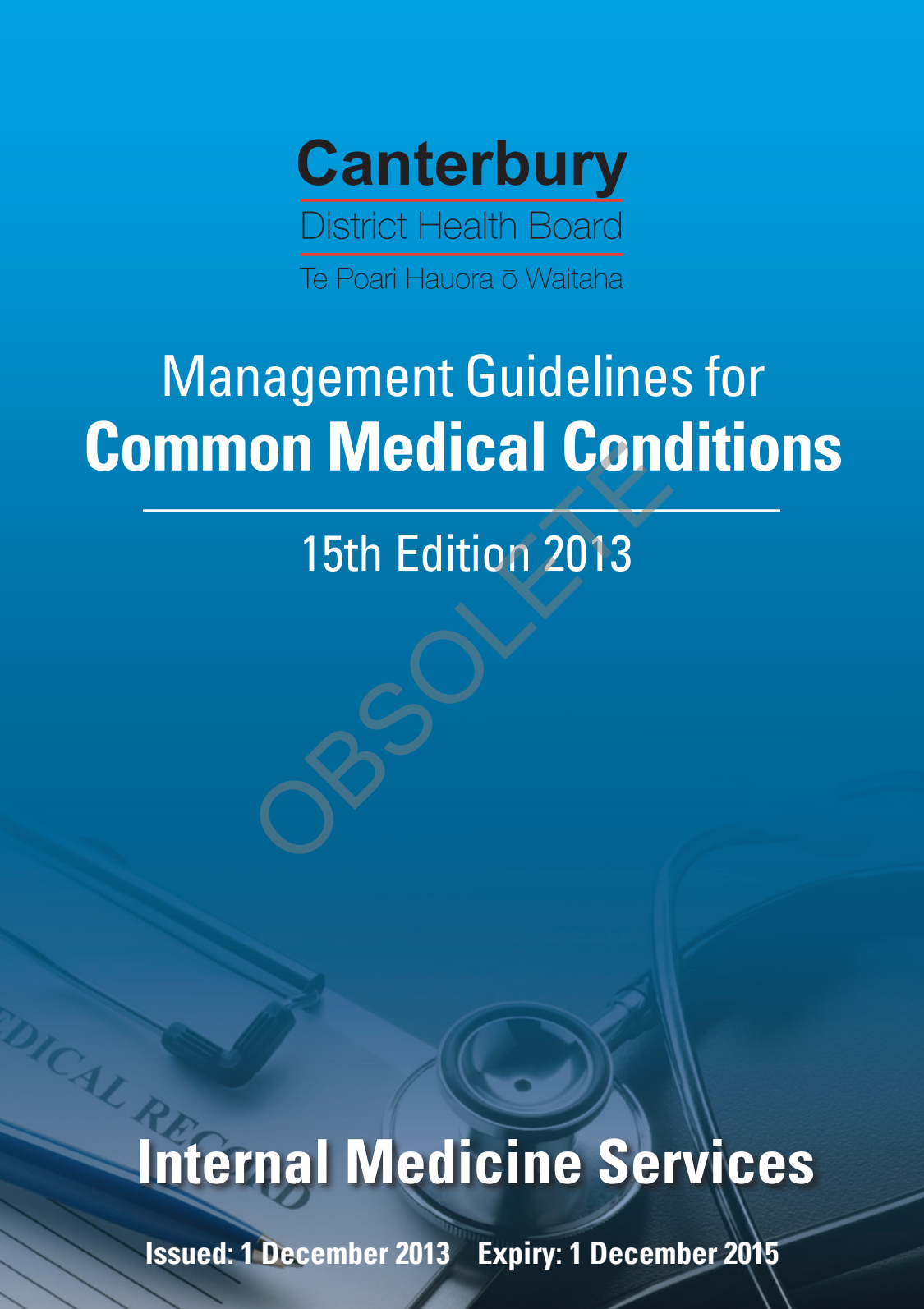
Canterbury
District Health Board
Te Poari Hauora ō Waitaha

Management Guidelines for
Common Medical Conditions

15th Edition 2013



Internal Medicine Services

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Management Guidelines for Common Medical Conditions

15th Edition 2013

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 - Fifteenth Edition 2013

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 standardized 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. Each subspecialty continues to produce recommendations for every 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 obtain as much accurate information about the patient as possible. Obtain 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 at CDHB. We are very grateful to Drs Ken Boon, Tamara Brodie, Thomas Evans, Niall Hamilton, Timothy Hii, Heather Isenman, Mike Liu, Aileen Ludlow, Clare Pate, Laura Sellers, Avinesh Shankar, Preechapon Tovaranonte, Emma Trowbridge, and Thomas Upton for their assistance with proof-reading, and to Helen Noble for her secretarial support. This edition has been produced by Emma Harding of Streamliners NZ Ltd (***www.streamliners.co.nz***) in the appropriate format for hard copy, intranet, and PDA/smartphone 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 PDA/smartphones. Please refer to mob.streamliners.co.nz for instructions on how to download the mobile versions.*

Note: *These guidelines must be used in conjunction with CDHB Preferred Medicines List ("the Pink Book")*

The following resources are also recommended:

- HealthPathways (http://www.healthpathways.org.nz) and its patient information site for the public of Canterbury, HealthInfo (http://www.healthinfo.org.nz).
- UpToDate - on the CDHB intranet.
- Harrison's Online - on the CDHB intranet.
- Cochrane Database.
- Ovid Medline.
- PubMed.
- eMedicine.
- Current medical journals.

See also: CDHB Early Warning Scores and Policy (online at http://cdhb.health.nz - search for "ews")

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

Note: *As at time of printing, the upgraded CDHB intranet is still "pending release", due early 2014. Some controlled documents may not be available on the old intranet. We have included search terms that will work as soon as the new intranet comes online.*

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1. Abbreviations

General

- **ABG:** arterial blood gases
- **alb:** albumin
- **ALP:** alkaline phosphatase
- **ALT:** alanine aminotransferase
- **AST:** aspartate aminotransferase
- **bili:** bilirubin
- **BiPAP:** bilevel positive airway pressure
- **Ca:** calcium [(1)]
- **CBC + diff:** Hb, PCV, MCV, WBC/differential, and platelets
- **CK:** creatine kinase
- **Cl:** chloride [(1)]
- **CPAP:** continuous positive airway pressure
- **CPR:** cardiopulmonary resuscitation
- **CRP:** C-reactive protein
- **CTPA:** CT pulmonary angiogram
- **CrCl:** creatinine clearance
- **CSF:** cerebrospinal fluid
- **CXR:** chest X-ray
- **DIC:** disseminated intravascular coagulation
- **DKA:** diabetic ketoacidosis
- **ESR:** erythrocyte sedimentation rate
- **FEV_1:** forced expiratory volume in 1 second
- **FiO_2:** fraction of inspired oxygen
- **fu:** fraction excreted unchanged in urine
- **FVC:** forced vital capacity
- **GGT:** gamma glutamyltransferase
- **INR:** international normalized ratio
- **K:** potassium [(1)]
- **LDH:** lactate dehydrogenase
- **LMWH:** low molecular weight heparin
- **LP:** lumbar puncture
- **Mg:** magnesium [(1)]
- **MJ:** megajoule, a million joules
- **MSU:** mid-stream urine
- **Na:** sodium [(1)]
- **NIV:** non-invasive ventilation
- **NSAID:** non-steroidal anti-inflammatory drug
- **PCA:** patient-controlled analgesia
- **PCR:** polymerase chain reaction
- **PML:** Preferred Medicines List
- **PO_4**: phosphate [(1)]
- **PTH:** parathyroid hormone
- **r-tPA:** recombinant tissue plasminogen activator
- **sat.O_2**: haemoglobin oxygen saturation (pulse oximetry)
- **SPE:** serum protein electrophoresis
- **STI**: sexually transmitted infection
- **USS:** ultra-sound scan
- **TFT:** thyroid function tests
- **VQ:** ventilation/perfusion scan

1. These items should be written in full in prescriptions and patient notes. They may be abbreviated only when requesting laboratory tests.

Drug Administration and Dosage

- **IV:** intravenous
- **IM:** intramuscular
- **subcut:** 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:** millilitre
- **L:** litre
- **mg:** milligram
- **g:** gram
- **kg:** kilogram
- **mmol:** millimole
- **mcmol:** micromole
- **pmol:** picomole
- **nmol:** nanomole

"international unit", "unit", and "milliunit" should not be abbreviated.

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 full word microgram **must** be used. Do not use µ or mcg.

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.
  - See Alcohol Withdrawal Scale (AWS) (search for "C240008" on the CDHB intranet).

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 IM maintenance. Oral therapy when appropriate.

Note: *Haloperidol may provoke seizures or hypotension. Give lower doses in the elderly.*

2.1.2 Early Withdrawal: AWS ≤10

- 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, see below.

2.1.3 Moderate to Severe Withdrawal: AWS >10 - Diazepam Loading

- For patients without concurrent disease give diazepam 20 mg orally every 1-2 hours for 3-4 doses (60- 80 mg) to achieve light sedation, and then stop. Re-assess after four hours.
- Because of the long half-life of diazepam, it is usually unnecessary to sedate further.
- If further doses are required due to the AWS score, give 5-10 mg every six hours if needed, and taper off after 3-5 days.
- A maximum daily dose of 120 mg of diazepam is generally sufficient, unless the case is especially severe, which should prompt the exclusion of other underlying conditions.
- If at high risk of seizures, consider also carbamazepine 400 mg PO stat then 200 mg TDS PO for 5 days. Some authorities recommend phenytoin or sodium valproate in this situation.

Note: *Consider dose reduction in the elderly, frail and those with liver impairment.*

2.1.4 Alcohol Withdrawal Seizures

- **Seizure treatment**: a previous history of withdrawal seizures denotes a significant likelihood of their recurrence. In those exhibiting seizures for the first time, blood sugar and neurological investigations (especially in head injury) should be undertaken before they are attributed to alcohol withdrawal. Treat seizures with:
  - 10 mg diazepam IV bolus injection, given slowly (can be repeated up to twice in first 30 minutes).
  - Thereafter give 10-20 mg diazepam by slow IV injection every six hours; or a maintenance dose of 1-3 mg per hour via continuous infusion. After 24 hours patients can be changed to oral medication.
  - For continuous seizures or status epilepticus consider IV anti-convulsants, such as IV phenytoin or IV valproate using IV loading dose and subsequent infusions.

Note: *Refer to the Neurology section for* **full seizure treatment details** *(see page 176).*

2.1.5 Wernicke's Encephalopathy

- If Wernicke's encephalopathy is present or suspected, give:
  - Thiamine 400 mg IV or IM TDS for 2 days, then 200 mg IV or IM daily for a further 5 days.
  - Vitamin B Complex 2 tabs PO BD. This preparation contains vitamins B1, B2, B3, and B6.
  - A multivitamin preparation once daily.
- **All patients** should be given **long term treatment** with thiamine 100 mg BD PO and Vitamin B Complex 1 tab BD PO and a multivitamin tablet daily.

2.2 Screening for Alcohol Related Problems

- This section provides guidance for the screening of alcohol related problems.
- 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 15 standard drinks per week.
  - Females: Over 10 standard drinks per week.
  - Male: Over 5 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 = 10 g alcohol

    = 330 mL (12 oz) beer, or

    = 110 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?
  - 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?
  - Most people have days when they drink more than usual; how many times in the last year have you drunk more than 6 standard drinks?

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 to the Alcohol and Other Drug Central Coordination Service.

2.3 Facilities available to help with alcohol related problems

Alcohol and Other Drug Central Coordination Service

Ph: 338 4437, Fax: 338 7427
35 Collins Street, Christchurch 8024

- All referrals for comprehensive alcohol and drug assessments/provision of treatment.
  - This service is for patients requiring more than brief intervention.
  - If a comprehensive assessment is required, your referral will automatically be sent to the most appropriate assessment service.
  - They will also advise on detoxification services available.
- You will be kept informed of the progress of your referral.
- Fax referrals using the AOD Referral Form.

Community Alcohol and Drug Service

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

- Phone this service for specific medical advice regarding prescribing for substance dependence/withdrawal.

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.
- Community services are at the same address.
- SixtyFive ALIVE - works jointly with other services to provide alcohol and drug treatment for patients >65 years.

Kennedy Detoxification Unit

Ph: 339 1139, Fax: 339 1142
Hillmorton Hospital, Christchurch

- Medical detoxification for South Island by referral only via Central Coordination Service or alcohol and drug treatment provider.

Thorpe House

Ph: 379 1682, Fax: 371 0602
Unit 1, 446 Cashel Street

- Non-medical detox

Home Detox Service

Ph: 379 1682, Fax: 371 0602
Unit 1, 446 Cashel Street

- For people over 18

Familial Trust

Ph: 981 1093; Fax: 974 9847
6 Wilsons Road, Christchurch

- Education/support for family members of people with addiction

Addiction Advocacy Service

Ph: 366 8288
PO Box 13496
Armagh, Christchurch 8141

Alcohol and Drug Helpline

Ph: 0800 787 797

3. Anaesthesia

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

- Contact the Anaesthetist concerned.
- If the Anaesthetist is 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

Preoperative 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.

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

Table 1 Routine Preoperative Investigations

Full Blood Count (CBC)	**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 70 mL/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
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 other procedures requiring 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 including warfarin and dabigatran.
- Diuretics/ACE inhibitors.
- Anti-inflammatory drugs.
- Antiplatelet drugs - if patient is taking any of these drugs for secondary prevention of cardiovascular events, the risks vs benefits of cessation should be ***discussed with the surgical team or Anaesthetist*** (see page 136).
- 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 Clinical Pharmacology Peri-operative Medications guideline (search for "peri-operative" on the CDHB intranet).

3.4 Management of Adult Postoperative Nausea and Vomiting (PONV)

PONV incidence and significance

Nausea and vomiting in the post-operative period is common and occurs in up to 80% of some patient groups. PONV can be highly distressing and many patients are more concerned about PONV than pain. It may cause significant morbidity.

Preoperatively anaesthetists aim to identify patients at high risk for PONV and give multimodal antiemetic prophylaxis/treatment intra-operatively.

The following information should assist ward doctors treating adult patients with PONV in the post-operative period.

Antiemetic drugs

Antiemetic drugs work principally by blocking afferent pathways between the gastrointestinal tract and the chemo-sensitive trigger zone, and between the vestibular apparatus and the vomiting centre. Several different receptor types have been identified:

Table 2 Antiemetic Drugs

Class	Receptor Blockers		Steroids	Anti-cholinergics
	5HT3	Histamine 1		
Antiemetic drugs	Ondansetron	Cyclizine	Dexamethasone	Scopolamine (Scopoderm™)
Recommended adult dose	4 mg IV 8 mg PO dispersible tablet	12.5 - 25 mg IV	4 mg IV	1.5 mg topical
Advantages	Non-sedating	Treats motion sickness	Some analgesia	
Side effects include	Headache Constipation	Sedation	Hyperglycaemia	Visual disturbance (rare)

Recommended approach

- First line: ondansetron 8 mg dispersible tablet PO or 4 mg IV q6h.
- Second line: cyclizine 12.5 - 25 mg IV (repeat q8h prn).

Note: *Risk of sedation, especially with opioids. Use with caution in older or debilitated patients.*

Other options to consider if symptoms have not improved within 20 minutes:

- Dexamethasone 4 mg IV: consider single dose (if not diabetic and if not already given intra-operatively).
- Scopoderm™ patch: 1.5 mg topical adhesive, effective for up to 72 hours with 2-4 hour latency for onset.

  Note*: Metoclopramide 10 mg IV is not effective for PONV prophylaxis and even in larger doses, metoclopramide does not appear to be an effective antiemetic in this situation.*
- **Or discuss PONV problems with the Duty Anaesthetist (pager 8120/8212) or Acute Pain Service (pager 8114).**

RMOs should:

- Use the safest, cheapest, lowest dose therapy to treat PONV.
- **Not** repeat doses of the same antiemetic drug (or drugs that act at the same receptor) within 6 hours.
- Consider surgical/neurological causes for PONV resistant to therapy.
- Consider other adjunctive treatments for PONV:
  - Use multimodal analgesia to lower total opioid doses
    - Regular paracetamol.
    - NSAIDs where appropriate.
    - If using IV tramadol, administer over 30 minutes (in 100 mL sodium chloride 0.9%).
- Maintain normal hydration in patients.
- Prevent/treat constipation and consider nasogastric drainage for bowel obstruction (discuss with Consultant).

Notes:

- Ondansetron by oral dispersible tablet has an oral bioavailability of ≈ 50% and is inexpensive, well-tolerated, and has proven efficacy as PONV prophylaxis.
- Dystonic reactions: excessive doses of dopamine receptor D_2 antagonists (e.g., metoclopramide, prochlorperazine, domperidone, droperidol) may cause extra-pyramidal symptoms or acute confusion. Treat with benztropine 1-2 mg IV, IM, or PO.
- **QTc prolongation/polymorphic VT/other arrhythmias:** many drugs (including ondansetron and droperidol) cause prolongation of the corrected QT interval. This may rarely predispose to torsades de pointes/polymorphic VT in susceptible individuals. Use with extreme caution in patients with known prolonged QT interval.

3.5 Perioperative Management of Diabetes

This will usually be supervised by the Anaesthetist. If not, refer to the guidelines in *Peri-Procedural Management of Diabetes* on page 89.

3.6 Guidelines for Perioperative Steroids in Patients already on Steroids

Refer to *Guidelines for Perioperative Steroids in Patients already on Steroids* on page 81.

3.7 Patients on Oral Anticoagulants Undergoing Surgery

For patients on oral anticoagulants such as warfarin or dabigatran, refer to *Patients on Oral Anticoagulants Undergoing Surgery* on page 135.

3.8 Guidelines for use of Anticoagulants if Spinal/Epidural Anaesthesia is used

Refer to *Guidelines for use of Anticoagulants if Spinal/Epidural Anaesthesia is used* on page 125.

3.9 VTE Prophylaxis in Surgical Patients

Refer to *VTE Prophylaxis in Surgical Patients* on page 123.

3.10 Patients on Antiplatelet Drugs Undergoing Surgery

Refer to *Patients on Antiplatelet Drugs Undergoing Surgery* on page 136.

4. Ancillary Services

4.1 Guidelines for Requesting Ancillary Services

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

4.2 New Zealand Blood Service

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

Blood Bank ☎ 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* (see page 22) including the management of Transfusion Reactions for further information.

4.3 Canterbury Health Laboratories

4.3.1 Service Provided

- The Core Laboratory functions 24 hours daily providing routine biochemistry, haematology, microbiology, and haemostasis testing.
- All other laboratories provide a routine diagnostic service between 0800 and 1700 hours, Monday to Friday.
- After hours, most laboratories provide an on-call service for urgent specialized tests not performed in the Core Biochemistry, Microbiology, and Haematology Laboratories.
- CHL also manages Point of Care Testing (POCT) for the CDHB.

