 vestibular nerve. The nystagmus is always unidirectional 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 (see below) is abnormal on the affected side. The acute vertigo can last up to a week. Balance recovery can take a month, and longer in older individuals. Benign positional vertigo can follow. The main differential diagnosis is cerebellar infarction.

Head Impulse Test

This is to test the normality or absence of the vestibulo-ocular reflex (VOR). The patient should be sitting upright staring at the examiner's nose. The examiner turns the head from the midline sharply to one side 30 degrees. The test is positive if the eyes make saccades to refix on the nose target. In the illustration the test to the left is normal (eyes fixed on target). Thrust of the head to the right results in corrective saccades to the left, indicating a right VOR abnormality.

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.

10.8.2 Central Causes of Vertigo

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.

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**.

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.

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.

10.8.3 Management of Vertigo

Benign Positional Vertigo

Repositioning treatment (see page 80). 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 PO under top lip, 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, pizotifen, sodium valproate

Investigation

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

10.9 Anaphylaxis

Definition - a severe, life-threatening, generalised or systemic hypersensitivity reaction.

- **Cardiorespiratory**: shock, bronchospasm, laryngeal oedema.
- **Skin**: pruritus, urticaria, flushing, angioedema.
- **Other**: headache, vomiting, abdominal pain, diarrhoea, feeling of impending doom.

It is important to document the clinical features that support the diagnosis of anaphylaxis.

Not all symptoms may be present. Patients with only non life-threatening symptoms (e.g., urticaria, external angioedema, abdominal pain) do not have anaphylaxis.

10.9.1 Immediate Management

- **ABC**
  - High-flow oxygen.
  - Lie patient flat and elevate legs.
- **ADRENALINE**
  - **0.5 ml of 1:1000 IM (0.5 mg).**
  - Repeat every five minutes if needed.
- Antihistamines: promethazine 25-50 mg IM (preferred) or via slow IV push; or cetirizine or loratadine both 20 mg PO.
- Hydrocortisone 200 mg IV (onset of action 4-6 hours).
- Intravenous fluids - normal saline to maintain blood pressure.
- Nebulised salbutamol 5 mg (bronchospasm).
- Nebulised adrenaline 2 ml of 1:1000 (2 mg) diluted to 4 ml in normal saline (stridor).
- **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.
  - **Call ICU.**

Notes:

- In **early anaphylaxis** (e.g., witnessed during desensitisation therapy), 0.3 mg of adrenaline IM may be appropriate initial therapy; this dose is equivalent to that present in self-injecting adrenaline devices.
- Patients on beta-blockers are more likely to have severe anaphylaxis and may respond poorly to adrenaline, with side-effects resulting from unopposed alpha-adrenergic stimulation. Initial adrenaline doses should be halved if this is known. Hypotension in patients on beta-blockers may respond to IV bolus and/or infusions of glucagon.

10.9.2 Short-term Management

- Observation:
  - A minority of patients will experience biphasic reactions, with recurrence of symptoms 6-12 hours later. Steroids decrease this risk.
- Confirm diagnosis if this is in doubt:
  - Tryptase is released by activated mast cells. Levels peak 1-2 hours after anaphylaxis onset, and return to normal in 6-8 hours. Some patients with anaphylaxis will have normal tryptase levels, but an abnormal level is useful if there is doubt about the diagnosis. Ideally measure tryptase 60-120 min post-anaphylaxis and again at 8 hours.
- Antihistamines and prednisone, for example:
  - prednisone 40 mg daily for 3 days
  - cetirizine 10 mg bd for 4 days

- Advice to patient:
  - Anaphylaxis plans can be downloaded from www.allergy.org.au.
  - Self-administered adrenaline should be considered when the trigger for anaphylaxis is unknown or repeat exposure is not avoidable (e.g., food and venom, but not drugs).
  - EpiPens can be bought over the counter from pharmacies without a prescription; prices vary ($150-$200). ACC will reimburse EpiPen costs in cases of anaphylaxis where the trigger is food or venom. ACC may not refund in cases where the trigger is unknown, and in this situation will not pay for patients to carry EpiPens prophylactically. Needles, syringes, and ampoules are cheaper but may be difficult for patients to administer accurately.
  - ADR reporting guidelines (http://intraweb.cdhb.govt.nz/cph/pml/intro_04_adr.html).
  - CARM (national pharmacovigilance) ADR reporting form (http://intraweb.cdhb.govt.nz/ADR/adr.htm).
  - Consider MedicAlert.

10.9.3 Medium-term Management

- Referral to Immunology Service (see page 248):
  - For all patients with anaphylaxis unless trigger known, and patient capable of managing attacks. Patients with severe and/or recurrent anaphylaxis should definitely be referred.
  - Management includes identification of trigger, education, advice re avoidance and (if possible) desensitisation therapy.
- Further testing:
  - Skin prick testing cannot be performed for at least 4 weeks after anaphylaxis, as exhaustion of the mast cells can result in false negatives. Some false negative antibody tests (EAST/RAST) can occur if done soon after the event.
- Anaesthetic reactions:
  - Patients with reactions related to anaesthesia should be referred to the Anaesthetic Department.

References:

Soar et al. (2008) Emergency treatment of anaphylactic reactions – guidelines for healthcare providers. Resuscitation, 77, 157-169.

Liberman et al. (2005). The diagnosis and management of anaphylaxis: an updated practice parameter. Journal of Allergy and Clinical Immunology, 115 (3), S483-523.

10.9.4 Immunology and Allergy: recommended referrals

Refer to Immunology and Allergy: recommended referrals on page 248.

10.10 Head Pain

10.10.1 Initial assessment and resuscitation

- Airways, Breathing, Circulation: brief appraisal

10.10.2 Complete assessment

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

10.10.3 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 head scan should precede lumbar puncture if focal signs, papilloedema, or an impaired level of consciousness is present.*

10.10.4 Important to exclude

- Subarachnoid haemorrhage
- Space occupying lesions
- Meningitis

10.11 Chest Pain

10.11.1 Initial assessment and resuscitation

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

10.11.2 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. Myocardial injury markers, including troponins and myoglobin, 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 repeated myocardial injury markers at 6 - 12 hours from the onset of symptoms. Other investigations that may be required include a CTPA or ventilation/perfusion scan, abdominal ultrasound, aortography, echocardiography.

10.11.3 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.

10.11.4 Important to exclude

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

10.12 Abdominal Pain

10.12.1 Initial assessment and resuscitation

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

10.12.2 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.

- Urine test strip 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.

10.12.3 Definitive management

According to the cause. Possible causes:

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

10.12.4 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.

10.13 Shortness of Breath

10.13.1 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 Venturi mask 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 231).

10.13.2 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 to assess pH and $PaCO_2$.
- CXR - particularly for pneumothorax, (is difficult to exclude clinically), pneumonia and cardiac failure.
- Other investigations as indicated.

10.13.3 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.

10.14 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.

10.14.1 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.

10.14.2 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.

10.14.3 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.

10.15 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.

10.15.1 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.*

10.15.2 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, LFTs, Na, K, Ca, CK, urine for myoglobin.

10.15.3 Definitive management

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

11. Endocrinology / Diabetes / Metabolic Disorders

11.1 Endocrinology Department Information

Main Office

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

Inpatient Services Ward 26

- Dr Tom Cawood, Dr David Cole, Dr Catherine Conway, Dr Penny Hunt, Dr Steven 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.

Thyroid Disorders - Nuclear Medicine Department.

- Medical Consultation (Drs Cawood/Hunt/Turner), ☎ 80890, Fax 80869

Children (<12 years)

- Endocrine disorders, growth, etc. through Department of Paediatrics (Dr Karen Mackenzie) in the first instance or Dr Penny 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 test nurses, ☎ 80934, Fax 81159

See Endolab Handbook (November 2008) for details on request, logistics and interpretation of endocrine tests.

See www.cdhb.govt.nz\chlabs\endo for information about screening for endocrine disorders and interpretation of endocrine test results.

11.2 Diabetes Service Information

Main Office

- 550 Hagley Ave, ☎ 80860, Fax 80171

Diabetes Physicians

- Dr Tom Cawood, Dr David Cole, Dr Catherine Conway, Dr Helen Lunt, Dr Peter Moore, Professor Russell Scott, Dr Steven Soule, and for paediatric patients, Dr Karen Mackenzie

Consultation and On-call Service

24 hours a day, seven days a week through the Christchurch Hospital operator. Urgent calls during working hours can be directed to the Diabetes Registrar (pager 8688 or 8660). Out of hours, contact the on-call Consultant through the Christchurch Hospital operator. For less urgent issues that can wait until working hours, contact the Diabetes Registrar via pager, and fax a written referral to 80171.

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. If recent glycaemic control is a concern, consider ordering an HbA1c to aid with assessment.

Other Services

- For a Diabetes Nurse Specialist, contact the Diabetes Centre, ☎ 80860, Fax 80171
- Nurse Maude Diabetes District Nurse (referral management centre), ☎ 375 4238, Mobile 027 437 5903, Fax 355 6085
- Outpatient Appointments, Fax 80171
- Diabetes Specialist Podiatrist – consider referral to High Risk Foot Clinic for patients with diabetes-related foot problems, including foot ulceration and Charcot foot, ☎ 80860, Fax 80171
- Lipid and Diabetes Research Group (Claire Chandler), ☎ 80449, Fax 80457

11.3 Adrenal Insufficiency

11.3.1 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.

11.3.2 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.

11.3.3 Investigations

- Na, K, creatinine, urea, glucose - may all be normal (hyponatraemia common). In later phases of primary adrenal deficiency, low Na and high K, high urea, lowish glucose.
- CBC + diff - may be eosinophilia and neutropaenia.
- 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 30 minutes after Synacthen - 0.25 mg IM/IV] - may be indicated. Contact Endocrine test nurses.
- **Interpretation:** In primary adrenal insufficiency plasma ACTH and renin are markedly raised. Plasma cortisol can be in the "normal" range but there is a diminished response to Synacthen. In pituitary failure (ACTH deficiency), plasma cortisol is inappropriately low for the clinical status, plasma ACTH is normal to low, 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). Consult Endocrine Team.
- In the setting of severe acute illness, a random cortisol >950 nmol/l makes adrenal insufficiency unlikely, <450 nmol/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.

11.3.4 Treatment

- If hypovolaemic, especially if primary adrenal insufficiency, fluid replacement with normal saline to restore blood pressure. 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 empiric antibiotics in cases of profound shock.
- Hydrocortisone 50-100 mg IV then 50 mg q8h 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 mg q8h 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, medic alert, and information sheet on management of acute illness.

11.3.5 Guidelines for Perioperative Steroids in Patients Already on Steroids

Note: Approximate equivalent doses: 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 below). Monitor fluid status, electrolytes and glucose daily.

Table 18: Perioperative guidelines for patient taking steroids

Patients currently taking steroids:
▪ ≤5 mg prednisone daily (and not known to be steroid deficient): ▪ Assume normal HPA response ▪ Additional steroid cover not usually required. ▪ **>5 mg 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 or fax 81159.**

11.4 Steroid Excess (Cushing's Syndrome)

Many symptoms/signs of steroid excess are nonspecific, e.g., obesity, hypertension, glucose intolerance/diabetes, and menstrual irregularity. Thin skin in adults and growth failure in children may be helpful clues. Screening tests for Cushing's include:

- 24hr urinary cortisol excretion or
- Low dose overnight dexamethasone test (1 mg oral dexamethasone at midnight, then measure plasma cortisol at 8:00 am next morning (normal is <100 nmol/l).

All tests for Cushing's syndrome can give false positive and negative results, so if high index of suspicion, consult Endocrinologists.

11.5 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 pregnant patients, measurement of total T4 is more accurate for assessing thyroid function. In acutely unwell hospital patients, total T4 is also recommended (use Endolab request form for total thyroid hormone levels).

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 a sustained 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 radioisotope thyroid scan is helpful to distinguish thyroiditis from toxic nodular disease or 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).

11.6 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.
  - If a fasting test is not possible, or there is a possibility of incomplete fasting, a non-fasting HbA1c is recommended. A fasting glucose of ≥5.5 mmol/l, or an HbA1c >6% requires further diagnostic testing.
- **Glycated haemoglobin (HbA1c):**
  - This is a useful test to measure average glycaemic control over the preceding 3 months. It can be misleading in those with abnormal red cell turnover (bleeding, transfusions, etc.) or haemoglobin variants (e.g., thalassaemia). It is reported as a percentage (typical target is 7%, but this needs to be individualized), but will soon also be reported in molar units (mmol/mol) and an average estimated glucose level will also be given.
  - If recent glycaemic control is uncertain, consider ordering an HbA1c, especially if inpatient review by a member of the diabetes team has been requested.

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. 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.)
- Supplemental SC fast acting insulin such as lispro (Humalog) or aspart (NovoRapid) insulin can be given in addition to twice daily Penmix. SC fast acting insulin should be prescribed before or with meals, e.g., 6 units if blood glucose is ≥ 15 mmol/l.
- Subcutaneous fast acting insulin injections with aspart (NovoRapid) or lispro (Humalog) are preferred to SC neutral insulin injections using Actrapid or Humulin R.
- As a rule of thumb, in an insulin-sensitive person, 1 unit of aspart (NovoRapid) or 1 unit of lispro (Humalog) will lower blood glucose by 3 mmol/l.
- Pen injectors (e.g., Novopen, Humapen) can be obtained from the Christchurch Hospital Pharmacy.

Table 19: Description of Insulins currently available in New Zealand

Type of Insulin	Brand Names	Description of Action	Duration of Activity *		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.
Glulisine	Apidra	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 insulin e.g., 30% neutral 70% isophane	Penmix30 Humulin 30/70	Biphasic (Short acting plus intermediate)	As for component insulins	**24**	Half an hour before breakfast and the evening meal.
Humalog Mix25 (25% Humalog, 75% Protamine suspension of Humalog)	Humalog Mix25	Biphasic (Fast acting plus intermediate)	As for component insulins		Take with food, usually with breakfast and with evening meal. Must prescribe clearly (do not confuse with Humalog).
Isophane (NPH)	Protaphane Humulin NPH	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	Lantus	Long acting	**4 - 24**	**>24**	Background (basal) insulin. Reduced risk of nocturnal hypoglycaemia.
Detemir	Levemir	Long acting	**4 - 18**	**>24**	Background (basal) insulin. Reduced risk of nocturnal hypoglycaemia.

* *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.*

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 (pre-meals and at bedtime).
- Patients on Penmix (or equivalent) should be tested before main meals and at bedtime.
- Patients on fast acting insulins (aspart (NovoRapid) or lispro (Humalog)) may need to do additional tests 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. Osmolarity (tonicity) is however usually elevated.
- Corrected Na can be calculated by adding 2.4 mmol/l for each 5.5 mmol/l rise in glucose above normal. Thus for each 10 mmol/l rise in serum glucose, an approximately 4 mmol/l fall in serum sodium is expected.
- It follows that a fall in glucose is usually associated with a rise in sodium, i.e., correction of both elevated glucose and associated dehydration will usually result in normalisation of the serum sodium.
- The most relevant measure of osmolarity in this setting is **effective osmolarity** (2xNa + glucose). See below for further explanation.

Osmolarity and osmolality

Osmolarity is the number of particles of a substance in a volume of fluid (e.g., mmol/l), and **osmolality** is the number of particles dissolved in a mass of fluid (e.g., mmol/kg). In clinical practice, these values are virtually the same. Strictly speaking, **osmolality** is the term used in the reports issued from the laboratory, and **osmolarity** is what is calculated from mmol/l of the venous solutes. "Effective" osmolarity is a calculation that excludes urea since this moves freely between extracellular and intracellular compartments.

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 >160 mcmol/l or eGFR <30 ml/min), 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.

Glitazones - induced fluid retention

- Glitazones (pioglitazone and rosiglitazone) can cause fluid retention. Minor ankle swelling is unlikely to be an indication for cessation of therapy. Glitazones should however be discontinued in patients presenting in heart failure.

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 with the patient and caregivers, 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.

11.7 Diabetic Ketoacidosis (DKA)

11.7.1 General Principles and Precautions

- DKA is defined by hyperglycaemia with positive plasma ketones and an arterial pH ≤7.30 and/or a plasma bicarbonate ≤15 mmol/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 101).

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

11.7.2 Common Causes of DKA

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

11.7.3 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.

11.7.4 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. Patients on long acting insulins such as glargine or detemir, who are suffering from mild/moderate metabolic disturbance only, can continue their long acting subcutaneous insulin at a reduced dose, e.g., 70% usual dose, whilst receiving IV Actrapid therapy.

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).
- *Effective osmolarity* (2xNa + glucose, in mmol/litre) should be monitored in severely unwell patients or in those with hypernatraemia. *Effective osmolarity* measures only osmolytes that draw fluid from one compartment. Urea, which diffuses freely across membranes, is excluded from the calculation. Serum effective osmolarity is closely related to mental status. Coma is usually present when *effective osmolarity* is >340 mosm/l.
- Patients who have access to a Medisense Optium or Optium Xceed meter can test for capillary beta hydroxybutyrate. Some wards (for example, AMAU) have access to this meter. Inpatient capillary beta hydroxybutyrate test strips can be prescribed on the drug chart through the hospital pharmacy. This test provides a better measure of changes in DKA status than does monitoring of urine ketones.
- Patients with DKA have a beta hydroxybutyrate measurement >1.2 mmol/l and often much higher. If beta hydroxybutyrate is <1.2 mmol/l and the patient is unwell, consider other diagnoses.
- 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

- Normal saline 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 normal saline 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.
- Aim to correct major metabolic disturbance slowly.
- Half normal saline is rarely needed. Many patients will experience a rise in Na following correction of hyperglycaemia. In occasional patients, initial IV therapy may result in Na continuing to rise above the normal range, however *effective osmolarity* (2xNa + glucose; see Monitoring (above) for further information) may be falling appropriately with treatment.
- When the blood glucose approaches 15 mmol/l, change to 4% dextrose and fifth normal saline.
- See below for K replacement. Many patients can receive K replacement using ready mixed 30 mmol KCl in 1 litre normal saline or 4% dextrose and fifth normal saline. Some patients will however require K added to normal saline. For safety reasons, K replacement must be given via an infusion pump.

Insulin

- Add 50u Actrapid to 50 ml normal saline 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 normal saline if glucose >15 mmol/l or 4% dextrose and fifth normal saline 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 100).

- When glucose has fallen to around 15 mmol/l, change IV fluid replacement to 4% dextrose and fifth normal saline or 5% dextrose.
- 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.

Table 20: 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 normal saline. 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 at least a 2 hour overlap from IV to SC insulin especially if intermediate or long acting insulin has just been given.
- In patients already on a long acting insulin (e.g., glargine, detemir) prior to admission, consider continuing this insulin throughout hospitalisation but at a reduced dose (70% usual dose).
- 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 **either** Penmix 30 before breakfast and the evening meal, **or** isophane NPH (Humulin NPH or Protaphane) at bedtime with aspart (NovoRapid) or lispro (Humalog) with main meals.
- If the patient is converting to SC aspart (NovoRapid) or lispro (Humalog) insulin, a small dose of long acting insulin such as isophane NPH (Humulin NPH or Protaphane) may also be required at the time the IV infusion is discontinued.

Use of sliding scale SC 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 hyper-amylasaemia often occur in DKA. The raised amylase may be the result of extra-pancreatic secretion and does not necessarily mean the patient has pancreatitis.
- IV bicarbonate is rarely, if ever, necessary. 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 10-20 mmol of potassium and review pH in one hour.
- Always refer the patient to the Diabetes Service to assess overall diabetes management.

11.8 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 measured 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%.

11.8.1 Investigations

As for DKA (see page 98) but include plasma osmolality.

11.8.2 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.
  - If severely unwell (measured serum osmolality >340 mosm/kg, glucose >50 mmol/l, significant intercurrent illness or comorbidities), 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**
  - Total body Na will be low, however serum Na is often elevated secondary to dehydration.
  - Initial therapy is **1 litre normal saline over 30-60 minutes**.
  - Subsequent therapy will be dependent on the patient's clinical state but is likely to be **2-3 litres normal saline** or **half normal saline at 500 ml/hr**. Serum Na may rise during treatment, however this is unlikely to be of major concern if effective osmolarity is falling (see Treatment on page 98 for an explanation of effective osmolarity), level of consciousness is improving, and IV therapy is producing a slow but steady improvement in metabolic status.

- Aim for slow metabolic correction, i.e., over >24 hours for less severely unwell patients and up to 72 hours for more severely affected patients.
- Run 5% dextrose in addition to normal saline or half normal saline 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 4% dextrose and fifth normal saline or 5% dextrose only.
- K replacement will probably not be needed initially, but, after a few hours rehydration, K may be needed at a rate averaging 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 (see page 258).

11.9 Perioperative Management of Diabetes

Peri-operative protocols are available on the wards (see C160011 insulin/dextrose infusion sheet) but no single protocol will work for all patients, hence individualized treatment plans may be required, usually supervised by the Anaesthetist. If the C160011 protocol is not suitable, here are some suggested regimens:

- If on oral agents, omit drug. Restart when eating for at least 12 hours.
- If patient has Type 1 diabetes and uses glargine (Lantus) insulin at bedtime, then give usual glargine dose on day before surgery, fast from midnight and omit meal-time rapid-acting insulin on day of surgery. Measure blood glucose 2-4 hourly pre- and post-operatively and every hour during surgery. Patient may need dextrose infusion if glucose dropping (e.g., <5 mmol/l), and may need additional infusion of rapid-acting insulin if becoming hyperglycaemic (e.g., >11 mmol/l), at the discretion of the Anaesthetist.
- If on insulin but not on glargine, omit morning subcutaneous insulin. Start infusion, using a pump, of 1 litre 5% dextrose, 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, hyponatraemia is likely to develop if this protocol is continued. Therefore 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.

11.10 Management of the Newly Diagnosed Patient with Diabetes

- Refer all patients to the Ward Dietitian.
- **The non-obese patient**: if the patient is not ketotic, they may safely be given a trial of diet, and adding an oral agent if the presenting glucose level is very high (unless contraindicated, metformin is usually the drug of choice for initial oral therapy) 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 ≥160 mcmol/l, eGFR <30 ml/min), 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. If recent glycaemic control is uncertain, consider ordering an HbA1c.

11.11 Hypoglycaemia

11.11.1 In Patients with Diabetes

This is commonly seen in patients on insulin or sulphonylureas. Manage as detailed below, but usually no need to draw blood for laboratory tests to investigate the cause of the hypoglycaemia. A mismatch of insulin or sulphonylurea to carbohydrate intake is the likely cause of hypoglycaemia. Consider worsening renal function or (rarely) hypocortisolism as possible contributors.

11.11.2 In Patients without Diabetes

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.

11.11.3 Management of Hypoglycaemia

- 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. Half a glass of lemonade or fruit juice may be an appropriate first step, depending on clinical circumstances such as degree of hypoglycaemia, amount of insulin taken, etc. The patient's capillary glucose should be checked every 10 minutes, and further lemonade/fruit juice given until glucose >3.5 mmol/l, and then longer acting carbohydrates given (or usual meal if available in less than 15 minutes).

- **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.

11.12 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 defence 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.

11.12.1 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, $NaHCO_3$ infusion
  - Hyperalimentation: IV or parenteral nutrition

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

11.12.2 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. Calculate osmolarity **[**(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.

11.12.3 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 10 mmol/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 4% dextrose and fifth normal saline, 77 mmol/l for half normal saline and 154 mmol/l for normal 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, e.g., 10 ÷ 3.9, plus insensible losses of 1.5 litres, giving total of 4 litres per 24h or 166 ml/h.
- Frequent clinical and biochemical reviews are essential in patients with severe and symptomatic hypernatraemia; repeat Na after 6-8 hours 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.

11.13 Hyponatraemia

11.13.1 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.

11.13.2 Causes

- These are many and varied.
- Remember to consider factitious causes:
  - Laboratory error - check anion gap (see page 110) 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).

11.13.3 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 family) is important in assessing the likelihood of plasma volume depletion (e.g., history of poor salt intake, nausea, vomiting and diarrhoea, recent use of thiazide diuretic).

11.13.4 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.

- **Laboratory:**

  Hypo-osmolar plasma, inappropriately concentrated urine (>100 mmol/kg), urine Na >20 mmol/l (**not** if water restricted), reduced uric acid, urea, creatinine.

Note:

- Hyponatraemia with raised plasma osmolality occurs in hyperglycaemia (see page 97).
- 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).

11.13.5 Management of Hyponatraemia

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 normal saline 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 1-2 mmol/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.
- Hypertonic (3%) saline - infuse 1-2 ml/kg of body weight/hour and check plasma Na every 2-4 hours to guide therapy.
- Investigate and treat underlying cause.

11.14 Hypercalcaemia

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

11.14.1 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.
- Immobilisation (ICU).

11.14.2 Symptoms

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

11.14.3 Investigations

Note: *The ionised calcium may be misleading and we do not advocate routine assay of ionised calcium.*

- In all cases of uncertain aetiology, request Na, K, Ca, Mg, PO_4, alk. phos., alb, creatinine, and PTH level - **before** giving hypocalcaemic drugs.
- Consider the tests listed below and Endocrinology consult, depending on the clinical context:
  - 25OH Vitamin D.
  - CXR (lung cancer, sarcoidosis).
  - CBC + diff and ESR.
  - Thyroid function tests.
  - Serum protein electrophoresis, immunoglobulin levels, and serum free light chain analysis (myeloma).
  - X-ray painful bones (metastases, myeloma), consider radionuclide bone scan.
  - Urine calcium to creatinine ratio (preferably fasting).
  - 1-25 diOH vitamin D and PTH related peptide assay may be helpful, the latter if malignancy suspected. Recommend consultation with Endocrinology as these assays are performed infrequently.

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

Table 21: Calcium Correction Formula

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

11.14.4 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 normal saline over 2 hours then 1 litre normal saline 6-8 hourly and reassess at regular intervals. Potassium supplements 10-20 mmol KCl per 500 ml may be required.
- Bisphosphonates
  - Pamidronate 60 - 90 mg in 0.5 litre normal saline IV over 2 hours or zoledronic acid 4 mg in 100 ml normal saline 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.
- 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.

11.15 Hypocalcaemia

Check albumin and if necessary adjust the calcium level (see page 107). 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).

11.15.1 Causes

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

11.15.2 Investigations

- Plasma Ca, PO_4, creatinine, Mg, alk. phos., albumin, 25 OH vitamin D and PTH levels.
- Urine calcium to creatinine ratio (preferably fasting).
- Consider malabsorption and lack of other fat soluble vitamins (A, E, and K) and assessment for osteomalacia.

11.15.3 Management

- If severely symptomatic:
  - Give calcium gluconate IV e.g., 10 ml of 10% solution as bolus over 2 minutes.
  - 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.
  - Start oral calcium e.g., 1000-2000 mg elemental calcium daily, e.g., Calci-Tab 500 1-2 BD or Calci-Tab effervescent 1-2 daily.
  - Start 1,25 diOH vitamin D (calcitriol) e.g., 0.25 mcg - 1 mcg/day

- Monitor serum calcium 12-24 hourly. Consult Endocrinologists.
- Magnesium deficiency:
  - If severe (Mg<0.5 mmol/l) and symptomatic, e.g., arrhythmia or tetany, consider 50 mmol Mg IV slowly over 8-24 hours.
  - Otherwise, oral Mg supplements 10-20 mmol/day (Mg chelate 50 mg tablets (2 mmol Mg/tablet); Mylanta suspension (5 mmol Mg/7.5 ml)).

11.16 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.

11.16.1 Causes

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

11.16.2 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.

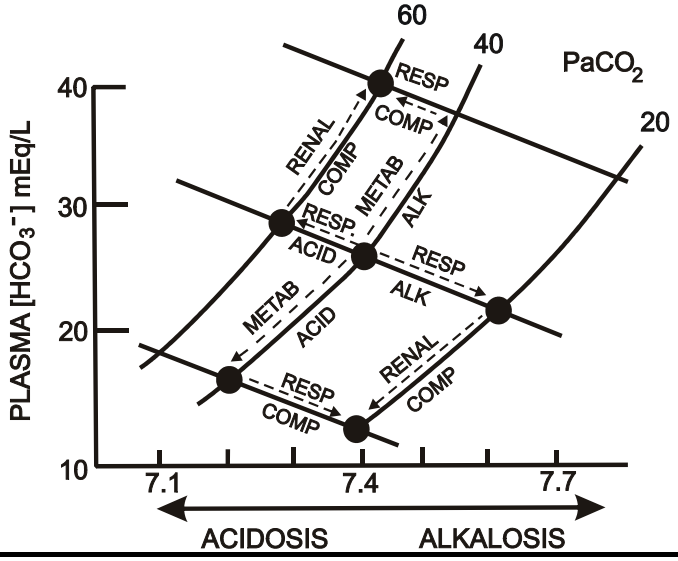
11.16.3 Follow-Up

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

11.17 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:



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.

11.17.1 Causes of Metabolic Acidosis

Table 22: 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.

11.17.2 Investigations

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

11.17.3 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.

11.17.4 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.

11.17.5 Investigations

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

11.17.6 Treatment

- Treat the underlying cause.

12. Fluids and Nutrition

12.1 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:** 70 mmol (~1 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 (normal saline)
   - **Standard premix**: sodium chloride 0.9% + 30 mmol/l KCl
   - **Dextrose 5%**
2. Colloids:
   - Gelatin succinylated
   - Pentastarch 6% and 10%
3. Blood Products:
   - RBC
   - Fresh frozen plasma (FFP)

General rules for IV fluids

The elderly and those with renal or cardiac dysfunction have difficulty excreting salt (sodium). It is critical to limit the infusion of intravenous fluids, particularly sodium chloride 0.9% in these patients 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, BP, JVP, urine output) 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: gelatin succinylated or pentastarch 6% or 10%
   - 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 29)

   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 Rh negative blood.
   - Keep blood and patient warm if massive transfusion necessary. If massive transfusion is required, contact the Transfusion Medicine Specialist ☎ 80310.

Maintenance fluids

- Ensure patient really needs IV fluids and is unable to manage with oral or NG fluids.
- Sodium chloride 0.9% or dextrose 5% 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.

12.2 Nutrition Support

12.2.1 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.

Nutrition 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.

Standard parenteral nutrition (PN) consists of 1.5, 2, and 2.5 litre bags containing glucose (200-320 g), amino acids (60-100 g), lipid emulsion (60-100 g), electrolytes, trace elements and multivitamins. Non-standard bags are also available at a higher cost.

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 and intolerant of enteral feeding.
- Mucositis following chemotherapy.
- Short bowel syndrome.
- Small bowel obstruction or prolonged ileus.

PN is an expensive therapy with risks to the patient. PN of less than 5 days is unlikely to benefit the patient.

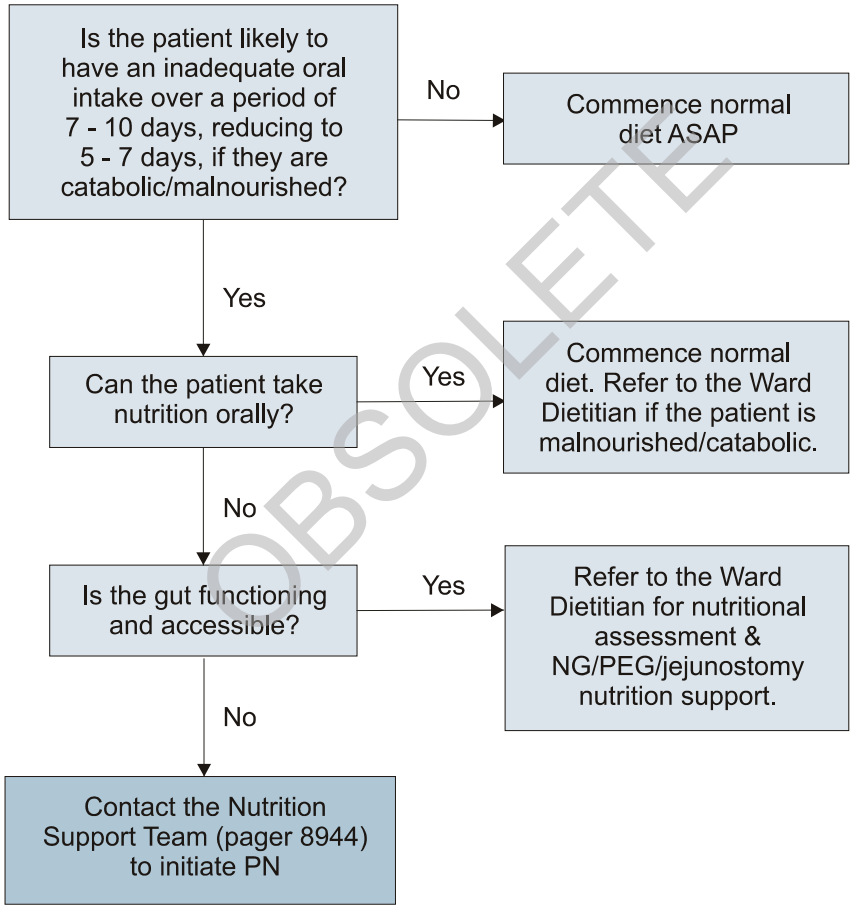
PN can be initiated once a peripherally inserted central catheter (PICC) or other central venous catheter has been placed and the Nutrition Support Team (pager 8944) or the ward dietitian has been consulted. PN is initiated at half rate for the first 6 hours. If blood glucose levels are satisfactory, and the patient is not at risk of refeeding syndrome (see page 115), increase to full rate as determined by the dietitian.

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.

For further useful information on nutrition support, refer to "Parenteral Nutrition FAQs". This is available on general surgical wards and in ICU, CHOC, BMTU, Ward 29, and on the intranet under **View Departments** > **Pharmacy Department** > **Frequently Asked Questions**.

If in doubt, contact the Nutrition Support Team (pager 8944) or your ward dietitian.

Table 23: Medical Review of Patient's Nutritional State



Notes:

- PICC or other central venous 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.
- Vitamin K must be provided via IV line 2 mg once weekly - separately.
- Patients referred after 11:00 Friday and in the weekend will receive a standard PN without trace elements and vitamins. Early referral is encouraged.

12.3 Refeeding Syndrome

Refeeding syndrome can be defined as the potentially fatal shifts in fluids and electrolytes that may occur in malnourished patients receiving artificial refeeding (whether enterally or parenterally). These shifts result from hormonal and metabolic changes and may cause serious clinical complications. The hallmark biochemical feature of refeeding syndrome is hypophosphataemia. However, the syndrome is complex and may also feature abnormal sodium and fluid balance; changes in glucose, protein, and fat metabolism; thiamine deficiency; hypokalaemia; and hypomagnesaemia.

Awareness of refeeding syndrome and identification of patients at risk is crucial as the condition is preventable and the metabolic complications are avoidable.

There is a high risk of developing refeeding problems if:

- One or more of the following:
  - BMI <16 kg/m^2.
  - Unintentional weight loss >15% within the last 3-6 months.
  - Little or no nutritional intake for more than 10 days.
  - Low levels of K, PO_4, or Mg **prior to feeding**.
- Or if two or more of the following:
  - BMI <18.5 kg/m^2.
  - Unintentional weight loss >10% within the last 3-6 months.
  - Little or no nutritional intake for more than 5 days.
  - A history of alcohol abuse or drugs including insulin, chemotherapy, antacids, or diuretics.

12.3.1 Management

For patients who are at risk of refeeding syndrome:

- Check K, Ca, PO_4, and Mg.
- Before feeding starts, administer thiamine 300 mg daily PO and give 'Vitamin B Complex Strong' 1-2 tabs PO TDS. This preparation contains Vitamins B1, B2, B3, and B6. For severely affected patients or if Wernicke's encephalopathy is suspected give thiamine 300 mg daily IV or IM in three divided doses for 2 days, and 'Vitamin B Complex Strong' in the above dosage. A multivitamin preparation should also be given once daily.
- Contact dietitian to start feeding.
- Rehydrate carefully and supplement and/or correct levels of potassium (suggest 2-4 mmol/kg/day PO or IV), phosphate (0.3-0.6 mmol/kg/day PO or IV), and magnesium (0.2 mmol/kg/day IV or 0.4 mmol/kg/day PO), unless pre-feeding levels are high. Calcium supplements may be needed.

  Note: *Approximate conversions mmol to mg are: phosphate 1 mmol ≈ 31 mg, potassium 1 mmol ≈ 39 mg, and magnesium 1 mmol ≈ 24 mg.*

  Note: *For further information on phosphate replacement and PN generally, refer to Management of Hypophosphataemia in "Parenteral Nutrition FAQs" (which is available on general surgical wards and in ICU, CHOC, BMTU, and Ward 29).*

- Monitor K, PO_4, Ca, and Mg for the first 2 weeks and amend treatment as appropriate.

Reference: Mehanna et al BMJ 2008;336: 1495-1498.

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

- Assoc. Prof. Murray Barclay, Dr Michael Burt, Dr Teresa Chalmers-Watson, Dr Bruce Chapman, Dr Steven Ding, Dr Richard Gearry, Dr Catherine 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 76):
  - History often unreliable.
  - Useful signs include:
    - Resting tachycardia.
    - Hypotension.
    - Postural BP drop >15 mm Hg.
- Stabilize patient and monitor:
  - Give normal saline IV, then blood when available.
  - Use Group O Rh negative blood in an emergency. See Collection of Blood from Blood Bank on page 29.

- 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 (25 - 50 mcg/h) for up to 72 hours. Consult Gastroenterologist. Administer prophylactic antibiotics, e.g., cefotaxime, ceftriaxone.
- 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.)

Peptic ulceration

- **Acute bleeding** from a peptic ulcer. High dose omeprazole infusion is beneficial in specific situations. This will be directed by the Gastroenterologist. This regimen may be followed:
  - Bolus omeprazole IV injection: 80 mg stat loading dose, followed immediately by continuous omeprazole IV infusion of 8 mg/hour for 70 hours.
  - Commence oral omeprazole 20 mg once daily at the end of the 70-hour infusion period.

  Alternatively, if the omeprazole infusion product is unavailable, use:
  - Bolus omeprazole IV injection: 80 mg stat, followed in 6 hours by omeprazole IV injection 40 mg every 6 hours. The **total** duration of IV treatment should be **72 hours**.
  - Commence oral omeprazole 20 mg once daily at the end of the IV treatment period.

  Administration of parenteral omeprazole:
  - **Bolus omeprazole IV injection**
    - When administering the **bolus** injection, it is important to use the **IV injection product**, **NOT** the IV infusion. These two formulations are different and are not interchangeable due to stability concerns.
    - Reconstitute each 40 mg vial with the solvent provided, according to the guidelines in the package insert.
    - Administer reconstituted vial by direct IV injection (into vein or side arm) over at least two and a half minutes at a rate not exceeding 4 ml/min.
  - **Continuous omeprazole IV infusion**
    - When administering the **continuous infusion**, it is important to use the **IV infusion product**, **NOT** the IV injection. These two formulations are different and are not interchangeable due to stability concerns.

- Reconstitute **two** 40 mg vials of omeprazole infusion with 10 ml normal saline each.
- Remove 85 ml from a Baxter 250 ml bag of normal saline (takes 15 ml bag overage into account). Add both reconstituted vials to this bag.
- The final diluted infusion product = 80 mg/200 ml normal saline. Run this at **20 ml/hour** (8 mg/hour). It will be sufficient to last for 10 hours.
- The infusion should be continued for 70 hours (unless stopped after a diagnosis is made at endoscopy). Therefore, **two** 40 mg vials of omeprazole IV infusion will need to be reconstituted and diluted every 10 hours, as directed above.

Helicobacter pylori

- **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.
  - Anticipatory.

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 TDS PO, IM, IV, but higher doses may be required.
  - Domperidone 10 mg QID PO. Preferred initial antiemetic agent for Parkinsonism.
- Phenothiazines:
  - Prochlorperazine 5 - 10 mg TDS PO, IM, PR. (Tabs 5 mg, buccal 3 mg, injection 12.5 mg, PR 5 mg and 25 mg.)
- Cyclizine 25 - 50 mg TDS PO, IM, IV.
- Sedatives and hypnotics may be used.
- Ondansetron (for approved indications). May cause constipation.

Note: *for vomiting in malignancy, refer to Management of Nausea and Vomiting (see page 26).*

13.4 Acute Diarrhoea (<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**: Norovirus. 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.
  - Acute diarrhoea is not an indication for colonoscopy.

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 litres 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 278).
  - 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 TDS PO 7-10 days. Is effective for relapse or recurrence. Alternative - vancomycin 125 mg PO QID.
  - 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 137).
  - 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 CT scan abdomen and **urgent 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.
  - Patients on immunosuppressive treatment - steroids, azathioprine, TNF alpha antibodies, etc., are at increased risk of infection.

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). If no response then consider:
  - Faecal softeners (e.g., docusate).
  - Lactulose has an osmotic effect but may cause excess flatulence and bloating.
  - 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 Picoprep or (if less severe) Movicol 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.

13.7 Acute Hepatocellular Dysfunction (Hepatitis)

13.7.1 History

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

13.7.2 Investigations

- USS 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. If acute Hepatitis B is possible, check Hepatitis B IgM core antibody.
- EBV & CMV serology.

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, e.g., 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 123), consider cefotaxime 1g q6h IV or ceftriaxone 2 g IV q24h 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).
- Alb, bili, alk. phos., AST, ALT, 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 123), aspirate for diagnostic purposes.
- Correct coagulation defects with vitamin K 10 mg IV slowly; consider fresh frozen plasma only if bleeding.

13.8.5 If encephalopathy suspected

- Purge with lactulose 10-30 ml TDS adjusted to produce three loose stools per day. Fleet enemas can also be used.
- Watch for alcohol withdrawal (see page 11).
- Consult Gastroenterologist promptly.
- Sometimes short term protein restriction and neomycin may be indicated.

13.9 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 serum-to-ascites albumin 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 an ascitic fluid white count of >250 x 10^6/l with neutrophils predominant. The initial treatment for proven or suspected bacterial peritonitis is cefotaxime 1 g q6h IV (use higher dose if the patient is unwell) or ceftriaxone 2 g IV q24h, and albumin 1.5 g/kg ± further dose of 1 g/kg on day 3.

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 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.

13.10 Acute Pancreatitis

See also the **Acute Pancreatitis Pathway** which is in the Surgical Wards and the Emergency Department.

13.10.1 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.*

13.10.2 Diagnosis

- Serum amylase is usually elevated at least 3 x above normal range in appropriate clinical setting. Other abdominal diseases may cause a lesser elevation of amylase.

13.10.3 Aetiology

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

13.10.4 Investigations

- Serum amylase. Serum lipase only required if amylase is normal and there is a strong clinical suspicion of pancreatitis, especially with a history of pain for more than 48 hours.
- CBC + diff.
- Na, K, Ca, PO_4, creatinine, glucose, LDH, bili, alk. phos., AST, ALT, GGT, CRP.

- Blood cultures.
- Abdominal ultrasound.
- Arterial blood gases.
- Lipid analysis if types I and V hyperlipidaemia.
- CXR.

13.10.5 Management

- Treatment of shock (see page 76).
- Pain relief - pethidine is first choice.
- Patients should eat and drink as tolerated.
- 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 24: Prognostic Factors in Acute Pancreatitis

On Admission	At 48 Hours
Age >55 years	Haematocrit decreased >10%
WBC >16 x 10^9/l	Urea increased >1.8 mmol/l
Glucose >11.1 mmol/l	Calcium <2.0 mmol/l
LDH >350 U/litre	PaO_2 <60 mm Hg
AST >250 U/litre	Fluid retention >6 litre

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. General Medicine Services

Main Office

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

Inpatient Services: Ward 23, Acute Medical Admitting Unit (Ward 24), Ward 29, Ward 30, Ward 31, and the Stroke Unit.

Clinical Director:

- Dr David Jardine, ☎ 86010

Service Manager:

- Margaret Krauss, ☎ 86218

Nursing Director:

- Pam Kiesanowski, ☎ 88996

Clerical Supervisor:

- Jill Grieg, ☎ 86010, Fax 81025

Secretaries:

- Mary Simes, ☎ 80155
- Susan Crysell ☎ 86005

Physicians:

- Tom Cawood, David Cole, Barry Colls, John Elliot, Valerie Fletcher, Greg Frazer, Dave Jardine, Libby King, David MacGregor, Sarah Metcalf, Nigel Millar, Peter Moore, John O'Donnell, Alan Pithie, Kirsten Ramsay, 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 10,000 patients per year.

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

Consultation Guidelines

An acute consult service is provided.

Contact the Acute Medical Registrar/Consultant via the hospital operator.

Outpatient Clinics

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

Rapid response clinics (Senior Registrar) 3 days per week.

15. Hyperbaric Medicine Unit

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 (HMU).

The CDHB intranet provides general information on the Hyperbaric Medicine Unit and HBOT under **View Departments** > **Hyperbaric Unit**.

15.1 Emergency Referrals

- Ring Christchurch Hospital operator (Internal ☎ 80000 or external 03 364 0640) and request the Hyperbaric Unit Duty Doctor.
- Give the operator your name, contact phone number, and location.
- For acute in-patient referrals between 0830 and 1600 hours, try ringing HMU first ☎ 80045.

  Note: *Trying to contact individual clinicians may result in delay.*

15.2 Acute Emergency Indications for Hyperbaric Oxygen

(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 (e.g., clostridial myonecrosis, streptococcal necrotising fasciitis); combined in a planned way with surgery and antibiotics.
- Carbon monoxide poisoning (see page 207) (possibly cyanide and H_2S also); smoke inhalation.
- 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.

15.3 Non-Emergency Referrals

Patient referrals and any further information or non-urgent enquiries should be directed 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.

Note: *Trying to contact individual clinicians may result in delay.*

15.4 Non-Acute Indications for Hyperbaric Oxygen

(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.

References:

http://www.hboevidence.com

Bennett MH. The evidence basis of diving and hyperbaric medicine - a synthesis of the high level clinical evidence with meta-analysis. Australian Digital Theses Program, University of New South Wales, 2006. http://www.library.unsw.edu.au/~thesis/adt-NUN/public/adt-NUN20060808.155338/.

Cochrane Database of Systematic Reviews (via CDHB intranet > Clinical Info > Databases).

16. Infectious Diseases

16.1 Infectious Diseases Department Information

Main Office

1st Floor, Riverside, ☎ 80951, Fax 80952

Inpatient Services Ward 30

- Prof Steve Chambers, Dr Sarah Metcalf, Dr Alan Pithie
- 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.
- Outpatient IV Antibiotic Service for short-term (cellulitis) and long-term antibiotics - pager 8839 for the IV Antibiotic Service Nurse, or contact the Infectious Diseases Service.

16.2 Meningitis

16.2.1 Clinical Features

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

16.2.2 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). Cryptococcus neoformans.
- Viral - especially mumps, enteroviruses, and herpes simplex (type 2).
- Other - amoebae, fungi (rare).

16.2.3 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 steroids, malignancy and HIV.
- CSF shunts.

16.2.4 Investigations

- Blood cultures - 2 sets before antibiotics given.
- Lumbar puncture. **Caution - refer to Management (see page 129) and lumbar puncture technique (see page 68).**

  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, viruses, amoeba - consult Microbiologists, if indicated.

16.2.5 Management

- **Lumbar puncture (see page 68).** 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, a **CT/MRI head scan** should be done **before** doing a lumbar puncture if:
  - There is clinical evidence of **raised intracranial pressure** (raised BP, decreased pulse, decreased level of consciousness), **seizures, papilloedema, focal neurological signs, or sinus or ear infections, or**
  - The patient is **immunosuppressed, or**
  - The symptoms have lasted **more than 5 days**.

  Note: *If a lumbar puncture cannot 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 25: 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

16.2.6 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% in New Zealand. 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 g 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 15 mg/kg loading dose using lean body weight. If renal function is normal, continue with either 0.5 g IV q6h, or 1 g q12h. If there is renal impairment, monitor vancomycin levels and seek advice. See also the Pink Book. **Consult Infectious Diseases Service.**
    - If severely penicillin or cephalosporin allergic, chloramphenicol 80-100 mg/kg/day IV 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 2.4 g q4h is the preferred treatment if the organism e.g., pneumococci, is sensitive.
  - **Steroids and Meningitis:** A 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 earlier 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 (MIC ≥0.12 mcg/ml), 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 600 mg 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 600 mg 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 278).
- 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), early morning urine 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 15 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 - herpes simplex (see treatment on page 136), mumps, and enteroviruses (PCR tests 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, Behçet's, sarcoidosis, and others. Accurate cytology essential.
  - Consult Infectious Diseases Service.
- **Encephalitis** - see Encephalitis in the Neurology section on page 163.

16.3 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". See Harrison's Online 17e Edition. 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 >24/min.
- WBC >12 x 10^9/l or <4 x 10^9/l or >10% bands; may have a non-infectious aetiology.

These criteria need to be interpreted in the context of the individual patient and any comorbidities 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.

16.3.1 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 - neutropaenia etc.

16.3.2 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.

16.3.3 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 66).
- 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 subcutaneous 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.

16.3.4 Management of Septicaemia

Fluids:

- Resuscitate with normal saline then alternate normal saline with 5% dextrose. 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 76). If you are not competent to place a CVP line (see page 64) do not attempt it.
- If patient remains hypotensive (systolic <80 mm Hg) 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.

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 109) 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 + gentamicin + metronidazole.
  - Intra-abdominal sepsis:
    - Amoxycillin + gentamicin + metronidazole.
    - Ceftriaxone + metronidazole.
  - Cellulitis (see page 134): Flucloxacillin.
  - Pseudomonas sepsis:
    - Piperacillin/tazobactam + tobramycin.
    - Ceftazidime + tobramycin.
  - Urinary tract:
    - Gentamicin.
    - Ceftriaxone.
  - Neutropaenic fever/sepsis:
    - Piperacillin/tazobactam (Tazocin) 4.5 g IV q8h plus gentamicin 5-7 mg/kg IV in 100 ml normal saline over 30 min q24h

    or, if there is a history of penicillin allergy,
    - Imipenem 500 mg IV q6h plus gentamicin 5-7 mg/kg IV in 100 ml normal saline over 30 min q24h.
- Oxygen therapy:
  - Give oxygen if patient hypoxic (see page 231).

16.4 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.

16.5 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 only if patient is very unwell (i.e., satisfies criteria for SIRS on page 131), 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 patients 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. Community acquired MRSA infections are increasingly recognised. Consider these in all patients with cellulitis which is not responding to beta lactam antibiotics.*

  - **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 anti-tetanus 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.

16.6 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. Community acquired MRSA infections are increasingly recognised. Consider these in all patients with cellulitis which is not responding to beta-lactam antibiotics. 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 130.

16.7 Herpes Simplex

16.7.1 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 PO 5 times daily 5 days.
    - If cannot swallow or unwell aciclovir 5 mg/kg IV q8h.
  - **Immunosuppressed patients:**
    - Aciclovir 400 mg PO 5 times daily 5-7 days. IV aciclovir as above if unwell.
  - **Recurrences:**
    - Normal host - minor attacks - povidone iodine cream 10% tds. Aciclovir cream of minimal benefit. Aciclovir 200 mg PO 5 times daily only of benefit if started early.
    - Immunosuppressed - seek advice.
  - **Prophylaxis:**
    - Aciclovir 400 mg PO BD.

16.7.2 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.

16.8 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 lead to faster healing, but no change to the incidence of post-herpetic neuralgia.
- There are a number of strategies for reducing/managing post-herpetic neuralgia including low dose amitriptyline and gabapentin. Gabapentin requires Special Authority if continued on discharge. A tricyclic must have been trialled prior to gabapentin for Special Authority to be granted.
- **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 PO 5 times daily 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 q8h 7-14 days.

- **Ocular zoster**:
  - Aciclovir 800 mg PO 5 times daily and **consult an Ophthalmologist**.
  - Sight-threatening disease aciclovir 10 mg/kg IV q8h.

16.9 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. All patients with HIV should be discussed with an Infectious Diseases Consultant.

16.9.1 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.

16.9.2 Antibody Testing

- Provide full explanation of test. Consult with Infectious Diseases if you are uncertain.
- Obtain oral 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 (DDMMYY).

16.9.3 Other Investigations

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

16.9.4 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 may be 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.
  - Thrombocytopaenia.
  - Leucopaenia/lymphopaenia.
  - Reduced CD4 T lymphocyte count.

16.9.5 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 mm Hg) 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. Add 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 clindamycin-primaquine.

- **Clindamycin - Primaquine** - Clindamycin 450 mg QID PO and Primaquine 15 mg once daily PO (check G6PD). Diarrhoea is a frequent side effect.

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 seizures 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 STIs, and decisions made about appropriate use of antiretroviral drugs and prophylactic antibiotic regimens.

16.10 Malaria

16.10.1 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.
- P. knowlesi is an emerging pathogen in South East Asia, especially Borneo and Malaysia:
  Morphologically resembles P.malariae, but may be associated with hyperparasitaemia and more severe disease.
- 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.**

16.10.2 Presentation

- Prophylaxis should be continued for up to four weeks depending on the drug used 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.*

16.10.3 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.

16.10.4 Management

- **P.vivax, P.ovale, P.malariae, and P.knowlesi.** 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: 1000 mg stat PO, 500 mg 6 hours later then 500 mg daily for 3 days.
    - If chloroquine is unavailable, use atovaquone/proguanil 1000 mg/400 mg (i.e., 4 tablets) once daily with food for 3 days.
  - Primaquine: check for G6PD deficiency. Quick screening tests are available. If normal, then primaquine is safe. If G6PD deficient, check with Infectious Diseases.
    - **P.vivax:** give primaquine 30 mg PO daily for 14 days in addition to chloroquine.
    - **P.ovale:** give primaquine 15 mg PO daily for 14-21 days in addition to chloroquine.
    - **P.malariae or P.knowlesi:** Primaquine treatment is not needed. Give chloroquine only.