4.3.2 Contact Telephone Numbers

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

4.3.3 Labelling of Specimens and Forms

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

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

For further information with regard to the requirements for individual tests and for interactions that may interfere with some assays, see **Test Manager** at www.chl.co.nz.

Minimum labelling requirements for specimens:

- Two patient identifiers:
  - Full name (surname and first name) **and**
  - NHI or DOB (preferably both).

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 authorized 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 CHL.

4.4 Radiology

- **Christchurch Hospital**: Emergency (general X-ray), Orthopaedic (general X-ray), and Main departments (general X-ray, ultrasound, CT, MRI, fluoroscopy and interventional). Subspecialty Radiology service 0800-1700 Monday to Friday , with 24 hour on-site MRTs.
- **Burwood Hospital**: general X-ray 0800-1700 Monday to Friday. MRT on-call 24/7. Patients requiring more specialized imaging will need to be sent to the main department at Christchurch Hospital. Radiologist on-site Friday morning only.
- **Christchurch Women's Hospital**: general X-ray and ultrasound 0800-1700 Monday to Friday. Patients requiring more specialized imaging will need to be sent to the main department at Christchurch Hospital. NICU MRT on-call.
- **The Princess Margaret Hospital:** 0800-1700 Monday to Friday on-site radiology services are provided by the Christchurch Radiology Group (CRG). General X-ray, ultrasound, and CT are available on-site. There is a CRG Radiologist on-call at all times.
- **Ashburton Hospital:** 0830-1700 Monday to Friday. General X-ray, ultrasound, and CT are available on-site. There is no regular on-site Radiologist cover. Please contact the department for report enquiries.
- **Radiologist on-call services:** there is 24/7 cover on-site at Christchurch Hospital by Radiology Registrars supported by Paediatric, Adult and Interventional on-call Radiologists. For general X-ray out of hours, contact the relevant site. For out of hours imaging for all other modalities for all sites (including Ashburton) you must contact the Radiology Registrar on-call via the Christchurch Hospital operator.

4.4.1 Picture Archiving and Communications System (PACS)

All imaging (except mammography) from Canterbury DHB, South Canterbury DHB and West Coast DHB is stored in the Canterbury DHB PACS. Radiology Department staff can assist with accessing imaging from other local providers' PACS and other DHBs. Reports for all publicly funded imaging in the greater Christchurch area, including imaging undertaken by private providers, are copied in to the Canterbury DHB Eclair results repository as part of TestSafe.

4.4.2 Consultation Forms

Paper or electronic forms are accepted. If using a paper form, ensure the patient's full name, date of birth and hospital number are included (a patient identification sticker is sufficient), and that your name, a contact pager number and the responsible Consultant's name are clearly written. If using the electronic order entry system, please select the correct patient encounter **and** the correct Consultant or team responsible for report sign-off, and make sure your role and contact details are up to date. The electronic order entry system will display messages on the status of any electronic orders placed, including triage, waitlisting and scheduling information. If the electronic order entry system is down, please use paper forms (they can be printed from the Radiology site on the intranet).

Adequate clinical information is mandatory to ensure the most appropriate examination is performed. Please include current clinical presentation, relevant past history that may be important to image interpretation (e.g., prior malignancy, surgery, smoking) **and** the question to be answered by imaging. If there has been relevant previous imaging by a site/provider who does not use the CDHB PACS, please provide details.

Please note that a contrast consent form is required for the majority of MRI and CT scans. The consent form must be filled in on the ward for patients unable to give consent e.g., patients who are confused or unable to communicate.

4.4.3 Radiology Reports

Radiology reports are distributed electronically for sign-off. Each department is responsible for identifying who is responsible for signing reports (emergency and inpatients reports should be signed within 24 hours, outpatient reports within 3 days). Reports that are not signed in a timely manner will be automatically escalated to an alternative responsible Consultant for sign-off.

4.4.4 Radiology subspecialty organization

Radiologists and Radiology Registrars practice under a subspecialty organization. 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.

4.4.5 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 specialized areas such as CT, MRI, ultrasound, and interventional radiology. The Registrar pager is 8911. Registrar office in Radiology is ☎ 89369.

4.5 Nuclear Medicine

Located on 2nd Floor, Riverside, Clinical Services Block, Reception: ☎ 80890, fax 80869.

Services Provided:

- Outpatient Imaging Service
  - ☎ 80867
  - 8:00 am - 4:30 pm Monday to Friday.
  - No out of hours service
- Therapeutic Service
  - Radio-iodine (I^{131}) therapies are provided by the Endocrine service (Thyroid management).
  - Other therapies: contact Dr O'Malley / Charge Technologist.

Personnel

- Clinical Leader: Dr Sue O'Malley, 027 241 1174, ☎ 80858
- Nuclear Medicine Physician: Dr Bill Avery, 027 511 1072, ☎ 80866
- Charge Technologist: Lynda Murray, ☎ 80863
- Physicist: Darin O'Keefe, 021 119 3744, ☎ 80855

Bookings

- Fax Nuclear Medicine Request form to 80869
- Bring by hand and discuss any urgent bookings or special clinical considerations

Nuclear Medicine Imaging Service

Detailed information about the nuclear medicine scans and the service provided, scan type, and any relevant patient preparation is available on the CDHB Intranet or externally at www.cdhb.health.nz/nuclear-medicine.

4.6 Pharmacy Services

Located on Ground floor opposite the Orthopaedic Outpatient Department at Christchurch Hospital.

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

Services Provided:

- A comprehensive clinical service to all wards in Christchurch Hospital, Christchurch Women's Hospital, The Princess Margaret Hospital, Burwood Hospital, and Hillmorton 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 prioritized and referred patients daily (Mon-Fri).
- 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 Transfusion Practice

5.1 Blood Transfusion Services

Blood Transfusion services are provided by the New Zealand Blood Service. In Christchurch they have two sites: the Blood Donor Centre processing and accreditation facilities at 87 Riccarton Road, and the Blood Bank (Crossmatching) 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.

See also: CDHB transfusion guidelines (search for "transfusion" on the CDHB intranet); NZBS clinical information (online at http://www.nzblood.co.nz/Clinical-information/Transfusion-medicine); Haematology Department Protocols and Guidelines (online at http://redbook.streamliners.co.nz).

5.2 Ordering of Blood

Note: The majority of transfusion errors are of a clerical nature.

- The same care and consideration must be taken with ordering blood transfusion as for the prescription of a dangerous drug.
- Blood must be ordered on the appropriate blood transfusion request form which must be completed as printed. No forms are acceptable unless they show the full particulars of the patient including surname, first names and patient identification number or date of birth which should be obtained from the identification bracelet. For group and hold or cross-match, send 6 mL blood in EDTA tube (pink top).
- A sample of the patient's blood must accompany the requisition form. All samples must be labelled by ballpoint pen 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.
- Blood is issued on demand within Christchurch and Christchurch Women's Hospitals and by request at other CDHB hospitals.
- A Christchurch audit revealed that 25% of patients were transfused to an excessively high haemoglobin level. If the haemoglobin is >110 g/L, a transfusion is rarely justified.
- In an adult, 1 unit of blood will raise the haemoglobin by 10 g/L.

5.3 Collection of Blood from Blood Bank

- Christchurch Hospital Blood Bank operates 24 hours a day, 7 days a week. Laboratory staff issue directly to Christchurch and Christchurch Women's Hospital on receipt of Form QMR022A.
- Blood is stored in blood fridges on site at Ashburton, The Princess Margaret and Burwood Hospitals. Blood is uplifted from these fridges by trained clinical staff using completed Form QMR022A.
- When blood is collected, the particulars on the compatibility label (i.e., 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.**

5.4 Administration of Blood

- Written consent must be obtained from patients or their guardians before transfusing blood or blood products. In an emergency, a medical officer can take legal responsibility to transfuse without consent.

- Blood must not be collected until it is needed for transfusion. Half an hour is the maximum interval permitted between collection and administration, and 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 blood products are administered, the particulars on the label must be checked with the particulars on the identification bracelet worn by the patient.
- A record of the transfusion should be kept in the patient's notes.

5.5 Transfusion Reactions

- Please report all transfusion reactions to Blood Bank using the Transfusion-Related Adverse Reactions Form. For all severe transfusion reactions contact the Transfusion Medicine Specialist (TMS) or Clinical Haematologist immediately.

Table 3 Guidelines for Management of Mild Adverse Transfusion Reactions

First Mild Reaction

Symptoms:

- Mild febrile reaction
  - Temperature increase <1.5°C from baseline
  - Stable haemodynamics
  - No respiratory distress and no other symptoms

OR

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

Action:

1) Check compatibility label and recipient identity.
2) Slow transfusion.
3) Call for medical assessment.
4) Medical staff may consider the need to prescribe paracetamol for pyrexia or antihistamines for urticaria.
5) Continue transfusion at a slower rate with increased monitoring, e.g., temp, pulse and BP at 15-30 minute intervals.
6) Send 1 x group and screen (EDTA) tube to Blood Bank + a completed Adverse Reaction Notification form (111F00901).
7) Document in patient's clinical notes.

If symptoms increase treat as a moderate or severe reaction.

Subsequent Transfusions and:

- Recurrence of mild febrile reactions

OR

- Recurrence of mild allergic reactions

Action:

1) Febrile reaction: consider giving premedication of an antipyretic (e.g., paracetamol).
2) Urticarial/allergic reaction: consider giving premedication of an antihistamine (e.g., oral phenergan). Slow transfusion administration rate.

Note: Hydrocortisone is not usually indicated.

Table 4 Management of Moderate and Severe Adverse Transfusion Reactions

Moderate and Severe Adverse Transfusion Reactions may include any of these:

Symptoms may include:

- Fever: ≥1.5°C from baseline with or without rigors / chills
- Unexpected tachycardia or change in blood pressure
- Acute breathlessness, desaturation, wheeze, stridor or cyanosis
- Facial oedema ± pharyngeal or laryngeal oedema
- Extensive erythematous or urticarial rash
- Acute pain up transfusion arm
- Chest or loin pain
- Severe apprehension
- JVP acutely elevated, onset of crepitations in lung
- Haemoglobinuria

Action if moderate or severe reaction is suspected:

1) Stop transfusion. THEN :
   - Check compatibility label and recipient identity information is correct.
   - Call for help: urgent medical review required.
   - Maintain ABC and monitor vital signs.
   - Comfort and keep patient informed.
2) Replace infusion set; administer saline to keep vein open and, or maintain blood pressure.
3) Treat and stabilize patient as per medical directives.
4) Obtain specimens based on clinical signs/symptoms (collect away from site of cannula):
   - Blood group serology: 1 x group and screen (EDTA) tube: send ASAP to blood bank with Adverse Reaction Notification form (111F00901) + infusion set + attached blood bag (sealed in a plastic bag).
   - If haemolysis suspected: send full blood count, blood film, coag screen to Haematology; Na, K, creatinine, haptoglobin, bilirubin, LDH to biochemistry and complete a ward urinalysis.
   - If sepsis is suspected: send blood cultures to microbiology.
   - If respiratory distress present: send blood gases to biochemistry.
5) Notify Blood Bank promptly by phone: Discuss further transfusion needs and/or any special requirements.
6) For all severe transfusion reactions: inform the NZBS Transfusion Medicine Specialist (TMS) or Clinical Haematologist immediately. They will provide clinical advice and support.
7) Document in patient's clinical notes.

Additional treatment: depends on cause, clinical state, test results and TMS or Clinical Haematologist consultation:

- Sepsis likely: broad spectrum antibiotics as per ***severe sepsis antibiotic guidelines*** (see page 147).
- Anaphylaxis/anaphylactoid reaction: as per ***anaphylaxis guidelines*** (see page 72).
- Transfusion associated circulatory overload (TACO): diuretics and oxygen, positive airway pressure.
- Transfusion related acute lung injury (TRALI): respiratory support. NZBS will initiate blood donor investigation.
- If HLA antibodies suspected: the TMS or Clinical Haematologist will advise.
- Recurrent severe allergic reactions: discuss with TMS or Clinical Haematologist. Use of washed cellular components may be required.
- Acute haemolysis: discuss with TMS or Clinical Haematologist. Maintain blood pressure, force diuresis and alkalinize urine.

5.6 Blood Products Available

See also CDHB Fluid and Medication Manual, Blood Components and Blood Products (online at http://cdhb.health.nz - search for "blood components").

Table 5 Blood Components	
Blood components available include: ▸ Red Cells Resuspended ($279) ▸ Whole Blood Plasma Reduced ($276) ▸ Cryoprecipitate ($390)	▸ Platelets Apheresis ($807) ▸ Platelet Pooled ($807) ▸ Fresh Frozen Plasma ($208)
1. Cryoprecipitate contains on average 1.3 g of fibrinogen per bag. 2. The above prices are for the period 2013 - 2014.	

Table 6 Blood Products[1]	
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-VF (Factors II, IX & X) ▸ Anti-D Immunoglobulin ▸ Tetanus Immunoglobulin ▸ Thrombotrol VF (Antithrombin III) ▸ Zoster Immunoglobulin ▸ C1 Esterase Inhibitor
1. Manufactured blood products have a NZBS label and are dispensed by NZBS directly to the requesting area. 2. Recombinant clotting factors are available from Pharmacy.	

5.7 Massive Transfusion Protocol (MTP)

- The New Zealand Blood Service (NZBS) has encouraged District Health Boards to develop protocols to enable the more efficient management of massive and acute bleeding.
- It is difficult to give exact criteria which should lead to activating the MTP. Acute blood loss of 1 litre or more with signs of hypovolaemic shock such as tachycardia and hypotension, particularly if the cause of bleeding cannot be immediately controlled, would be the sort of situation to initiate the MTP.
- An MTP should include a schedule that integrates the rapid provision of red cells, coagulation factors, and platelets, with the appropriate blood tests required to monitor the patient's progress. In addition, tranexamic acid 1 g IV stat followed by an IV infusion of 1 g over 8 hours is indicated for bleeding associated with massive trauma (the CRASH-2 trial), but must be given as soon as possible and definitely within 3 hours of injury. Tranexamic acid may be helpful in other causes of massive bleeding (seek Consultant advice).
- The CDHB has developed its own MTP (search for "mtp" at http://cdhb.health.nz). It can be activated by contacting the Blood Bank, tel 80310.
- In other DHBs, contact your local Blood Bank/NZBS to access the local MTP.

Reference: CRASH-2 Trial. Lancet.2010: 376(9734): 23-32.

5.8 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 (online at http://redbook.streamliners.co.nz).

6. Cardiology

6.1 Cardiology Department Information

Main Office

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

Inpatient Services

- Five inpatient teams on Wards 12, 14, & CCU

Cardiologists

- Dr Sally Aldous, Dr James Blake, Dr Paul Bridgman, Dr Ian Crozier, Dr Matt Daly, Assoc Prof John Elliott, Dr John Lainchbury, Dr Dougal McClean, Dr Iain Melton, Dr Aniket Puri, 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).

Primary Care Liaison

- Dr Joan Leighton (GP).

Outpatient Consultations

- Clerk, ☎ 81138

Consultation Guidelines

Chest pain, dyspnoea, documented angina, myocardial infarction, heart failure, valvular heart disease, arrhythmias, syncope, congenital heart disease. Please refer hypertension problems to General Medicine.

Other Services

- Cardiac catheters, coronary angioplasty, aortic valvuloplasty, trans-arterial aortic valve implantation, structural heart interventions (e.g., patent foramen ovale, patent ductus arteriosus), stress echo, trans-oesophageal echo, electrophysiology studies, radiofrequency ablation for tachyarrhythmia, pacemaker/defibrillator implantation
- Cardiology Day Ward, ☎ 81071, fax 81127
- 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)

6.2 Heart Failure

6.2.1 Definition

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

6.2.2 Management

Management requires each of the following:

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

6.2.3 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, severe anaemia.

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, particularly atrial fibrillation.
- 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.

6.2.4 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, acute or previous infarction).
- Myocardial injury markers: troponin and CK if the heart failure is acute or there has been a sudden deterioration.
- Na, K (urgently if ECG or rhythm abnormal), creatinine, Mg, Ca, PO_4.
- CBC.
- Echocardiography to assess LV function, valves, RV pressure estimate (urgent if hypotensive or if tamponade or bacterial endocarditis suspected).
- Thyroid function tests.
- ABGs if there is also COPD and there are concerns regarding CO_2 retention.
- 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 30).

  Note: *It can take 4 hours for BNP to become abnormal if heart failure is acute.*

6.2.5 Acute Heart Failure Therapy

Correct any contributing factors such as arrhythmia, infection etc.

- Always consider potentially reversible causes of severe heart failure (e.g., myocardial infarction, acute valvular regurgitation, pulmonary embolism) and institute appropriate therapy without delay.
- Acute pulmonary congestion, pulmonary oedema:
  - Sit patient upright.
  - Oxygen at 4-6 L/min only if the patient is hypoxic with a persistent sat.O_2 <92%.
  - 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 - 120 mg IV - repeat as necessary to initiate diuresis. The effective dose will vary and the larger dose may be needed if patient is on frusemide maintenance treatment or has renal impairment.
- Glyceryl trinitrate spray under tongue. Repeat doses of nitrate every 5 minutes while the blood pressure remains high.
- If patient does not respond to initial treatment then ***nitrate infusion*** (see page 32), continuous positive airway pressure (CPAP) by face mask, and haemodynamic monitoring in ICU or CCU should all be considered.
- CPAP is useful if the patient remains breathless and 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.
- In less distressed patients, oral frusemide may be sufficient but usually IV frusemide is required. Be alert to poor absorption from an oedematous GI tract in patients with chronic heart failure and peripheral oedema or ascites.

- 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), and stopping negative inotropic agents.
- For patients in atrial fibrillation or flutter, consider digoxin.
  - Digoxin - indicated only for control of ventricular response in atrial fibrillation and atrial flutter in acute heart failure. It has value as a third line agent in chronic heart failure in sinus rhythm.
    - Give 0.5 mg digoxin initially (IV if in heart failure or nauseated).
    - Give further 0.25 mg increments every 4 hours to complete a loading dose of 1 mg. Elderly and renally impaired patients are prone to toxicity and may only require a total loading dose of 0.75 mg. Some younger patients may require a further two 0.25 mg increments to give a total loading dose of 1.5 mg. Assess clinically before giving each increment. Do not give a loading dose if recently on digoxin.
  - Consider maintenance dose of 62.5 micrograms to 250 micrograms per day.
  - Monitor clinically for efficacy. As with many drugs, plasma concentrations are poorly correlated to therapeutic effect.
  - In renal failure and the elderly reduce the maintenance dose.
  - Plasma concentrations may be used to monitor for suspected digoxin toxicity.
    - In atrial fibrillation, avoid concentrations >2 nmol/L.
    - In chronic heart failure, concentrations >1.5 nmol/L are associated with poorer clinical outcomes, with 0.7 - 1 nmol/L preferred.