- **P.falciparum.** All patients should be admitted.
  - **Oral therapy**:
    - The Artemisinin derivatives are now considered the most effective treatment and should be used if available. Artemether/Lumefantrine 20 mg/120 mg per tab. Give 4 tabs stat and another 4 tabs 8 hours later on day 1, then 4 tabs BD on days 2 and 3,

      OR, quinine 600 mg q8h PO for 7 days **plus** doxycycline 100 mg BD PO for 7 days.
    - Oral atovaquone/proguanil may be appropriate in some patients with mild/moderate disease. Give atovaquone/proguanil 1000 mg/400 mg (i.e., 4 tablets) once daily with food for 3 days.
  - **IV therapy**: Quinine dihydrochloride 20 mg/kg (maximum dose 1400 mg) in 5% dextrose by infusion over 4 hours. Monitor ECG during infusion. **Do not give as a bolus**. Loading dose not required if antimalarials have been given in the previous 24 hours. Hypoglycaemia may occur. Check blood glucose levels 6 hourly while on parenteral quinine. From 8 hours post the loading dose give quinine 10 mg/kg over 4 hrs by IV infusion q8h until the patient can swallow (maximum dose 2100 mg/24 hours).
  - **Treat seizures with diazepam.**

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

16.10.5 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.

16.11 Immunisation Information for the International Traveller

Information on immunisation for overseas travel is available from the following internet web page: www.fit-for-travel.de.

16.12 Causes of Fever in the Returning Traveller

These include malaria as well as enteric fever, dengue fever, typhus, Legionnaires' disease, tuberculosis, amoebic liver abscess, hepatitis (viral), and viral haemorrhagic fever.

16.12.1 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.

17. Nephrology

17.1 Nephrology Department Information

Main Office

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

Inpatient Care Ward 14

- Dr Nick Cross, Prof Zoltan Endre, Dr John Irvine, Dr David McGregor, Dr Martin 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

- 550 Hagley Avenue, ☎ 80610

17.2 Renal Failure - Acute

Acute renal failure (ARF) is defined by a recent 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.

- Early diagnosis to identify reversible causes and rapidly progressive disease.
- 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!

17.2.1 Causes

- Pre-renal:
  - Hypovolaemia or hypotension.
- Renal:
  - Nephrotoxins including drugs and chemicals (commonly radiocontrast agents).
  - 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.

17.2.2 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 and creatinine concentrations and osmolality may sometimes be helpful. Calculate the fractional excretion of sodium (FENa) from serum and urine, sodium and creatinine concentrations. Nursing staff should test urine for blood, protein and glucose using a urine test strip.
- CBC + diff, Na, K, urea, creatinine, bicarbonate, Cl, and coagulation profile. The biochemical tests should be done at least daily. Plasma potassium may need checking more often. Assess pH if HCO_3 <18 mmol/l.
- 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 (see page 254). 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.

17.2.3 Management

- Stop any potentially nephrotoxic drugs.
- Ensure optimal hydration with appropriate fluid - blood or normal saline. Consider central venous pressure monitoring in the elderly or those with heart disease. When the patient has been rehydrated give 600 ml plus urine output and other losses per 24 hours, either as oral fluid or 5% dextrose. Replace sodium losses as normal saline 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 26: Treatment of Hyperkalaemia

Plasma Potassium	Treatment
5.5-6.5 mmol/l	Resonium-A 15-30g PO or PR q6h. Consider loop diuretics if non-oliguric.
6.5-7.5 mmol/l	As above. Do ECG. Give: • 100 ml of 50% dextrose IV over 15-30 minutes plus 10 units actrapid insulin IV, or • 100 ml of 8.4% $NaHCO_3$ over 4 hours provided patient is not fluid overloaded.
>7.5 mmol/l	As above but also give 10-30 ml 10% calcium gluconate IV as a separate infusion (to reduce risk of arrhythmias). Consult re immediate dialysis.

Note: *Treatment of hyperkalaemia with insulin and glucose or with bicarbonate produces only temporary reduction in potassium.*

- 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 27: 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 and early sepsis.		

17.3 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 62). 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 quinapril 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 Quinapril and Enalapril Dosage on page 37 for dosage recommendations**.

17.4 Renal Function - Assessment

- The plasma creatinine concentration alone is not a sufficiently accurate predictor of glomerular filtration rate, particularly for small or elderly patients. It is also inaccurate by any formula when the creatinine is changing (non-equilibrium).
- **Cockcroft and Gault** (Nephron 1976, 16:31-41) developed a simple bedside formula to predict the creatinine clearance without having to collect urine and using the variables of plasma creatinine concentration, body weight, sex and age.

  The formula is as follows:

Table 28: Creatinine Clearance Calculation

$$CrCl\ (\text{ml/min}) = \frac{(140 - \text{age}) \times \text{lean body weight (kg)}}{\text{plasma creatinine (mcmol/l)} \times 0.8} \quad (\times 0.85 \text{ if female})$$

  - 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 Cockcroft and Gault. Estimated GFR is reported in ml/min (normal >90, although there is an age-related decline) 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.

17.5 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 women 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.

17.5.1 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 bacterial colony 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.

17.5.2 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.

17.5.3 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 increasing incidence of bacterial resistance, trimethoprim may no longer be appropriate.

  Suggested regimens are:

Table 29: Drug Guidelines for Cystitis

Single dose
▪ Trimethoprim 600 mg(1)
▪ Norfloxacin 800 mg
3 day course
▪ Trimethoprim 300 mg daily
▪ Nitrofurantoin 50 mg TDS (ineffective for Proteus)
▪ Norfloxacin 400 mg BD
▪ Amoxycillin 250 mg TDS (for enterococcus faecalis)
(1) *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 (e.g., >3 in 6 months) with normal renal function and a normal urinary tract merit consideration for low dose prophylactic antimicrobial therapy.
- Try simple measures - treatment of cervical erosion or vaginitis, increase in fluid intake, increase the frequency of micturition, post coital voiding, application of an antiseptic cream to the periurethral area prior to intercourse (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 nocte.

  The above drugs should be taken after emptying bladder and before retiring. If patients have renal insufficiency, cefaclor 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, atrophic vaginitis should be considered and treated appropriately, e.g., intravaginal oestrogens.

17.6 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.

17.6.1 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**).

17.6.2 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 131); 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.

17.6.3 Management

If the patient is dehydrated and/or vomiting, give IV normal saline.

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.

18. Neurology

18.1 Neurology Department Information

Main Office

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

Consultant Staff

- Prof Tim Anderson, Dr Stuart Avery, Dr John Fink, Dr Deborah Mason, Dr Philip Parkin

Inpatient Services

These are provided by a team comprising one of the Neurologists (on a rotational basis), a Registrar and a House Physician.

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.

18.2 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

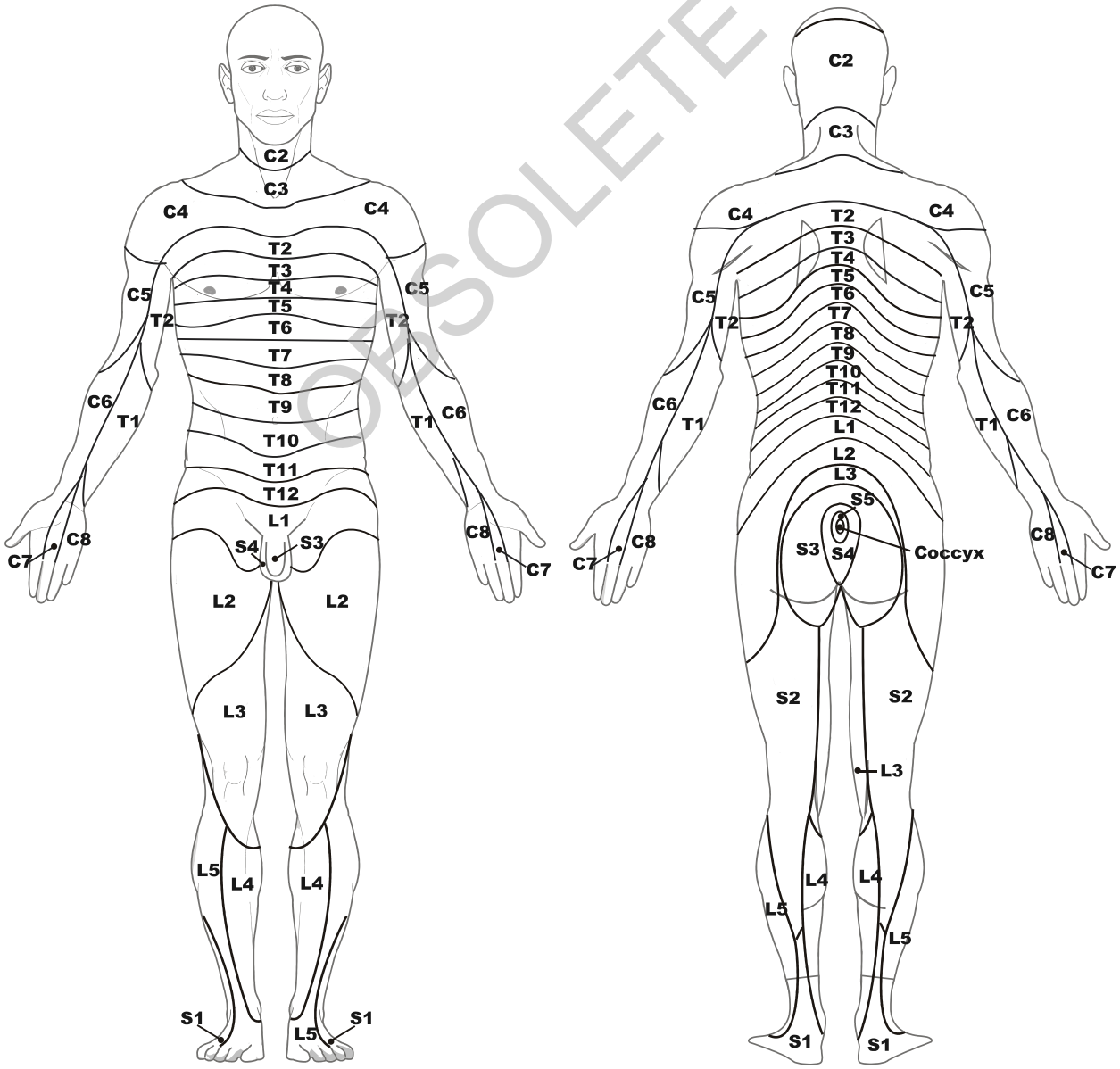
- The principal spinal segments responsible for the most commonly tested reflexes are:
  - Biceps jerk: C5, C6 (Musculocutaneous nerve)
  - Brachioradialis reflex: C5, C6 (Radial nerve)

- 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. 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

18.2.1 Dermatome Distribution



18.3 Stroke

18.3.1 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 12).
- 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.

18.3.2 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)

- These patients should be admitted to the Neurosurgical ward, not ASU. Refer to Subarachnoid Haemorrhage on page 158.

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**.

18.3.3 Investigations

- Include:
  - CBC + diff (polycythaemia, thrombocytosis).
  - CRP or ESR (arteritis).
  - Na, K, creatinine.
  - Glucose.
  - HbA1c.
  - Lipids.
  - ECG.
  - CT head scan.

  Note: *Cerebral haemorrhage is 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). See Transient Ischaemic Attacks on page 156.
  - 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.

18.3.4 Acute Management of Ischaemic Stroke

- **Thrombolysis** with tissue plasminogen activator for acute ischaemic stroke.
  - May be considered for highly selected patients **within 4.5 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.

  The Stroke Unit Network of NZ Stroke Protocols (including thrombolysis protocols) may be downloaded from http://www.stroke.org.nz/forum/index-pro.html.
- **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 swallowing assessment: either dysphagia screening tool administered by a stroke unit nurse trained in its use or speech language therapy (SLT) 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), but correct maintenance of sodium/water homeostasis is the greater imperative. Subcutaneous route may be a good option if hypotonic fluids are needed.
  - 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. Detailed protocols are available in the ASU.

  Note: If repeated boluses of intravenous antihypertensive medications are required for adequate BP control beyond 24 hours of stroke onset then additional medications should be prescribed orally. Consideration should be given to NGT placement for this purpose if taking medications by mouth is not possible or considered too unsafe.
  - 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 >48h. This decision should be made in conjunction with the multidisciplinary team.
- DVT prophylaxis:
  - Full-length graduated compression stockings are **not** to be used routinely. These have been shown to be **ineffective** at preventing DVT after stroke and can cause tissue damage [Lancet. 2009;373:1958-65].
  - Subcutaneous low molecular weight heparin (LMWH) should be considered after 48h for patients at high risk from DVT such as immobile patients unable to lift one leg off the bed, obese patients, or those with past history of DVT/PE or known thrombophilia. Give enoxaparin 40 mg SC daily, less in renal impairment. The best timing for initiation of LMWH after stroke is not known. LMWH use is associated with increased ICH when initiated in the acute phase (<48h); the risk beyond 48h is not well quantified. Fatal PE is rare in the first week after stroke but peaks at the end of week 2. A decision regarding the use or otherwise of LMWH for immobile stroke patients should be documented by the end of week 1. Aspirin should be continued. Hydration and mobilisation remain cornerstones of DVT prophylaxis.
- 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.

18.3.5 Acute Management of Intracerebral Haemorrhage

Many aspects of acute management of intracerebral haemorrhage (ICH) are similar to ischaemic stroke, particularly the benefit of stroke unit care, maintenance of homeostasis, and early rehabilitation (see page 152). **There are some important differences, however:**

- Avoid aspirin, heparins, thrombolytic agents.
- Full coagulation screen is required urgently.
- If taking warfarin, urgent action to reverse the anticoagulant effect is required - see detailed guideline on page 154.
- Acute BP management: the threshold for considering use of IV antihypertensive agents in the acute phase of stroke is lower in patients with ICH (>180/105) compared with ischaemic stroke (>220/120). Antihypertensive agents as for ischaemic stroke on page 152.

  ***Note**: there is evidence a lower target of 140 mm Hg systolic may be safe in acute ICH but not yet any evidence that this confers additional clinical benefit to the patient.*
- Neurosurgical referral should be considered for potentially life-threatening ICH in previously neurologically well patients who have:
  - Cerebellar ICH.
  - Superficial supratentorial ICH.

Investigations

- CT head scan - "hypertensive" deep ICH can be usually diagnosed on clinical grounds with plain CT and does not usually require additional investigation:
  - Further vascular imaging is generally not indicated in patients if:
    - ICH in deep (basal ganglia) location - territory of the penetrating arteries.
    - Patient is clinically hypertensive or has a history of treated hypertension.
    - Patient is >45 y in age.

  An angiographic study following ICH of patients fulfilling the above criteria revealed no additional abnormalities.

- Investigation for possible underlying vascular disorders may be indicated in other patients. The first investigation is usually MRI. The best timing for this investigation depends on clinical factors (including the patient's prognosis for survival) and the size and location of the ICH. Neurological/Neurosurgical or Neuroradiological advice is recommended.

  Investigations for vascular cause are usually deferred until the patient is clinically stable from their acute ICH (this does not apply to patients with subarachnoid haemorrhage, which is a neurosurgical emergency).

- CBC + diff.
- Coagulation screen.

Intracerebral Haemorrhage while on Warfarin: Reversal of the Warfarin-related Coagulopathy

Background

- ICH while taking warfarin is life-threatening with a mortality of between 43-70% at 30 days.
- ICH volume and further expansion of ICH are both independent predictors of mortality.
- ICH volume is not maximal at the outset but expansion of a primary ICH can continue for several hours (without warfarin). If taking warfarin at the time of bleed, this continued bleeding can continue for 24-48 hours.
- Most warfarin related ICHs occur with the INR within the "therapeutic" range.
- Warfarin causes functional deficiencies of several different clotting factors which require replacement. Furthermore, this needs to occur **urgently** to reduce ICH expansion.

Reversal Guidelines

- **All** patients with a warfarin-related ICH and an elevated INR (>1.2) should have rapid reversal of the coagulopathy.
- **Do ALL of the following:**
  - **Cease warfarin.**
  - **Give 5-10 mg Vitamin K intravenously .**
  - **Prothrombin complex concentrate (Prothrombinex-HT is available in New Zealand) 25-50 IU/kg intravenously.**
  - **Fresh frozen plasma (150-300 ml) IV.**

Notes:

- Vitamin K takes 6-24 hours to be effective.
- Fresh frozen plasma contains all the relevant clotting factors but requires large volumes (2 or more litres) to adequately replace clotting factors.
- Prothrombin complex concentrate acts rapidly (within 15 minutes) and is accessed through contacting New Zealand Blood Service doctor on call.
- Prothrombinex used in NZ may not contain sufficient factor VII, hence concurrent use of a small amount of FFP as well.
- Monitoring:
  - INR alone is not useful for monitoring the effectiveness of clotting factor replacement. It is only useful for monitoring warfarin use in steady state situations.
  - Monitoring should be done immediately after treatment using a coagulation screen (INR, APTT, thrombin time and fibrinogen). If still abnormal, more coagulation factors should be given immediately.
  - If normal recheck in 4-6 hours (reflecting shortest half life of factor VII and vitamin K onset of action).
  - If normal again, then recheck at 24 hours, or sooner if patient clinically unstable.

- The risk of thrombotic events during this short term reversal appears very low, even in patients with prosthetic heart valves.

Longer Term Management

- This requires an individual assessment of the risks and benefits of restarting warfarin or not. Most should not restart warfarin, but it is dependent on indications, location and severity of bleed, comorbidities, age and concurrent medications. See also the guidelines on page 270.

Reference: Australasian Society of Thrombosis and Haemostasis guidelines, Med J Aust 2004; 181 (9): 492-497.

18.3.6 Secondary Prevention of Ischaemic Stroke

- **Aspirin 75-150 mg daily** for all patients with ischaemic stroke not treated with warfarin, unless contraindicated.
- **Dipyridamole 150 mg bd** in addition to aspirin reduces the risk of recurrent stroke and should be considered for all patients.
- **Statin lipid-lowering therapy**. Simvastatin 40 mg daily reduces the overall risk of recurrent vascular events after stroke and is our current recommendation after stroke. One recent trial showed that atorvastatin 80 mg daily reduced the risk of recurrent stroke; this treatment is not generally available on a funded basis. 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 treatment 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 mm Hg lowering of systolic BP, even for "non-hypertensive" patients.
  - 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 2-3 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. See Transient Ischaemic Attacks on page 156.
  - Patients benefit most when endarterectomy is performed **early** after symptoms.
  - Some patients with ipsilateral 50-70% stenosis might benefit from endarterectomy and should also be referred to vascular surgery for urgent assessment.
- **Smoking cessation advice** should be given to all current smokers (see page 172).

18.3.7 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 163). However:
  - Corticosteroids are **not** helpful for post-infarction brain oedema.
  - Surgical decompression can be life-saving, particularly for cerebellar 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).

18.3.8 Rehabilitation

- Rehabilitation efforts should commence as soon as possible after stroke, e.g., mobilisation out of bed within the first 24 hours.
  - 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.

18.3.9 Transient Ischaemic Attacks (TIAs)

- The American Heart Association definition of TIA is now: "a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction."
  - The arbitrary 24 hour limit has been removed [Stroke 2009;40:2276-2293].
  - Most (60%) TIAs under the previous "24h" definition resolve completely within **one hour** and only 14% were of >6 hours duration. The longer the duration of symptoms, the greater the probability of brain infarction on MRI.
  - In practical terms, if the patient you are assessing acutely has **any** residual symptoms or signs you should diagnose "stroke" and manage accordingly. You do not need to wait 24 hours to diagnose "stroke".
- All patients presenting with TIA should be started immediately on appropriate secondary prevention medications (see below).
- Some patients are at very high risk of early stroke after TIA and need urgent investigations. The risk of stroke within the next 7 days after TIA can be estimated using the ABCD2 score:

Table 30: ABCD2 - Prediction of Stroke Risk after TIA

ABCD2 - Prediction of Stroke Risk after TIA				
	ABCD2 items (Score: 0-7)			**Points**
A	**Age**: ≥60 years			1
B	**Blood pressure**: ≥140/90 mm Hg			1
C	**Clinical features**:			
	unilateral weakness or			2
	speech impairment without weakness			1
D	**Duration** of symptoms:			
	≥60 minutes or			2
	10 - 59 minutes			1
D	**Diabetes**: (on medication/insulin)			1
Risk of Stroke According to ABCD2 Scores				
ABCD2 Score:		**0 - 3**	**4 - 5**	**6 - 7**
Proportion of all TIAs		34%	45%	21%
Stroke Risk (%) at				
2 days		1.0	4.1	8.1
7 days		1.2	5.9	11.7
90 days		3.1	9.8	17.8

Patients at high risk:

- Include those with ABCD2 scores of 4 or more, crescendo TIAs, atrial fibrillation or who are taking anticoagulants.
- Require urgent investigations and Specialist assessment as soon as possible but definitely within 24 hours.
  - Urgent CT head.
  - Carotid ultrasound within 24h.

  Urgent (same/next day) outpatient ultrasound is now available for high-risk patients (ABCD2 4-7). Contact the Acute Neurology Registrar to facilitate booking.
    - One outpatient Carotid USS slot is held daily (Mon-Fri) at 3.00 pm for this purpose. If today's slot is taken already, the Neurology Registrar can assist by arranging follow-up of next-day USS results and next-day neurology review if this will prevent an otherwise unneeded admission.
    - The referring team should ensure secondary prevention medical treatment is started before discharge.

  Patients referred for urgent ultrasound who have a >50% ipsilateral stenosis detected will be automatically referred to vascular surgery.

  *Patients who have definite posterior circulation symptoms only or who would not be considered surgical candidates under any circumstances do **not** require carotid ultrasound.*
  - Institution of secondary prevention measures before discharge.
  - Lifestyle advice including smoking cessation advice (see page 172).

Patients at low risk:

- Include those with ABCD2 scores of less than 4 (1.2% 7-day stroke risk) or those who present **more than one week** after TIA symptoms.
- NZ TIA guidelines suggest Specialist assessment and investigations within 7 days.
  - CT head prior to discharge is reasonable to rule out other diagnoses.
    - If same-day CT is not available the patient should still be prescribed antiplatelet drugs before outpatient CT is performed.
  - Secondary prevention medications should be prescribed before discharge.

- "Semi-urgent" carotid ultrasound can be requested as outpatient - these are usually performed within 7-14 days.
- Follow-up can be arranged either with the patient's GP or a Specialist clinic.

Secondary Prevention

- As soon as the diagnosis is confirmed all people with TIA should have their risk factors addressed and be established on an appropriate individual combination of secondary prevention measures including:
  - Anti-platelet agent(s) - aspirin, aspirin plus dipyridamole or clopidogrel.
  - Blood pressure lowering therapy, e.g., ACE inhibitor + diuretic.
  - Statin - usually simvastatin 40 mg/day.
  - Warfarin - if atrial fibrillation or other cardiac source of emboli.
  - Nicotine replacement therapy or other smoking cessation aid (see page 172).
- Follow-up, either in primary or secondary care, should occur within one month so that medication and other risk factor modification can be reassessed.

Vertebrobasilar TIA

- Consider subclavian steal syndrome. Check BP in both arms.
- Carotid ultrasound is not required.

Note: A more detailed TIA management protocol is available in AMAU and ED.

18.4 Subarachnoid Haemorrhage

18.4.1 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%.

18.4.2 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.

18.4.3 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 subarachnoid haemorrhage is confirmed on the CT then a CT angiogram (CTA) should immediately follow. It should also be noted that an MRI scan with T2 and FLAIR sequences has as high, if not higher, diagnostic yield for subarachnoid blood and that an MRA can also be performed at the same time.
- If CT or MRI 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 68) could prove fatal.*

Remember: *There are only three ways to diagnose a subarachnoid haemorrhage: lumbar puncture (see page 68), cranial imaging (CT or MRI scan) or post mortem.*

- Once the diagnosis of subarachnoid haemorrhage has been made the patient may require additional intracranial angiography (DSA). Neurosurgical intervention to clip an aneurysm or excise an AVM (craniotomy) or neuro-interventional treatment to coil an aneurysm or embolise an AVM, will then be undertaken as appropriate.

18.4.4 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.
- Referral to a Neurosurgeon is required in all cases.
- Nimodopine (IV or oral) and magnesium sulphate (IV) (as per Neurosurgery protocols) will now usually be commenced to help counteract cerebral ischaemia and any neurological deficit associated with impaired autoregulation/vasospasm from the subarachnoid blood. Dexamethasone may be indicated in "poor grade" patients.
- 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 mm Hg 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.

18.4.5 Christchurch Hospital Admission Arrangements

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

18.5 Status Epilepticus

18.5.1 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.

18.5.2 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

- Hypoglycaemia
- Stroke
- Cerebral trauma or surgery
- Drug toxicity
- Encephalitis, meningitis
- Hypoxic brain damage
- Sepsis/infection
- Alcohol or benzodiazepine withdrawal
- Renal failure

- **Chronic Disorders**
  - Poor drug compliance in a patient with pre-existing epilepsy
  - Change in antiepilepsy drug therapy
  - Chronic alcoholism
  - Cerebral tumour or other structural lesion affecting the brain.

18.5.3 Investigations

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

18.5.4 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 benzodiazepine
  - Underlying cause requiring intensive therapy, e.g., sepsis, encephalitis.
- Insert IV line and take bloods (above).
- Administer IV benzodiazepine:
  - **Diazepam**: the emulsion formulation has now been discontinued. The plain formulation can be used but is irritant to veins and should therefore be given into a large vessel, such as an antecubital vein. Give 5 mg IV bolus, then 2 mg/minute if needed until seizures stop or to 20 mg maximum.
  - An alternative to diazepam is **midazolam** 0.1 - 0.3 mg/kg IV bolus over no less than 4 minutes. It has a short half-life and can be followed by IV infusion at the rate of 0.75 - 5 mcg/kg/minute.
- If patient has not been on prescribed antiepileptic medication prior to developing status, start **IV phenytoin**:

Table 31: Phenytoin IV Infusion

Phenytoin IV Infusion[1]

▪ Usual IV dose in Status Epilepticus (Adults)
  ▪ 15 mg/kg phenytoin in 100 ml normal saline (if >1000 mg required, dilute in 250 ml normal saline) 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.

(1) *Phenytoin precipitates in all IV solutions except normal saline, 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.
- 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.

18.5.5 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).
- Alternatives to IV phenytoin (such as when a patient is already on phenytoin and in whom the plasma levels are therapeutic) include:
  - Sodium valproate: loading dose of 20-25 mg/kg IV in 100 ml normal saline infused over 20-30 minutes. Then give a maintenance dose of 1 mg/kg/hour.
  - Phenobarbitone: loading dose of 10 mg/kg IV at a rate not exceeding 60 mg/minute (max dose 1 g). Then give a maintenance dose of 0.5-1 mg/kg/hour. Maximum dose (loading and maintenance) 2 g with close respiratory monitoring.