Table 7 Digoxin

- 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 52. 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.

1. See also ***digoxin poisoning*** on page 232.

Inotropic support:

- 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 this can be done in 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, each contains 250 mg in 20

mL) in 5% glucose to give a total volume of 500 mL (1 mg/mL) and run at 10 mL/hour (approximately 2.5 microgram/kg/min). Increase dose as required to achieve clinical response. Doses up to 10-15 microgram/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 microgram/kg/min) by IV infusion. Can be increased up to 7.5-10 microgram/kg/min if necessary (2 hourly steps of 2.5 microgram/kg/min).

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

- **After load reduction:**
  - In the acute situation, where oral therapy may not be suitable, use intravenous vasodilators such as ***glyceryl trinitrate*** (see page 32) or sodium nitroprusside. Nitroprusside will need close monitoring possibly including Swan Ganz and arterial pressure in CCU or ICU.
  - Correct any contributing factor such as arrhythmias, infection, etc.

6.2.6 Chronic Heart Failure Therapy

- **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.
  - 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 52. The starting dose of cilazapril for a patient with normal renal function is 0.5 mg to 1.25 mg/day.

Table 8 Quinapril and Enalapril Dosage in Renal Failure

Creatinine Clearance (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 potassium 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.
- Thiazide diuretics (e.g., metolazone 2.5 mg once daily, bendrofluazide 2.5 mg once daily)
  - These agents can induce a profound diuresis in a patient with persisting oedema who is already on frusemide and a potassium sparing diuretic and can be extremely useful. They need to be given as a single 'one off' dose and their effect monitored.
- **Further Management**
  - Daily weight to monitor the effects of diuretic therapy. It is essential to have a baseline weight with which to compare.

- Repeat CXR prior to discharge or if dyspnoea and/or clinical features fail to respond.
- Consider echo in all new patients with a diagnosis of heart failure.
- Consider echo if a pericardial effusion is suspected.
- Low molecular weight heparin (enoxaparin 20-40 mg subcut q24h). Start on admission. Consider full heparinization then warfarin in those with chronic atrial fibrillation.
- Potassium supplements will be needed with most diuretics. Requirements may be reduced or unnecessary in renal failure, with ACE inhibitor treatment, or when using potassium sparing diuretic therapy.
- Re-evaluate the primary cause of the heart failure - attempt to confirm the primary disease process and exclude aggravating factors. This may include cardiac catheterization in selected cases.
- Beta-blocker drugs do not have any role in the management of acute heart failure. However carefully titrated administration of beta-blockers reduces mortality in stable chronic heart failure associated with systolic dysfunction.

6.2.7 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 >120 pmol/L supports diagnosis of heart failure as the cause of breathlessness.

Notes:

- Normal range in healthy subjects is <30 pmol/L. In this situation, heart failure is unlikely.
- Values greater than 120 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 can be useful to guide treatment.
- BNP does not add to the diagnosis in a patient with overt heart failure.
- BNP may be abnormal in patients with RV strain secondary to pulmonary hypertension.

6.3 Myocardial Infarction

6.3.1 Definition

The diagnostic criteria for acute myocardial infarction are elevated biochemical markers of myocardial necrosis (e.g., 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.

6.3.2 Causes

Acute coronary occlusion due to:

- Coronary artery plaque rupture and thrombosis.
- Spontaneous coronary dissection (rare; occurs mainly in young women).
- Emboli (rare).

Note: *Other cardiac conditions such as myocarditis and stress cardiomyopathy (Takotsubo cardiomyopathy) may mimic myocardial infarction.*

Note: *Troponins may be elevated in other conditions. See* **Troponin Testing** *on page 34.*

6.3.3 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.

6.3.4 Investigations

- The initial ECG on presentation determines the management strategy:
  - If it shows ST elevation, the patient should be considered for immediate reperfusion (discuss with Cardiologist). Often no further tests are required prior to instigating treatment.
  - Otherwise, the diagnosis will rest on serial ECGs and myocardial injury markers.
    - Serial ECGs on 3 occasions 6 hours apart and before discharge. Repeat ECG when pain resolved or if pain recurs. Consider performing right sided leads for ST elevation, i.e., look actively for right ventricular infarction. May be useful in inferior infarction with heart block.
    - Cardiac Injury Markers: A *troponin* (see page 34) and creatine kinase (CK) should be done on admission and at 8 to 12 hours.
    - CXR can usually wait until normal working hours or prior to discharge. Indications for urgent X-ray 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).
- **If suspicion of aortic dissection, arrange an urgent CT scan and inform the Cardio-Thoracic team - see** *Thoracic Aortic Dissection* **on page 44.**

6.3.5 Management of ST Elevation Myocardial Infarction (STEMI)

- 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.
- **Remember "time is muscle" - expedite treatment and assess suitability for reperfusion by angioplasty urgently (thrombolysis is rarely given if angioplasty is available).**
- **Contact Catheter Laboratory and transfer patient** - any patient with acute myocardial infarction is at risk of an acute arrhythmia and should be discussed with the Consultant. Normally a patient can remain in ED but if there are going to be delays the patient should transfer to CCU and thrombolysis should be considered.
- Stable patients with suspected myocardial infarction require a telemetry bed or admission to CCU. For advice on admission contact the Cardiology Registrar on call.
- **IV access** - IV insertion on admission. Flush 4-6 hourly with 0.9% sodium chloride.
- **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.
- **Antiplatelet therapy:**
  - Give aspirin to all patients, 300 mg chew and swallow stat, then 150 mg daily. If ticagrelor is also to be given, the dose of aspirin should not exceed 100 mg daily.

- If the patient is scheduled for angioplasty, give ticagrelor 180 mg PO stat, then 90 mg BD PO unless the patient is deemed a high bleeding risk (CrCl <30 mL/min or severe liver impairment).
- If the patient is scheduled to receive thrombolysis, or is deemed a high bleeding risk, give clopidogrel 600 mg PO stat then 75 mg daily provided the patient is under 75. If over 75, give clopidogrel 300 mg PO stat, then 75 mg PO daily.

- **Oxygen** - should only be administered if the sat.O_2 <92%.
- **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% glucose in a non-PVC bag (200 microgram/mL). Start infusion at 10 microgram per minute (3 mL/h). Increase infusion rate by 10 microgram/minute every 3 to 5 minutes. Boluses of 10 - 20 microgram can be given until pain relieved or BP falls (can go as low as 90 mm Hg systolic if otherwise well).

6.3.6 Current Indications for Thrombolysis

- Where angioplasty is not available.
- If there is a delay in access to catheter laboratory, it can be considered (e.g., if both labs are occupied or if the patient has been assessed in a rural setting >45 minutes from hospital). An r-tPA (e.g., tenecteplase) is recommended.
- A combined approach, thrombolysis and angioplasty, may be appropriate when access to the catheter lab is delayed (>60 minutes). In this situation half dose tenecteplase is administered along with IV unfractionated heparin. Give an IV bolus of unfractionated heparin of 80 units/kg with a maximum dose of 10,000 units. The patient can then be transferred to the catheter laboratory when this is available.

6.3.7 Thrombolytic Therapy

The Christchurch Cardiology department runs a 24 hour service for ST elevation myocardial infarction.

The current favoured thrombolytic agent is **tenecteplase**.

Contraindications to Thrombolysis

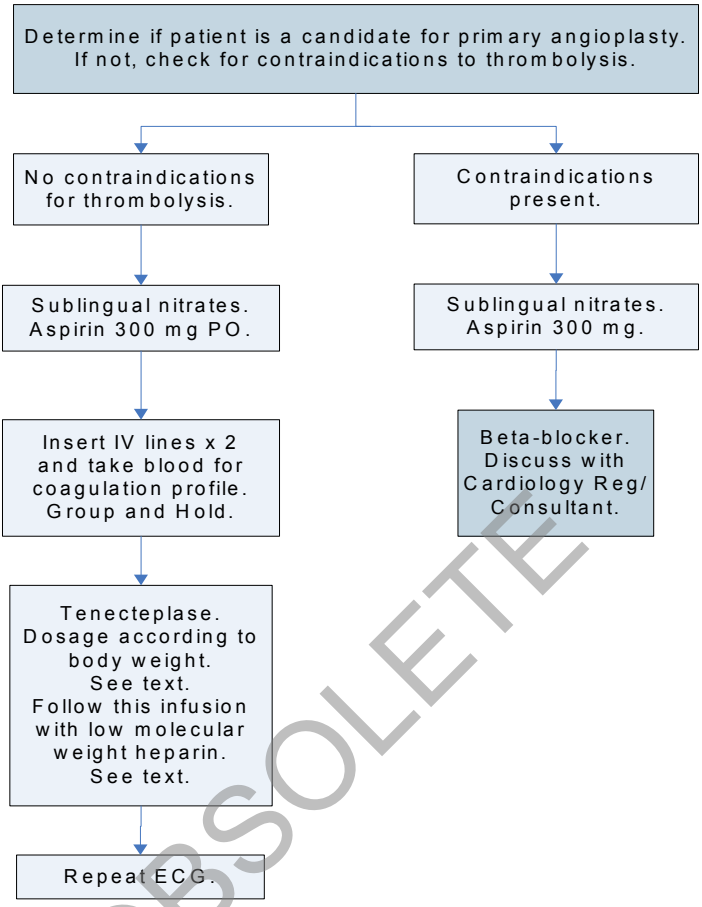
Absolute Contraindications:

- Any prior intracranial haemorrhage.
- Known structural cerebral vascular lesion.
- Known malignant intracranial or spinal neoplasm or arteriovenous malformation.
- Ischaemic stroke within 3 months, except if ischaemic stroke is being treated by thrombolysis.
- 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 (e.g., warfarin with an INR >2: the higher the INR, the higher the risk of bleeding).



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 subcut q12h for 48 hours post thrombolysis. Give first dose after completion of tenecteplase **if no abnormal bleeding has occurred**.

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

6.3.8 Further Guidelines on Management of STEMI

- Continue antiplatelet drugs as appropriate.
- If the patient has had an angioplasty, do not give heparin. Where thrombolysis is contraindicated and angioplasty is not done, give a low molecular weight heparin, e.g., enoxaparin 1 mg/kg subcut q12h. The usual duration of

enoxaparin treatment is 48 hours. The dosage of LMWH will need to be reduced if there is significant renal impairment (CrCl <60 mL/min) or for extremes of weight. See *the VTE section* on page 131.

- Anti-Xa monitoring is recommended if the duration of LMWH therapy is >48 hours. Treatment for longer than 48 hours 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 severe renal impairment or if reversal of the heparin effect is likely to be needed, consider a continuous infusion of unfractionated heparin and monitor the APTT (see *Unfractionated Heparin Dosage* on page 132). 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 - 95 mg PO daily), 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 32) may also be helpful.

6.3.9 Troponin Testing

- **Cardiac troponins are highly cardiac specific. High-sensitivity 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).

6.3.10 In-Hospital Management following Myocardial Infarction

- Mobilization protocols - these protocols are available in CCU and Cardiology Ward. Some patients can be discharged as early as three days after admission.
- Investigation after myocardial infarction:
  - 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.
  - Coronary angiography should be considered in all patients, including non-ST elevation MI and post infarction angina.
- Medical therapy should be tailored to each individual patient, but should include aspirin and clopidogrel (or ticagrelor) unless contraindicated (see *Management of ST Elevation Myocardial Infarction* on page 31 for dosage recommendations). Beta-blockers should also be given unless contraindicated, and ACE inhibitors given 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.

6.3.11 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).

6.4 Cardiogenic Shock

6.4.1 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).

- Consider immediate angioplasty and insertion of aortic balloon pump if feasible.
- If inferior MI consider RV infarction. Check RV leads on 12-lead ECG.
- Consider emergency echo (contact Cardiologist). This will identify the cause of the hypotension and guide treatment (RV infarction, LV infarction, acute VSD, acute papillary rupture, tamponade).
- Consider inotropes, e.g., dobutamine or dopamine. See *Inotropic Support for infusion details* on page 28. Discuss with Cardiologist.
- About 20% of patients with cardiogenic shock have low LV filling pressures (e.g., RV infarction or patients on diuretic therapy) and may benefit from fluid infusions (250 mL bolus 0.9% sodium chloride, 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, pulmonary embolism.

6.5 Acute Coronary Syndromes

6.5.1 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 30).

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.

6.5.2 Causes

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

6.5.3 Investigation and Management

This is similar to the treatment of acute myocardial infarction except that thrombolysis is not indicated. Angioplasty may be indicated for persistent or recurrent rest pain with ECG changes (discuss with Cardiologist).

- 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 a higher risk of subsequent complications.
- 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 subcut q12h 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. The dosage of LMWH will need to be reduced if there is significant renal impairment (CrCl <60 mL/min) or for extremes of weight. See *the VTE section* on page 131.
- Anti-Xa monitoring is recommended if the duration of LMWH therapy is >48 hours. Treatment for longer than 48 hours 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.
- Start aspirin. Consider adding clopidogrel if ticagrelor is not to be given. Ticagrelor should be started if the patient is being scheduled for angiography/angioplasty. Seek Cardiologist advice. See *Management of ST Elevation Myocardial Infarction* on page 31 for dosage recommendations.
- Start nitrate and a beta-blocker (or calcium antagonist if beta-blocker is contraindicated). 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.
- Oxygen should be considered only if sat.O_2 <92%.
- Telemetry monitoring if troponins are raised or the patient's condition is unstable; monitor for 48 hours.
- 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.

Reference: Non-ST-elevation acute coronary syndromes: New Zealand management guidelines. Cardiac Society of Australia and New Zealand. NZMJ. 2012 Jun 29;125(1357):122-47.

6.6 Cardiac Arrhythmias

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

6.6.1 Classification

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

6.6.2 Aetiology

- Common in the presence of structural cardiac disease, especially after 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 some anti-arrhythmics.
- Hyperthyroidism.

6.6.3 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.

6.6.4 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 potassium, magnesium, calcium; acidosis and hypoxia. Metabolic factors may contribute to the initiation/perpetuation of the arrhythmia.
- Thyroid function tests. Subclinical or overt hyperthyroidism increases the risk of heart failure.

6.6.5 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. 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. A temporary pacemaker may be required if there is recurrent syncope, non-sustained ventricular tachycardia, severe bradycardia (<30/min) or cardiovascular compromise.
- Symptomatic A-V block not associated with infarction usually merits placement of a permanent rather than temporary pacemaker.

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% glucose (= 4 microgram/mL) and start at 1 mL (4 microgram) per minute but then run as slowly as possible (0.5-10 microgram/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 they 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 stepwise 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 or hypotension.*
- If on beta-blockers and no structural cardiac disease present give flecainide 2 mg/kg (max 150 mg) diluted in 20 mL 5% glucose over 10 minutes IV (telemetry required) or consider further beta-blockade (make sure patient is not asthmatic).
- If unsuccessful or if the patient is hypotensive, 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 anaesthetized, 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 or adenosine therapy 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, Mg, thyroid function tests.
  - Control rate with either digoxin, calcium channel blocker, beta-blocker.
  - Digoxin - loading dose:
    - Give 0.5 mg digoxin initially (IV if in heart failure or nauseated).
    - Give further 0.25 mg increments every 4 hours to complete a loading dose of 1 mg. Elderly and renally impaired patients are prone to toxicity and may only require a total loading dose of 0.75 mg. Some younger patients may require a further two 0.25 mg increments to give a total loading dose of 1.5 mg. Assess clinically before giving each increment. Do not give a loading dose 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 LMWH, initial dose of enoxaparin is 1 mg/kg subcut q12h. The usual duration of enoxaparin treatment is 48 hours. The dosage of LMWH will need to be reduced if there is significant renal impairment (CrCl <60 mL/min) or for extremes of weight. See *the VTE section* on page 131.
  - Anti-Xa monitoring is recommended if the duration of LMWH therapy is >48 hours. Treatment for longer than 48 hours 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.
  - Oral anticoagulants may not be required if heparin started within 12 to 24 hours of onset of atrial 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. Otherwise start warfarin or dabigatran. Seek advice.
- Emergency electrical cardioversion:
  - Indicated if there is cardiac compromise with hypotension, angina 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 or dabigatran 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:

    Drug causes include:
    - 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 potassium.
    - Low magnesium.

    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.

Note: *Refer to the CDHB Clinical Pharmacology bulletin on Drug-Induced QT Interval Prolongation.*

- **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. See also ***Investigations for Poisoning/Drug Overdose*** on page 225.

  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 below).

- **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% glucose 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.

6.7 Cardiac Arrest

Commence basic life support unless the patient's status is ***DNACPR*** (see page 42). Use the ***ABCs of CPR*** (see page 63). Call for the **Clinical Emergency Team** and resuscitation trolley. Consider precordial thump if monitored or witnessed and a defibrillator is not immediately available.

REMEMBER:

- External chest compression at 100/min.
- **30 compressions to 2 ventilations for both 1 and 2 person CPR. Minimize interruptions to chest compressions.**
- Use oropharyngeal airway with ambu bag and face mask or supraglottic airway rather than intubate unless you are confident of success. If you insert an endotracheal tube, basic life support must not stop for more than 20 seconds. Give uninterrupted chest compressions at 100/min once intubated with a ventilation rate of 6-10/min.
- When the defibrillator arrives identify the rhythm utilizing the electrode pads.
- Position pads 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.