- 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 of administration in the treatment of epilepsy - absorption is erratic and unpredictable.
- The most straightforward way of managing status is with one antiepileptic drug (such as IV phenytoin) rather than using multiple drugs. Ensure full dosage with adequate blood concentration before discounting a drug as ineffective.

18.5.6 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.

18.6 Epilepsy: Patients Presenting with their First Seizure

18.6.1 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.

18.6.2 Investigations

- CBC + diff, ESR.
- Glucose, Na, Ca.
- Cranial imaging is generally required in all patients. MRI should be chosen if the history or clinical findings suggest a 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).

18.6.3 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 epilepticus 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, lamotrigine are the drugs of first choice for tonic-clonic or for partial (focal) seizures.

18.6.4 Driving

- **All** patients must be advised that the NZ Transport Agency driving stand-down requirement is twelve months following 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.

18.7 Raised Intracranial Pressure

18.7.1 Clinical Features

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

18.7.2 Causes

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

18.7.3 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.

18.7.4 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 129) and Lumbar Puncture (see page 68).*

18.8 Encephalitis

18.8.1 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.

18.8.2 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.

18.8.3 Investigations

Important differential diagnoses include meningitis, severe sepsis, cerebral neoplasia, SLE, toxic metabolic encephalopathy (see Stupor and Coma on page 165).

- 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 brain scan) - 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.

18.8.4 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. Prognosis correlates with level of consciousness at commencement of therapy.
- Steroids may be appropriate for selected cases of either herpetic or non-herpetic encephalitis particularly if there is evidence of raised intracranial pressure.
- Anticonvulsant therapy will be necessary in some patients.
- Close neurological observation to detect signs of increasing intracranial pressure.

18.9 Spinal Cord Compression

18.9.1 Causes

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

18.9.2 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.

18.10 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.

18.10.1 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. Refer to details of the urgent reversal of warfarin-related coagulopathy on page 154.**

18.11 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.

18.11.1 Emergency Management / Resuscitation

Refer to Emergency Management/Resuscitation (see page 75).

18.11.2 Causes

The causes of coma are:

- Drug overdose: one third
- Intracranial lesions, haemorrhage, infarction, tumour: one third
- Toxic/metabolic/infective: one third

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

- **Structural**
  - 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 thrombocytopaenic 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.*

18.11.3 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 76) 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.

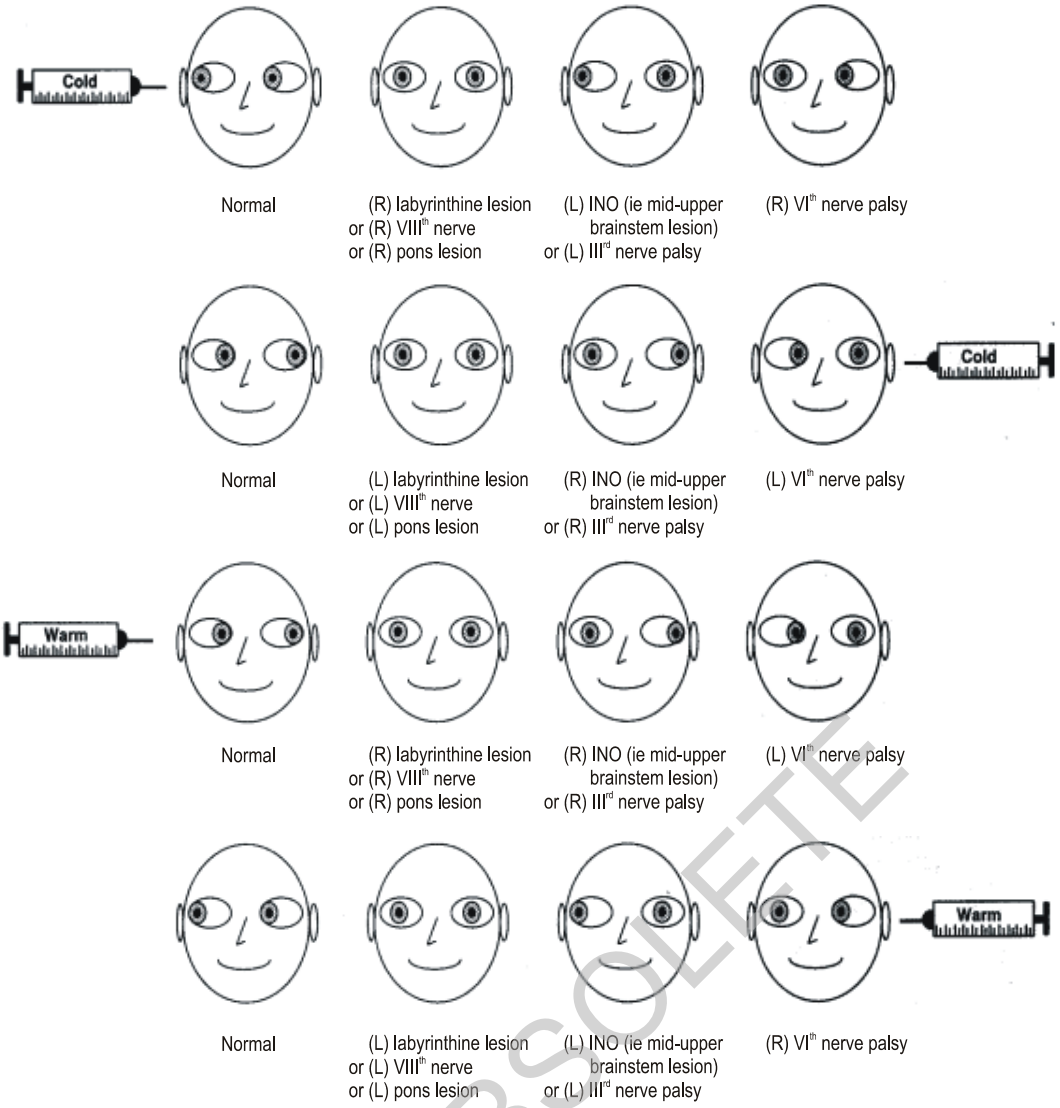
- **Eye Movements**

  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 169).
    - The expected response in an 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 169). 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.*

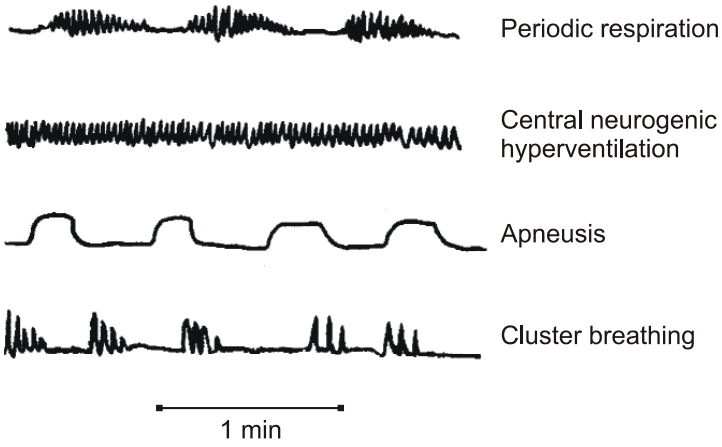
Table 32: Caloric Responses in Coma



Respiratory Pattern

- See the table on page 170.
- The pattern of respiration has less localising value than the neuro-ophthalmic 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 33: 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 Responses** 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.

18.11.4 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 199)).
- 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 68) if safe to do so.
- EEG may be considered to identify psychogenic unresponsiveness or partial complex status epilepticus.

18.12 Facial Nerve (VII) Palsy

18.12.1 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.**

18.12.2 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.

18.12.3 Bell's Palsy

- Consider alternative explanation for unilateral facial weakness: UMN lesion, zoster infection (see below), sarcoid, compression.
- The weakness is usually maximal on the first day.
- 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'.
- Provided no contraindications, a 10-day course of steroids if commenced within 72 hours, improves an already favourable prognosis. Prednisone 60 mg/day for 5 days, then taper over the next 5 days.
- Bilateral VII palsy: this is **not** "Bell's palsy". Suspect: sarcoidosis, Guillain Barre syndrome, myasthenia, myopathy. Neurological opinion advised.

18.12.4 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).

18.12.5 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.

19. Nicotine Dependent Patients

ABC Strategy for Smoking Cessation - all health professionals need to initiate this and document on form C120001.

A: **A**sk all patients for their smoking status and document it.
B: provide **B**rief advice to quit.
C: refer to or provide **C**essation support.

Current Smokers

Inpatients identified as current smokers should be offered appropriate Nicotine Replacement Therapy (NRT), whether or not they wish to quit long term. This should enable them to be smokefree during the in-patient stay or at least reduce their smoking. This may be crucial, especially for those admitted with cardio-respiratory illness and/or needing oxygen treatment.

- Inform all patients of the CDHB Smokefree Policy and offer NRT.
- Be supportive and non-judgmental.
- Assess the patient's willingness to quit.

Note that:

- 1/40 people who smoke will successfully quit on brief advice from a doctor.
- Over 2/3 of smokers want to quit and nearly 50% of smokers try to quit each year.
- Successful abstinence takes an average of 14 attempts - relapse is a learning opportunity.
- It is estimated that only 1/20 of attempts are made with optimal treatment and support.
- NRT doubles the chances of success.

19.1 Nicotine Replacement Therapy (NRT) Products and Dosage

NRT will reduce nicotine withdrawal symptoms but the pharmacokinetic properties of the respective preparations must be considered; peak blood levels are achieved within seconds when smoking cigarettes, but not for several hours when a nicotine patch is administered. NRT gum, lozenges, and inhalers have faster onset of action and achieve high levels of nicotine concentration in around 15-30 minutes. The nicotine inhaler delivers nicotine to the oral mucosa - not the lung!

- Patches (21 mg, 14 mg and 7 mg), lozenges (2 mg and 1 mg), and gum (4 mg and 2 mg) are available for hospital inpatients and subsidised in the community.
- Inhaler (10 mg) is available for selected inpatients but is not subsidised in the community.
- People who are strongly addicted or whose symptoms are not well controlled on a single product may require a combination of products (e.g., patch and gum, or patch and lozenges). Combination treatments are safe and more effective than single products.
- Strength of addiction is assessed by number of cigarettes smoked per day and time of the very first cigarette - or by using the Fagerstrom tool.
- Generally, NRT should be used for 8-12 weeks, although very heavy smokers may require longer.

Table 34: NRT Dosage Guidelines

NRT Dosage Guidelines	
>10 cigarettes/day	Nicotine patch 21 mg/24h plus nicotine gum or lozenge for PRN use(1).
<10 cigarettes/day	Nicotine gum or lozenge for PRN use(1). If patient is nil by mouth or cannot tolerate an oral product, use 14 mg nicotine patch/day. This may need to be increased to a 21 mg patch if the patient still has a desire to smoke.

(1) *The choice of PRN preparation is dependent on patient preference. The dose of gum and lozenge depends on time to first cigarette after waking in the morning:*

- if <30 minutes, then 4 mg gum or 2 mg lozenge
- if >30 minutes, then 2 mg gum or 1 mg lozenge

Monitoring

- Each patient should be assessed once per duty for their urge to smoke.
- If patients on NRT develop nicotine withdrawal symptoms, their dose is likely to be insufficient.
- Nicotine withdrawal symptoms include depressed mood, irritability or anger, insomnia, increased appetite, anxiety, decreased heart rate, difficulty concentrating, restlessness.

Notes regarding NRT dosage

- Cigarette consumption is not a good measure of dependence as people who have recently cut down are likely to compensate by smoking the fewer cigarettes more intensively.
- Patients 12 years of age and older who smoke >10 cigarettes or more per day can use NRT as per table above. Young patients smoking <10 cigarettes per day should be offered gum (2 mg) or lozenges (1 mg) in the first instance with monitoring of their urge to smoke on a daily basis. If their craving is not controlled by these products, they can be offered higher dose products or patches.
- **Contraindications:** same as for smoking, i.e., acute MI, unstable angina pectoris, severe arrhythmias, recent CVA. However, these are **relative** contraindications; 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.*
- NRT does not contain the toxic substances found in cigarette smoke, such as carbon monoxide, cyanide, ammonia, vinyl chloride, and tar. It does not produce dramatic surges in blood nicotine levels, and does not produce strong dependence.
- Whether the mother smokes or uses NRT, nicotine passes through the placenta to the foetus, and via breast milk to the baby. However NRT is preferable for the reasons explained above. Provide PRN products (lozenges or gum) to pregnant women.

Discharge - Provision of NRT and Cessation Programmes

At discharge or in the outpatient service, prescribe NRT either by using a standard prescription or a Quitcard, provide NRT information pamphlets if required, and refer to a cessation programme. A full programme of up to 8 weeks NRT (including co-therapy, e.g., 8 weeks of high dose patches, plus 4 weeks of gum or lozenges to be taken as a top-up) can be put onto one Quitcard.

- PEGS smoking cessation programme - available through most GPs (minimal costs).
- Aukati Kaipaipa 'by Maori, for Maori and their whanau' (free programme) - phone 0800 425 700.
- Smokechange for pregnant women and their partners - phone 0-3 379 9947.
- QUITLINE – provides a range of cessation services - phone 0800 778 778.

There will be a charge of $3 per type of NRT (not by amount). The charge is the same for NRT provided by standard prescription or by Quitcard. Parkside Pharmacy Ltd (main foyer Christchurch Hospital) continues to provide NRT free to patients. A referral to the patient's GP and/or cessation service provider should be made at the same time.

Other pharmacological management options

- Nortriptyline - subsidised.
- Bupropion - subsidised.
- Varenicline - not subsidised.

Practitioners should refer to the product information for contraindications and precautions.

Combination therapy with NRT can be considered in highly nicotine dependent individuals.

20. Obstetrics and Gynaecology

20.1 Obstetrics and Gynaecology Department Information

- Delivery Suite, third floor, ☎ 85711
- Gynaecology Assessment Unit (GAU), 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 GAU 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 Registrar or Consultant directly, via the telephone operator. (The Consultant is not always on site in the hospital out of normal working hours.)

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

20.2 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 - Gynaecology Assessment Unit (GAU)
- Gestation >22 weeks: obstetrics

20.3 Menorrhagia

Heavy bleeding per vaginum with negative beta-HCG.

20.3.1 Aetiology

- Dysfunctional uterine bleeding.
- Bleeding secondary to trauma (vaginal laceration/blunt force trauma). Consider sexual abuse and ask about this. If confirmed, refer to 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.

20.3.2 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.

20.3.3 Management

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

Reference: Heavy Menstrual Bleeding. NICE. January 2007 http://guidance.nice.org.uk/.

20.4 Genital Tract Infection

Table 35: 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	Treat partner Screen for other STI
Chlamydia	PCR from endocervical swab First void urine	Azithromycin 1 g stat **or** Doxycycline 100 mg bd 7 days **or** Erythromycin 500 mg QID 7 days in pregnancy	Screen and treat partners Screen for other STI
Gonorrhoea	Endocervical swab Urethral/anal swab Throat swab	Ciprofloxacin 500 mg stat PO 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 400 mg TDS PO for 5-7 days Catheterise if needed	Screen for other STI
Pelvic Inflammatory Disease	Swabs for all of above Image for abscess - ultrasound or CT	Amoxicillin/clavulanic acid + doxycycline ± metronidazole **or** Gentamicin and clindamycin	

Reference: Treatment and sequelae of pelvic inflammatory disease. American Journal Obstetrics and Gynaecology (2005); 106:573-580.

20.5 Gestational Proteinuric Hypertension (Pre-eclampsia)

Normally appears beyond 20 weeks gestation. Once present it will progress at a variable rate until the foetus 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.

20.5.1 Clinical signs

- Hypertension.
- Proteinuria.
- Rapid development of oedema.
- Headache and visual disturbance.
- Hyperreflexia/clonus.
- Epigastric tenderness.
- Altered level of consciousness; seizures.
- Placental abruption.

20.5.2 Investigations

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

20.5.3 Management

- **Call Obstetric Registrar**.
- If BP ≥160 mm Hg systolic or ≥100 mm Hg diastolic, institute antihypertensive therapy:
  - Loading dose of methyldopa 1 gm orally and then 250-500 mg 6 hourly.
- **If urgent reduction in BP necessary (BP** ≥170 mm Hg systolic or ≥110 mm Hg diastolic), the options, in addition to implementing the above, 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**
  - Hydralazine 5 mg IV repeated as required (beware of precipitating hypotension). Total dose 30 mg.

  Whichever option is used, the foetus needs to be monitored by CTG if the blood pressure is being acutely lowered.
- These drugs should be given until the BP falls below 160/110. Seek Specialist advice.
- 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**).

Reference: Society of Obstetric Medicine Australia and New Zealand '2008 Guidelines for the management of hypertensive disorders of pregnancy'; available as a PDF download at http://www.somanz.org.

20.6 Ovarian Hyperstimulation Syndrome

20.6.1 Aetiology

Follows IVF or ovulation induction.

20.6.2 Assessment

Examine woman for signs of:

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

20.6.3 Investigations

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

20.6.4 Management

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

21. Older Persons Health Specialist Service

21.1 OPHSS Department Information

The Older Persons Health Specialist Service (OPHSS) 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 Jackie Broadbent, Dr John Elliot, Dr Val Fletcher, Dr Nigel Gilchrist, Dr Carl Hanger, Dr Julie Kidd, Dr Sarah Hurring, Dr Anne Roche, Dr Andrew Sidwell, Dr John Thwaites, Prof Tim Wilkinson

Consultant Psychiatrists

- Dr Chris Collins, Dr Matthew Croucher, Dr Brian Deavoll, Dr Ken Fox, Dr Jeff Kirwan, Dr Dominic Lim, Dr Colin Peebles, Dr Jo Reeves

Medical Officers

- Dr Angela Harding, Dr Gerald Johnstone

Consultation and On-call Service

- There is a duty Physician on call at all times for the OPHSS, contactable through the Princess Margaret Hospital operator. Every clinical area in the CDHB has a nominated OPHSS 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 OPHSS Physician or Psychiatrist for:

- Specialist medical or psychiatric opinion about an elderly patient.
- Assessment for rehabilitation and for ongoing management by Older Persons Health Specialist Service.
- Assessment for entry to residential care - rest home, hospital, or dementia-care facility.
- Please refer to the Guidelines for Referral (OPHSS 0116, OPHSS 0117) and use the OPHSS Referral form (QMR0050).

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 OPHSS 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). OPHSS specialises in the recognition and management of these issues, both before and as they arise, to maximise the health and independence of elderly people.

Department Guidelines

Refer to the CDHB intranet under:

- **Divisions** > **Older Persons Health Specialist Service**, and
- **RMO World** > **OPHS RMO Guidelines**.

21.1.1 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.

21.2 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 cares 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 regimen; 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, hypnotics, and complementary and/or alternative medicines.
- **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 element 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 Mini Mental State Examination (MMSE) if cognitive impairment is suspected, and consult Confusion Assessment Method (CAM) (see page 210) 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.

21.3 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.

21.4 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 209) 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 Mental Status Quotient (MSQ) (see page 148) routinely, the MMSE if cognitive impairment is suspected, and the CAM Screen (see page 210) 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.
- To treat significant distress arising from agitation or psychotic symptoms, use haloperidol 0.25 - 0.5 mg BD PO. Regular dosing is preferred to PRN. 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.
- In complex cases, further advice can be obtained through the Delirium Service (☎ 66788, fax 66998).

21.5 Continence Problems

Urinary Incontinence

- Attempt to ascertain whether the patient has stress or urge incontinence.
- Consider medication as possible contributor.
- Check MSU.
- Perform abdominal, rectal, and vaginal examinations to exclude faecal impaction, prostatism, urinary retention, atrophic vaginitis, etc.
- Measure bladder residual volume by portable 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 detrusor overactivity 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.

21.6 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.

22. Ophthalmology

22.1 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, Assoc Prof Mark Elder, Dr Sean Every, Dr Ainsley Morris, Dr Allan Simpson, Dr Rebecca Stack, Dr Ken Tarr, and Dr Rob 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.

22.2 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.

22.3 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 252) for more details.

23. Orthopaedic Medicine

23.1 Orthopaedic Medicine Department Information

Main Office

- Older Persons Health, The Princess Margaret Hospital.

Inpatient Services

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

Staff

- Dr Nigel Gilchrist, Dr Sarah Hurring, Dr Andrew Sidwell, Dr John Thwaites

Consultation and On-Call Service

- Consultants can be contacted by cell phone through the operator at TPMH or Christchurch Hospital. The Orthopaedic Medicine 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.

23.2 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 Orthopaedic Medicine 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 while immobile unless contraindicated.
- Aspirin may be an alternative.
- All patients should have TED stockings unless contraindicated.
- Warfarin may be indicated in some patients - discuss with Consultant.

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 before and after surgery. For further information, refer to Nutrition Support (see page 113).

Analgesia

Refer to the guidelines on analgesia on page 189. 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 120). 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 on page 267.

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 209). 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 management of osteoporosis (see page 186).

24. Osteoporosis

24.1 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.

24.2 Osteoporosis

A condition of reduced bone mass and strength resulting in fractures. The most important consideration is an individual's absolute risk of fracture. This can be estimated using the FRAX (http://www.shef.ac.uk/FRAX/tool.jsp?locationValue=1) or Dubbo Osteoporosis Epidemiology Study (http://www.fracturerisk calculator.com) risk calculator tools.

- **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, smoking, 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 compared to age matched normal control). The World Health Organisation defines osteoporosis as T score < -2.5 and low bone mass as 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, consider calcium and vitamin D.
- **T score > -1**: don't treat.
- **If on supra-physiological steroid therapy, treat if T score < -1.5**.

24.2.1 Investigations

All patients should have Ca, PO_4, albumin, alk. phos., 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, urine calcium/creatinine ratio.

24.2.2 Treatment

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

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

  Calcium carbonate (Calci-Tab 500 or 600) one bd with food or Calci-Tab effervescent 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 is recommended for most patients on bisphosphonates: calciferol forte 50,000 IU monthly. If taken daily, a dose of at least 800 IU is required.

  Calcium and vitamin D reduce fractures and falls 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, with no significant effect on peripheral fractures.
  - **Alendronate**: 70 mg once a week 30 mins before breakfast with water, remain upright after taking tablet for at least 30 minutes and until after breakfast. More effective than etidronate, 50% reduction in all fractures, but **special authority required**. Fosamax Plus now contains 800 IU per day of vitamin D. Initial loading with cholecalciferol however is still required in those who have a probable low vitamin D level, and some patients may require a higher maintenance dose than that provided by Fosamax Plus.

    Consider a period off treatment after 5 years of alendronate if bone density has improved out of the osteoporotic range and there are no further fractures. This could be most relevant in younger patients with potential long term exposure to bisphosphonates.
  - **IV bisphosphonates - pamidronate and zoledronic acid**: 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.

    To avoid possible hypocalcaemia it is important to ensure that patients are vitamin D replete before administering an IV bisphosphonate. IV zoledronic acid is licensed for use in osteoporosis. It has similar effects to alendronate on fracture risk. IV pamidronate is 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. The evidence base favours zoledronic acid and generally this should be the first line IV bisphosphonate.
    - Zoledronic acid 5 mg in 100 ml normal saline over 15-30 minutes, once yearly
    - Pamidronate 30-60 mg in 250 ml normal saline over 1-2 hours, 3 monthly

    If GFR <30 ml/min zoledronic acid, and probably also pamidronate, are contraindicated. 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 following the first dose but usually GI tract side effects are not seen. Osteonecrosis of the jaw is extremely rare in patients treated with bisphosphonates for osteoporosis and probably no more common than in the normal population. We do not recommend a change from standard dental treatment for these patients. Bisphosphonates are not licensed for use in premenopausal women and their use requires careful assessment of risk versus benefit.

    If pamidronate is used for acute pain in those with vertebral fractures unresponsive to conventional analgesia, a dose of 60-90 mg in 250 ml normal saline may be superior to 30 mg.

- **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, HRT may be safe and effective in patients under 60 years or within 10 years of menopause. In older individuals, risks of vascular disease and breast cancer need to be considered and therefore HRT is not commonly 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.

25. Pain Management

25.1 Pain Management Contact Information

Pain Management Centre (Burwood)

- Prof. Edward Shipton, Clinical Director - for chronic pain problems, ☎ 99831

Acute Pain Management Service (APMS) (Christchurch Hospital)

- Dr Hamish Horton, Coordinator, APMS
- Richard Craig, Specialist Nurse, pager 8114

Palliative Care

The Christchurch Hospital Palliative Care Service is located within Oncology.

- Main Office (voice mail), ☎ 81473 (internal) or ☎ 364 1473 (external)
- Referral fax, ☎ 86233 (internal) or ☎ 378 6233 (external)
- General fax, ☎ 80759 (internal) or ☎ 364 0759 (external)
- 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 Palliative Care Guidelines on the intranet (under **Clinical Information and Resources** > **Palliative Care Service and Guidelines**).

25.2 Principles

Pain is a symptom that 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 or changed to a more appropriate analgesic. 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 215 since the treatment of these patients is covered by legislation.

25.3 Severe Acute Pain

Opioids are the most potent analgesics and should be used 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.
- Oxycodone is an alternative opioid to morphine that may have advantages with respect to some side effects including sedation/drowsiness, as well as being safer in renal impairment.
- Pethidine is still used occasionally, mainly where there is 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, oxycodone and methadone. Pethidine toxicity with convulsions can be an issue.
- Tramadol can be used as a second 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. May cause confusion in the elderly, and may cause the Serotonin Syndrome (see page 203).
- Fentanyl, a synthetic opioid, can also be used, especially where severe pain is anticipated in certain procedures. Discuss with Consultant. (Transdermal fentanyl is not appropriate for the management of acute pain.)
- Local anaesthetic agents can be useful for providing sensory block of specific dermatomes, e.g., femoral nerve block for fractured femur and abdominal wound catheters.

A wide range of modalities is used to manage severe acute pain. These include:

- Patient controlled analgesia (PCA).
- Regional nerve and wound blocks.
- Intrathecal morphine.
- Epidural infusions.

Perioperative care has been improved with newer anaesthetic and analgesic techniques, development of minimally invasive surgery, and drugs to reduce surgical stress. Fast-track surgery or enhanced postoperative recovery programmes have been developed by combining these techniques with evidence-based adjustments to the use of nasogastric tubes, drains, and urinary catheters, preoperative bowel preparation, and early initiation of oral feeding and mobilisation. This needs 'the right care, delivered to the right patient at the right time, by the right person, in the right way'.

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 either the APMS or Palliative Care Service ☎ 81473 (or page via the operator) - whoever is deemed most appropriate to involve. In-depth prescribing guidelines for oxycodone and transdermal fentanyl are available in the Palliative Care Guidelines.

25.3.1 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 below for general guidelines) and chart according to hospital protocols.
- Reassess at regular intervals and adjust prescription accordingly.

25.3.2 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, blood pressure, respiratory rate, and $\pm O_2$ saturations before and at appropriate intervals after the administration of the opioid. See Frequency of Observation during Acute Pain Medication on page 192 and Pain and Sedation Scores on page 192.
- Request an urgent medical review if the pain protocol/prescription is not fully effective.

25.3.3 Management of Complications

Manage appropriately any untoward effects (see Management of Severe Complications on page 192 and Management of Opioid Side Effects on page 195).

Table 36: Dosage Guidelines for Systemic Opioids in Acute Pain

Drug	Route	Dose For Adults	Notes & Dose Intervals
Morphine	IM or SC	0.15 mg/kg	3-6 hourly.
	IV over 1 minute	0.03 mg/kg	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	Use 1 mg/ml solution **with** infusion or syringe pump.
Tramadol	IV	50-100 mg by slow IV	4-6 hourly. Max 600 mg/day.
Pethidine (when intolerant of morphine)	IM	1.5 mg/kg	2-3 hourly.
	IV over 1 minute	0.3 mg/kg	Can be repeated at 4 min intervals until desired effect achieved and repeated as required.
Fentanyl	Transdermal patch	Commence with smallest patch as instructed by Specialist - size 25, 50, 75, 100 mcg/hr	Takes approximately 24 hours to reach steady state. **Not** recommended in acute pain unless under Specialist supervision.

Notes:

- The oral and rectal routes are not usually recommended for severe breakthrough 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 orally. Doses of **up to** 60 mg morphine sulphate (2 ml of 30 mg/ml) OR 120 mg morphine tartrate (120 mg/1.5 ml) 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 subcutaneous syringe pump). Refer to the Christchurch Hospital Palliative Care Guidelines.
- Parenteral oxycodone is not generally recommended in acute pain management.

Table 37: Frequency of Observation during Acute Pain Medication

Route	At least(1)
IM/SC	1 hour after each dose
IV	Repeat observations at 10 mins then hourly for 2 hours.
IV infusion	1 hourly

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

Table 38: 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 39: 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.)*

25.3.4 Adjuncts to Opioids for Severe Acute Pain

NSAIDS remain the standard approach, but are relatively contraindicated where there is a bleeding disorder, renal dysfunction, 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. Generally speaking, 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.

25.4 Chronic/Persistent Pain in the Cancer/Palliative Setting

- 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). It may be preferable to use oxycodone first line in mild-moderate renal impairment.
  - Patients maintained on morphine may develop 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 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 Guidelines, 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 $^{1}/_{6}$th of total daily morphine dose.

  If several breakthrough doses needed per day, increase morphine sulphate SR dose.

Incident Pain:

- Use morphine elixir, $^{1}/_{6}$th of total daily morphine dose, before activity that causes pain. Adjustment of the morphine sulphate SR dose is generally not recommended.

Alternative Opioids to Morphine:

- Oxycodone.
- Transdermal fentanyl (a special authority application is required. There are certain prerequisites, and approval is for 3 months only).
- Methadone.

Oxycodone is increasingly used in palliative care patients, fentanyl and methadone less so. See the Opioid Conversion Guide below. For advice regarding indications and prescribing, refer to the Palliative Care 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.

25.4.1 Opioid Conversion

Table 40: Palliative Care Opioid Conversion Guide

Conversions are always approximate Observe the patient closely			
morphine oral	:	morphine subcut	
2	:	1	
morphine oral	:	oxycodone oral	
2	:	1	
oxycodone oral	:	oxycodone subcut	(1)
1.5 to 2	:	1	
oxycodone oral	:	morphine subcut	(2)
1.5	:	1	
morphine subcut	:	oxycodone subcut	
1	:	1	
codeine oral	:	morphine oral	(3)
10	:	1	
tramadol oral	:	morphine oral	(4)
evidence regarding conversion ratio is conflicting			
(1) *Conversion may be slightly less than 2:1*			
(2) *Conversion may be slightly more than 1.5:1*			
(3) *If maximum dose of codeine (240 mg/day) is ineffective, convert to morphine 5 mg q4h PO*			
(4) *When converting from maximum dose of tramadol (400 mg/24 hr), titrate with an initial dose of morphine elixir (or tablets) 10 mg q4h (or OxyNorm 5-10 mg q6h)*			

25.4.2 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/SC q8h (can be given as a single nocte dose).
- Cyclizine 50 mg PO q8-12h. Can be given IV q8h or as SC infusion over 24 hours (maximum dose of 150 mg/24 hrs).

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 **domperidone** (has fewer side effects, and extrapyramidal reactions are very rare).

Methotrimeprazine 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 q6h for severe nausea & vomiting which has not responded to the first line antiemetics. Caution: constipation is a side effect when used for more than a few days.

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 should be used routinely when taking opioids. Movicol requires an application for a special authority number as it incurs a part charge in the community (SA0891 - Macrogol 3350). It is recommended for faecal impaction (up to 8 sachets per 24 hours) or can be used as chronic treatment (up to 3 sachets daily). Refer to Christchurch Hospital Palliative Care Guidelines.

25.4.3 Adjuvant Analgesics

- Paracetamol or NSAIDs are effective for pain, especially for bone or soft tissue injury.
- Corticosteroids are useful for pain related to nerve compression or cerebral oedema.
- Dextropropoxyphene, a component of Di-gesic, is **not** favoured because of the long duration of effect and potential for abuse.
- Tricyclic antidepressants and anticonvulsant agents are useful in neuropathic pain (burning, shooting).
- Gabapentin is an effective agent with limited adverse side-effects. A special authority application is required. The patient must have first tried and failed or been unable to tolerate treatment for pain with a tricyclic antidepressant. Initial approval is for 3 months. Subsequent approval, once proven effective, is for 2 years. Dose modification is required in renal impairment.
- Occasionally nerve blocks, transcutaneous nerve stimulation (TENS), or intraspinal catheter may be required.

For more information on adjuvant analgesics and neuropathic pain management in palliative care, see Palliative Care Guidelines.

For complex pain problems, 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.

25.5 Acute Persistent Pain and Chronic Pain in the Non-Cancer/Non-Palliative Setting

Acute Pain Management

- Good management of acute pain reduces the chance of developing persistent pain.
- Frequent pain assessment is essential to good pain management and to quality assurance.
- Measure pain, "the fifth vital sign". Pain needs to be measured alongside temperature, blood pressure, heart rate, and respiratory rate.

Risk Factors for Acute Persistent Pain

- Preoperative factors: young females, pain before surgery, preoperative chronic pain, preoperative fear and anxiety, re-operations, and low income, low self-rated health and lack of education.
- Intraoperative factors: the site (thoracotomy, sternotomy, major limb amputation), and extent of the surgery.
- Postoperative factors: unrelieved pain, severe pain, and the amount of analgesics consumed in the first few days.

Identify patients with high risk factors for developing persisting post surgical pain, and follow up after discharge.

Table 41: Pain After Surgery

Types of surgery	Estimated incidence of chronic postoperative pain (%)
Amputation	30 – 50
Coronary artery bypass	30 – 50
Thoracotomy	30 – 40
Breast surgery	20 – 30
Inguinal hernia repair	10
Caesarean section	10

Chronic Pain

Chronic pain is pain that persists and lasts beyond the usual healing period. Chronic pain is a major public health problem.

25.5.1 Pharmacological Treatment

- Note allergies, drug intolerances, contra-indications, and adverse effects.
- Analgesics should be individually tailored.
- Start low and go slow (except in cancer/HIV pain).
- Provide multimodal analgesia with a baseline of regular paracetamol.

Primary Analgesics

- Paracetamol: oral dose is 1000 mg four times daily in the adult patient.
- Non-steroidal anti-inflammatory drugs (NSAIDS): Ibuprofen (200 mg eight hourly), diclofenac (50 mg eight hourly), and naproxen (250 mg twelve hourly).
- COX 2 inhibitors: Celecoxib (100 mg twelve hourly) and etoricoxib (60 mg daily). The only injectable COX 2 inhibitor is parecoxib (40 mg).
- Tramadol: start oral tramadol immediate release 50 mg six hourly or tramadol slow release 50 mg twelve hourly.
- Strong opioids:
  - These are generally not used in chronic non-malignant pain.

- Cancer pain of moderate to severe intensity should be managed with the systemic administration of strong opioids.
- Full opioid agonists include morphine, oxycodone, methadone and fentanyl (usually used transdermally in cancer pain).
- Consult the Senior Registrar/Consultant if strong opioids are required.

Note: Pethidine is no longer considered a first-line analgesic.

Secondary Analgesics (or Co-Analgesics)

- Antidepressants: start with amitriptyline 10 mg nocte.
- Anti-epileptics (anti-convulsants): start with carbamazepine 100 mg twelve hourly.
- Gabapentin: starting dose is 100 mg eight to twelve hourly. A tricyclic must have been trialled prior to gabapentin for Special Authority to be granted.

Peripheral Neural Blockade

- Local anaesthetic blocks (0.2% ropivacaine, 0.25% bupivacaine) can be used for diagnostic purposes or act as an aid to physical therapy with corticosteroids (triamcinolone acetate 40 mg, methyl prednisolone acetate 40 mg). Consult the Senior Registrar/Consultant.

Biopsychosocial Model

- Patients' understanding and interpretation of symptoms (beliefs and cognition) can modulate their pain experience. Patients with psychological risk factors (fear avoidance, catastrophising, pain behaviour, depression), can be identified and early preventative measures instituted. Refer the patient to the Pain Management Centre, Burwood Hospital.

Table 42: Summary - Recommendations in the Prevention and Management of Acute Persistent and Chronic Pain

Measure pain - the fifth vital sign	
Risk Factors: Identify patients with high risk factors for developing persisting post surgical pain and follow up after discharge.	
Preoperative risk factor	Female, younger age Pain before surgery Preoperative chronic pain Preoperative anxiety and fear Site (e.g., thoracotomy, sternotomy, major limb amputation) and extent of surgery Low income, low self-rated health, lack of education.
Postoperative risk factor	Unrelieved pain Severe pain Amount of analgesics consumed (7 days) Re-operations
Chronic pain is a major **public health problem**.	
Use low dose **multimodal** pharmacological analgesia with a baseline of regular paracetamol.	
Use **secondary analgesics** in chronic pain.	Use antidepressants (tricyclics) and anticonvulsants (gabapentin and pregabalin) as first line co-analgesics.
Use the **biopsychosocial** approach in the patient with persistent or chronic pain.	

26. Poisoning / Drug Overdose

26.1 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 74)

   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., insecticides**
     - 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 may be considered.

   The options include:

   1. Activated charcoal
   2. Gastric lavage
   3. Whole bowel irrigation

   Note: *Usually gut decontamination is not required.*

26.1.1 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.

26.1.2 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.

26.1.3 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 Picoprep at approximately 2 litres per hour orally until the rectal effluent is clear (usually 4-6 hours).
- Contraindications: ileus, perforation, obstruction.
- Cautions:
  - Picoprep 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.

26.1.4 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 (☎ 80900) or via the intranet.
- The Emergency Medicine Physician, Clinical 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 7248 and ask for Poisons Centre.

26.1.5 Investigation

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

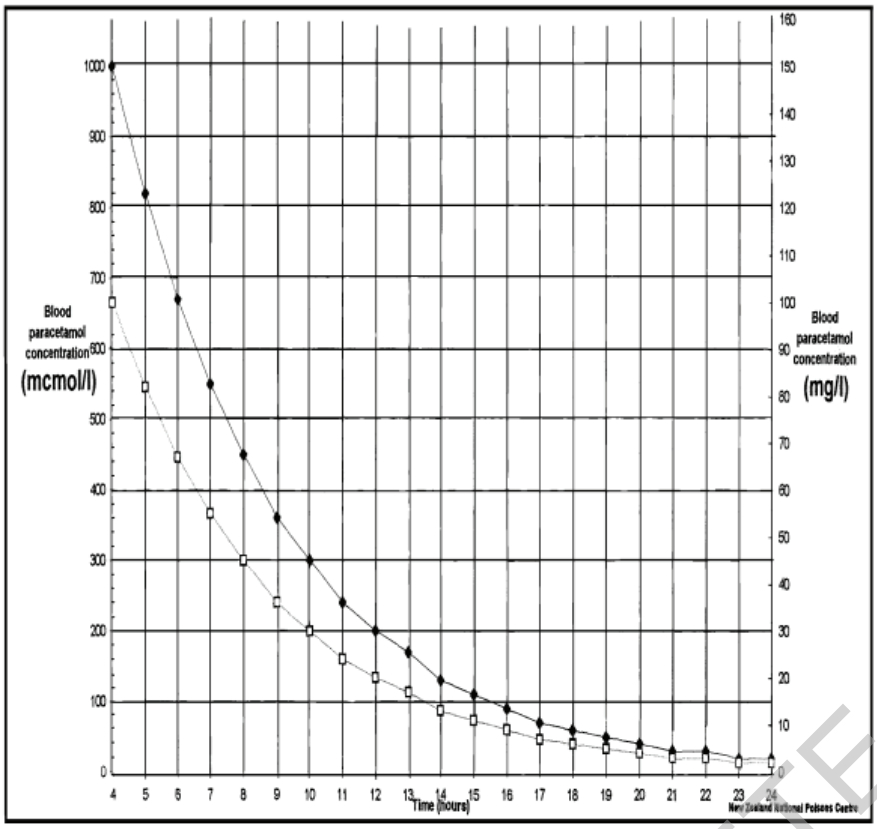
26.2 Paracetamol

- 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. Foetal death.
  - 4 days to 2 weeks: Resolution or hepatic failure.

  Treatment is directed by the nomogram (see page 201).

Table 43: 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 44: N-acetylcysteine Dosage in Paracetamol Poisoning

N-acetylcysteine:
INITIALLY: 150 mg/kg in 200 ml 5% dextrose over 60 minutes
THEN: 50 mg/kg in 500 ml 5% dextrose over 4 hours
THEN: 100 mg/kg in 1000 ml 5% dextrose 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.

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.*

26.3 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 5 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 203).

26.4 Monoamine Oxidase Inhibitors

- Examples of MAOI 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 MAOI 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 benzodiazepines 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 203).

26.5 Fluoxetine/Paroxetine/Citalopram (SSRIs), Venlafaxine (SNRI)

(Selective Serotonin Re-uptake Inhibitors - SSRIs, and Serotonin Noradrenaline Reuptake Inhibitors - SNRI)

- To date overdoses with these drugs alone have been mostly benign although deaths have occurred and seizures have been reported and are significantly more common with SNRI overdosage.
- Serious toxicity and death can occur when taken with MAOIs and they may increase the toxicity of any tricyclics ingested at the same time (see Serotonin Syndrome on page 203).
- In most cases CNS depression will predominate.
- Treatment is supportive.

26.6 Serotonin Syndrome

- May occur with combination of drugs such as MAOIs with SSRIs, SNRIs, clomipramine, other tricyclic antidepressants, lithium or pethidine, tramadol.
- Diagnosis requires such a combination of drugs, 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.
- Cyproheptadine is a potential antidote. Give 12 mg orally (not available IV) but discuss with senior medical staff first, as it is not always appropriate.

26.7 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.

26.8 Neuroleptic Malignant Syndrome

- An idiosyncratic reaction to neuroleptic drugs, e.g., haloperidol, chlorpromazine, prochlorperazine, metoclopramide - similar to serotonin syndrome, but with a slower onset.
- 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. May need cardiovascular and respiratory support.

26.9 'Fantasy' (GHB; Gamma Hydroxybutyrate)

- 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 and patients 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 (T½).
- 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.

26.10 'Ecstasy' (MDMA - methylenedioxymethamphetamine)

- 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 0.5-10 mcg/kg/min titrated to response.
  - Hyperthermia (see page 89) - aggressive cooling measures.
  - IV fluids to ensure adequate hydration.
  - Observe for at least 6 hours after resolution of symptoms.

26.11 N-Benzylpiperazine (BZP, main ingredient in most party pills)

- Essentially a designer CNS stimulant.
- Usually taken orally but is sometimes injected.
- Slow absorption with onset of effects after 2 hours and peak levels probably between 2 and 6 hours.
- Long duration of effects:
  - Neurological - stimulation, euphoria, agitation, insomnia, anxiety, panic attack, stimulant psychosis, collapse, coma, seizure

- Cardiovascular - tachycardia, chest pain, prolonged QT (no dysrhythmias reported yet)
- Renal - acute renal failure
- Gastrointestinal - anorexia, nausea, vomiting (sometimes up to 36 hours post ingestion), hepatotoxicity (rarely)
- Other - hyperthermia, rigidity, facial dyskinesias, rhabdomyolysis, urinary retention, hyponatraemia, coagulopathy (rare)

- **Treatment** - individuals should be treated symptomatically.
  - Rehydration if required
  - Benzodiazepines for agitation/anxiety or seizure
  - Cyclizine 50 mg or ondansetron 4 - 8 mg PO is recommended for nausea/vomiting
  - Observe for at least six hours post ingestion if symptoms are moderate (small risk of late seizure).

26.12 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.

26.13 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). Repeat doses are often 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.

26.14 Lithium

- Well absorbed orally. Low protein/tissue binding. Eliminated solely by the kidneys.
- CNS depression or stimulation. CVS stimulation progressing to conduction defects and heart 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.

26.15 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 IV or IM to a maximum of 2 mg. May need pacing.
  - Tachyarrhythmias.
    - Magnesium sulphate 2-4 g IV.

  Note: If cardioversion required, it may precipitate ventricular tachycardia/ventricular fibrillation, or asystole. Use low energy (10-25 joules).
  - Hyperkalaemia: Standard treatment (see page 143), 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 ventricular tachycardia, ventricular fibrillation, 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 normal saline.
    - 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 - following Fab fragments treatment:**
    - 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.*

26.16 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 indications below).
- Consider the diagnosis in all burns, smoke inhalation, coma or attempted suicide cases.

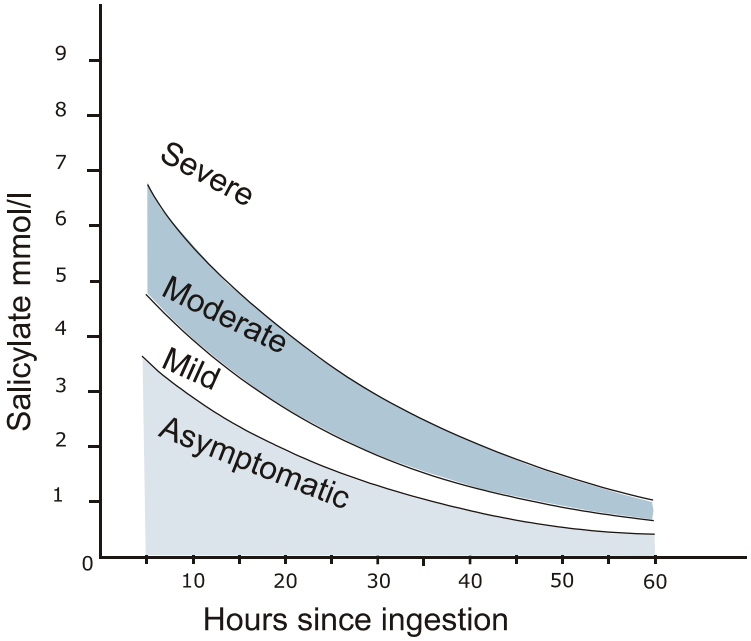
Treatment

- 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, but not 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 126).

26.17 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 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 45: 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 5% dextrose, 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.

26.18 Alcohol

See Alcohol Related Problems (see page 11).

27. Psychiatry

27.1 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 operator ☎ 66000 (external number 337 7899).
- **Psychiatric Services for the Elderly (PSE)**: refer to Older Persons Health Specialist Service Information on page 178 for contact details.

27.2 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.

27.3 Delirium

Refer also to 'Guidelines for Care of Patients with Delirium', Canterbury DHB 2002, Ref. 0020, which is available on the wards.

27.3.1 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 210) and a cognitive screening test, Mental Status Quotient (MSQ) (see page 148) or Mini Mental State Examination (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, rest home, or GP.

Table 46: 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 unpredictably 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.

27.3.2 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.

27.3.3 Common Causes

Systemic Disease

- Toxic:
  - Drugs:
    - Medication toxicity/withdrawal (see table below)
    - 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 47: 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, antimuscarinic antiparkinsonians, antispasmodics, tricyclic antidepressants, neuroleptics.
- **Cardiac:** antiarrhythmics, some antihypertensives, digoxin.
- **Gastrointestinal:** H_2-antagonists, proton-pump inhibitors (occasionally), prochlorperazine, metoclopramide.
- **Miscellaneous:** anticonvulsants, corticosteroids, dopaminergic antiparkinsonians, lithium, antibiotics (occasionally), pro-serotonergic drugs ('serotonergic syndrome').

27.3.4 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 initial 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 11) **or benzodiazepine withdrawal delirium** (see page 215): 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 as an adjunct to haloperidol may be appropriate for severe agitation, anxiety, or sleep disruption.
  - Resolution of delirium may take up to several weeks.

27.4 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.

27.5 Preferred Antipsychotic Drugs

Range of dosage suggested:

Table 48: Oral/parenteral doses of antipsychotic drugs

Drug	Acute (mg)		Daily Dose
	IM	Oral	(mg)
Risperidone	N/A	0.5 - 2 mg	6 mg
Olanzapine	10 mg (not within 2 hours of lorazepam)	10 mg (wafer or tab)	20 - 30 mg
Lorazepam	1 - 2 mg	1 - 2 mg	8 mg

Common side effects:

- **Low potency** (e.g., chlorpromazine).
  - Sedation.
  - Hypotension.
  - Marked anticholinergic effects; dry mouth; constipation; urinary retention.
- **High potency** (e.g., 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 antipsychotics** (e.g., risperidone, olanzapine)
  - Agitation (initially).
  - Metabolic syndrome (including weight gain).

  Note: There are fewer extrapyramidal effects with atypical antipsychotics.
- **Treatment of extrapyramidal 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 improvement, reduce dosage frequency.
- Write clear instructions to nursing staff about indications for 'repeat' dosage.

27.6 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.

Management

- Selective serotonin re-uptake inhibitors (SSRIs) are first choice in all age groups, e.g., citalopram, fluoxetine, paroxetine - 20 mg mane. Use 10 mg initially in the elderly. Citalopram has fewer drug interactions, thus is generally preferred in the medically ill.
- Serotonin noradrenaline reuptake inhibitors (SNRIs) (e.g., venlafaxine) are second choice between the ages of 21-65 years; 75 mg start; max dose 300 mg. Check blood pressure.
- Tricyclics are third choice; nortriptyline is the most tolerable of tricyclics. Dosage range 50 -150 mg nocte, but start low and titrate slowly upwards. Caution in elderly. Check serum therapeutic level.
- Check serum sodium with SSRI/SNRI drugs as hyponatraemia may occur, especially in older patients.
- Beware of sensitivity to the side effects of antidepressant medications in the medically ill, particularly the tricyclics.
- ECT should not be forgotten as an option (if fit for GA). Discuss with Consultant Psychiatrist.
- Tricyclic antidepressants may have additional analgesic activity. SSRIs do not.
- The physically ill should not be allowed to suffer an untreated depressive disorder.
- SSRIs may render codeine ineffective (exception citalopram).

27.7 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. Patient supports in the community should also be considered.

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 (see Guidelines to the Mental Health (Compulsory Assessment and Treatment) Act 1992 on the internet). 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.*

27.8 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 at 2-3 minute intervals. 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 20-30 mg methadone. Do not give more than 40 mg, then reduce over 10-14 days. Clonidine is 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 11).*

27.9 Management of Patients on the Methadone Programme

Methadone prescribing and supply

- It is illegal to prescribe drugs of abuse to a patient. The Christchurch Methadone Programme (CMP) and Community Alcohol and Drug Service are available for consultation in the management of these patients.
- It is an offence for a medical practitioner to prescribe controlled drugs for the treatment of dependence unless a practitioner is approved or authorised under the Misuse of Drugs Act 1975.
- The prescribing and administration of methadone to patients on a methadone programme is governed by strict guidelines.
- SMOs, RMOs, nursing staff, and pharmacists working in CDHB hospitals need to be aware of the relevant regulations contained in the Ministry of Health publication, *Practice Guidelines for Opioid Substitution Treatment in New Zealand, 2008*, which is available on the Ministry of Health website. The following aspects of these guidelines are drawn to your attention.

  Exception: *The 2008 restrictions do not apply to those using methadone for chronic pain, such as those in palliative care, and who are not enrolled in a methadone programme.*
- Patients who are enrolled with a methadone programme are not to receive methadone in any CDHB hospital until their daily dose (in milligrams) has been confirmed with their methadone 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 has 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, and will suspend the community prescription. This is so that extra supplies of methadone cannot be collected by a third party while the patient is admitted.

- For out of town clients receiving methadone, firstly contact the originating programme to confirm dosage, find out when it was last dispensed, and determine if the client is in possession of any methadone takeaway doses not yet consumed. Arrange an authority form to allow scripting while in hospital. Ensure that the dispensing pharmacy in Christchurch is notified of admission and halts the script (this is to prevent clients attempting to obtain further doses in the community resulting in methadone overdose as has occurred in the past).
- No methadone is to be prescribed or dispensed until confirmation of the last consumed dose. This is to protect against accidental overdose. Please check with the dispensing pharmacy to determine whether the client has had their dose on the day of admission and if any takeaways were provided. If the client presents in the afternoon or evening they have probably already attended their pharmacy, although you should not assume this. If unsure, do not prescribe methadone until the following day when the situation can be clarified. If you have any concerns or questions please contact CMP for further advice.
- Prescribers of methadone to patients receiving methadone 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. It is recommended that you consult with the methadone programme doctors.
- 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.

Driving Considerations

- For all patients prescribed methadone, it is important to consider the implications of them driving, as per Section 18 of the Land Transport Act. Patients who are on a stable dose of methadone and are not prescribed or using other drugs which could affect their reaction time, motor coordination, or sleepiness, are deemed safe to drive. However if the patient is prescribed benzodiazepines, opiates, or other drugs which could affect the above, the patient should be advised not to drive.

28. Respiratory Medicine

28.1 Respiratory Medicine Department Information

Main Office

2nd Floor, Riverside, ☎ 80280, Fax 80914

Inpatient Services

Three inpatient teams on Ward 25:

- Resp 1 - Dr Peter Thornley / Dr Mike Epton / Dr Richard Laing
- Resp 2 - Dr Chris Drennan / Dr Michael Hlavac / Dr Libby King
- Resp 3 - Dr Bronwen Rhodes / Dr Lutz Beckert / Dr Greg Frazer

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, severe COPD/asthma, pleural effusion of unknown cause, pulmonary mass lesions; complicated pneumonia, or other lung infiltrates of uncertain aetiology, pneumothorax, bronchiectasis, suspected TB, or significant haemoptysis.