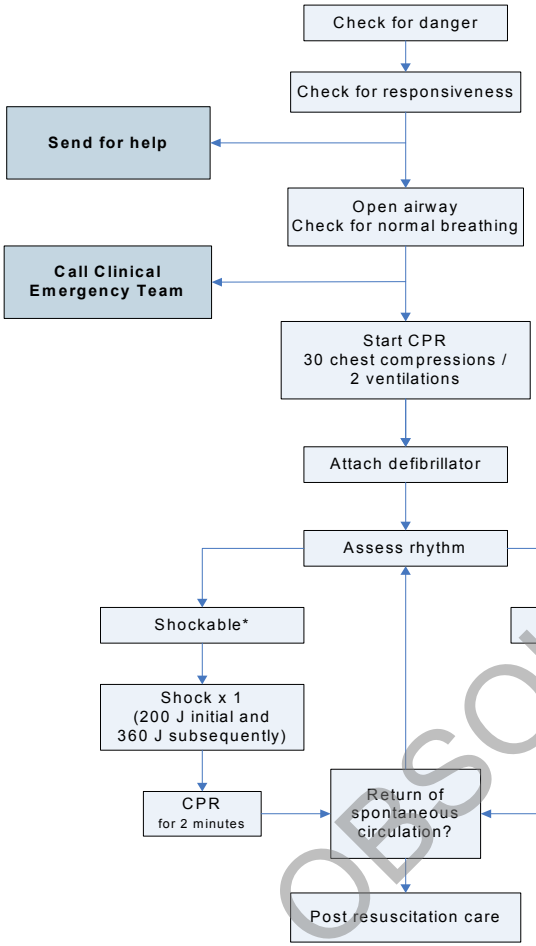
6.7.1 Identify the Cardiac Rhythm

- **Ventricular fibrillation (VF):**
  1. Defibrillate immediately using initial setting of 200 joules. Give one shock.
  2. Perform CPR for 2 minutes.
  3. If still in VF give one shock at maximum setting (360 joules), then 2 minutes CPR.
  4. After second shock if still in VF give adrenaline 1 mg IV. Continue one shock / 2 minutes CPR sequence.
  5. After third shock if still in VF give amiodarone 300 mg IV bolus. Continue one shock / 2 minutes CPR.
  6. Repeat adrenaline every 3-5 minutes.
  7. 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., organized 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. 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.*
  1. Perform CPR. Briefly check rhythm every 2 minutes. Minimize 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. Percussion (fist) pacing. For patients in cardiac arrest, percussion (fist) pacing is not recommended. However it may be considered in haemodynamically unstable bradyarrhythmias until an electrical pacemaker (transcutaneous or transvenous) is available.
  2. Consider atropine 0.6 mg IV and repeat if necessary.
  3. Consider an adrenaline infusion.

Table 9 Adult In-Hospital Resuscitation



During CPR

- Airway adjuncts (LMA / ETT)
- Oxygen
- Waveform capnography
- IV / IO access
- Plan actions before interrupting compressions (e.g., charge manual defribillator)
- Drugs

Shockable *

- Adrenaline 1 mg after 2nd shock (then every 2nd cycle)
- Amiodarone 300 mg after 3rd shock

Non Shockable

- Adrenaline 1 mg immediately (then every 2nd cycle)

Consider and Correct

Hypoxia

Hypovolaemia

Hyper / hypokalaemia (metabolic disorders)

Hyper / hypothermia

Tension pneumothorax

Tamponade

Toxicity

Thromboembolus

Post Resuscitation Care

Re-evaluate ABCDE

12 lead ECG

Treat precipitating causes

Re-evaluate oxygenation and ventilation

Temperature control (cool)

Authorized by the CDHB Resuscitation Coordinator 2011.

6.7.2 Post-Arrest Management

- Maintain basic life support unless the patient has an adequate spontaneous circulation and respiration.
- Titrate inspired oxygen to achieve sat.O_2 of 94-98%.
- Monitor ECG and transfer when stable to CCU or ICU, depending upon level of consciousness and requirement for artificial ventilation.

6.7.3 Information for Doctors Regarding Resuscitation Decisions

Cardiopulmonary resuscitation (CPR) can be lifesaving, particularly in the hospital context where effective CPR can be commenced very quickly. Cardiac arrest may be due to an acute and potentially reversible event and CPR can be effective at restoring cardiorespiratory function and allowing recovery.

CPR is the default option within all health care facilities in New Zealand unless an advance decision is made that CPR should not be attempted.

More commonly however, cardiac arrest is the final event in the dying process and for many patients with advanced illness, CPR has virtually no prospect of success and may be either unsuccessful or leave the patient worse off.

DNACPR stands for "Do Not Attempt Cardiopulmonary Resuscitation" and is the medical order in the CDHB which indicates that CPR should be withheld, allowing the death of that patient from natural causes or inevitable progression of their illness.

DNACPR status must be clearly displayed in the patient's notes. If this order is not apparent then as indicated the default position is to carry out CPR.

For further details, see *Cardiac Arrest* on page 40.

6.8 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 patient on telemetry whose status is DNACPR (do not attempt CPR).

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 CDHB guidelines for IV Administration of Drugs that require Cardiac Monitoring (search for "cardiac monitoring" at http://cdhb.health.nz).
  - 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 normalization 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 CDHB guidelines for IV Administration of Drugs that require Cardiac Monitoring (search for "cardiac monitoring" at http://cdhb.health.nz).
  - Monitor for at least 6 hours post drug administration.
  - These patients can be observed on AMAU using a bedside monitor.

6.9 Hypertension

Refer to *Hypertension* (see page 110) in the General Medicine section.

6.10 Rheumatic Fever

Refer to *Rheumatic Fever* (see page 148) in the Infectious Diseases section.

6.11 Thoracic Aortic Dissection

6.11.1 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.

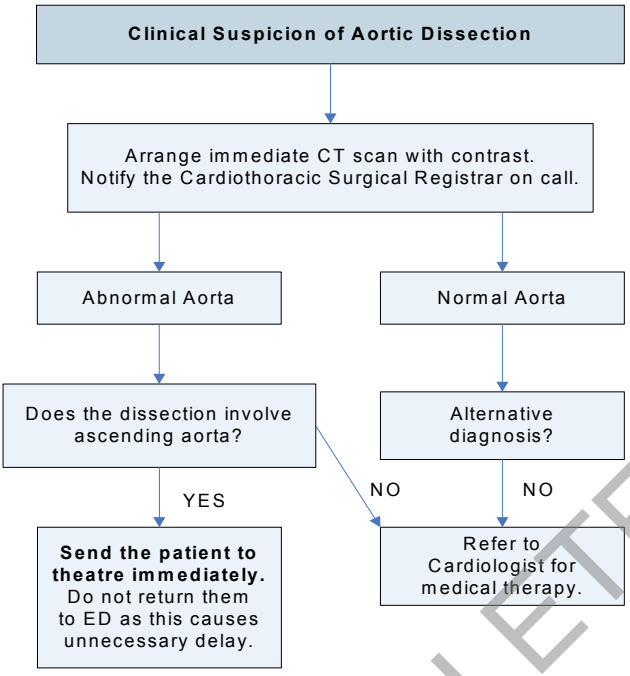
6.11.2 Aetiology

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

6.11.3 Investigations

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 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.*

6.11.4 Treatment - Type A

- Utilize 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, Na, K, creatinine. Cross-match six units. An ECG should also be done.
- 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 blood pressure and the force of contraction. This is best done with vasodilators and concurrently with 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. 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 32) or a sodium nitroprusside infusion. 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 prochlorperazine 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?"**

6.11.5 Treatment - Type B

- Refer to Cardiologist and Consultant Vascular Surgeon. These patients are generally managed by vascular surgery but may occasionally be admitted to CCU for control of labile hypertension. 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.

6.12 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.

6.12.1 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.

6.12.2 Treatment

- Initial therapy - benzylpenicillin 2.4 g IV q4h **and** gentamicin 1 mg/kg IV q8h **and** flucloxacillin 2 g IV q4h-q6h. If penicillin allergy, seek advice.
- If a prosthetic valve is present, the initial therapy should be vancomycin 20-30 mg/kg IV as a loading dose (max 1.5-2 g), then 1 g q12h IV with peak and trough levels around the third dose (refer to the vancomycin dosing guidelines in the Pink Book) **and** gentamicin 1 mg/kg IV q8h (refer to the gentamicin in Endocarditis guidelines in the Pink Book) **and** rifampicin 450 mg BD PO.
- 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.

6.13 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.

6.13.1 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.

6.13.2 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 **need prophylaxis**.
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.

6.13.3 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 49). 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 catheterization).

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

6.13.4 Education and Identification of At-Risk Patients

District Health Boards and other organizations 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.

6.13.5 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., amoxicillin) 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)
Amoxicillin 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 catheterization) 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 - amoxicillin (clindamycin or clarithromycin if penicillin allergy). ▸ Gastrointestinal, hepatobiliary, genitourinary or obstetric/gynaecological infections – amoxicillin (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).

7. Clinical Pharmacology

7.1 Clinical Pharmacology Department Information

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

Main Office

- Ground Floor Parkside, Department of Medicine, ☎ 81858, fax 81003.

Consultants

- Prof Murray Barclay, Prof Evan Begg, Assoc Prof Matt Doogue

Consultation Service

- For consultations, fax 81003.
- For urgent consultations, contact the Registrar ☎ 88354, pager 8524.
- For Drug Information ☎ 80900.

Consultation Guidelines

Any clinical pharmacology issues, such as: interpretation of drug concentrations, advice on therapeutic drug monitoring and toxicology; adverse drug reactions and drug interactions; complex polypharmacy; guideline development; drug utilization/costs; drug information.

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

Clinical Pharmacology Intranet

The Clinical Pharmacology site provides ready access to key medicines resources, including:

- The Preferred Medicines List (PML, the Pink Book).
- The NZ Formulary.
- MIMS: access to the MIMS formulary online and for PDA/smartphone.
- 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 Utilization Review: drug expenditure, campaigns.
- Bulletins/Guidelines: clinical pharmacology bulletins/guidelines (including Perioperative Medication).

To access this site, search for "clinical pharmacology" on the CDHB intranet

7.2 Drug Information Service

Staff

- Tracey Borrie, Pam Buffery, Elle Coberger, Julie Knight, Marie-Claire Pow.
- ☎ 80900, pager 8264, fax 80902.
- Email: druginfo@cdhb.health.nz

Function

- To answer patient-related drug information enquiries from health professionals on a wide range of topics including:
  - Drug safety in pregnancy and breastfeeding.
  - Drug use in liver or renal impairment.
  - Pharmacokinetics.
  - Drug concentration monitoring.
  - Adverse effects.
  - Identification and funding/access issues.

- 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 does not:

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

7.3 The Preferred Medicines List (PML), known as the Pink Book

The Pink Book guides local prescribing and includes common daily dose ranges, costs, PHARMAC restrictions, and advice about prescribing. It lists the "preferred" drugs used at CDHB 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.
- Drugs with PHARMAC restrictions should be used within the restriction.

The Antimicrobial 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.
- Common polypharmacy toxicities (including serotonin toxicity).
- Drug metabolism (including Cytochrome P450) and interactions.
- Drug profiles, for commonly used drugs.

7.4 Drug Utilization Review

Staff

- Jane Vella-Brincat, ☎ 89971

Functions

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

7.5 Dose Individualization and Drug Concentration Monitoring

Individualization 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. Contact the Clinical Pharmacology Registrar for assistance.

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

7.5.1 Dose Reduction in Renal Impairment

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

How to reduce the dose:

1) Choose the dose-rate (DR) that you would use in this patient if renal function were normal (DR(normal)). DR is mg/day or similar.
2) **Estimate renal function**. Either calculate creatinine clearance (CrCl) or use the estimated GFR (eGFR).

   Note: *eGFR is provided by Canterbury Health Laboratories (using the CKD-EPI formula). Like its predecessor, the MDRD formula, this is unreliable when the creatinine is changing or at extremes of weight.*

   Calculate CrCl using the modified Cockcroft and Gault equation as follows:

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

   Note: Use actual body weight if this is less than the ideal body weight.

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

   Note: *The creatinine must be stable for the estimated renal function to be valid (whether using the eGFR result or the Cockcroft and Gault result). In addition, the further the patient is from normal (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. Alternatively the estimated glomerular filtration rate (eGFR) supplied by the laboratory can be used for patients whose BMI is close to the normal range.*
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 maximize compliance.

8. Clinical Procedures

This section describes the current policy and practice 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.health.nz or the Clinical Skills website or visit the Clinical Skills website (search for "CSU" online at http://cdhb.health.nz).

8.1 Intravenous Cannula Insertion and Care

Any procedure that 'breaks' the protective skin surface has the potential to introduce infection. It is important for RMOs 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.
  - 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. Use a tourniquet or sphygmomanometer.
  - Prior to insertion, prepare site with antiseptic solution. We recommend 2% chlorhexidine and 70% isopropyl alcohol. Alcohol alone is not adequate. Hirsute arms may need clipping. The antiseptic must remain in contact with the skin for at least 30 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 stylet and connect previously primed administration set or luer plug. Ensure puncture site is clean and dry (using sterile gauze swab) before covering site.
  - To stabilize the cannula, use a sterile prepackaged transparent dressing with sterile tapes which will stabilize the cannula and act as a dressing (these are found on the IV trolley).

IV Cannula
Insertion date / /
Time

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.*

- **Failure to insert an IV line**
  - It is important to recognize 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/nursing team.
- **Care of IV Cannula**
  - Examine daily. Replace routinely every 48-72 hours, or at the first signs of phlebitis. Clinical examination detects only some infected catheters. Septic thrombophlebitis may cause ongoing bacteraemia after removal of the catheter, and may need surgical drainage.
  - Both nursing **and** medical staff are responsible for assessing the need for cannula change.
- **Suspected Cannula Infection**
  - Disconnect giving set from the cannula.
  - Remove dressing and take swab of cannula site.
  - Remove the catheter and cut off subcutaneous portion using sterile scissors. Place in a sterile container. Send to Microbiology Laboratory.
  - Clean the cannula exit site with antiseptic solution as above, leave for 30 seconds, and apply dressing.
  - 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 23.
  - **In the event of a needle stick injury, if the patient is known to be HIV positive, or there is reason to suppose that there is an increased risk of HIV positivity, contact the on-call Infectious Diseases Physician (not the Registrar) immediately.**
  - For further information, refer to the CDHB Peripheral Cannulation Handbook 2010.

8.2 Central Venous and PICC Lines

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

CVADs are 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. For further information refer to CDHB CVAD Handbook (ref no: 3022).

8.3 Intravenous Line Sepsis

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

Common Errors

- Failure to wash hands before inserting the cannula.
- Placing non-sterile dressings and tape over puncture site
- Inadequate disinfection of site.
- Leaving in situ too long (>72 hours).
- No record of insertion date.
- Failure to replace lines inserted under emergency conditions (e.g., by ambulance staff, or in Emergency Department).
- Failure to seek help when you have had 2 or 3 attempts to insert an IV line.

8.4 Blood Culture Collection

- Blood cultures should ideally be taken via the **vacutainer compatible (safety) butterfly method** (see below), 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 below), aseptically inoculate the **aerobic** bottle first.
  - If using the *syringe method* (see page 55), aseptically inoculate the **anaerobic** bottle first.
- Taking 2 to 3 blood culture sets 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.

8.4.1 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
- Vacutainer hub, sharps container, tape, and bandaid/plaster.
- Plastic transport container, 1 for each bottle.

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

- Wash hands before procedure.
- Thread the vacutainer compatible safety butterfly onto the Vacutainer hub.

- 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 10 cm above proposed venepuncture site 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.
- Put on non-sterile gloves.
- 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 hub 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. While the needle is still in the vein, activate the push button to retract the needle. Withdraw the butterfly from the vein. Apply plaster. **Immediately discard entire unit into sharps container**.
- **Place filled bottle into plastic transport container and send to lab immediately**. Do not refrigerate.

8.4.2 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 10 cm above proposed venepuncture site 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 skin wipe 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.
- **Place filled bottle into plastic transport container and send to lab immediately**. Do not refrigerate.

8.5 Chest Aspiration

Diagnostic pleurocentesis may be undertaken by medical staff with appropriate experience. You must see at least one performed and then do one yourself under supervision before attempting a chest aspiration on your own. **If you are uncertain about whether to carry out a chest aspiration, contact the Respiratory Team on call.**

8.5.1 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. It is essential to have the assistance of a nurse when performing a chest aspiration.
- Obtain the most recent chest X-ray.
- A lateral decubitus CXR will allow identification of free fluid. Pleurocentesis may be performed safely if 10 mm width free fluid is identified on a lateral decubitus CXR. **However an USS is preferred pre aspiration and is essential if loculated fluid is suspected.**
- 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 (1% lignocaine), 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 264) for advice on which tests to do on the pleural fluid obtained.

Note - when to use ultrasound:

- **Ultrasound is preferred for all patients. However it is essential if the effusion is difficult to locate by clinical examination, or if it appears to be loculated.**
- **If you are unable to obtain fluid with a 22G 38 mm needle, seek Respiratory Service advice before proceeding. It is unwise to use a longer or larger gauge needle without further imaging.**

8.5.2 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 55). 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.

8.6 Insertion of Intercostal Tubes

The insertion and management of intercostal tubes is a complex and specialized area which should only be undertaken by trained staff. 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. See Spontaneous Pneumothorax - Treatment for further advice.

See also: *Intercostal Tubes* on page 267.

8.7 Joint Aspiration

Joint aspiration must only be performed by trained staff.

Explain the procedure to the patient. Obtain verbal consent and document in the clinical notes.

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 269.

8.8 Lumbar Puncture

See also: *Meningitis* on page 143, and *Subarachnoid Haemorrhage* on page 175.

- 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 or if platelets $<50 \times 10^9$/L or if INR/APTT is abnormal.
  - **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 22G pencil-point lumbar puncture needles although some departments favour 25-26G. A 22G needle is easier to use, enables accurate measurement of CSF opening pressure, and allows a sufficient flow of CSF to obtain the volume of CSF that may be required. The smaller needle may however be associated with a lower complication rate.

  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.

  Note: *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.

  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.*
- 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%.
- Aim for the L3-4 or L4-5 disc spaces. Use strict aseptic technique and chlorhexidine/alcohol for skin sterilization. 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, check the pressure if this is required. The hips should be partly unflexed since any pressure on the abdomen may falsely elevate the CSF pressure.
- Then 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

  Notes:
  - 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 tuberculosis is suspected, an additional 1 mL of CSF is required for further analysis.
- 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, especially if a pencil-point needle is used.
- 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.**

8.9 Urethral Catheterization

Refer to the guidelines in the *Urology section* (see page 281).

9. Common Emergency Presentations

9.1 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, Mark Gilbert, Amanda Holgate, Rob Ojala, Scott Pearson, David Richards, Claire Taylor, Martin Than.
- Secretary, ☎ 89614
- Triage Nurse, ☎ 80274 (waiting room) ☎ 88254 (resuscitation/ambulance area)

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 from the General Medical and General Surgical services should generally see their patients in the AMAU and SARA respectively, if space there permits. If a patient has been referred by a GP direct to that team, a brief assessment will be done by nursing staff on arrival to the ED, and if the patient meets the Early Warning Score (EWS) criteria, then the patient shall be sent to the AMAU or SARA. If the patient does not meet the EWS criteria, a Senior ED Doctor may review the patient and then send them to the AMAU or SARA if they feel it safe and appropriate to do so.
- The same process should generally apply to patients who have been seen by the ED Doctors and then referred on.
- There will be exceptions to these rules and they should be negotiated between a Senior ED Doctor and the inpatient Registrar or Consultant.
- Fast track admission pathways are in place and will be used when appropriate by the ED.
- 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 placement.

9.2 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.

9.3 Early Care of Trauma

For a more comprehensive manual on trauma management at CDHB, including Trauma Call Activation Criteria and Trauma Team composition and responsibilities, see the CDHB Trauma Guidelines (search for "trauma guidelines" on the CDHB intranet).