Other Services

- Respiratory Laboratory, ☎ 80874, Fax 80878
  - For pulmonary physiology tests and blood gases
  - Some tests require Respiratory Physician approval
- (Cardio-)Respiratory Outreach Service, ☎ 88303, Fax 80849
  - For home-based service and education
  - Domiciliary oxygen service
  - Maori Respiratory Educator
- Respiratory Education Service
  - Glenys Martin, Respiratory Education Nurse, ☎ 81140, Fax 81260
- Pulmonary Rehabilitation Coordinators: c/o Christchurch Hospital Physiotherapy Department, ☎ 80680
- Respiratory Outpatients
  - Enquiries and appointments, ☎ 80280, Fax 80914
  - Clinic Nurse, ☎ 80463, Fax 81260
- Respiratory Research Group
  - Julie Cook, Research Manager, ☎ 81157, Fax 81184
- Sleep Unit
  - Technical Director, ☎ 81089, Fax 81089

Departmental guidelines are available on the CDHB intranet under **Clinical Information and Resources** > **Respiratory Services Guidelines and Protocols**.

28.2 Respiratory Failure

28.2.1 Definition

Respiratory failure is defined as occurring with PaO_2 <60 mm Hg, or $PaCO_2$ >50 mm Hg 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.

***Note**: A kilopascal (kPa) is the SI unit of pressure. 1 mm Hg equals 0.13 kPa and 1 kPa equals 7.5 mm Hg.*

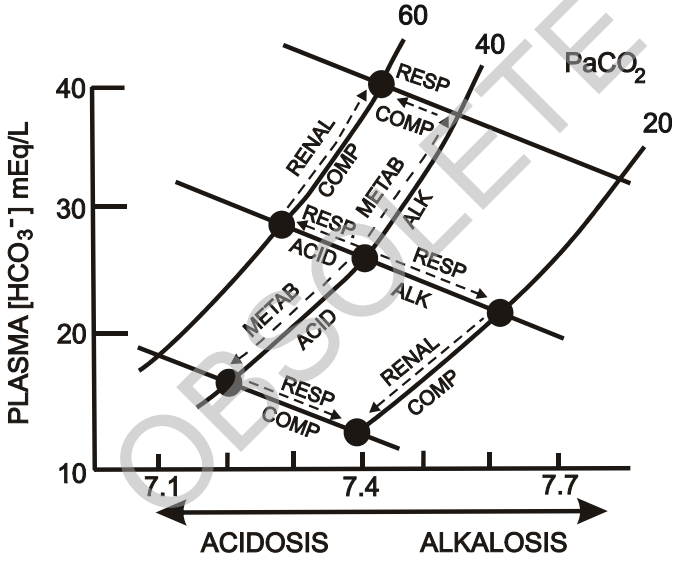
28.2.2 Classification

- 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 109):



28.2.3 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.

Table 49: 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.

Table 50: 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.

28.3 Obstructive Sleep Apnoea

28.3.1 Assessment of Patients with Suspected 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 type 2 diabetes. OSA is associated with excessive daytime sleepiness and sufferers are at increased risk of motor vehicle accidents. OSA is also becoming increasingly recognised as an important risk factor for cardiovascular disease.

Patients who present with a history of loud snoring and excessive daytime sleepiness should be considered for investigation of 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. Other clinical features which may suggest OSA include a history of disturbed or unrefreshing sleep, sleepiness-related accidents, resistant hypertension, and nocturnal cardiac arrhythmias.

For patients with suspected OSA, two initial screening tests are recommended to facilitate timely and appropriate management:

- Epworth Sleepiness Score (ESS, see table on page 220).
- Overnight oximetry to estimate a desaturation index (DI). This can be requested through the Sleep Unit using a Sleep Studies Request Form (C270075).

An ESS of >10 is considered abnormal, with a score of >16 indicative of pathological daytime sleepiness. An elevated DI (≥10) is relatively specific for OSA. However it should be noted that oximetry is insufficiently sensitive to exclude OSA, and may miss up to 30% of patients with significant disease. If there is a high level of clinical concern regarding OSA but normal oximetry, further assessment (which may include more detailed testing such as a Level 3 or Level 1 Sleep Study - full overnight polysomnography) should be considered.

In general, if the ESS and DI are elevated, or if there is a high degree of concern regarding OSA even if the DI is <10, then patients should be referred to the Sleep Disorders Unit for clinical evaluation. Referrals can be made on either a standard inpatient consultation form or using a Sleep Studies Request Form.

Table 51: 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	**2** = *moderate* chance of dozing
1 = *slight* 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	

28.3.2 Management of Obstructive Sleep Apnoea

Obesity is the most important risk factor for OSA, and weight loss strategies should be explored for all overweight OSA patients. Other lifestyle measures which may be of benefit include minimizing supine sleep, and avoiding precipitants of upper airway obstruction such as alcohol and nocturnal benzodiazepines.

The most common initial treatment for OSA is CPAP (Continuous Positive Airway Pressure). A CPAP unit generates air pressure which provides a pneumatic splint to the upper airway which is delivered via a nasal or full-face mask during sleep. CPAP has been shown to effectively normalize the breathing disturbance in OSA and significantly reduce the driving and cardiovascular risk. All requests for CPAP must be made through the Sleep Disorders Unit and approved by a Sleep Physician. It should be noted, however, that not all patients referred may qualify for hospital funded CPAP therapy.

Another option for treatment of OSA is a MAS (Mandibular Advancement Splint). This may be the preferred treatment for patients with mild OSA or those who are intolerant of CPAP. Upper airway surgery also has an important role in the treatment of OSA, particularly for those patients where there is a clearly defined anatomical abnormality such as enlarged tonsils or retrognathia (small lower jaw). Referral for these treatment options should be undertaken in conjunction with a Sleep Physician.

28.4 Chronic Obstructive Pulmonary Disease

28.4.1 Summary

- Smoking is the most important risk factor for COPD, and should be addressed on every possible occasion.
- 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 requires spirometry with bronchodilator testing. The diagnosis of COPD rests on the demonstration of airflow limitation which is not fully reversible. The severity assessment of COPD requires spirometry testing.
- If airflow limitation is fully or substantially reversible, the patient should be treated as for asthma.
- Many patients with COPD have comorbid conditions for which one should screen.

- Currently, the interventions for management of chronic COPD that have good evidence include pulmonary rehabilitation, long term oxygen for those with significant hypoxaemia, tiotropium, and inhaled corticosteroids for those with severe disease and frequent exacerbations.
- An individual plan for the long term management of this most common chronic respiratory condition should be developed in conjunction with the patient and the GP.

28.4.2 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.

28.4.3 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).

28.4.4 Investigations

- Arterial blood gases (pulse oximetry alone is **not** adequate).
- CXR.
- Sputum Culture and microscopy.
- CBC + diff, U+E.
- ECG.
- Consider BNP to assess contribution of LV dysfunction.

28.4.5 Severity Assessment in COPD

Make an immediate assessment of severity (see table on page 222) and initiate treatment accordingly. Confirm the diagnosis, identifying precipitating factor(s) and estimate the degree of usual functional impairment. **Referring to old notes** for information about previous functional status, spirometry, and **blood gas analysis** may be helpful. Old notes may also contain previous discussions with patients about ceiling of care, and wishes about resuscitation and ventilation.

Table 52: 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	Normal	Falling (<7.3)
* If the HCO_3 level is raised and pH normal this suggests chronic CO_2 retention.			

28.4.6 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. This requires admission to the Non-Invasive Ventilation (NIV) unit on Ward 25 or the ICU (see page 223).
- 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% by Venturi mask or 0.5 - 2 l/min by nasal prongs, aiming for O_2 saturations of 88-92%) is indicated for hypoxaemia. Prescribe oxygen therapy, including device, flow rate, and target $Sat.O_2$.
- Assess and document smoking status. If the patient is heavily nicotine addicted, suggest the use of nicotine replacement therapy - regardless of the patient's intention or readiness to quit smoking. See the section on Nicotine Dependent Patients (see page 172).

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 mm Hg or $Sat.O_2$ 88-92% (0.5 - 2 l/min by nasal prongs or 24-28% by Venturi mask). For more information, see Oxygen Therapy on page 231. 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 or there is concern the patient may be retaining CO_2.

Note: Nebulised treatment is simpler to administer, but does carry a risk of spread of droplet infections such as influenza or SARS. An aerosol inhaler should be used with a spacer device in the acute setting if possible. If nebuliser therapy is used, this should be changed to inhalers at the earliest convenient time.

- Give oral prednisone 40 mg (IV hydrocortisone 200 mg if unable to take orally).
- Review ABG:
  - If pH <7.35 and $PaCO_2$ >45 mm Hg, all patients should be considered for ventilatory support and must be discussed with the Respiratory Physician on call. See Non-Invasive Ventilation on page 223.
- Consider oral antibiotics (IV if unable to take orally) 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 235).
- 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, but if you believe IV aminophylline is indicated, discuss with a Respiratory Physician. If inhaled treatments do not improve the situation consider ICU, Non-Invasive Ventilation. Always 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 mm Hg) on arterial blood gas analysis.
- 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 admitting 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 an Acute On-call 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 - ☎ 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 comorbidity.
- 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 231).
- 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. Consider inhaled bronchodilator therapy with a spacer as soon as convenient. (Note that nebulizer treatment may potentially spread droplet infection.)
- 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.
- Aim for early mobilization, to avoid further deconditioning.

Monitor Progress

- Oxygen therapy:
  - Monitor Sat.O_2 and aim to maintain >90%.
  - Monitor for hypercapnia (symptoms of drowsiness and/or confusion). If there is a risk of hypercapnia this should be documented and oxygen should be prescribed to achieve a target Sat.O_2 of 88-92%.
  - 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 GP 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.
- Assess for comorbidities.
- 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). Facilitate a referral to a cessation programme such as the PEGS (Preparation, Education, Giving Up and Staying Smokefree) programme, or Quitline. Consider prescribing nicotine replacement therapy via a Quitcard prescription. See Nicotine Withdrawal on page 172.
- Consider nutritional supplement (requires a Pharmac Special Authority application) and advice for underweight patients.
- Advise influenza vaccination each autumn.
- Encourage regular exercise.
- 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.

Note: Pathways for the integrated management of a number of chronic medical conditions, including COPD and other respiratory diseases, are being developed as part of the Canterbury Initiative. These pathways can be accessed via the HealthPathways website (www.healthpathways.org.nz).

References:

The COPDX Plan: Australian and New Zealand Guidelines for the management of Chronic Obstructive Pulmonary Disease 2009. http://www.copdx.org.au/

NICE UK guideline on management of COPD in adults in primary and secondary care. http://www.nice.org.uk or Thorax 2004 Feb; 59; suppl 1:1-232.

GOLD: Global Initiative for the diagnosis, management, and prevention of COPD 2008. http://www.goldcopd.org/

28.5 Asthma

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

The aims of hospital management are:

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

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

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.

28.5.1 Guidelines for Assessing the Severity of Acute Asthma

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

Table 53: 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 litre
Respiratory rate	Normal	18-25	>25 or <10
Pulse rate	<100	100-120	>120
Oximetry	>94%	90-94%	<90%
PaO_2	Test not necessary	<80 mm Hg	<60 mm Hg
$PaCO_2$	Test not necessary	<40 mm Hg	≥40 mm Hg

DANGER SIGNS: *Exhaustion, confusion, cyanosis, bradycardia, unconsciousness, silent chest on auscultation, signs of respiratory muscle fatigue (indrawing of lower costal margin, abdominal paradox).*

28.5.2 Immediate Management

Specific treatment is dependent on severity. All patients should be treated with nebulised bronchodilator in the first instance. Other therapy is added depending on the response and reassessment of severity. Nebuliser treatment should be changed to an inhaler with spacer as soon as practicable.

Table 54: Management of Mild-Severe Asthma

For management of life-threatening asthma, see Life Threatening Asthma on page 228.

MILD Asthma: Management

- Repeated doses of salbutamol inhaler with a spacer device, or 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 Sat.O_2 >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 Sat.O_2 >95%.
- Add intravenous access. Prednisone 40 mg orally stat then daily. If unable to take orally, give IV hydrocortisone 200 mg stat then q6h (for 24 hours). Fluids - normal saline 1 litre 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 - see Life Threatening Asthma on page 228.

- Increase oxygen to high flow system.
- Contact ICU team and Respiratory Physician.
- Add IV salbutamol 250 mcg loading dose then salbutamol infusion.

Monitoring:

- Resuscitation or Intensive Care Unit. Nurse and doctor to stay with patient at all times.

(1) *It is essential that all nebulised bronchodilators are given with oxygen 6-8 litres/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.*

Note: *The evidence for magnesium therapy, either IV or nebulised, is currently inadequate to recommend its use in acute severe asthma.*

Note: *IV aminophylline has not demonstrated improved outcomes when compared to IV salbutamol in the treatment of acute asthma, and is associated with significantly more frequent side effects.*

Table 55: 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 (5 mg/5 ml salbutamol made up to 100 ml with 5% dextrose, 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.

28.5.3 Subsequent Management of Acute Asthma Episode

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 mm Hg.
    - 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 mm Hg or rising and acidosis.
  - 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.
    - Smoking status, with nicotine replacement and smoking cessation advice and support if required. Current smoking is associated with reduced effectiveness of inhaled corticosteroids.
  - 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.

28.5.4 Severity Assessment

- The risk of a severe or fatal asthma attack is higher when any of the following features are present:
  - 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.

28.5.5 Asthma 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?
  - Does the patient have a written Asthma Self-management Plan? Did they follow the instructions contained in the 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 Clinical Nurse Specialist (Respiratory Outpatient Unit) 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.
- Clinical Nurse Specialist - Respiratory Outpatient Unit.
- General Practitioner/Practice Nurse.
- Respiratory Educators/Health Promoters - Asthma Canterbury, 275 Cashel Street, PO Box 13 091, Christchurch. Email office@asthmacanty.org.nz. ☎ (03) 366 5235, fax (03) 366 5209.

28.5.6 Exhaled Nitric Oxide

Exhaled nitric oxide is a new breath test for detection of eosinophilic airway inflammation, usually in the context of asthma. High levels predict increased likelihood of response to inhaled steroids. Exhaled nitric oxide is useful for diagnosis of asthma, and for titrating steroid treatment. There may be a role for assessment of compliance with inhaled steroid treatment. For assistance with ordering exhaled nitric oxide assessment, and interpreting the result, please contact the Acute Respiratory Physician on call.

28.5.7 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. This high dose must be reviewed on follow-up.
  - 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 (e.g., salmeterol) 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. Long-acting $beta_2$ agonists should be added only in patients who are already taking inhaled steroids.
  - $Beta_2$ 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, indigestion, insomnia.

Note: Patients prescribed inhaled aerosolized corticosteroids should be encouraged to use a large volume spacer device.

28.6 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.

Note: Some individuals with chronic respiratory disease may tolerate moderate hypoxaemia for significant periods of time without acute symptoms. If the patient is well and asymptomatic, acute oxygen therapy may not be required (see section on Long Term Oxygen Therapy, Domiciliary Oxygen on page 233).

28.6.1 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.

28.6.2 Indications

- PaO_2 less than 60 mm Hg or $Sat.O_2$ <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.

28.6.3 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.

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

28.6.4 Administration

- Oxygen is a drug and must be prescribed on the drug administration chart indicating the **flow rate**, the **device to be used**, and the **target oxygen saturations**.
- 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.
- **Standard mask (Hudson)**
  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.
- **Variable concentration mask (24-50%) (Venturi 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).
- **High flow humidified oxygen (e.g., via a Fisher and Paykel)**
  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.

28.6.5 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 litre/min steps.
  - For variable concentration masks in percentage increments.
  - For standard masks in 2 litre/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.

Note: The predicted oxygen percentages supplied by masks and nasal cannulae are not precise - they will depend on the patient's respiratory minute volume, i.e., the degree of "dilution" by room air.

28.6.6 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, in some cases, 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.

Note: There is limited availability of transcutaneous CO_2 monitoring on the Respiratory Ward/Sleep Service.

28.7 Long Term Oxygen Therapy, Domiciliary Oxygen

28.7.1 Long Term Oxygen Therapy (LTOT) (16-24 hours daily)

The aims of LTOT are to:

- Correct hypoxaemia without introducing dangerous hypercapnia.
- Improve survival.
- Reduce polycythaemia.
- Improve neuropsychological status.
- Improve sleep quality and prevent nocturnal hypoxaemia.
- Prevent right heart failure.
- Improve quality of life.
- Reduce health cost.

Note: LTOT is not a treatment for breathlessness, as such.

Indications for LTOT:

- COPD PaO_2 <55 mm Hg in the stable state (usually defined as approximately 6 weeks after admission/exacerbation).
- COPD PaO_2 55-60 mm Hg with evidence of polycythaemia, clinical cor pulmonale, or pulmonary hypertension (in the stable state).
- Restrictive lung disease with PaO_2 <55 mm Hg.

Notes:

- Periods of hypoxaemia may be tolerated quite well by patients during a stable phase of their illness. Patients should receive appropriate education about this treatment in order to avoid unnecessary anxiety.
- LTOT is not offered to current smokers, or those who are unable to clearly demonstrate abstinence from smoking (cessation advice must be provided).
- To initiate LTOT, fax a referral to the Respiratory Physician (☎ 80914) and to Respiratory Outreach, fax 80849. Please complete the form fully in order to allow rapid determination of patient suitability.
- The flow rate for LTOT should be titrated to achieve a target $Sat.O_2$ of around 90-92%. The flow rate may need to be increased at night time or during exercise. Specify the indication, the oxygen requirements, and the urgency of the referral. Domiciliary oxygen must be sanctioned by a Respiratory Physician. LTOT generally will be provided by using an oxygen concentrator – the highest possible flow rate is 5 l/min.

28.7.2 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 mm Hg), 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. Referral to a Respiratory Physician is required before STOT or LTOT is given.

STOT is not a treatment for breathlessness. If disabling breathlessness is the reason for delay in hospital discharge, consider referral for advice to Respiratory Services.

28.7.3 Portable Oxygen

Portable O_2 must be approved by a Respiratory Consultant. There is a limited availability.

28.8 Spirometry

- Spirometry testing should be considered for all patients with symptoms or signs of lung disease such as cough, dyspnea, wheezing, hyperinflation. This test can be useful to assess the severity of disease, progression of respiratory disease, or response to treatment.
- The first step for interpreting spirometry is to assess the FEV_1/FVC ratio.
  - If the 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 spirometry is either normal or a restrictive defect may be present.
- The next step for interpretation is to assess the FVC.
  - When the FVC is above the lower limit of normal, a restrictive pattern is excluded.
  - When the FVC is below the lower limit of normal, a restrictive pattern is suggested which should be confirmed with total lung capacity (TLC) measurement.
- Spirometric restrictive patterns are correct only 50% of the time. Confirmation should be sought.

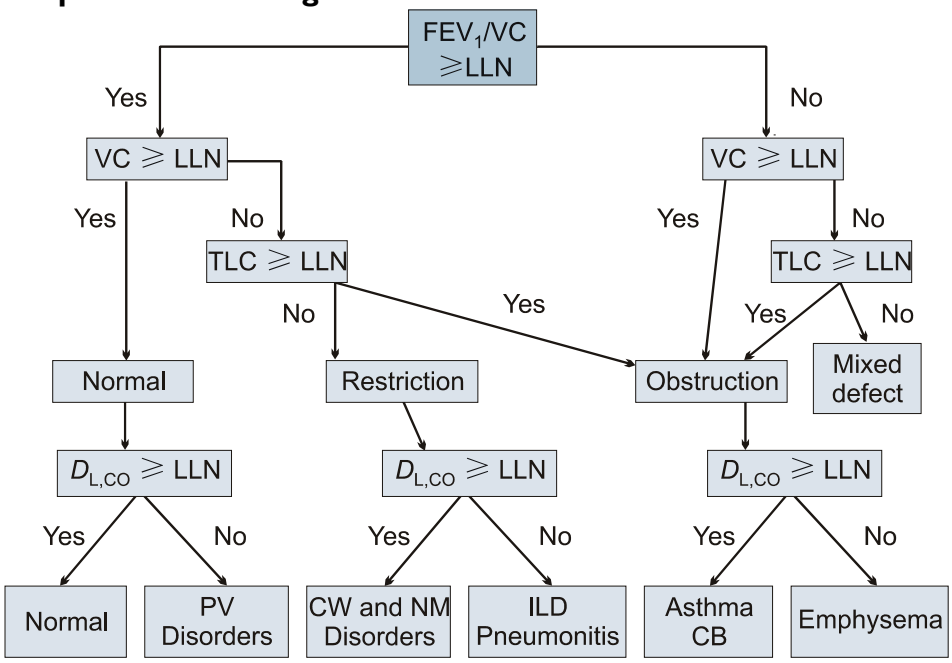
Once you diagnose obstructive lung disease on the FEV_1/FVC pattern, the FEV_1% predicted can be used to assess the severity of obstructive lung disease. The American Thoracic Society/European Respiratory Society (ATS/ERS) recommends the following severity classification in their 2005 standardization of lung function testing document.

Table 56: Severity of any spirometric abnormality based on the forced expiratory volume in one second (FEV_1), from ATS/ERS

Degree of severity	FEV_1% predicted
Mild	>70
Moderate	60 - 69
Moderately severe	50 - 59
Severe	35 - 49
Very severe	<35

- Occasionally it might be useful to test for reversibility of airway obstruction after the administration of a bronchodilator. 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). ATS/ERS define an improvement in the FEV_1 or FVC of 12% and at least 200 ml after bronchodilator administration as significant reversibility and asthma should be considered.
- Occasionally more complex lung function tests are useful to investigate a patient problem in detail. ATS/ERS suggest the following flow diagram in interpreting lung function testing. Prior to requesting these tests, they should be discussed with the respiratory physiology laboratory.

Table 57: Interpretation of Lung Function Tests



Reference: From ATS/ERS Pellegrino et al., 2005 Interpretative strategies for lung function tests. Eur Respir J 26, 948-968.

Note: LLN: lower limit of normal; PV: pulmonary vasculature; CW: chest wall; NM: neuromuscular; ILD: interstitial lung disease; CB: chronic bronchitis.

28.9 Community Acquired Pneumonia

28.9.1 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 pneumoniae 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)**.

28.9.2 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.

28.9.3 Investigations

- Refer to the description of the CURB-age score (see page 236).
- All patients with severe CAP (CURB-age score 3-5) should have blood drawn for blood culture - 2 sets before antibiotics (10 ml 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 (particularly in severe cases):
  - Urinary pneumococcal antigen.
  - Legionella - options include sputum for culture (contact lab), urinary antigen, serology (acute and convalescent), PCR on sputum and serum.
  - ZN stain and culture for TB.
  - Stains for Pneumocystis jiroveci (previously known as P.carinii) in induced sputum.
- Oximetry (or ABGs for severe cases or where there is chronic respiratory or cardiac disease.)
- Serology is unlikely to alter clinical management and is not recommended in routine practice.
- Throat and nasopharyngeal swabs for viral antigen detection and culture especially if influenza is suspected.

28.9.4 Additional Investigations

- Pleurocentesis (see page 66):
  - 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 parapneumonic effusion suspected.
- Bronchoscopy. Indications include:
  - Immunosuppressed patient.
  - Life threatening pneumonia.
  - Multiple CXR changes.
  - Deterioration despite appropriate initial treatment.
  - Contact the Respiratory Physician on call.

28.9.5 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.

Table 58: CURB-age Score

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 mm Hg or diastolic BP <60 mm Hg.
- Age = ≥65 years.

If **CURB-age** score 0-1 (Mild CAP):

- Low mortality (<2%).
- Consider outpatient management so long as no significant comorbidity and adequate social supports.
- Use single agent oral antibiotic e.g., beta-lactam, macrolide, 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 amoxicillin 1 g q8h** (alternatives - IV benzylpenicillin or clarithromycin).
  - Older patients (>50yrs) COPD, smokers: **IV Augmentin 1.2 g q8h** (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.** Take levels following the initial dose of gentamicin.

Notes:

- When using clarithromycin, watch for drug interactions, e.g., warfarin.
- IV clarithromycin causes phlebitis and should be diluted in 250 ml normal saline 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 the patient's condition is deteriorating then start rifampicin 600 mg q12h PO or ciprofloxacin 750 mg q12h PO and contact Respiratory or Infectious Diseases Physician.
- If influenza diagnosed in the setting of CAP consider neuraminidase inhibitor therapy, e.g., oseltamivir if duration of symptoms is <48 hrs.

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:
  - Haemodynamic stability.
  - Temperature settling.
  - Improved respiratory status.
- Able to tolerate oral therapy.
- Return to premorbid mental status.

Discharge planning

- **Duration of therapy**
  - There is little scientific evidence for optimal duration of antibiotic therapy for CAP.
  - **Inpatient observation whilst on oral therapy in the absence of other medical issues is usually unnecessary.**
  - 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 should be arranged with either the GP or hospital team at 6 weeks to document recovery and exclude ongoing complications or alternate diagnoses.
  - **The role of the routine 6 week CXR remains uncertain, but is generally recommended.**

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.

28.9.6 Common Complications

- Parapneumonic effusion - seen in up to 40% of cases. Should always be aspirated to exclude empyema and complicated parapneumonic effusions (see page 242).
- Large simple parapneumonic effusions (>1/3 of hemi-thorax), all complicated parapneumonic effusions and all empyemas should be immediately referred to the Respiratory Service.

28.9.7 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.

28.10 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 hospital acquired pneumonia.
- Medical patients may become more unwell very quickly - the diagnosis should be suspected in any medical patient developing a fever.

28.10.1 Investigations

- Sputum sample - involve a physiotherapist if necessary.
- Blood cultures - 2 sets. 10 ml in each bottle.
- WBC + diff.
- CXR.

28.10.2 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 mmol/l, hypotension, PaO_2 <55 mm Hg 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.*

Note: *When using clarithromycin, watch for drug interactions, e.g., warfarin.*

References:

British Thoracic Society Guidelines. Thorax 2001; 56 (Suppl IV), http://www.brit-thoracic.org.uk. This site also gives access to the October 2009 update of the CAP Guidelines 2001.

Infectious Disease Society of America/American Thoracic Society Consensus Guidelines on the Management of

Community-Acquired Pneumonia in Adults. Clin Inf Dis 2007;44:S7-S72, http://www.idsociety.org/
American Thoracic Society Document: Guidelines for the Management of Adults with Hospital-acquired, Ventilator-associated, and Healthcare-associated Pneumonia. Am J Resp Crit Care Med 2005;171:388-416. http://www.idsociety.org/

28.11 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.

28.11.1 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.

28.11.2 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.

28.12 Pleural Effusion

See BTS Guidelines published in Thorax 2003;58 suppl II http://www.brit-thoracic.org.uk

- *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).*

28.12.1 Classification

The differentiation between **exudates and transudates** is the essential first step in the diagnostic evaluation.

Diagnostic pleurocentesis 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 or with loculated effusions to the Respiratory Service early, **before** attempting therapeutic thoracocentesis.

28.12.2 Investigations

- Diagnostic pleurocentesis. May be undertaken by medical staff with appropriate experience. Ultrasound guided aspiration should be considered, in particular if the effusion is small or loculated. Use a 20 ml syringe with a 22G needle under sterile conditions. See Clinical Procedures (see page 66).
- Measure plasma total protein, glucose and LDH levels for comparison with pleural fluid.

28.12.3 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.

28.12.4 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).