- The principles of the Emergency Management of Severe Trauma 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, including any specialized investigations that are needed.
  - **Definitive Care**: the treatment to 'fix' the injury.

9.3.1 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 (with C-spine control).
   - Oro/nasopharyngeal airway.
   - Oro/nasotracheal intubation.
   - Cricothyroidotomy.
3) Recognize 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) Administer high flow oxygen.
3) Consider chest decompression or drain where appropriate.

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, external jugular vein, central venous access, or cutdown depending on the situation and the clinical skills of the operator. Take blood for CBC+diff, cross match, Na, K, creatinine, glucose, coagulation profile 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 22). Activate the *Massive Transfusion Protocol* (see page 25).
5) Monitor the patient with an ECG and BP monitor and a pulse oximeter.

Disability

1) Determine the level of consciousness - AVPU or *GCS* (see page 63). 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.

9.3.2 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 281).

9.3.3 Radiology

Note: *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.

Cases where C-spine X-rays are not routinely needed:

- Nearly all multisystem trauma patients require CT imaging. Many patients require a "trauma CT scan" 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.
- C-spine X-ray may also be omitted if the patient requires a CT head (do a CT head **and** neck), or if it is clear that they will require a CT regardless of the X-ray result (in which case proceed straight to the CT).
- In some patients it is possible to clinically clear the neck using the "NEXUS" (see www.mdcalc.com/nexus-criteria-for-c-spine-imaging) or "Canadian C-Spine" rules (see www.mdcalc.com/canadian-c-spine-rule), in which case no X-ray is needed.

9.3.4 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. Remember the core purpose of a FAST scan is to rule out life-threatening intra-abdominal bleed as a site for major blood loss in the haemodynamically unstable patient to aid immediate

disposition and treatment. A normal FAST scan does **not** rule out all abdominal injuries in every patient, and should not be used to do this.

9.3.5 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 prioritize 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. **P**regnancy.
- **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 immobilization of the cervical spine and remove the front of the semi-rigid collar. Inspect the neck and palpate posteriorly.
  - If not already performed, and clinically indicated, ask for a cross-table lateral cervical spine X-ray (see above). Regardless of the result, keep the collar on.
- Chest
  - Re-evaluate the chest as in the Primary Survey.
  - Ask for a CXR if not already done. 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 as clinical examination is unreliable.
- 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. Assess neurovascular status of each limb.
  - Adequately splint any injuries.
- Neurological examination
  - Assess the *Glasgow Coma Scale* (see below).
  - Look for any localizing signs.
  - Re-evaluate the pupils.
  - Assess the neurological level of injury (myotomes and dermatomes) as appropriate.

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
	Localizes pain/purposeful movement	5
	Withdraws from pain	4
	Abnormal flexion	3
	Abnormal extension	2
	None	1
Score	**Total Possible**	**15**

9.4 The ABCs

9.4.1 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.
  - Nasopharyngeal airway.
  - 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).

9.4.2 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.
  - Pleural disease - pneumo/hydro/haemo thorax.

9.4.3 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).

Pitfalls in assessment of circulation:

Elderly patients, pregnant patients, athletes and patients on cardiac medications or with a pacemaker may have the normal signs of hypovolaemia masked, and caution should be used when assessing these groups.

9.5 Altered Level of Consciousness

See also: *Stupor and Coma* on page 181.

In general terms decreased level of consciousness and coma is caused by either intracranial or extracranial abnormalities. Use the *GCS* (see page 63) or AVPU score to ascertain the level of consciousness.

9.5.1 Initial assessment and resuscitation

Note that supportive care with attention to the ABCs is paramount while the diagnostic possibilities are being considered. Once a diagnosis is reached, definitive management can be undertaken.

- Airway (commonly impaired by altered level of consciousness), breathing, circulation.
- Consider the "three coma antidotes":
  - **Thiamine** 100 mg IV or IM, if there is the possibility of Wernicke's encephalopathy. Give before glucose.
  - **Glucose** - check capillary blood glucose, if hypoglycaemia confirmed give 50 mL of 50% glucose (dextrose) solution IV.
  - **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.

9.5.2 Definitive Management

According to the *cause* (see page 181).

Extracranial causes

Rarely have focal neurology and often gradual onset.

- **A** **A**irway compromise.
- **B** **B**reathing abnormalities (hypoxia and hypercapnia).
- **C** **C**irculation abnormalities (hypo- and hypertension).
- **D** **D**rugs (sedatives, ethanol, tricyclic antidepressants, opiates etc.).
- **E** **E**ndocrine and metabolic abnormalities.
  - Consider glucose, sodium and calcium abnormalities, hepatic encephalopathy, renal failure, hypo- and hyperthermia, hypo- and hyperthyroidism and Wernicke's encephalopathy.
  - Attention to and correction of any ABC abnormalities, along with specific treatment for toxic ingestions and metabolic abnormalities, is required.

Intracranial causes

Often have focal neurology and often abrupt onset. Will need an urgent CT head and referral to Neurosurgery if any suspicion of a focal lesion as it may be amenable to operative treatment.

- Trauma - extradural, subdural, subarachnoid, or intraparenchymal bleeds or diffuse axonal injury.
- Seizures and post ictal states:
  - Benzodiazepines, phenytoin and valproate as indicated.
- Infective - meningitis or encephalitis:
  - CT should precede lumbar puncture, but where meningitis is suspected early antibiotics are mandatory.
- Vascular - intracranial bleed or brain stem infarct.
- Mass lesion e.g., bleed into tumour or subdural haematoma:
  - Urgent referral to Neurosurgery as indicated.
- Hysterical coma (psychogenic altered level of consciousness):
  - Diagnosis of exclusion.
- Assess the *Glasgow Coma Scale* (see page 63).

9.6 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 with large bore IV cannulae. Trendelenberg position. Patient should be managed in an area capable of monitoring and with resuscitation capability. A urinary catheter 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.

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.
- ED ultrasound may be used to determine whether there is right heart strain or pericardial effusion.
- 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.
- Ultrasound of the IVC may be a quick way of assessing volume status in the Emergency Department or ICU - this needs a practitioner trained in this technique.
- CVP may also 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).

  Note: *There is a trend in penetrating trauma to limit fluid resuscitation until surgical arrest of the bleeding can be achieved - especially in young healthy adults. Overtly aggressive resuscitation may cause haemodilution of coagulation factors and hypertension which disturbs fragile clots. Aim for a systolic blood pressure >90 and treat hypertension with analgesia if needed. Discuss early with a Surgeon as to whether surgical correction of blood loss is indicated.*
- Crystalloid 'splints' the circulation temporarily before extravasating, therefore more will usually be required (another 2000 mL).
- For Class III or Class IV shock (see table below), 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 22. Type specific blood may be available from the blood bank prior to full cross-match.
- Consider the Massive Transfusion Protocol (available on the CDHB website) if there is massive bleeding with either shock or abnormal coagulopathy. In general if there is not an adequate response to 3 units of blood then the protocol should be activated.
- Don't forget localized control (pressure on external bleeding, surgery for internal bleeding).

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

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

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%)

9.7 Syncope

Definition - a transient loss of consciousness. See also *Altered Level of Consciousness* on page 64.

Overview

After initial stabilization as needed, the aim is to diagnose the cause. In about 50% of patients the cause is obvious (e.g., GI bleed, epilepsy, hypoglycaemia, or classic vasovagal syncope). However in approximately 50% of people we will not ascertain the cause early on. In this undifferentiated group we aim to divide them into a "High Risk" group with respect to cardiac syncope, which should be referred, and a "Low Risk" group which can usually be discharged.

Indicators that may point to **high risk** syncope:

- Cardiac condition (ischaemic heart disease, conduction issues including permanent pacemaker in situ, aortic stenosis).
- Cardiac symptoms (e.g., palpitations or chest pain).
- Multiple episodes of syncope, or syncope in the horizontal position.
- Syncope on exertion.
- No vasovagal type prodrome (sudden onset).
- Drug overdose or toxicity.
- Advancing age.
- ECG abnormalities.

9.7.1 Initial assessment and resuscitation

- Airway
- Breathing
- Circulation

Consider telemetry if you are concerned that an arrhythmia is a likely cause of syncope. Refer to *Telemetry Guidelines* on page 43.

9.7.2 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. Include a medication history, particularly hypotensive drugs, e.g., alpha-blockers, ACE inhibitors, diuretics.

Examination

- The examination should include careful palpation of pulse rate, 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.
- Listen for murmurs, particularly the ejection murmur of aortic stenosis and hypertrophic obstructive cardiomyopathy.
- Assess for other possible causes of syncope, e.g., GI bleed, pulmonary embolus, sepsis etc.

Investigations

- 12 lead ECG - the most important test in undifferentiated syncope. Look for acute ischaemia, arrhythmias, and conduction abnormalities (e.g., heart block, bundle branch block, long QT, hypertrophic cardiomyopathy, Brugada syndrome, etc.).
- Random blood sugar, Na, K, creatinine, CBC + diff when indicated.

Note: *Troponins are not routinely required unless clinically indicated.*

9.7.3 Possible causes

Possible causes, in order of prevalence:

- Vasovagal syncope (90%)

  Usually occurs when the torso is upright, has a prodrome, and may be triggered by needlestick phobia, standing in warm crowded rooms, or postural hypotension (particularly in the elderly). The commonest cause of postural hypotension in the elderly is drugs.

  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, ruptured ectopic pregnancy.
- 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 symptomatic cardiac condition. It carries a poor prognosis, so if suspected, the patient should be admitted and monitored.
- Epilepsy (0-5%)

  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
  - TIA/stroke is an exceedingly rare cause of syncope and can be clinically excluded if there is no focal neurology. Syncope may occur at the onset of sub-arachnoid haemorrhage.

9.8 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.

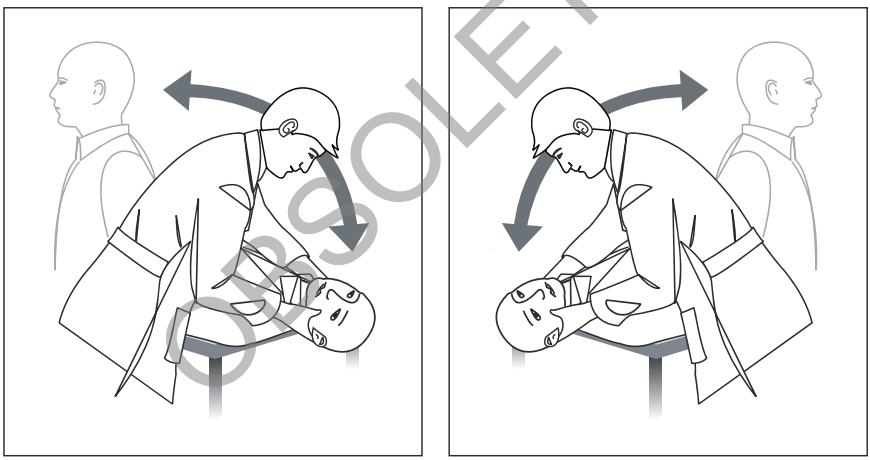
9.8.1 Peripheral Causes of Vertigo

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 Canalith Repositioning Procedure* (see page 70).
- 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.

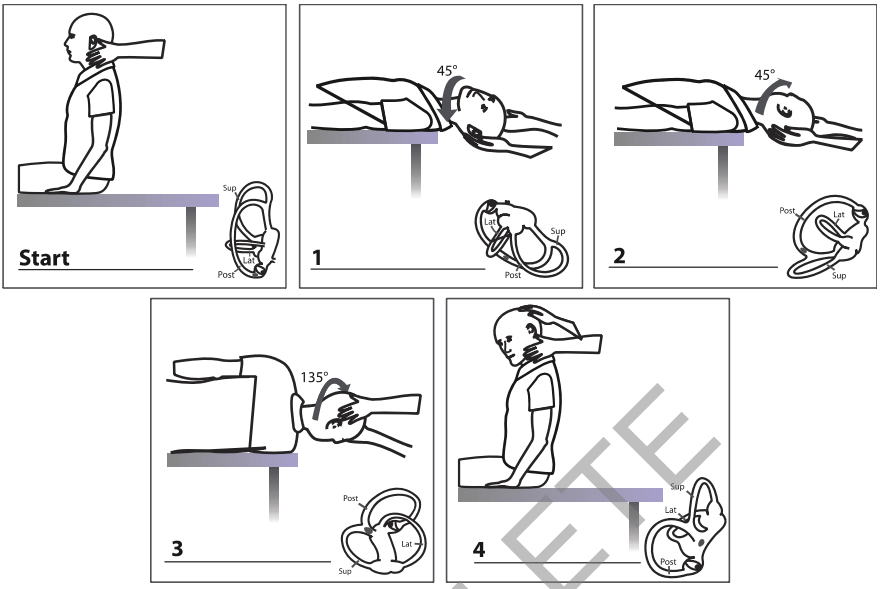


The patient sits with legs extended near the end of the examination table. The examiner turns the head 45 degrees to one side and lies the patient down so the head is below the table. For a positive test the patient must experience acute vertigo and have brisk torsional nystagmus which is anticlockwise 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 69). If no response, cease. If positive, repeat the Epley Canalith Repositioning Procedure once.

Special care is required for patients with back and neck problems.

Vestibular Neuritis (Neurolabyrinthitis)

Acute vertigo is the only symptom, due to sudden unilateral vestibular failure (probably viral) of the superior and/or inferior 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.

9.8.2 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.

9.8.3 Management of Vertigo

Benign Positional Vertigo

Repositioning treatment (see page 70). Absence of vertigo on positional test, non-response to treatment or non-typical nystagmus should alert to a possible central cause.

Symptomatic Relief of an Acute Episode

- Prochlorperazine IM or PO under top lip (3 mg buccal tab). Suppositories if continues for days. An alternative drug is cyclizine.
- Migraine: sumatriptan or non-steroidal anti-inflammatory.

Vertigo Prophylaxis

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

Investigation

This is guided by the clinical findings, but the three most relevant investigations are MRI (8th nerve pathology, infarction/ischaemia, demyelination, tumour), pure tone audiogram, and electrocochleography for Meniere's disease.

9.9 Anaphylaxis

Definition - a severe, life-threatening, generalized 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.

9.9.1 Immediate Management

Stop administration of precipitant if possible, assess reaction severity and treat accordingly.

- **ABC:**
  - High-flow oxygen - airway/ventilation support if needed.
  - Lie patient flat and elevate legs.
  - IV access - large bore.
- **ADRENALINE:**
  - **0.5 mL of 1:1000 IM (0.5 mg) to lateral thigh.**
  - Repeat every three to five minutes if inadequate response, immediate life threat or deteriorating.
  - If **hypotensive** give sodium chloride 0.9% bolus (20 mL/kg) under pressure (and repeat as necessary).
  - **Bronchospasm:** give nebulized salbutamol 5 mg.
  - **Stridor:** give nebulized adrenaline 5 mL of a 1:1000 solution (5 mg).
  - **IV** adrenaline is indicated if the situation is life threatening with circulatory collapse, and 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.
  - **Call ICU.**
  - Hydrocortisone 200 mg IV (onset of action 4-6 hours).

Notes:

- In **early anaphylaxis** (e.g., witnessed during desensitization therapy), 0.3 mg of adrenaline IM may be appropriate initial therapy; this dose is the same as 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.
- Antihistamines generally do not help with life threatening anaphylaxis. They may be considered for treatment of urticaria. Likewise H2 blockers (e.g., ranitidine) are useful in urticaria not anaphylaxis.

9.9.2 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).

- Self-injecting adrenaline devices (EpiPens and Anapens) can be bought over the counter from pharmacies without a prescription; prices vary ($125-$200). ACC will reimburse 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 these devices prophylactically. Needles, syringes, and ampoules are cheaper but may be difficult for patients to administer accurately.
- Adverse Drug Reactions (ADR) reporting guidelines (search for "ADR guidelines" on the CDHB intranet).
- Centre for Adverse Reactions Monitoring (CARM) (national pharmacovigilance) ADR reporting form.
- Consider MedicAlert.

9.9.3 Medium-term Management

- Referral to *Immunology Service* (see page 269):
  - 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) desensitization 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. False negative antibody tests (EAST/RAST) can also 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.

Brown et al. (2006). Anaphylaxis: Clinical concepts and research priorities. Emergency Medicine Australasia, Vol. 18, issue 2, 155-169

9.9.4 Immunology and Allergy: recommended referrals

Refer to *Immunology and Allergy: recommended referrals* on page 269.

9.10 Head Pain

9.10.1 Initial assessment and resuscitation

- Airways, Breathing, Circulation: brief appraisal.

9.10.2 Complete assessment

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

9.10.3 Definitive management

According to the cause. Possible causes include:

- Trauma - scalp, skull fracture, intracranial haematoma.
- 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.*

9.10.4 Important to exclude

- Subarachnoid haemorrhage
- Space occupying lesions
- Meningitis

9.11 Chest Pain

9.11.1 Initial assessment and resuscitation

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

9.11.2 Complete assessment

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

Acute Coronary Syndrome will often present with atypical features and cannot always be excluded on history alone.

Investigations usually include CXR and ECG but may require pulse oximetry/arterial blood gases and markers of myocardial damage. Myocardial injury markers, including troponins, are the most useful screening tests for an acute myocardial infarction.

Remember, cardiac markers are slow to rise after myocardial damage 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 repeat myocardial injury markers at 8 - 12 hours from the onset of symptoms. Other investigations that may be required include a CTPA or ventilation/perfusion scan, abdominal ultrasound, aortography, echocardiography.

9.11.3 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 (if there is chest pain and another area of pain outside the chest, think dissection, e.g., chest and abdomen, chest and limb, chest and head).
  - Oesophagus - inflammation, spasm, rupture
  - Abdominal - peptic ulceration, pancreatitis, cholecystitis etc.
  - Psychogenic.

9.11.4 Important to exclude

- Ischaemic heart disease
- Aortic dissection
- Pulmonary embolism
- Pneumothorax
- Pneumonia

9.12 Abdominal Pain

9.12.1 Initial assessment and resuscitation

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

9.12.2 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., myocardial infarction, 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 (any abdominal pain in males mandates a testicular exam).
  - Auscultation: bowel sounds, bruit.
  - Rectal examination (looking for masses, blood - not useful in assessing appendicitis).

Investigations

Guided by findings above - not all are routinely indicated.

- Urine dipstick for protein and blood. Urine microscopy and culture.
- Pregnancy test - either urine or blood.
- CBC + diff.
- Urea, creatinine, Na, K, Ca, glucose, amylase, bili, AST, ALT, GGT, ALP.
- Erect CXR if perforation is suspected. Supine/erect abdominal X-ray or CT scan if obstruction is suspected.
- Ultrasound - liver, biliary system, pancreas, kidneys, ureters, pelvis, aorta.
- CT abdomen.

Note: *Faecal loading on a plain AXR does* ***not*** *confirm constipation as the diagnosis.*

9.12.3 Definitive management

According to the cause. Possible causes:

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

9.12.4 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.

9.13 Shortness of Breath

9.13.1 Initial assessment and resuscitation

- Airways, Breathing, Circulation: may require supplemental oxygen.
- Supplemental oxygen therapy:
  - Titrate according to PaO_2 or pulse oximeter (aim for sat.O_2 >92% if pre-existing normal lungs).
  - 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 sat.O_2 of 90% in long standing COPD.

9.13.2 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). Refer to *Pulse Oximetry* (see page 256).
- Peak Expiratory Flow rate to assess asthma severity - tables of normal values can be found on the internet.
- Arterial blood gas to assess pH, PaO_2 and $PaCO_2$.
- CXR - particularly for pneumothorax (is difficult to exclude clinically), pneumonia and cardiac failure.
- Other investigations as indicated.

9.13.3 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.

9.14 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.

9.14.1 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").
  - Remove any wet/cold clothes.
- **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.
  - **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.

9.14.2 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, occult sepsis, the inactive, cerebrovascular disease, trauma, cardiovascular disease, diabetic ketoacidosis, hypoglycaemia etc.

Examination, Investigations: for traumatic injuries, underlying disease and complications of cold. Complications may include pulmonary oedema, bradycardia, hypotension and ventricular arrhythmias.

9.14.3 Definitive management

- Moderate/severe hypothermia is best managed in ICU.
- Treatment of the underlying disease and complications, including antibiotics if suspicious of sepsis.
- Hypothermia is 100% reversible, i.e., the patient has the potential to return to exactly the condition they were in prior to becoming cold.

9.15 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.

9.15.1 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.*

9.15.2 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.

9.15.3 Definitive management

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

10. Endocrinology / Diabetes / Metabolic Disorders

10.1 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 Belinda Schouten, 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/Schouten), ☎ 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

Within the hospital, details on request, logistics and interpretation of endocrine tests are available - search for "endocrinology" on the CDHB intranet.

HealthPathways also has succinct clinical information for medical staff, and HealthInfo has a number of patient information resources.

10.2 Diabetes Service Information

Main Office

- 550 Hagley Ave, ☎ 80860, fax 80171

Diabetes Physicians

- Dr Juliet Berkeley, Dr Tom Cawood, Dr David Cole, Dr Catherine Conway, Dr Chris Florkowski, Dr Helen Lunt, Dr Peter Moore, Professor Russell Scott, Dr Steven Soule

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, 8660 or 8414). Out of hours, contact the on-call Consultant through the Christchurch Hospital operator. For less urgent issues that can wait until normal 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

- Diabetes Nurse Specialist. Diabetes Centre, fax 80171, ☎ 80860
- Community Diabetes Nurse Specialist. Diabetes Centre, fax 80171, ☎ 80860
- District Nurse. Care Coordination Centre, fax 355 5225, ☎ 355 5066
- Outpatient Appointments, fax 80171
- Diabetes Specialist Podiatrist - for referral of patients with diabetic foot ulceration or Charcot foot, ☎ 80860, fax 80171

10.3 Adrenal Insufficiency

10.3.1 Causes

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

10.3.2 Clinical Features

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

10.3.3 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 ☎ 80934.
- **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 in-between values (450-950) a cortisol increment of <200 nmol/L after Synacthen suggests adrenal insufficiency and the need for supplemental steroids. Consult Endocrine team.

10.3.4 Treatment

- If hypovolaemic, especially if primary adrenal insufficiency, fluid replacement with sodium chloride 0.9% to restore blood pressure. May require 1 L or more over 2 hours. May require 5-25% glucose to raise glucose 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 (e.g., daily dose rapidly reduced to 50-75, 30-50 mg/day on successive days depending on metabolic status) then gradually down to a long term maintenance level of 15-20 mg PO 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 (e.g., more than 5-7.5 mg prednisone/day) for conditions other than cortisol deficient states, who are admitted with acute illness, sepsis etc., may require a doubling of the dose (e.g., 20 mg prednisone/day for 1-2 days) then reduce rapidly to normal maintenance doses. If unable to take oral steroids, consider parenteral hydrocortisone e.g., 50 mg q8h for 1-2 days and monitor electrolytes, mental status, blood pressure. Reduce steroid dose rapidly as clinical state allows to maintenance levels.
- All patients with adrenal insufficiency should have a steroid card, medic alert, and information sheet on management of acute illness.

10.3.5 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 microgram beclomethasone or >750 microgram 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.

10.4 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, proximal weakness and broad (>1 cm) purple striae 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 0800 hours 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.

10.5 Adrenal Incidentaloma

An adrenal incidentaloma is a mass lesion >1 cm diameter discovered by chance on ultrasound, CT, or MRI.

Consider non-contrast CT adrenals to give Hounsfield units (a measure of radiodensity). Refer to Adrenal Incidentaloma Clinic via Endocrinology, fax 81159, including copy of scan report, medical conditions and current medications.

10.6 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 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 milliunit/L usually have primary hypothyroidism requiring treatment with thyroxine. If the patient is elderly or has ischaemic heart disease, start thyroxine 25-50 microgram per day, increase by 25 microgram every 4-8 weeks; if young, start 100 microgram daily; repeat TFTs in 6 weeks. In the recovery phase after acute non-thyroidal illness, TSH may transiently show a slight elevation, usually <6 milliunit/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. Request T3 levels, thyroid antibodies, baseline FBC and LFTs. A radionuclide thyroid scan is helpful to distinguish thyroiditis from toxic nodular disease or Graves' disease. Consult Thyroid Physicians. Treat thyrotoxicosis with carbimazole 5-30 mg daily depending on severity; repeat TFTs in 4-6 weeks. Warn all patients about the rare but serious complication of agranulocytosis. Consider beta-blocker for symptom control. If radionuclide scan shows no uptake, stop carbimazole, treat as thyroiditis.

  Patients with suppressed TSH (<0.2 milliunit/L) and normal Free T4/T3 have sub-clinical thyrotoxicosis; recommend general practitioner repeats TFTs in 2 months.
- **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). Hypothyroidism is treated with cautious thyroxine replacement therapy. Thyrotoxicosis can be difficult to treat - consult Thyroid Physicians.
- **For further guidance on treatment of thyroid disorders, refer to HealthPathways.**

10.7 Diabetes - General Comments

- **Diabetes terminology** - The preferred terminology is Type 1 and Type 2 diabetes instead of insulin dependent and non-insulin dependent diabetes. Some patients may have other forms of diabetes such as Maturity Onset Diabetes of the Young (MODY).
- **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 hospitalized 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 (see below) 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 HbA1c. An HbA1c ≥50 mmol/mol is diagnostic of diabetes mellitus if confirmed on a second sample. A diagnosis of diabetes can also be made on two fasting results ≥7 mmol/L, or random glucose ≥11.1 mmol/L.
  - HbA1c levels above 41 mmol/mol are suspicious of diabetes and dictate further testing or monitoring.
- **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, recent pregnancy, etc.) or haemoglobin variants (e.g., thalassaemia). Typical target is 53 mmol/mol (7%), but this needs to be individualized.
  - If recent glycaemic control is uncertain, consider ordering an HbA1c, especially if inpatient review by a member of the diabetes team has been requested.

10.7.1 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 Humalog Mix25™, 60% in the morning and 40% at the evening meal, at a total dose of 0.3 units/kg per 24 hours. For example a 100 kg patient might be prescribed Humalog Mix25™, 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 subcut fast acting insulin such as lispro (Humalog™), glulisine (Apidra™) or aspart (NovoRapid™) insulin can be given in addition to the twice daily Humalog Mix25™. Subcut 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 subcut neutral insulin injections using Actrapid™ or Humulin R™.
- As a rule of thumb, in an insulin-sensitive person, 1 unit of aspart (NovoRapid™), 1 unit of glulisine (Apidra™), or 1 unit of lispro (Humalog™) will lower blood glucose by 3 mmol/L.
- Patients using 3 mL insulin cartridges can obtain pen injectors (e.g., Novopen™, Humapen™) from the Christchurch Hospital Pharmacy or from the Diabetes Centre during normal working hours.
- **Patients using subcutaneous insulin pump therapy:**
  - Patients who routinely use a continuous subcutaneous insulin pump may be admitted to the ward. If the admission is for hyperglycaemia, the insulin in the pump, the tubing and the cannula should be changed. A consult to the Diabetes Centre should be made to check the pump if there are any concerns about the pump or the infusion rates.
  - In the event of DKA, they can be managed as per the standard protocol with or without the subcutaneous pump continuing in the background.

Table 19 Description of Insulins currently available in New Zealand

Type of Insulin	Brand Names	Description of Action	Duration of Activity (1) (hours after injection)		Common outpatient use (NB: all insulins listed here can be used with a pen injector)
			Peak	Time to disappearance	
Aspart	NovoRapid™	Fast acting	**1.5**	**6**	TDS 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**	TDS half an hour before food in addition to a bedtime intermediate or long-acting insulin.
Premixed insulin (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	**24**	Take with food, usually with breakfast and with evening meal. Must prescribe clearly (do not confuse with Humalog).
NovoMix 30 (30% aspart 70% aspart protamine)	NovoMix® 30	Biphasic (Fast acting plus intermediate)	As for component insulins	**24**	Take with food, usually with breakfast and with evening meal.
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.

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

10.7.2 Patient autonomy

- Most patients on insulin are competent at diabetes self-care, including self-adjustment of insulin. Maintenance of this autonomy should be encouraged during hospitalization.

  Changes in insulin dose should therefore be made in consultation with the patient.

10.7.3 Ward capillary blood glucose testing

- Many patients require frequent testing when admitted acutely or during the perioperative period.
- Once the patient's condition has stabilized, four times a day testing is usually adequate (pre-meals and at bedtime).
- Patients on premixed insulins 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.

10.7.4 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 sodium can be calculated by adding 2.4 mmol/L for each 5.6 mmol/L rise in glucose above a level of 5.6 mmol/L. 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 normalization of the serum sodium.
- The most relevant measure of osmolarity in this setting is **effective osmolarity** (2xNa + glucose). See below for further explanation.

10.7.5 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.

10.7.6 Metformin - induced lactic acidosis

- Lactic acidosis is a rare but potentially fatal complication of metformin treatment.
- Metformin should be avoided or used in a reduced dose in patients who are at increased risk of lactic acidosis. This includes patients with renal impairment (serum creatinine >160 micromol/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.

10.7.7 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 stabilize, after discharge?
- Have you prescribed the right sort of insulin? Most patients use 3 mL cartridges - some patients use 10 mL vials or disposable pens.
- 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. Some general practitioners are very confident with insulin initiation and dose adjustment. Consider using Primary Care in suitable patients.

10.8 Diabetic Ketoacidosis (DKA)

10.8.1 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.

- Some patients with Type 1 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 state*** (see page 88). Seek advice.

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

10.8.2 Common Causes of DKA

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

10.8.3 Baseline Investigations

- Glucose.
- K, Na, Cl, 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.
- Venous pH and bicarbonate, rather than arterial pH, may be sufficient if patient has mild DKA only.
- CBC + diff.
- Cultures of blood and urine and any other material as indicated.
- CXR.
- ECG.

10.8.4 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/L) 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, sodium, potassium, 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

- The usual first choice of rehydrating fluid is sodium chloride 0.9%.
- 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 1 L sodium chloride 0.9% 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.
- Sodium chloride 0.45% is rarely needed. Many patients will experience a rise in sodium following correction of hyperglycaemia. In occasional patients, initial IV therapy may result in sodium continuing to rise above the normal range, however *effective osmolarity* (2x sodium + glucose; see ***Monitoring*** (see page 86) for further information) may be falling appropriately with treatment.
- When the blood glucose approaches 15 mmol/L, change to 4% glucose and sodium chloride 0.18%.
- **See below for potassium replacement**. Many patients can receive potassium replacement using ready mixed 30 mmol potassium chloride in 1 L sodium chloride 0.9% or 1 L 4% glucose and sodium chloride 0.18%. Some patients will however require further potassium added to sodium chloride 0.9%. For safety reasons, potassium replacement must be given via an infusion pump.

Insulin

- Add 50 units Actrapid™ to 50 mL sodium chloride 0.9% 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 sodium chloride 0.9% if glucose >15 mmol/L or 4% glucose and sodium chloride 0.18% if glucose is ≤15 mmol/L.
- Increase or decrease the insulin infusion rate according to the rate of fall of glucose.
- Aim to normalize glucose over >24 hours, no faster.
- When the glucose has fallen to around 20 mmol/L, slow down the rate of infusion of insulin (see the sliding scale below).
- When glucose has fallen to around 15 mmol/L, change IV fluid replacement to 4% glucose and sodium chloride 0.18% or 5% glucose.
- 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/L)	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 1 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 centralized 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, potassium replacement will be required within two hours of commencing insulin, or sooner if baseline potassium is low.
- The key to adequate potassium replacement is regular monitoring.
- Most patients can have pre-mixed 30 mmol/L potassium chloride in sodium chloride 0.9%. Occasionally, patients with severe total body potassium depletion will require greater concentrations.
- Discontinue potassium replacement once the patient is eating or potassium 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 subcut 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 hospitalization but at a reduced dose (70% usual dose).
- If the patient has newly diagnosed Type I diabetes, estimate the likely subcut insulin requirements from the previous 24 hours IV insulin requirement.
- All patients changing over to subcut insulin should be commenced on an insulin regimen which includes a long-acting component, for example **either** Humalog Mix25™ 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 subcut 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.

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

Additional Notes

- Do not strive for rapid correction of hyperglycaemia - the underlying principle is to avoid hypoglycaemia and correct salt and water loss.
- 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. Give 1 mmol sodium bicarbonate 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.

10.9 Hyperglycaemic Hyperosmolar Non Ketotic State

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%.

10.9.1 Investigations

As for *DKA* (see page 86) but include plasma osmolality.

10.9.2 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 sodium 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 sodium will be low, however serum sodium is often elevated secondary to dehydration.
  - Initial therapy is **1 L sodium chloride 0.9% over 30-60 minutes**.
  - Subsequent therapy will be dependent on the patient's clinical state but is likely to be **2-3 L sodium chloride 0.9%** or **sodium chloride 0.45% at 500 mL/hr**. Serum sodium may rise during treatment, however this is unlikely to be of major concern if effective osmolarity is falling (see *Treatment* on page 86 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% glucose in addition to sodium chloride 0.9% or sodium chloride 0.45% when the blood glucose is <15 mmol/L and sodium <150 mmol/L. If glucose is <15 mmol/L but sodium >150 mmol/L, change to 4% glucose and sodium chloride 0.18% or 5% glucose only.
  - Potassium replacement will probably not be needed initially, but, after a few hours rehydration, potassium may be needed at a rate averaging 10-20 mmol/hr. (Total body potassium deficiency will be less marked than in diabetic ketoacidosis). For safety reasons, potassium infusions of >30 mmol potassium chloride/L need to be given via an infusion pump.
- **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 subcut 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 122).

10.10 Peri-Procedural Management of Diabetes

Peri-procedure protocols are available on the wards (search for "C160011" or "C260110" on the CDHB intranet), but no single protocol will work for all patients, hence individualized treatment plans may be required, usually supervised by the Anaesthetist.

Note: *These protocols may not be suitable in some specialty areas such as Nephrology. Seek advice.*

If the C160011 and C260110 protocols are not suitable, here are some suggested regimens:

- **If patient has Type 1 diabetes**, uses glargine (Lantus) insulin at bedtime, and is undergoing a procedure likely to result in only 1 missed meal, 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 hourly pre- and post-operatively and every hour during surgery. The patient may need to commence a peri-procedure insulin infusion sliding scale if becoming hyperglycaemic (e.g., >11 mmol/L), at the discretion of the Anaesthetist. See table below.

  If on insulin but not on glargine, omit morning subcutaneous insulin. Start IV fluids and an insulin infusion sliding scale (see table below). Measure blood glucose 2 hourly pre- and post-operatively and every hour during surgery.
- **If patient has Type 2 diabetes**, and is on oral hypoglycaemic medications, omit these. Restart when eating for at least 12 hours.

  If the patient is on insulin (± oral hypoglycaemic medications), omit morning insulin and oral hypoglycaemic medications on the day of surgery. Measure blood glucose 2 hourly pre- and post-operatively and every hour during surgery. Only start an insulin infusion if the patient's glucose level rises above 12 mmol/L.

Table 21 Peri-Procedure Insulin Infusion Sliding Scale

IV fluid guidelines: 1000 mL bag sodium chloride 0.18% and glucose 4% with 30 mmol potassium chloride IV to run at 83 mL/hour (12 hourly) reviewed daily. Consider using Plasma-Lyte 148™ and 5% Glucose with prolonged infusion use.

Prescription: 50 units Actrapid™ insulin made up to 50 mL with sodium chloride 0.9% IV to run according to blood glucose sliding scale below.

Blood glucose (mmol/L)	Insulin infusion rate
<3.5	Stop insulin infusion. Recheck the blood glucose after 15 minutes. If patient has clinical signs of hypoglycaemia (confused, semi- or unconscious) administer 50 mL of 50% glucose IV. Recheck glucose at 10 - 15 minute intervals. Recommence sliding scale insulin once blood glucose ≥4 mmol/L.
4	0.5 mL/hr
4.1 - 7	1 mL/hr
7.1 - 9	2 mL/hr
9.1 - 11	3 mL/hr
11.1 - 14	4 mL/hr
14.1 - 17	5 mL/hr. If persistently at this rate for 6 hours, for medical review.
17.1 - 20	6 mL/hr. If persistently at this rate for 6 hours, for medical review.
>20	For medical review. Consider DKA.

- If there is any delay in the surgery or the patient does not resume normal intake promptly post-operatively, hyponatraemia may develop if this protocol is continued. Therefore sodium, potassium, and creatinine will need to be closely monitored, at least 12 hourly. Seek advice under these circumstances from the Diabetes Service.

10.11 Management of the Newly Diagnosed Patient with Type Two 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 micromol/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.

10.12 Hypoglycaemia

10.12.1 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.

10.12.2 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 glucose. If venous glucose confirms hypoglycaemia (<3 mmol/L), consult the Endocrine team.

10.12.3 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 glucose (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% glucose 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.