28.12.5 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; the 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 tuberculous aetiology is suspected, consider a closed pleural biopsy before drainage of the fluid (refer Respiratory Services). Otherwise consider a CT thorax first and if there is focal nodularity/mass lesions, consider either an image-guided biopsy or a thoracoscopy (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. Immuno-cytochemistry is important to differentiate between metastatic carcinoma or pleural malignant mesothelioma.
  - 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. Pleural adenosine deaminase activity (ADA) has shown some promise for the diagnosis of TB.
- Haematocrit if heavily blood-stained effusion (use EDTA tube). If >50% of peripheral blood haematocrit = haemothorax (see page 245).
- Rheumatoid factor - rheumatoid effusion.
- Triglyceride - if "milky" appearance - chylothorax.
- Amylase - pancreatitis, ruptured oesophagus, malignancy.

- Other investigations and treatment options include:
  - CT scan. This should be contrast enhanced.
  - Closed or image guided pleural biopsy.
  - Intercostal drain.
  - Thoracoscopy. Options of local anaesthetic thoracoscopy or surgical thoracoscopy are available at Christchurch Hospital.
  - Note: Malignant mesothelioma can be very difficult to diagnose using cytology alone and a thoracoscopy should be considered for those cases.

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 thoracoscopy.
- If a complicated parapneumonic effusion or empyema is suspected, **do not delay Respiratory Specialist input**.

28.12.6 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 repeat 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 is indicated. The evidence that streptokinase can lyse adhesions is equivocal but may be considered in selected cases. Again seek urgent advice. There are ongoing clinical trials using alteplase/DNase. Thoracoscopy or thoracotomy may be required.

28.13 Spontaneous Pneumothorax

See BTS guidelines for the management of spontaneous pneumothorax. Thorax 2003;58 (suppl II):ii39-ii52. http://www.brit-thoracic.org.uk.

28.13.1 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.

28.13.2 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.
  - Abnormal deviation of the trachea.

28.13.3 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 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.

28.13.4 Treatment

- Discuss case with Respiratory 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.
  - Haemo-pneumothorax.
  - Reaccumulation after >2500 ml removed.
- **Technical competence for pleural aspiration or chest drain insertion is essential.**
  - As a general rule chest drains 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 or in certain situations in the second intercostal space in the anterior mid-clavicular 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 generally better than smaller tubes (10-14 French). Smaller tubes will be more comfortable for the patient, however there is a greater risk of kinking or blockages and the airflow (=drainage) rate may prove to be insufficient, in particular in patients with a secondary pneumothorax. 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.

28.13.5 Follow-Up

All patients must have a follow-up CXR at 10-14 days to ensure that the pneumothorax has resolved. Smokers must be strongly advised to quit. In recurrent pneumothoraces, a pleurodesis procedure should be considered, and 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).

28.14 Intercostal Tubes

The insertion and management of intercostal tubes is a complex and specialised area. Internal medicine patients requiring an intercostal tube should be referred to the Specialist Respiratory or Cardiothoracic surgical team for care 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.

28.14.1 Indications

- Pneumothorax.
- Pleural effusion.
- Parapneumonic effusion/empyema.
- Haemopneumothorax.

28.14.2 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. A CT scan should be done first. An ultrasound 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).

28.14.3 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.

- Assessment should include the following:
  - Check the insertion site, all tubes and connections for patency. Check if there is a 3-way tap. Ensure the position of the drain is still correct.
  - Check for swinging, i.e., movement of the water column during deep breathing.

  Note: A tube on suction will not swing and any bubbling seen is due to the suction. However, if not on suction, any bubbling through the water seal chamber, especially on coughing, suggests a 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.
- Never advance a chest tube after the insertion procedure itself although 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).
  - Call the Respiratory Physician if unsure.

28.14.4 Removal of chest drains

- See special protocol in Respiratory Services and Cardiothoracic Surgical Services.
- A CXR should usually 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.

28.15 Haemothorax

- If a heavily blood stained effusion is noted, use an EDTA (purple top) 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 instances a large bore chest drain is required (>28 French).

28.16 Tuberculosis (TB) - Early Recognition and Initial Management

TB is managed within the departments of Respiratory and Infectious Diseases. The following is a guide to the timely recognition and early management of TB for the generalist pending Specialist referral. Remember that TB is a notifiable disease (see page 278).

28.16.1 Incidence

The annual incidence of TB in New Zealand is 12 per 100,000 amounting to 20 to 30 cases annually in Canterbury. There are marked differences between ethnic groups with higher incidences of TB in Maori (x4) and Pacific Islanders (x8), compared to Pakeha.

28.16.2 Diagnosis

The diagnosis of TB may be delayed unless the possibility of TB is considered.

- **Risk situations for TB include:**
  - Previous residence in an endemic area which includes most of the world with the exception of North America, Western Europe and Australasia. About 50% of cases in New Zealand acquired TB overseas.
  - Prolonged close contact with an infectious case, usually domestic contact over weeks or months.

- Inadequate treatment for any reason of previously active TB, including a drug regimen that did not include both isoniazid (available since 1950) and rifampicin (available since 1965).
- Radiographic or pathological evidence of previously unrecognised, and therefore untreated, naturally remitting TB, such as abnormal calcification on radiograph or granulomatous inflammation on surgically resected tissue.
- Immunosuppression by disease or medication.

- **Clinical scenarios - common clinical presentations of TB include:**
  - Chronic respiratory symptoms with chest radiograph abnormality particularly bilateral upper zone pleuropulmonary fibrosis with cavitation, calcified pulmonary nodules, or calcified intrathoracic lymph nodes.
  - Persistent or recurrent pleural effusion.
  - Lymphadenopathy or lymph node suppuration.
  - Chronic spinal osteomyelitis, persistent septic arthritis.
  - Chronic diarrhoea with/without ascites.
- **Less common presentations of TB include:**
  - Persistent pneumonia or pleural effusion.
  - Pyrexia of unknown origin.
  - Constitutional clinical features and blood dyscrasia especially in the elderly.

28.16.3 Respiratory isolation

Suspected cases of infectious TB should be placed in respiratory isolation pending confirmation of the diagnosis. Suspected cases of Multi Drug Resistant Tuberculosis should be placed immediately in high level respiratory isolation in a negatively ventilated isolation suite.

Pulmonary TB should be considered infectious and requiring respiratory isolation if:

- Sputum smear positive for AFB.
- Extensive CXR consolidation, particularly if cavitated.
- Prominent cough.
- Occurring with profound immunosuppression.
- Laryngeal tuberculosis.

***Note**: Extrathoracic TB and pleural TB without pulmonary involvement is **not** infectious.*

28.16.4 Multi Drug Resistant TB

Multi Drug Resistant TB should be suspected in cases:

- From areas of high endemicity such as Africa, Asia, Indian subcontinent, South America and Eastern Europe.
- Previously treated but relapsed disease.

28.16.5 Collection of Specimens

In cases of suspected TB an early and vigorous attempt should be made to collect relevant specimens for mycobacterial culture:

- Sputum, induced sputum or bronchoalveolar lavage at bronchoscopy. (A protocol for the safe and effective collection of induced sputum is available from Respiratory Services or the Physiotherapy Department).
- Pleural fluid aspirate.
- Joint fluid aspiration.
- Lymph node aspirate or biopsy.
- Early morning urine.

***Note:** Make sure that the laboratory is informed that the samples need to be analysed for TB.*

28.16.6 General Information

- Certain antibiotics, not normally used to treat TB, have antimycobacterial activity and should be avoided if possible in suspected cases before adequate diagnostic specimen collection. Antibiotics with antimycobacterial activity include quinolones, aminoglycosides and co-amoxyclav (Augmentin).
- TB commonly has a naturally remitting and relapsing course. Chronic radiographic abnormality such as fibrosis or calcification does not necessarily indicate inactive disease.
- Tuberculin testing neither confirms nor excludes a diagnosis of active TB.

Reference: Guidelines for Tuberculosis Control in New Zealand 2003 www.moh.govt.nz.

29. Rheumatology and Immunology

29.1 Rheumatology and Immunology Department Information

Main Office

3nd Floor, Riverside, ☎ 80953, Fax 80201

Inpatient Services Ward 23

- Dr Peter Chapman, Dr Miriam Hurst, Dr John O'Donnell, Dr Lisa Stamp

Consultation and On-call Service

- The on-call Registrar and Consultant can be contacted via the Christchurch Hospital operator on ☎ 364 0640.

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 - Immunology secretary, ☎ 80950, Fax 81241

The department is closely associated with the Immunology Laboratory, Canterbury Health Laboratories.

29.2 Immunology and Allergy: recommended referrals

- Anaphylaxis:
  - See Anaphylaxis (see page 83).
- Angioedema/urticaria:
  - If severe enough to require admission and/or recurrent. Single episodes of urticaria that have resolved and there is no obvious trigger are unlikely to benefit from further investigation.
- Drug reaction:
  - Where symptoms are severe (e.g., anaphylaxis, serum sickness, Stevens-Johnson Syndrome) or likely to compromise necessary treatment (e.g., history of penicillin reaction in a patient with enterococcal endocarditis). Patients with reactions during anaesthesia are reviewed by the Anaesthetic Department.
- Immunodeficiency:
  - Patients with a history of severe infections, recurrent infections, and/or infections with unusual organisms.

29.3 Acute Swelling of a Single Joint

The cause of the acute swelling must be established before any rational form of treatment can be given.

29.3.1 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.

29.3.2 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 ml 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-B27.
- Swab throat, cervix, urethra, anus (should be cultured at bedside to grow N. gonorrhoea). Do chlamydia trachomatis nucleic acid testing on cervical and urethral swabs in women and first-catch urine in men.
- Culture faeces (Yersinia, Salmonella, Campylobacter).
- Ferritin if haemochromatosis suspected.

29.3.3 Treatment

Septic Arthritis:

- Splint joint and give analgesia.
- Use appropriate antibiotic.
  If Gram positive cocci seen or staphylococci suspected (S. aureus is the most common organism), give flucloxacillin 2 g IV q4-6h.
  If allergic to penicillin give cephazolin 2 g IV q8h if allergy mild or vancomycin if allergy severe (see page 133).
- Repeat aspiration of synovial fluid daily when effusion is recurrent.
- Consult Orthopaedic and Infectious Diseases Services. 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 therapeutic options include NSAIDs, steroids, or colchicine:

- **NSAIDs:** may be used in the absence of contraindications such as previous peptic ulceration or renal disease. Caution is advised in the elderly. Naproxen 750 mg stat then 500 mg BD until the inflammation has settled.
- **Steroids:** if the joint is easily accessible to injection and you are competent to carry out this procedure, intra-articular steroids should be given. Otherwise give oral steroids which are the first choice in the elderly, in patients with renal impairment, or those with any other contraindication to NSAIDs. Prednisone 20-40 mg PO daily until the acute attack has resolved followed by a slow reduction over 14 days to avoid rebound attacks.
- **Colchicine:** should not be used in the elderly or those with renal impairment (eGFR <50 ml/min), due to the high risk of toxicity. Colchicine is generally only effective when prescribed within 24 hours of the onset of attack. Large doses of colchicine are inappropriate. The recommended dose is 1 mg followed by 0.5 mg 6 hourly to a maximum of 2 mg per 24 hours. Many patients will not tolerate even this dose - remember - diarrhoea is sign of toxicity not a side effect.

***Note**: a cumulative oral dose of 6 mg over four days should not be exceeded. Additional colchicine should not be administered for at least three days after a course of oral treatment.*

Fatal and non fatal cases of colchicine toxicity have been reported with concomitant use of P-glycoprotein and CYP3A4 inhibitors such as cyclosporin, clarithromycin, erythromycin, verapamil, diltiazem, ketaconazole, HIV protease inhibitors etc. Toxicity can also be increased by daily consumption of grapefruit juice, hepatic and renal impairment, statins, fibrates and digoxin.

Prevention of recurrent gout

After an acute attack of gout has subsided, consideration must be given to the cause of the hyperuricaemia. When urate-lowering drugs such as allopurinol are commenced the initiation period should be covered by NSAIDs, prednisone or rarely colchicine for 12 weeks or longer as urate-lowering drugs can precipitate acute attacks of gout.

Allopurinol dose should initially be adjusted according to renal function (see table below). Dose should be started low and increased weekly to the recommended dose according to creatinine clearance. Only sustained reduction of serum urate to <0.36 mmol/l will prevent gout. If recommended dose of allopurinol fails to achieve this target urate, consideration should be given to a gradual increase in allopurinol dose. Review closely for possible side effects including allopurinol hypersensitivity syndrome.

Table 59: Allopurinol Dosage

Creatinine clearance (ml/min)	Maintenance dose allopurinol
0	100 mg every 3 days
10	100 mg every 2 days
20	100 mg/day
40	150 mg/day
60	200 mg/day
80	250 mg/day
100	300 mg/day
120	350 mg/day
140	400 mg/day

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.

29.4 Polymyalgic Syndrome/Systemic Inflammatory Disease

- A doctor 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 produce polymyalgic symptoms so the diagnosis of polymyalgia rheumatica should be made with caution and only after careful consideration of other potential causes.
- The list below is to be used as a prompt for the consideration of possible diagnoses and how they might be investigated.

29.4.1 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).

29.4.2 Clinical

Clinical assessment with frequent reviews should be the main guide to investigation.

29.4.3 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).

29.4.4 Investigations

- CBC + diff.
- CRP or ESR.
- Na, K, creatinine, urate, Ca, PO_4, alb, bili, alk. phos, AST, ALT.
- CK, thyroid function tests.
- Urinalysis including microscopy.
- Blood cultures (x3 sets with 10 ml 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-cyclic citrullinated peptide (anti-CCP) antibodies. Serum protein electrophoresis, immunoglobulins, and serum free light chain analysis.
- 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. Also women suffering from abdominal obesity may have an elevated CRP (usually <15-20 mg/l) with no other explanation.*

29.5 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 usually 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.

29.5.1 Investigations

- CRP or ESR.
- CBC + diff.
- Urinalysis, microscopy, protein, culture.
- Na, K, creatinine, urate, Ca, PO_4, alb, bili, alk. phos, AST, ALT.
- CK, thyroid function tests.
- Other investigations as determined by clinical assessment.

29.5.2 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, 12.5 mg for 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, 5-7.5 mg daily, for a period of 6 months or more before complete steroid withdrawal at 1 mg per month. A synacthen test is advised at 5 mg daily, to guide further withdrawal.
- 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 serum Vitamin D levels may need to be measured to ensure no deficiency exists. Patients requiring higher or prolonged doses of prednisone should be referred for a baseline bone density study and consideration of bisphosphonates (see Osteoporosis on page 186).

29.6 Giant Cell Arteritis (GCA)

GCA 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 (see page 186).

29.7 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, infliximab, adalimumab) and IL-1 are associated with an increased risk of infection.
- Specific infections associated with TNF blockade:
  - Mycobacterium tuberculosis (especially with infliximab).
  - Streptococcus 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.
  - The on-call Rheumatologist/Immunologist should be consulted if suspected infection in any patient known to our service on anti-TNF or other biological therapy.

29.8 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/ Rheumatologist/Nephrologist.

Table 60: 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/dsDNA, C3, C4
Mixed cryoglobulinaemia	Cryoglobulins, RF, C3, C4, SPE
B: Non Vasculitic	
Renal vein thrombosis/PE	Radiological
TTP	Blood film
C: Infective (a rare cause)	
Mycoplasma	IgM antibody
Legionella	Sputum for culture (contact lab), urinary antigen, serology (acute and convalescent), PCR on sputum and serum
Mycobacterium	Sputum ZN stain
S. pneumoniae	Sputum Gram stain
(1) *Request "urgent vasculitis screen".*	

30. Spinal Injuries

Burwood Spinal Unit Consultants 03 383 6850

- Dr Xianghu Xiong (Medical Director)
- Dr Rick Acland, Dr Angelo Anthony, Dr Raj Singhal.

30.1 Introduction

Traumatic spinal cord injuries (SCIs) are managed by the Burwood Spinal Unit team. Contact Burwood Hospital for the on-call Registrar or Consultant.

Non-traumatic spinal cord injuries need thorough investigations (refer to Spinal Cord Compression on page 164). Consultations should be made to Specialties as appropriate (e.g., Neurosurgery, Neurology, Orthopaedics, or Oncology).

Traumatic instabilities are defined as inability to weight bear or mobilise without risk of significant pain or neurological damage due to major structural damage to the spine if not adequately treated. Examples include odontoid, Hangman's, or teardrop fractures, or **any dislocation or fracture dislocation in the cervical or thoraco-lumbar spines**.

30.2 Traumatic Spinal Cord Injury

30.2.1 Investigations

- X-ray: AP and lateral films; cervical spine must include C7, otherwise "swimmer's view" or oblique views are needed.
- CT: for fractures/fracture-dislocation, very useful to determine stability.
- MRI: for significant cord injuries, checking discs and haematoma; or patients with objective neurological deficits but no obvious bony injuries.
- MRA: when vertebral arteries are potentially compromised (cervical SCIs with severe traumatic brain injury, fractures and/or dislocations involving foramen transversorium).

30.2.2 Management

Patients with cervical spinal cord injuries (tetraplegia) or high level thoracic cord injuries (paraplegia) with other major trauma such as chest, abdominal injuries, or multiple fractures need to be cared for and monitored in ICU. Other patients will be admitted via Orthopaedic Trauma Unit to the Burwood Spinal Unit.

With any significant spinal cord injuries:

1. Bed rest with log-roll (spinal care).
2. Ensure airway, breathing and circulation - treat neurogenic shock (with features of low blood pressure but bradycardia) with goal of maintaining systolic pressure >90 mm Hg, or mean arterial pressure >85 mm Hg). Check perfusion and urine output.
3. Start prophylactic anticoagulation 24 hours after acute spinal cord injury, e.g., enoxaparin 60 mg SC once daily. For spinal injuries without cord injury, give enoxaparin 40 mg SC once daily. Withhold anticoagulant 12 - 24 hours prior to surgical intervention.
4. Use prophylactic medications (ranitidine or omeprazole) to prevent stress ulcers.
5. Indwelling urethral catheter for bladder drainage and urine output monitoring.
6. Daily digital rectal bowel check and evacuation if needed.
7. Close monitoring, nursing care (especially skin care), and physiotherapy input.

30.3 Autonomic Dysreflexia in Spinal Cord Injury

Definition: Autonomic dysreflexia (AD) is a potentially life-threatening condition that can occur in anyone with a spinal cord injury at or above T6.

- Sudden and significant increase in blood pressure.
- Pounding headache.
- Bradycardia (may be a relative slowing so that the heart rate is still within normal range).
- Profuse sweating and flushing of the skin at or above the level of injury especially face, neck and shoulders.
- Piloerection or goose bumps at or above the level of injury.
- Cardiac arrhythmia, atrial fibrillation, premature ventricular contractions and atrioventricular contraction abnormalities.
- Blurred vision.
- Appearance of spots in the visual fields.
- Nasal congestion.
- Feelings of apprehension or anxiety.
- Minimal or no symptoms, despite a significant elevated blood pressure (silent autonomic dysreflexia).

Note: *If AD is not recognised and treated* ***promptly*** *the hypertension may escalate to dangerous levels resulting in intracranial haemorrhage, seizures, cardiac arrhythmia or death.* ***This is a medical emergency.***

30.3.1 Management

- Recognise the signs and symptoms of autonomic dysreflexia (AD).
- Check the patient's blood pressure (BP).

  A patient with a spinal cord injury above the T6 level often has a normal systolic BP in the 90-110 mm Hg range. Therefore an elevation of 20-40 mm Hg above baseline may be a sign of AD.
- If signs and symptoms of AD are present but no raised BP evident and the cause has not been identified, refer to medical staff.
- If BP is elevated, immediately sit the patient up if the patient is supine (as their condition allows).
- Loosen any clothing or constrictive devices.
- Monitor the BP and pulse frequently (every 2-5 minutes).
- Quickly survey the patient for the instigating cause, beginning with the urinary system.
- If an indwelling urinary catheter is **not** in place, catheterise the patient.
- If patient has an indwelling urethral or supra-pubic catheter, check the system along its entire length for kinks, folds, constrictions, obstructions and for correct placement. If a problem is found correct it immediately.
  - If the catheter is draining and the BP remains elevated, suspect faecal impaction (see below).
  - If the catheter is not draining and the BP remains elevated, remove and replace catheter in conjunction with reference to the above.

  Important: *if there is difficulty passing a catheter please* ***contact*** *the on-call* ***Urology Service*** *or* ***Burwood Spinal Unit medical staff on call.***

  Note: *monitor the patient's BP during bladder drainage. Sudden large fluid loss could cause hypotension if the patient has been given drugs to lower BP.*
- If acute symptoms of AD persist, including elevated BP, suspect faecal impaction (see below).
- If BP is ≥150 mm Hg systolic, consider administering glyceryl trinitrate (GTN) spray x 2 sublingually.

  Note: monitor patient for signs of hypotension.

- If faecal impaction is suspected and BP <150 mm Hg, with a gloved hand, instil a 2% lignocaine gel generously into the rectum.
- Wait 2 minutes then with a gloved hand insert a lubricated finger into the rectum and check for the presence of faeces.
- If AD becomes worse, stop immediately and insert lignocaine gel into the rectum again. Wait 20 minutes and check for faeces.
- If the causative factor of AD has not yet been found, check for less frequent causes, e.g., pressure area, UTI, ingrown toenail, or anything below the level of injury which could cause irritation if normal sensory and motor function were intact.

***Important: if no cause can be identified and hypertension persists**, seek further medical assistance **IMMEDIATELY**. This is now an acute medical emergency. Burwood Spinal Unit: discuss with the Acute General Medical Consultant and transfer the patient to the Emergency Department at Christchurch Hospital.*

Reference:

Consortium of Spinal Cord Medicine. Clinical Practice Guidelines: Acute Management of Autonomic Dysreflexia: Individuals with Spinal Cord Injury Presenting to Health-Care Facilities. Journal of Spinal Cord Medicine. 2002.

31. Thrombosis, Embolism, and Anticoagulation

31.1 Venous Thromboembolism: Deep-Vein Thrombosis (DVT), and Pulmonary Embolism (PE)

31.1.1 Prophylaxis of DVT/PE

Medical DVT Prophylaxis

Certain medical conditions such as stroke, myocardial infarction, heart failure, COPD, pneumonia, etc. 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 40 mg SC daily, providing there are no contraindications (e.g., active bleeding). The dose may need to be reduced in renal impairment - discuss with Consultant.
  - The duration of prophylactic treatment with heparins must be individualised. It should cover the obvious risk period such as immobilisation, but must be stopped as soon as the perceived increased risk has passed.
- **Myocardial Infarction**

  Early studies showed an incidence of DVT 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.
- **Stroke**
  - Early mobilisation and optimal hydration should be maintained from the outset.
  - All patients should have the adequacy of the lower limb arterial circulation, sensation and fragility of the skin assessed by the admitting medical staff.
  - The cause, ischaemic or haemorrhagic, needs to be established.

  For patients with ischaemic stroke only:
  - Full-length graduated compression stockings are **not** to be used routinely. These have been shown to be **ineffective** at preventing DVT after stroke and can cause tissue damage [Lancet. 2009;373:1958-65].
  - Subcutaneous low molecular weight heparin (LMWH) should be considered after 48h for patients at high risk from DVT such as immobile patients unable to lift one leg off the bed, obese patients, or those with past history of DVT/PE or known thrombophilia. Give enoxaparin 40 mg SC daily, less in renal impairment. The best timing for initiation of LMWH after stroke is not known. LMWH use is associated with increased ICH when initiated in the acute phase (<48h); the risk beyond 48h is not well quantified. Fatal PE is rare in the first week after stroke but peaks at the end of week 2. A decision regarding the use or otherwise of LMWH for immobile stroke patients should be documented by the end of week 1. Aspirin should be continued. Hydration and mobilisation remain cornerstones of DVT prophylaxis.
- **Congestive Cardiac Failure and Related Medical Conditions**

  Elderly medical patients, especially those with cardio-respiratory failure, infections and immobility are prone to DVT. Low molecular weight heparin (e.g., enoxaparin 40 mg SC daily) may reduce this risk.
- **Spinal injuries** (see page 255)

Reference: Fitzmaurice and Murray. Thromboprophylaxis for Adults in Hospital BMJ 2007; 334: 1017-1018.

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; presence of factor V Leiden, prothrombin mutation, or 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, pelvic, or abdominal 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). Dosage reduction may be required in renal impairment.**
- **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:**
  - Enoxaparin 20 mg SC two hours pre-op, then daily.

    See guidelines below for use of LMWH if spinal/epidural anaesthesia likely.
- **High Risk:**
  - Enoxaparin 40 mg SC 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).
- Any subsequent dose is given at least **6 - 8 hours 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).

Reference: Fitzmaurice and Murray. Thromboprophylaxis for Adults in Hospital BMJ 2007; 334: 1017-1018.

31.1.2 Clinical Features of DVT and PE

- Suspect DVT if there is swelling, 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, then haemoptysis, pleuritic chest pain, and pleural effusion may occur.

31.1.3 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 years, has had recurrent thromboses or there is a family history of thrombosis, **consider** activated protein C resistance (Factor V Leiden), anti-thrombin III deficiency, protein C or protein S deficiencies, prothrombin gene mutation, or antiphospholipid antibody syndrome. These tests should be taken before treatment is started.

31.1.4 Risk Assessment for DVT

Table 61: Clinical Model for Predicting the Pretest Probability of Deep-Vein Thrombosis*

Clinical Characteristic	Score
Active cancer (patient receiving treatment for cancer within the previous six months or currently receiving palliative treatment)	1
Paralysis, paresis, or recent plaster immobilisation of the lower extremities	1
Recently bedridden for 3 days or more, or major surgery within the previous 12 weeks requiring general or regional anaesthesia	1
Localised tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling at least 3 cm larger than that on the asymptomatic side (measured 10 cm below tibial tuberosity)	1
Pitting oedema confined to the symptomatic leg	1
Collateral superficial veins (nonvaricose)	1
Previously documented deep-vein thrombosis	1
Alternative diagnosis at least as likely as deep-vein thrombosis	-2

* *A score of two or higher indicates that the probability of deep-vein thrombosis is **likely**; a score of less than two indicates that the probability of deep-vein thrombosis is **unlikely**. In patients with symptoms in both legs, the more symptomatic leg is used.*

Reference: Wells et al. NEJM 2003; 349: 1227-1235.

Note: It is essential to work out the pretest probability for DVT and to write this in the patient's notes.

31.1.5 Investigations for Suspected DVT

Assess the clinical probability of DVT (refer to Risk Assessment for DVT on page 260), and get D-dimer result. In the future the green D-dimer request form will be replaced with a mandatory electronic request form available via Eclair. This will automatically calculate pre-test risk from the input data and provide advice to guide further investigation and management.

This advice is also given below.