10.13 Hypernatraemia

Hypernatraemia (serum sodium>145 mmol/L) is due to a deficiency of water relative to solute (sodium) in the extracellular fluid and always represents a hyperosmolar state. Thirst and release of antidiuretic hormone 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 sodium:

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

10.13.1 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. Hypernatraemia is only sustained if there is impaired thirst or lack of access to water.
- **Depletion of hypotonic fluid** i.e. loss of relatively more water than Na.
  - Renal loss:
    - Loop diuretic, osmotic diuresis (glucose, urea, mannitol).
    - Post-obstructive diuresis, polyuric phase AKI (acute kidney injury).
    - Intrinsic renal disease.
  - GI tract loss:
    - Vomiting, NG drainage, diarrhoea, laxatives.
  - Skin loss:
    - Burns, excessive sweating.
- **Solute excess** (uncommon)
  - Sodium: ingestion of sodium chloride, sea water, sodium bicarbonate infusion
  - Hyperalimentation: IV or parenteral nutrition

10.13.2 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.
- Calcium. Calculate osmolarity **[**(2 x Na) + urea + glucose**]** (all mmol/L).
- Urine osmolality, sodium 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.

10.13.3 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% glucose - the lower the osmolality of the fluid, the lower the volume required for correction.
- Avoid sodium chloride 0.9% unless frank circulatory collapse.
- Remember to allow for ongoing fluid losses both incidental and obligatory.
- Use the following approach to estimate the **amount of excess water required** to return the serum sodium to 140 mmol/L in 60 kg patient with serum sodium 168 mmol/L:
  - 3 mL of electrolyte-free fluid/kg of lean body weight will lower serum sodium by 1 mmol/L.
  - thus in a 60 kg patient 180 mL electrolyte free fluid (3 mL/kg x 60 kg) will lower serum sodium by 1 mmol/L.

  if initial serum sodium is 168 mmol/L and target sodium is 140 mmol/L the electrolyte free fluid requirement is therefore 5 L or [180 mL x (168-140)]
- As a guide to the **rate of fluid infusion**, use the following approach:
  - Assume serum sodium will decrease by 1 mmol/L if 3 mL/kg infused per hour.
  - If desired decrease in serum sodium is 10 mmol/24hr, then initial rate of free water administration should be approximately 75 mL/hr, derived as follows:

    (3 mL/kg/hr x 60 kg x 10 mmol/L/day/24h per day)
  - To account for 40 mL/hr of obligate water loss in sweat and stool and 50 mL/hr of ongoing urinary free water loss, the total water prescription is 165 mL/hr (75+40+50).
- These are guidelines and frequent clinical and biochemical reviews are essential in patients with severe and symptomatic hypernatraemia; repeat sodium after 4-6 hours initially.
- For acute cranial diabetes insipidus (CDI) desmopressin (synthetic AVP) should be given parenterally in a dose of 1-4 microgram 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 microgram daily by intranasal spray.

10.14 Hyponatraemia

10.14.1 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 hyponatraemia is chronic.

10.14.2 Causes

- These are many and varied.
- Remember to consider factitious causes:
  - Laboratory error - check *anion gap* (see page 97) and calculate osmolarity **[**(2 x Na) + urea + glucose**]** (all mmol/L).
  - Hypertonic hyponatraemia is commonly seen with hyperglycaemia and reflects *osmotic shifts of water from the intracellular to extracellular space* (see page 85).
  - Drip arm specimen.
  - Pseudohyponatraemia (hyperlipidaemia or hyperproteinaemia). You may need to get a direct reading of sodium (contact Biochemistry).

10.14.3 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, diarrhoea, recent use of thiazide diuretic).

10.14.4 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.

Notes:

- *Hyponatraemia with raised plasma osmolality occurs in hyperglycaemia* (see page 85).
- Congestive heart failure and cirrhosis 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.

10.14.5 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 sodium (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 sodium <130 mmol/L.
- If **volume deficient** give sodium chloride 0.9% IV provided congestive heart failure/cirrhosis excluded.
- If **not volume deficient** (e.g., SIADH), main treatment is water restriction (500-1000 mL/day; allow water intake equal to urine output). Ensure adequate sodium and potassium intake (IV saline may be needed especially if plasma sodium <120 mmol/L). Oral sodium supplements may be useful (Slow Sodium™ - 10 mmol sodium per tablet, 4-6 tablets daily) - consult Endocrinology.
- In all cases aim to restore plasma sodium 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 3% saline. Infuse 1-2 mL/kg body weight/hour and check plasma sodium every 2-4 hours to guide therapy. Consult Endocrinology before use.
- Investigate and treat underlying cause.

10.15 Hypercalcaemia

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

10.15.1 Causes

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

10.15.2 Symptoms

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

10.15.3 Investigations

- In all cases of uncertain aetiology, request Na, K, Ca, Mg, PO_4, ALP, alb, creatinine, and PTH level - **before** giving hypocalcaemic drugs.
- **If PTH level is suppressed**, consider the tests listed below and Endocrinology consult, depending on the clinical context:
  - 25-hydroxy vitamin D.
  - CXR (lung cancer, lymphoma, 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 dihydroxy vitamin D and parathyroid hormone related peptide assay may be helpful, the latter if malignancy suspected. Recommend consultation with Endocrinology who can authorize these infrequently performed tests.

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

Table 22 Calcium Correction Formula

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

10.15.4 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, 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 L in 24 hours orally and IV. Monitor closely to avoid fluid overload. Start with 1-2 L sodium chloride 0.9% over 2 hours then 1 L sodium chloride 0.9% 6-8 hourly and reassess at regular intervals. Potassium supplements 10-20 mmol potassium chloride per 500 mL may be required, but use premix bags of potassium chloride 30 mmol/L in sodium chloride 0.9% if possible.
- Bisphosphonates
  - Zoledronic acid 4 mg in 100 mL sodium chloride 0.9% IV over 15 minutes, provided eGFR is >35 mL/min, **or** pamidronate 90 mg in 0.5 L sodium chloride 0.9% IV over 2 hours.
    - Ensure no extravasation occurs (irritant to tissues). Fever and aching may occur for 2-3 days and can be lessened with regular paracetamol.
    - Plasma calcium falls progressively with nadir at 3-5 days.
    - Ongoing monitoring of calcium is necessary as hypercalcaemia is likely to recur if the underlying cause is not identified and treated.
- 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. Consider if calcium >2.9 mmol/L, renal calculi, renal impairment, osteoporosis, or age <50 years. Consult Endocrinology.

10.16 Hypocalcaemia

Check albumin and if necessary adjust the calcium level (see above). If corrected calcium is <2 mmol/L investigation is needed - provided chronic renal failure is not present. Symptoms may not be prominent if problem is long standing. Check Chvostek and Trousseau signs, and history of fits, tetany, cataracts and previous thyroid surgery.

10.16.1 Causes

- Hypoparathyroidism or resistance to parathyroid hormone.
- Renal failure.
- Vitamin D deficiency.
- Low magnesium states; may be caused by proton pump inhibitors, alcohol, diarrhoea etc.
- Pancreatitis, rhabdomyolysis.

10.16.2 Investigations

- Plasma Ca, PO_4, creatinine, Mg, ALP, alb, 25-hydroxy 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.

10.16.3 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 5% glucose 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., calcium carbonate 1.25 g BD (500 mg calcium per tablet) or calcium carbonate effervescent (1000 mg calcium) 1-2 daily.
  - Start 1,25 dihydroxy vitamin D (calcitriol) e.g., 0.25 microgram - 1 microgram/day
  - Monitor serum calcium 12-24 hourly, aiming for corrected calcium 2 - 2.2 mmol/L. Consult Endocrinology.
  - Magnesium deficiency:
    - If severe (magnesium <0.5 mmol/L) and symptomatic, e.g., arrhythmia or tetany, consider 50 mmol magnesium IV slowly over 8-24 hours.
    - Otherwise, oral magnesium supplements 10-20 mmol/day (magnesium chelate 500 mg capsule containing 100 mg elemental magnesium (4 mmol magnesium/capsule); Mylanta suspension (6.9 mmol magnesium/10 mL)).

10.17 Hypertriglyceridaemia

- Levels >10 mmol/L require immediate medical attention : the major risk is pancreatitis.
- Triglyceride levels may be as high as 50-100 mmol/L. The serum is typically lipaemic and examination of the retinal vessels reveals lipaemia retinalis. Patients may present with eruptive xanthomata.

10.17.1 Causes

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

10.17.2 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 normalize glucose.

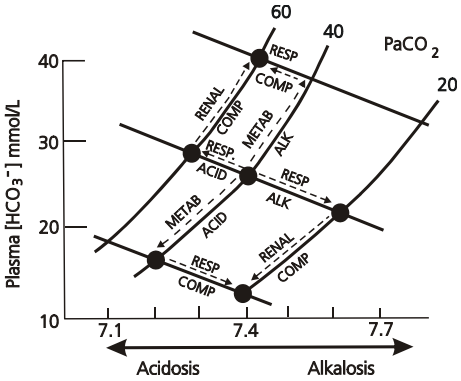
10.17.3 Follow-Up

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

10.18 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.

10.18.1 Causes of Metabolic Acidosis

Calculate Anion Gap

Table 23 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.

Investigations

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

Treatment

- **Treat underlying cause.**
- Recent evidence suggests that in most forms of metabolic acidosis, the use of sodium bicarbonate offers no benefit and may even be harmful.
- If sodium bicarbonate is used give 1 mmol/kg and review pH in one hour. Do not overcorrect, aim for pH of 7.1.
- Specific indications for sodium bicarbonate include methanol and tricyclic antidepressant poisoning - maintaining a normal pH probably reduces toxicity.

10.18.2 Causes of Metabolic Alkalosis

- Volume deficit (sodium conservation is coupled to bicarbonate reabsorption and therefore metabolic alkalosis is sustained).
  - Vomiting, gastric suction.
  - Diuretics (not acetazolamide, potassium-sparing).
- Mineralocorticoid excess:
  - Cushing's Syndrome.
  - Primary hyperaldosteronism.
  - Bartter's syndrome (decreased sodium chloride absorption in kidney leading to increased renin/aldosterone).
- Severe potassium depletion.
- Milk-alkali syndrome - chronic excess soluble calcium salts plus alkali cause a nephropathy which impairs bicarbonate 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.

Investigations

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

Treatment

- Treat the underlying cause.

11. 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 chloride, hydrogen and potassium
- 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:

Fluid	Electrolyte Content
Sodium chloride 0.9%	150 mmol/L Na + Cl
Sodium chloride 0.9% with 30 mmol/L potassium chloride	150 mmol/L Na + Cl + 30 mmol/L K
Sodium chloride 0.45%	75 mmol/L Na + Cl
Glucose 4% sodium chloride 0.18%	30 mmol/L Na + Cl
Compound sodium lactate	131 mmol/L Na + 112 mmol/L Cl + 5 mmol/L K
Plasma-Lyte 148™ either with water or glucose 5%	140 mmol/L Na + 98 mmol/L Cl + 5 mmol/L K
Glucose 4% sodium chloride 0.18% with 30 mmol/L potassium chloride	30 mmol/L Na + Cl + 30 mmol/L K
Glucose 5%	
Glucose 5% with 20 mmol/L potassium chloride	20 mmol/L K

***Note**: Plasma-Lyte™ fluid preparations are not routinely used.*

2) Colloid: gelatin succinylated 4%.
3) Blood Products:
   - Red cells.
   - Fresh frozen plasma (FFP).
   - Albumin 4%.

General rules for IV fluids

The elderly and those with renal or cardiac dysfunction have difficulty excreting salt (sodium). It is important 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: 1.5 to 3 times the amount of blood lost.
   - Large volumes of sodium chloride 0.9% can cause hyperchloraemic metabolic acidosis, therefore consider changing to compound sodium lactate or Plasma-Lyte 148™.
   - Short half-life (note that only 20% remains in the intravascular space after 2 hours).
2) Colloid: gelatin succinylated 4%.
   - Colloids to be used with caution in septic and anaphylactic/cardiogenic shock but use should be discussed with more senior medical staff in the first instance.
   - Albumin 4% may be used following discussion with senior medical staff.
3) Blood.

   The only fluid available that will carry oxygen! Indicated if the patient is anaemic or haemodynamically unstable as a result of blood loss or in uncontrolled bleeding.
   - Best to use fully typed and cross-matched blood (6 mL EDTA tube; crossmatch takes 30 min if no antibodies found). Group specific uncross-matched blood takes 10 min. Refer to *Blood Transfusion Practice* (see page 22).
   - 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 Blood Bank ☎ 80310 to activate the *Massive Transfusion Protocol* (see page 25).

Maintenance fluids

- IV or subcutaneous fluids if unable to manage with oral or NG fluids.
- Sodium chloride 0.9% or glucose 5%, or glucose 4% and sodium chloride 0.18%, depending on cardiac and renal function and plasma sodium concentration. Avoid IV glucose solutions for patients at risk of refeeding syndrome.
- Daily weighing gives accurate assessment of fluid balance.
- Sodium chloride 0.9% or glucose 5% at 60 to 80 mL/hour. Glucose 4% and sodium chloride 0.18% may also be used. Modifications if:
  - Normal kidney function: use sodium chloride 0.9% 30 mmol/L potassium chloride premix at 60 to 80 mL/hour. No potassium if acute renal failure or potassium >5 mmol/L.
  - Fluid overload: cease all maintenance fluids.
  - Hypernatraemia; sodium >150 mmol/L: give glucose 5% and aim to reduce the sodium level by 8 to 12 mmol/L per 24 hours.
- Prevent or correct any electrolyte imbalance.

12. Gastroenterology

12.1 Gastroenterology Department Information

Main Office Investigative Unit

- 2nd Floor, Riverside, general enquiries and Endoscopy unit ☎ 81991, fax 80419

Inpatient Services

Ward 26, ☎ 89260

- Prof Murray Barclay, Dr Michael Burt, Dr Teresa Chalmers-Watson, Dr Bruce Chapman, Dr Steven Ding, Dr James Falvey, Assoc Prof Richard Gearry, Dr Gary Lim, 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, oesophageal food bolus obstruction (urgent referral), colonoscopy and 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, endoscopic ultrasound, Fibroscan.

12.2 Haematemesis

12.2.1 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.

12.2.2 Management

See also: Upper Gastrointestinal Bleeding Pathway in the Emergency Department (search for "C240071" on the CDHB intranet).

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 65):
  - History often unreliable.
  - Useful signs include:
    - Resting tachycardia.
    - Hypotension.
    - Postural BP drop >15 mm Hg.
- Stabilize patient and monitor:
  - Give sodium chloride 0.9% IV, then blood when available.
  - Use Group O Rh negative blood in an emergency. See *Collection of Blood from Blood Bank* on page 22.
- Initial investigations:
  - Crossmatch 6 units of resuspended red cells.
  - CBC + diff.
  - Coagulation profile.
  - Na, K, creatinine, urea, LFTs.

Urgent surgical consultation if:

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

Gastroenterology consultation

- The prognosis is relatively favourable if the age is <60 with no shock or co-morbidities and the likely cause is a Mallory-Weiss tear. The outlook is poor for older patients >80, with haemodynamic instability and multiple comorbidities.
- Urgent consultation is generally indicated, especially if there are poor prognostic features.
- 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.

12.2.3 Therapy

Varices

- Consult Gastroenterologist.
- Terlipressin 1-2 mg IV bolus stat, then 1 mg q4h as an IV bolus. Use with caution if known ischaemic heart disease or other vascular disease due to vasoconstrictor effect.
- Octreotide is an alternative. Commence an IV infusion of octreotide using a 50 microgram bolus followed by a continuous infusion (25 - 50 microgram/hour) for up to 72 hours.
- Administer prophylactic antibiotics, e.g., cefotaxime, ceftriaxone.
- Urgent variceal ligation or occasionally sclerotherapy.
- Sengstaken-Blakemore or Linton tube and transfer to ICU if bleeding not controlled by endoscopy. (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:
- **Regimen:**
  - Bolus omeprazole IV injection: 80 mg stat loading dose

    Followed immediately by:
  - Continuous omeprazole IV infusion: 8 mg/hour for 72 hours.
  - Oral omeprazole 20 mg once daily should be commenced at the end of the 72-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**.
  - Oral omeprazole 20 mg once daily should be commenced at the end of the IV treatment period.
- **Administration:**

  Bolus omeprazole IV injection
  - When administering the **bolus** injection, it is important to use the **IV injection product**, NOT the IV infusion product. These two formulations are different and are not interchangeable due to stability concerns.
  - Reconstitute each 40 mg vial with the diluent provided, according to the guidelines in the package insert, i.e., 4 mg/mL.
  - 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 (5 minutes for 80 mg).

  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 a 40 mg vial of omeprazole infusion, add to an infusion bag according to the guidelines in the package insert (Dr Reddy's is 100 mL glucose 5%) and infuse over 5 hours (8 mg/hr).
  - Repeat for a total continuous infusion of 72 hours (unless stopped after a diagnosis is made at endoscopy).

- **Use of bolus dose intravenous omeprazole in other clinical situations**

  Intravenous omeprazole is **only** indicated if patients are unable to take oral or enteral formulations (i.e., difficulty swallowing, vomiting, non-functioning gut). In these cases patients should receive IV bolus omeprazole in a dose equivalent to what they would be expected to receive orally.

Helicobacter pylori

- **Eradication therapy** for **Helicobacter pylori** when this has been identified. Give oral treatment with omeprazole 20 mg BD + amoxicillin 1 g BD + clarithromycin 500 mg BD for 7 days. If penicillin allergy, substitute metronidazole 400 mg BD for amoxicillin.

Other regimens are available for treatment failures. Consult Gastroenterology.

12.3 Vomiting

12.3.1 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.

12.3.2 Complications

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

12.3.3 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 patients with parkinsonism.
- Phenothiazines:
  - Prochlorperazine 5 - 10 mg TDS PO, IM, PR. (Tabs 5 mg, buccal 3 mg, injection 12.5 mg, PR 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 in the Oncology section** *(see page 205). For post-operative vomiting, refer to* **Management of Adult Post-operative Nausea and Vomiting (PONV)** *(see page 17).*

12.4 Acute Diarrhoea (<2 weeks)

12.4.1 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.

12.4.2 Examination

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

12.4.3 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**: Request cultures for Salmonella, Shigella, Yersinia, Aeromonas, Campylobacter and Plesiomonas. (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.

12.4.4 Management

- Enteric isolation procedures required if infection suspected (contact Infection Control).
- IV fluids may be required. Remember faecal losses of electrolytes may be very high. 100-120 mmol sodium and 5-15 mmol potassium may be lost per litre of stool. An adult may lose more than 2-3 L of fluid per day.
- Avoid constipating drugs (especially in children) as these may prolong symptoms.
- Antimicrobials are not indicated for the majority of infective diarrhoeas.
- **Specific infections:**
  - Salmonella/Shigella/Campylobacter are usually self-limiting and antibiotics should only be used when illness is severe with systemic upset/septicaemia. These are ***notifiable diseases*** (see page 284).
  - 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 152).
  - 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 inhibitors, etc., are at increased risk of infection.
- Patients hospitalized with active inflammatory bowel disease should receive thromboprophylaxis with LMWH unless contraindicated. Discuss with Consultant if uncertain.