- **A negative D-dimer** in a patient with an "unlikely" pre-test probability score **excludes a clinically significant DVT.** If the pre-test probability is high, consider an ultrasound (USS).
- **A positive D-dimer** in a patient with suspected DVT needs to be correlated with the pre-test probability score **and** an ultrasound scan (USS) of the affected leg(s). Then proceed as follows:

- **If the clinical probability is unlikely**:
  - and the USS is **normal**, DVT is for practical purposes excluded. If however the symptoms persist and an alternative diagnosis is not apparent by 48-72 hours, consider a repeat USS. If positive at 72 hours, treat with anticoagulant therapy.
  - and the USS is **positive**, treat with anticoagulant therapy.
- **If the clinical probability is likely**:
  - and the USS is **normal** or **equivocal**, consider CT venography (± CTPA), discuss with Radiologist. If there is any delay, consider anticoagulant therapy.
  - and the USS is **positive**, treat with anticoagulant therapy.

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, and/or consider anticoagulation.
- 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 clinical picture and may also be visualised by ultrasound.

31.1.6 Risk Assessment for PE

Table 62: The Revised Geneva Score

Variable	Points
Risk factors	
• Age >65 years	**1**
• Previous DVT or PE	**3**
• Surgery (under general anaesthesia) or fracture (of the lower limbs) within 1 month	**2**
• Active malignant condition (solid or haematologic malignant condition, currently active or considered cured <1 year)	**2**
Symptoms	
• Unilateral lower-limb pain	**3**
• Haemoptysis	**2**
Clinical signs	
• Heart rate:	
◦ 75-94 beats/min	**3**
◦ ≥95 beats/min	**5**
• Pain on lower-limb deep venous palpation and unilateral oedema	**4**
Clinical probability	
• Low	0-3 total
• Intermediate	4-10 total
• High	≥11 total

Reference: Le Gal et al. Ann. Int. Med. 2006; 144: 165-171.

Note: It is essential to work out the pretest probability for PE and to write this in the patient's notes.

31.1.7 Investigations for Suspected PE

- All patients with suspected PE should have their pre-test probability assessed using the Revised Geneva Scoring system (see page 261).
- All patients with suspected PE should have bloods taken for a D-dimer assessment. In the future the green D-dimer request form will be replaced with a mandatory electronic request form available via Eclair. This will automatically calculate pre-test risk from the input data and will act as a pathway to guide further investigation and management. This advice is also given below.
- 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 266.
- Out of normal working hours, and if it is clinically safe to delay establishing the diagnosis, consider anticoagulation overnight and investigate the following morning.

31.1.8 Action to Take When Results are Available

If the D-dimer is negative and:

- the patient has a low or intermediate 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 should 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. A VQ scan could also be considered if CTPA is equivocal or indeterminate for technical reasons. **In pregnancy**, a perfusion-only scan may be more appropriate than a CTPA. Discuss with an Obstetric Physician.

  An electronic request form will soon become available via Eclair to order CTPA and VQ scans. This will automatically calculate pre-test risk from the input data and will act as a pathway to guide further investigation and management. The data should already be present from the D-dimer request with the exception of the D-dimer result.

  The same guidance is given here.

- **Patients with a low/intermediate pre-test probability** and a negative CTPA or normal VQ scan: excludes clinically significant PE.
- **Patients with a low/intermediate pre-test probability** and an intermediate probability VQ: investigate further with CTPA scanning.
- **Patients with a low/intermediate 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 USS 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, http://www.brit-thoracic.org.uk.

31.2 Management of DVT and PE

31.2.1 Management of DVT

The standard treatment consists of LMWH and warfarin (see the table below).

31.2.2 Management of PE

Pulmonary embolism:

- Anticoagulate (see the table below).
- Oxygen.
- Analgesia as required.
- Patients with signs of right ventricular dysfunction should be considered for urgent investigations with a CTPA and/or echo, as they may benefit from thrombolysis therapy. Refer to Thrombolytic Therapy for PE on page 266.

Table 63: Drug Therapy for DVT or PE

- **Low molecular weight heparin**, e.g., enoxaparin 1 mg/kg q12h SC. **See below for dosage modifications.**
  - **Duration:** until INR >2 for 2 consecutive days (normally given for at least 5 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 265 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 re-thrombosis 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 265).

Treatment of DVT/PE in pregnancy. Warfarin is teratogenic. Start the patient on heparin and seek advice about further anticoagulation.

Reference: British Thoracic Society Guideline. Thorax 2003;58:470-484, http://www.brit-thoracic.org.uk.

Note: The following information needs to be considered before starting heparin/warfarin therapy.

- CBC + diff.
- Na, K, creatinine, alb, bili, alk. phos., GGT, AST.
- **INR/APTT in all patients**. In patients under 45, or with recurrent DVT/PE or with a positive family history take 2 citrate (blue top) tubes for anticardiolipin antibodies, lupus anticoagulant, protein C, protein S, APC resistance and antithrombin III (thrombophilia screen). Consider also prothrombin gene mutation.
- **Heparin**
  - Low molecular weight heparins (LMWH) have more predictable pharmacokinetics, a longer half-life compared to unfractionated heparin, and a lower rate of thrombocytopaenia (HIT).

- The risk of haemorrhagic complications is also less than with unfractionated heparin, but it is still a significant problem and fatal haemorrhage may occur.
- Dose reduction of LMWH is required in certain situations. Unfractionated heparin dosage should not be altered in relation to renal function and should be monitored with APTT testing in the usual way.

  Renal impairment: LMWH dose reduction is required if the creatinine clearance (CrCl) is <60 ml/minute. The first dose should be calculated on the actual body weight but subsequent doses should be reduced as follows:

  - CrCl 50-60 ml/minute give 70% of the weight-based dose.
  - CrCl 40-50 ml/minute give 60% of the weight-based dose.
  - CrCl 30-40 ml/minute give 55% of the weight-based dose.

  If the creatinine clearance is 30 ml/minute or less, discuss with Consultant. Unfractionated heparin should be given in this situation with close monitoring. Unfractionated heparin can be reliably reversed by protamine sulphate if abnormal bleeding occurs.

  Extremes of weight <45 kg or >150 kg: LMWH dosage should be monitored by anti-Xa levels at 150 kg or more. Patients <45 kg should also have anti-Xa monitoring. These patients should be given the first three doses of LMWH based on the actual weight and an anti-Xa peak level should be taken three hours after the third or fourth doses; subsequent LMWH dosage may need to be modified when the anti-Xa result is known.
- Anti-Xa monitoring should also be used when LMWH is given for the treatment of DVT/PE in pregnancy, or if LMWH is given for seven days or more.
- Interpretation of anti-Xa levels. This is a controversial area as the correlation between anti-Xa levels and the risk of bleeding or recurrent thrombosis is not exact. There is general agreement that in the treatment of DVT with LMWH the anti-Xa level should be between 0.4 and 1 units/ml. Consult Haematology for advice if anti-Xa levels and the clinical findings appear inconsistent.
- A number of low molecular weight heparins are currently available for the treatment of DVT. CDHB recommends enoxaparin.
- Studies have shown that **outpatient treatment of DVT and less extensive PE** with LMWH is safe and effective. Use the referral form provided and contact the Haemostasis Nurse (☎ 81246) for this service.

- **Warfarin**
  - **Check INR 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 INR 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 comorbidities could be started on warfarin 10 mg on day 1 and 10 mg on day 2 if INR <1.5. Otherwise follow the nomogram below.**

Table 64: 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.

Table 65: 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
Mechanical valves:		
• Aortic valve replacement	2-2.5	life long
• Mitral valve replacement	2.5-3	life long
Arterial disease	3-4	life long

(1) *Recurrence despite prothrombin ratio between 2 and 3.*

31.2.3 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).
- **Gastrointestinal**: cimetidine, omeprazole.

Some Drugs Expected to Decrease Warfarin Effect(1)

- **Antibacterials**: rifampicin.
- **Cardiovascular**: colestipol.
- **Central Nervous**: barbiturates, carbamazepine, phenytoin.
- **Others**: Vitamin K, Vitamin K rich foods, e.g., avocados, broccoli.

Herbal medicines(1) may interact with oral anticoagulants (e.g., gingko, fish oils - increased warfarin effect; e.g., St. John's wort, ginseng - reduced warfarin effect). Check with Ward Pharmacist or Drug Information (☎ 80900) if unsure.

(1) *Care may be required for other drugs or herbal medicines not listed.*

31.2.4 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.
- A massive PE is defined as having:
  - Haemodynamic compromise, and right heart strain, i.e., raised JVP, RV dilatation on CT, and an echo showing pulmonary hypertension.

  In this situation, the 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.
- Such patients should be in either CCU or ICU.
- **If** the investigations have confirmed PE and the above criteria are satisfied, give tPA (tenecteplase). Give single bolus IV, dosage according to weight:

Weight	Dose of Tenecteplase
<60 kg	30 mg
60 - 70 kg	35 mg
70 - 80 kg	40 mg
80 - 90 kg	45 mg
>90 kg	50 mg

- The combination of thrombolysis **concurrent** with low molecular weight heparin increases the bleeding risk.
- **If no abnormal bleeding occurs with tenecteplase, follow with low molecular weight heparin and warfarin as recommended in Drug Therapy for DVT or PE** on page 263.

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 mm Hg or DBP >110 mm Hg).
- 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 INR >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. http://www.brit-thoracic.org.uk.

31.3 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.

Table 66: 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:
▪ **Withhold warfarin for 4 days prior to operation day**. The aim is to allow INR to drop to <1.5 on day of surgery. ▪ 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** ▪ Commence IV unfractionated heparin when INR <2. Stop 6 hours prior to surgery. ▪ **Test INR on day of surgery**. If still ≥1.5 discuss with Surgeon and Anaesthetist.
After Surgery:
▪ 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. ▪ Continue with heparin until INR >2.

If DVT or PE >1 month ago or Acute Arterial emboli[2] >1 month ago

Before Surgery:
▪ **Withhold warfarin for 4 days prior to operation day**. The aim is to allow INR to drop to <1.5 on day of surgery. ▪ Commence on LMWH at **prophylactic dose** e.g., enoxaparin 40 mg SC daily. Last dose given on the day BEFORE surgery. ▪ Test INR on day of surgery. If INR ≥1.5 discuss with Surgeon and Anaesthetist.
After Surgery:
▪ Continue with LMWH at prophylactic dose after procedure, preferably on day of surgery. ▪ Restart warfarin (patient's usual daily dosing) 12-24 hours after the surgery. Ensure therapy commenced only after discussion with Surgeon and/or Anaesthetist. ▪ Continue with heparin until INR >2.

If in Atrial Fibrillation

Before Surgery:
▪ **Withhold warfarin for 4 days prior to operation day**. The aim is to allow INR to drop to <1.5 on day of surgery. ▪ Test INR on day of surgery. If INR ≥1.5 discuss with Surgeon and Anaesthetist.
After Surgery:
▪ 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 only [3]** 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 TIAs, CVA, systemic emboli, atrial fibrillation, severe LV systolic dysfunction, recurrent CHF, previous thromboembolism, hypercoagulable conditions.*

***Haemostasis Service, Haematology**: ☎ 81246. This service comprises a Haematologist (Dr Mark Smith) and nurse specialists 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.*

31.4 Treatment of Anticoagulant Overdosage

31.4.1 Heparin Overdosage

- 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 of unfractionated heparin estimated to be remaining in the circulation (maximum single dose 50 mg). 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 units of heparin. 1 mg enoxaparin may be partially neutralised by 1 mg protamine sulphate. A return of anti-Xa effect may be seen 3 hours after LMWH reversal, due to continuous absorption of LMWH from the subcutaneous depot. It may be necessary to give protamine intermittently to achieve and maintain neutralisation of subcutaneous LMWH for 12-24 hours. The patient must be carefully monitored. Seek Consultant advice.

  ***Caution:** Excess protamine sulphate may act as an anticoagulant itself.*

31.4.2 Warfarin Overdosage

Consider why the patient is on warfarin. For those patients with artificial heart valves, discuss with Cardiologist or Cardiothoracic Surgeon.

The Australasian Society of Thrombosis and Haemostasis have prepared the following consensus guidelines (Medical Journal of Australia 2004. Vol 181(9):492-7):

Table 67: Guidelines for the management of an elevated international normalised ratio (INR) in adult patients with or without bleeding

Clinical setting	Action
INR higher than the therapeutic range but <5 bleeding absent	• Lower the dose or omit the next dose of warfarin. Resume therapy at a lower dose when the INR approaches therapeutic range. • If the INR is only minimally above therapeutic range (up to 10%), dose reduction may not be necessary.
INR 5-9(1) bleeding absent	• Cease warfarin therapy; consider reasons for elevated INR and patient-specific factors. • If bleeding risk is high, give vitamin K (1-2 mg orally or 0.5-1 mg intravenously). • Measure INR within 24 hours,(2) resume warfarin at a reduced dose once INR is in therapeutic range.
INR >9 bleeding absent	• Where there is a low risk of bleeding, cease warfarin therapy, give 2.5-5 mg vitamin K orally or 1 mg intravenously. Measure INR in 6-12 hours, resume warfarin therapy at a reduced dose once INR <5. • Where there is a high risk of bleeding,(3) cease warfarin therapy, give 1 mg vitamin K intravenously. Consider Prothrombinex-HT (25-50 IU/kg) and fresh frozen plasma (150-300 ml), measure INR in 6-12 hours, resume warfarin therapy at a reduced dose once INR <5.
Any clinically significant bleeding where warfarin-induced coagulopathy is considered a contributing factor	• Cease warfarin therapy, give 5-10 mg vitamin K intravenously, as well as Prothrombinex-HT (25-50 IU/kg) and fresh frozen plasma (150-300 ml), assess patient continuously until INR <5, and bleeding stops.(4) OR • If fresh frozen plasma is unavailable, cease warfarin therapy, give 5-10 mg vitamin K intravenously, and Prothrombinex-HT (25-50 IU/kg), assess patient continuously until INR <5, and bleeding stops.(4) OR • If Prothrombinex-HT is unavailable, cease warfarin therapy, give 5-10 mg vitamin K intravenously, and 10-15 ml/kg of fresh frozen plasma, assess patient continuously until INR <5, and bleeding stops.(4)

(1) *Bleeding risk increases exponentially from INR 5 to 9; INR ≥6 should be monitored closely.*

(2) *Vitamin K effect on INR can be expected within 6-12 hours.*

(3) *Examples of patients in whom the bleeding risk would be expected to be high include those with active gastrointestinal disorders (such as peptic ulcer or inflammatory bowel disease), those receiving concomitant antiplatelet therapy, those who underwent a major surgical procedure within the preceding two weeks, and those with a low platelet count.*

(4) *In all situations carefully reassess the need for ongoing warfarin therapy.*

31.4.3 Intracerebral Haemorrhage while on Warfarin

For reversal of the warfarin-related coagulopathy for patients with intracerebral haemorrhage while on warfarin, see the Stroke section on page 154. See also Warfarin on page 269.

31.4.4 Bleeding Following Thrombolytic Agents

Abnormal haemorrhage may be very difficult to correct at least for some hours. If fibrinogen level is low, cryoprecipitate may help. Discuss with Haematologist.

31.5 Acute Limb Ischaemia

31.5.1 Common Causes

- Arterial embolism
  - Usually no previous history of peripheral occlusive arterial disease.
  - Sudden onset usually in a patient in atrial fibrillation but sometimes following myocardial infarction.
- Arterial thrombosis in situ
  - Often associated with previous history of occlusive arterial disease.
  - Examination findings may suggest widespread vascular disease (e.g., absent contralateral pulses).

31.5.2 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 Poikilothermy (cold). This is usually obvious with emboli but can be more subtle with acute-on-chronic ischaemia.

31.5.3 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 ± moderate paraesthesia ± moderate weakness.

     Consider angiography ± intra-arterial thrombolysis.
   - **Rutherford III**: Ischaemic rest pain, profound paraesthesia, and profound 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 may 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.

Reference: Rutherford RB et al. J Vasc Surg 1997; 26:517-538.

32. Urology

32.1 Urology Department Information

Main Office

- 3rd Floor, Parkside West, Level 3, ☎ 81009, Fax 80936

Inpatient Care

- Ward 14 (Adult)
- Ward 21 and 22 (Paediatrics)

Spinal Injury Care

- Burwood Hospital

Consultant Urologists and On-call Registrar

- Consultants: Peter Davidson (peter@urology.co.nz), Sharon English (sharon@urology.co.nz), Frank Kueppers (frank@urology.co.nz), Jane MacDonald (janemacdonald@urology.co.nz), Stephen Mark (stephen@urology.co.nz)
- There are three Urology Registrars and one fellow (two training positions).
- To contact the Urological Service on call, contact the Registrar or Fellow via the operator. Contact the Consultant Urologist via cell phone through the Christchurch Hospital operator. Most Consultants do not regularly clear CDHB email. The Consultants use their addresses at Urology Associates as listed above.
- Secretaries: Rose Webber (rosew@cdhb.govt.nz, ☎ 81009), Sue Lockie (☎ 81009).

32.2 Renal Colic

32.2.1 History

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

32.2.2 Examination

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

32.2.3 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.

32.2.4 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 273).

32.2.5 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.

32.2.6 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 >200 mcmol/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).

32.2.7 Subsequent Management

- The majority of patients do not need admission and will pass a stone.
- 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.
- Any stone in a solitary kidney.
- Creatinine >200 mcmol/l.
- Admission may be required in other circumstances. Discuss with Consultant.

When discharged:

- Send a referral to the Urology Outpatient 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.
- Routine prescription of an alpha blocker (doxazosin 2 - 4 mg/day - but consider a lower dose in elderly patients) assists passage of ureteric calculi.

32.3 Macroscopic Haematuria

32.3.1 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?

32.3.2 Examination

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

32.3.3 Investigations

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

32.3.4 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.

32.4 Microscopic Haematuria

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

- CBC + diff, creatinine, Na, K.
- MSU.
- Request "CT Haematuria".
- Flexible cystoscopy.
- Urinary cytology.

32.5 Testicular Torsion

32.5.1 History and Examination

- Sudden onset of testicular pain or lower abdominal pain. Associated nausea and vomiting. No flu-like symptoms or dysuria.
- Significant testicular tenderness or swelling associated with high lying or horizontal testis.

32.5.2 Investigations

- No investigations are warranted in the acute presentation of possible testicular torsion as this may delay definitive diagnosis.
- In cases where there is intermittent pain, a Doppler ultrasound may be useful.

32.5.3 Management

- Make urgent referral for acute testicular exploration, detorsion and bilateral testicular fixation.

32.6 Acute Urinary Retention

32.6.1 History

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

32.6.2 Examination

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

32.6.3 Investigation

- CBC + diff, creatinine, electrolytes, PSA.

32.6.4 Management

- Catheterise patient (see below): urine specimen to laboratory.
- Do not have more than 2 attempts to pass a urethral catheter.
- If unsuccessful a supra-pubic catheter may need to be inserted.
- If they drain more than 1.5 litres, monitor fluid and electrolyte balance.
- Send referral to Urology Department for consideration of TURP.

32.7 Urethral Catheterisation

32.7.1 Indications for Urethral Catheterisation

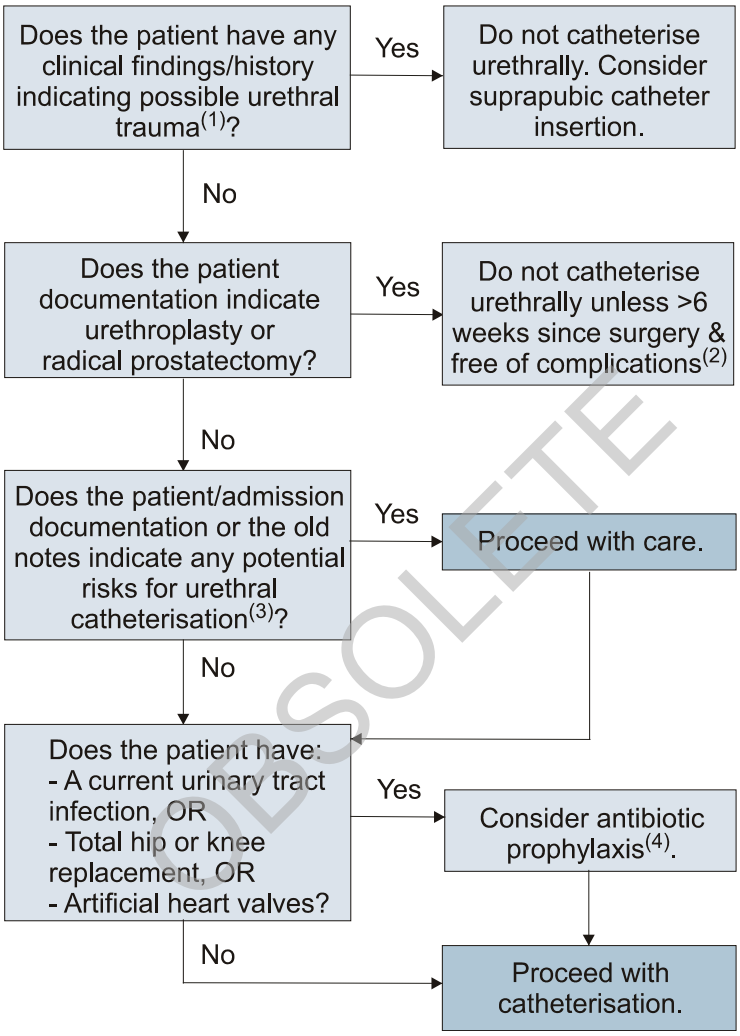
- Acute retention.
- Unconscious or sedated patients unable to void.
- In the operative and peri-operative setting.

- 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.

32.7.2 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.*

Table 68: Algorithm for Male Urethral Catheterisation



Notes

(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) *If history of:*

- Urethroplasty or radical prostatectomy <6 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 >6 weeks

  Proceed with urethral catheterisation with care using 14 Fr 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 Infective Endocarditis Prophylaxis (see page 56). For other situations, seek Consultant advice.*

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 (bigger is usually better, e.g., 18-20 Fr).**
- **Set up equipment following recommended best practice.**
- **Insert catheter as per recommended best practice.**

32.7.3 Insertion of Urinary Catheter

Choose catheter size and type according to reason for catheterisation. Choose the 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.

32.7.4 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.

33. Notifiable Diseases (New Zealand)

33.1 Notifiable Diseases (Health Act 1956)

Under section 74 of the Health Act (1956) all doctors **must** inform the Medical Officer of Health of all notifiable diseases. For some notifiable diseases, such as meningitis, this is because rapid follow-up of contacts by the public health service is imperative. Any doctor who does not inform public health services of notifiable cases **immediately**, may be held responsible for any deaths which occur as result of delaying prophylactic antibiotic treatment of contacts. Should such a tragedy occur, it would be very difficult to defend any charges of negligence, given that doctors who delay notifying public health services are clearly in breach of the Health Act.

Moreover, the Act specifies that the Medical Officer of Health should be notified **on suspicion**. This means doctors should not wait until laboratory results are available before notifying, unless the test carried out is simply to exclude a remote but serious possibility. Please note that laboratories in New Zealand are **also** required to notify, but this does not exempt clinical doctors from notifying **immediately on suspicion**.

In any case, if a positive result for a notifiable disease **is** received by a doctor they should notify the Medical Officer of Health even if the patient has been discharged. They must not assume the GP will follow up, even if the GP has been "copied in" to laboratory results.

IF IN DOUBT – NOTIFY

There are notification forms with every ward clerk which can be faxed to the Community & Public Health Division of CDHB (fax 379 6484).

Please note that under the Health Act, where an infectious notifiable disease is suspected in a patient, the Clinical Charge Nurse should **also** be informed, along with any precautions which should be taken by staff dealing with the patient.

33.2 Tuberculosis (TB Act 1948)

All forms of tuberculosis (see page 245) are notifiable to the Medical officer of Health under the TB Act (1948). In addition, under section 4 of this Act it is a legal obligation of **every** doctor to inform the Medical Officer of Health **in writing** of the intended discharge of a person infected with tuberculosis.

33.3 Sexually Transmitted Infections (Venereal Diseases Regulations 1982)

Sexually transmitted infections are **not** notifiable to the Medical Officer of Health **except** when:

- A patient does not attend for follow-up treatment twice, or is overdue by more than a week for a single follow-up, or
- The patient has syphilis or gonorrhoea and the treating doctor is aware of the names and/or descriptions of contacts which require follow-up.

33.4 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 other communicable diseases not on this list.*

33.4.1 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
- Cryptosporidiosis
- Giardiasis
- Hepatitis A
- Legionellosis
- Listeriosis
- Meningoencephalitis - primary amoebic
- Salmonellosis
- Shigellosis
- Typhoid and paratyphoid fever
- Yersiniosis

Section B - Infectious Diseases Notifiable to Medical Officer of Health

- Acquired Immunodeficiency Syndrome (AIDS)
- Anthrax
- Arboviral diseases
- Brucellosis
- Creutzfeldt-Jakob disease and other spongiform encephalopathies
- Diphtheria
- *Enterobacter sakazakii* invasive disease
- *Haemophilus influenzae* b
- Hepatitis B
- Hepatitis C
- Hepatitis (viral) - not otherwise specified
- Hydatid disease
- Influenza - Highly Pathogenic Avian Influenza (HPAI) and Influenza A (H1N1) - "swine flu"
- Leprosy
- Leptospirosis
- Malaria
- Measles
- Mumps
- *Neisseria meningitidis* invasive disease
- Pertussis
- Plague
- Pneumococcal invasive disease
- Poliomyelitis
- Rabies
- Rheumatic fever
- Rickettsial diseases
- Rubella
- Severe Acute Respiratory Syndrome (SARS)
- 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, early childhood service worker, etc)* ***or*** *single cases of chemical, bacterial, or toxic food poisoning such as botulism, toxic shellfish poisoning (any type) and disease caused by verocytotoxic* E.coli.

33.4.2 Diseases Notifiable to Medical Officer of Health (other than Notifiable Infectious Diseases)

Notifiable to the Medical Officer of Health

- Cysticercosis
- Taeniasis
- Trichinosis
- Decompression sickness
- Lead absorption equal to or in excess of 10 mcg/dl (0.49 mcmol/l)(1)
- Poisoning arising from chemical contamination of the environment

(1) ***Note:*** *Blood lead levels to be reported to the Medical Officer of Health (10 mcg/dl or 0.49 mcmol/l) are for environmental exposure. Where occupational exposure is suspected, please notify OSH through the Notifiable Occupational Disease System (NODS).*

33.4.3 Notifiable Diseases Under Tuberculosis Act 1948

Notifiable to the Medical Officer of Health

- Tuberculosis (all forms)

34. External Emergency Plan/Mass Casualty Incident Plan - Christchurch Hospital

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 the Emergency Department 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 bruce.hall@cdhb.govt.nz.

35. 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 in 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 2009 - November 2011 the stripe is

LIGHT GREEN

The Blue Book will be updated every second year in November at which time owners of the previous edition 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 the Revenue Manager, Gerard Thomas (03 364 0640 ☎ 80034).

The Medical Education and Training Unit/RMO Unit and the Revenue Manager maintain distribution lists 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 next 2 years.

36. Index

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37. List of Tables