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.*

12.5 Constipation

12.5.1 General Measures

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

12.5.2 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 Picosalax 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.

12.6 Jaundice

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

12.6.1 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 intrahepatic cholestasis. Check drug history, especially antibiotic use, NSAIDs and antipsychotics.

12.6.2 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.

12.7 Acute Hepatocellular Dysfunction (Hepatitis)

12.7.1 History

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

12.7.2 Investigations

- USS of the liver and biliary tract.
- INR, and Echis ratio if INR prolonged.
- Tissue auto antibodies, ANA, serum protein electrophoresis, caeruloplasmin.
- Hepatitis A, B, and C serology. If acute Hepatitis B is possible, check Hepatitis B IgM core antibody.
- EBV & CMV serology.

12.8 Liver Failure

Where this is suspected commence treatment early.

12.8.1 Clinical and Biochemical Features

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

12.8.2 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 *ascites* on page 107), consider cefotaxime 1 g 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.)

12.8.3 Investigations

- Arterial lactate and ABG in acute liver failure.
- Na, K, urea, creatinine (hepatorenal syndrome).
- Glucose (may require IV glucose infusion).
- Alb, bili, ALP, 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), smooth muscle/antinuclear antibodies, immunoglobulins.
- Blood cultures.

12.8.4 Treatment

- Acute liver failure, commence N-acetylcysteine infusion.
- Treat any underlying cause (e.g., bleeding varices, sepsis).
- Stop all offending drugs. Avoid benzodiazepines.
- Correct hypokalaemia, hypotension, hypoglycaemia.
- If *ascites present* (see below), aspirate for diagnostic purposes.
- Correct coagulation defects with vitamin K 10 mg IV slowly; do not give fresh frozen plasma unless actively bleeding (requires Consultant approval).

12.8.5 If encephalopathy suspected

- Avoid all benzodiazepines and sedatives.
- Purge with lactulose 10-30 mL TDS adjusted to produce three loose stools per day. Enemas can also be used.
- Watch for *alcohol withdrawal* (see page 11).
- Neomycin or rifaximin may be indicated.
- Consult Gastroenterologist promptly.

12.9 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.

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 a minimum of 10 g albumin for every litre of ascitic fluid removed.

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.

12.10 Acute Pancreatitis

See also the Acute Pancreatitis Pathway which is in the Surgical Wards and the Emergency Department (search for "C240280" on the CDHB intranet).

12.10.1 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.*

12.10.2 Diagnosis

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

12.10.3 Aetiology

- Gallstones.
- Alcohol.
- Idiopathic.
- Drugs.
- Types I and V hyperlipidaemia.

12.10.4 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, ALP, AST, ALT, GGT, CRP.
- Blood cultures.
- Abdominal ultrasound.
- Arterial blood gases.
- Lipid analysis for types I and V hyperlipidaemia only if no other aetiology found.
- CXR.

12.10.5 Management

- Treatment of *shock* (see page 65).
- Pain relief - early consideration of patient-controlled anaesthesia (PCA). Contact the Acute Pain Management Service or the Duty or on-call Anaesthetist.
- Patients should eat and drink as tolerated.
- Oxygen therapy- serial blood gases (ARDS, acidosis).
- Correct electrolytes and calcium disturbances.
- Antibiotics - only if co-existing cholangitis.
- Surgical consult.
- Consider **urgent ERCP** if (severe) coexisting cholangitis. 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 units/L	PaO_2 <60 mm Hg
AST >250 units/L	Fluid retention >6 L

12.11 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.

Notes:

- 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 epithelialized. Place a Foley urinary catheter to maintain the tract which starts to close within 1-2 hours. Seek advice as above.

13. General Medicine Services

13.1 General Medicine Department Information

Main Office

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

Inpatient Services

- Christchurch Hospital - Ward 23, Acute Medical Admitting Unit (Ward 24), and Ward 26

Clinical Director:

- Dr David Jardine, ☎ 86010

Service Manager:

- Dave Nicholl, ☎ 86218

Nursing Director:

- Mark Crawford, ☎ 88996

Secretaries:

- Mary Simes, ☎ 80155
- Susan Crysell ☎ 86010
- Annetta Walker, ☎ 86005

Physicians:

- Juliet Berkeley, Mark Birch, Tom Cawood, David Cole, John Elliot, Valerie Fletcher, Ben Franks, Leighanne Hughes, Dave Jardine, Libby King, David MacGregor, Sarah Metcalf, Nigel Millar, Peter Moore, Alan Pithie, Anne Roche, Russell Scott, Andrew Sidwell, Steven Soule, Anthony Spencer, Chris Warren.

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 12,000 patients per year. There is also an AMAU Team and an Acute Stroke Unit.

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 Birch, Fletcher, Hughes, Jardine and Spencer.

Rapid response clinics (Senior Registrar) 4 days per week.

Department Guidelines

Refer to the General Medicine "Green Book" (search for "green book" on the CDHB intranet).

13.2 Hypertension

13.2.1 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.

13.2.2 Aetiology of secondary hypertension

- **Renal:** Acute or chronic disease, renovascular disease and volume overload (especially dialysis patients).
- **Endocrine:** Cushing's syndrome, phaeochromocytoma, hyperaldosteronism, hyperthyroidism, acromegaly.
- **Neurological:** Raised intracranial pressure.
- **Diabetes Mellitus:** Both Type I and II patients are commonly hypertensive.
- **Coarctation of the Aorta**.
- **Respiratory:** Obstructive sleep apnoea.
- **Drugs:** NSAIDs, steroids, sympathomimetics including non-prescription drugs, alcohol, liquorice, cocaine, erythropoietin, cyclosporin. Clonidine withdrawal.
- **Obesity**.

13.2.3 Investigations

- Blood pressure measurement: Systolic BP is more accurately measured than diastolic and correlates more reliably with end-organ damage. Measure lying and standing BP. Blood pressure is variable and so several measurements should be made over a period of days or weeks before deciding on long term treatment. Elderly patients with postural hypotension should not be treated. Consider not only the magnitude of systolic BP but also end-organ damage, cardiovascular risk, and side-effects of treatment. Whitecoat hypertension in hospital patients is very common and if there is uncertainty consider a 24 hour ambulatory BP.
- CBC and blood film for microangiopathic changes.
- ECG and CXR.
- Urinalysis (diptest for proteinuria/haematuria, microscopy for cells and casts).
- Na, K, Cl, creatinine, glucose, TFTs.
- Other tests for secondary causes (e.g., if patient <40 years, has resistant hypertension, or has clinical features that suggest a secondary cause):
  - Phaeochromocytoma: obtain 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).
  - Hyperaldosteronism: aldosterone-renin ratio. ACE-inhibitors, AT2 blockers and thiazide diuretics can falsely lower the aldosterone-renin ratio.
  - Cushing's syndrome: 24 hour urine cortisol or low dose overnight dexamethasone suppression test.
  - Renal disease: renal ultrasound for renal size and calcification.
  - Renal artery stenosis: renal MRA or CT angiogram. Vascular intervention is generally only appropriate in younger patients with fibromuscular dysplasia.

13.2.4 Management of Acute Hypertensive Crisis

Monitor blood pressure frequently:

- The excessive use of powerful IV antihypertensive 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 sodium chloride 0.9% 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 100 mg in 500 mL 5% glucose, starting at 0.5 microgram/kg/min and titrating against BP. Only given in the CCU or ICU.

Note:

- Do not treat acute cerebrovascular accidents with IV therapy - oral therapy is best as this will result in a slower reduction in blood pressure and preserve cerebral autoregulation. Refer to ***Acute Management of Ischaemic Stroke*** on page 170.
- 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.

14. General Surgery

14.1 Department of General Surgery Information

Main Office

- 1st Floor, Hagley Outpatients
- General enquiries ☎ 81582, fax 80352

Inpatient Services

- Ward 15 ☎ 89150
- Ward 16 and Surgical Assessment and Review Area (SARA) ☎ 89160
- Ward 17 ☎ 89172
- Mr Saxon Connor, Mr Grant Coulter, Ms Birgit Dijkstra, Mr Tim Eglinton, Mr Richard Flint, Prof Frank Frizelle, Mr John Frye, Mr Steve Kelly, Miss Philippa Mercer, Mr Ross Roberts, Mr Gregory Robertson, Mr Robert Robertson, Mr Richard Tapper, Ms Josie Todd, Mr Chris Wakeman, Mr Malcolm Ward.
- Breast Nurses, pager 8898 ☎ 81804
- Colorectal Nurse, pager 8095 ☎ 81687

Consultation and On-Call Service

24 hours a day, seven days per week.

For urgent referrals/consults please contact the Acute Surgical Registrar on pager 8600 or the Acute Surgeon of the day via the hospital operator.

Outpatient Clinics

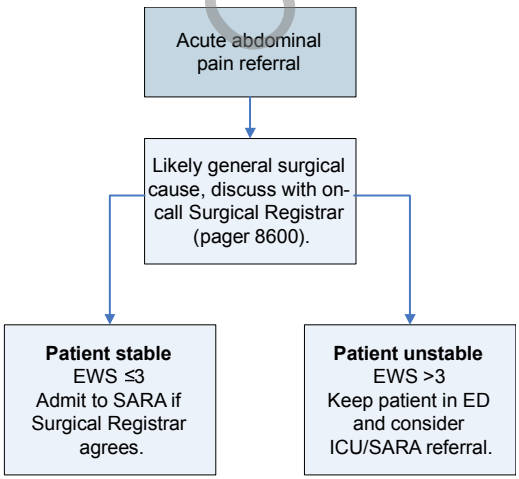
Fax referrals to 80952.

14.2 Acute Abdominal Pain

- See also Common Emergency Presentations - *Abdominal Pain* (see page 74).
- If the abdominal pain is likely to have a surgical cause, discuss the patient with the on-call Surgical Registrar (pager 8600).

14.2.1 Initial Assessment of Abdominal Pain

This will reflect the general clinical situation and the severity and stability of the patient's condition.



Subsequent management depends on the results of further assessments.

If unstable - resuscitate (see page 65)

- Get as much history and examination as is practicable.
- IV access (no smaller than 18 gauge), IV fluids.
- Analgesia.
- ABG.
- Urine output, indwelling urinary catheter.
- Consider CXR, abdominal X-ray, ECG, cardiac injury markers, CBC + diff, group and hold or cross-match. Na, K, creatinine, LFTs, amylase.

  Note: *Generalized peritonitis may proceed directly to theatre.*

  Note: *Inform the on-call Consultant of all acute cases going to theatre.*
- **When stable, or if presents in a stable condition, transfer to SARA if Surgical Registrar agrees**.

14.2.2 Further Assessment of Abdominal Pain

- Obtain a full history and examination.
- Consider the following investigations:
  - CBC, urea, Na, K, creatinine, CRP.
  - Coagulation screen (bleeding disorder, anticoagulants, jaundice).
  - MSU (dipstick, microscopy, culture and sensitivities).
  - Cardiac injury markers (chest/upper abdominal pain).
  - ECG (chest/upper abdominal pain).
  - Group and hold, cross-match (bleed, signs of anaemia, recent major surgery).
  - Imaging:
    - CXR (perforation, pneumonia).
    - Supine abdominal X-ray (bowel obstruction, renal colic).

    Discuss further imaging investigations (CT, USS) with Registrar/Consultant.
  - LFTs (upper abdominal pain, biliary history).
  - Amylase, lipase (pancreatitis).
  - Beta-HCG (pre-menopausal women).
  - ABG (ischaemic bowel, pancreatitis, unwell).
  - Lactate (ischaemic bowel).

14.2.3 Management of Acute Abdominal Pain

- If a definite diagnosis is established after the initial assessment, follow the appropriate pathway - *Acute Appendicitis* (see page 115), *Acute Cholecystitis* (see below), *Acute Pancreatitis* (see page 107), *Small Bowel Obstruction* (see page 118), *Suspected Diverticulitis* (see page 117).
- If none of these diagnoses is likely, then further investigation and treatment as directed by the General Surgical team.
- Appropriate referrals for other diagnoses, i.e., Cardiology, Respiratory Services etc. as indicated.

14.3 Acute Cholecystitis

14.3.1 Causes, History and Examination

- The most frequent cause is bacterial infection secondary to gallstones. It may also occur in malnutrition including anorexia nervosa.
- A history of biliary colic may be obtained, although an attack of acute cholecystitis may occur without prior biliary tract disease.
- Pain and tenderness in the right hypochondrium. Pain may be referred to the right shoulder. Fever and jaundice may be present.

14.3.2 Investigations

- CBC + diff, INR, APTT, Na, K, creatinine, LFTs, amylase or lipase, CRP.
- Upper abdominal USS.

Then review.

14.3.3 Management

- If no cholecystitis, consider other causes and investigate.

If cholecystitis confirmed:

- IV antibiotics: IV amoxicillin 1 g q6h, IV gentamicin (refer to the gentamicin/tobramycin dosing guidelines in the Pink Book), and IV metronidazole 500 mg q8h.
- Nil by mouth and IV fluids.
- 4 hourly observations, fluid balance chart.
- Analgesia.
- *VTE prophylaxis* (see page 123) (e.g., enoxaparin).

Acute Laparoscopic Cholecystectomy

- Book for surgery after discussion with Surgeon.
- Check ready for surgery, i.e. consent, CXR, ECG, laboratory results.
- Post-operative care:
  - Routine post-op observation.
  - Analgesia.
  - IV fluids until drinking.
  - Commence diet as tolerated.
  - *VTE prophylaxis* (see page 123).
  - Discuss with the Surgeon whether to change from IV to oral antibiotics.
  - Discharge home with appropriate script for analgesia, discharge summary.
  - Follow-up with GP unless Surgeon requests to see in clinic.

Acute Open Cholecystectomy

- As above but post-operative analgesia may include wound catheters, patient-controlled analgesia (PCA).

Conservative treatment

- Once settled, discharge on oral analgesia and antibiotics, usually amoxicillin/clavulanate 625 mg PO TDS.
- Book elective laparoscopic cholecystectomy.

14.4 Acute Appendicitis

See also *Acute Abdominal Pain* on page 113 and Common Emergency Presentations > *Abdominal Pain* on page 74.

14.4.1 History

- Full examination including rectal examination.
- Check for RIF tenderness, rebound guarding, signs of peritonism or peritonitis.
- Fever, tachycardia, oral foetor.

14.4.2 Investigations

- CBC + diff, Na, K, creatinine, amylase, LFTs.
- Urinalysis.
- In females, consider obstetric and gynaecological causes. Investigations as appropriate, e.g., pregnancy testing.

Imaging:

- More likely to be helpful if history/signs equivocal and in female patients.
- Abdominal X-ray + pelvic ultrasound.
- Consider MRI/CT - discuss with Consultant.

14.4.3 Management

- **Appendicitis**: appendicectomy (laparoscopic or open).
  - Prophylactic antibiotics: single doses of gentamicin 2 mg/kg IV and metronizadole 500 mg IV 1 hour pre-op.
  - *VTE prophylaxis* (see page 123).
- **Appendix mass or abscess**: conservative treatment with antibiotics ± radiological drainage:
  - Conservative (medical) treatment including IV antibiotics: IV amoxicillin 1 g q6h, IV gentamicin (refer to the gentamicin/tobramycin dosing guidelines in the Pink Book), and IV metronidazole 500 mg q8h.
  - Clinical review at least twice daily. Check for signs of peritonitis or peritonism and reconsider options.
  - Assess the need for intervention - this is a Consultant decision.
- **Appendicitis not confirmed**: seek alternate diagnosis.

Discharge:

- When patient is freely mobile.
- Tolerating fluids and food without nausea/vomiting.
- Pain is managed satisfactorily.

Check need for ongoing antibiotics, analgesia, and clarify follow-up arrangements.

14.5 Lower GI Bleeding

14.5.1 Background

- A lower GI bleed may present with substantial rectal bleeding.
- This should be distinguished from minor outlet rectal bleeding.
- The differential diagnosis can range from angiodysplasia, diverticular disease, polyps, bowel cancer to eating beetroot.
- Bleeding from the lower bowel usually stops spontaneously and management is usually supportive until this occurs. These patients rarely need emergency surgery.

14.5.2 Investigation and Management

- *Resuscitate* (see page 65).
- CBC + diff, Na, K, creatinine, LFTs, INR, APTT.
- Assess coagulation history, comorbidities, relevant history, including medications (oral anticoagulants, antiplatelet drugs, NSAIDs etc.). Correct as needed.
- If patient **stable**:
  - Close observation until bleeding stops. Decide on follow-up investigations such as colonoscopy, CT colonography.
- If patient **unstable or re-bleeds**:
  - CT angiography:
    - If no active bleeding, watch and wait, with possible rescanning if further major bleed.
    - If active bleeding, consider a further angiogram with embolization, or surgery.
  - Colonoscopy. There is a role for colonoscopy, however this is usually when there is a target lesion, i.e., a post polypectomy bleed.
  - Labelled red cell scan. Used selectively when there are repeat bleeds if the cause of bleeding has not been identified by CT angiography.

14.6 Perianal Abscess

14.6.1 History and Examination

- Painful perianal lump, fluctuance, surrounding cellulitis, systemic sepsis.
- Consider Crohn's disease if there is a history of altered bowel habit and/or abdominal pain. Check for family history of Crohn's disease.
- Make sure the infection is not secondary to neutropaenia, check CBC + diff.

14.6.2 Management

Initial Management

- Nil by mouth, IV fluids, analgesia.
- *VTE prophylaxis* (see page 123) if indicated.
- IV antibiotics if cellulitis, amoxicillin/clavulanate 1.2 g q8h. Consider gentamicin ± metronidazole.

Surgical Management

- Perianal abscesses usually require incision and drainage in theatre:
  - GA, lithotomy position.
  - Rigid sigmoidoscopy to check for proctitis.
  - Incise abscess adequately to drain all pus. Send pus for culture.
- Biopsy if Crohn's disease is possibility.
  - If a fistula is obvious consider seton ligature.
  - Only lay open a fistula with senior surgical input.
  - Loosely pack cavity with alginate.

Postoperative Management

- Continue antibiotics only if surrounding cellulitis and/or systemic sepsis.
  - Amoxicillin/clavulanate 1.2 g q8h IV initially. Then change to amoxicillin/clavulanate 625 mg PO TDS when afebrile and cellulitis improving.
  - If the patient deteriorates or fails to improve, inadequate drainage should be considered. The case should be discussed with the Consultant Surgeon and repeat examination under anaesthetic and/or MRI considered.
- After discharge:
  - District nurse dressing for large wounds (ongoing packing is usually not required).
  - GP follow up at 2 and 6 weeks with referral to surgical OPD if not healing/persistent fistula.
  - Complicated disease (e.g., Crohn's disease, identified fistula with seton) will require surgical OPD follow-up at six weeks.

14.7 Suspected Diverticulitis

Diverticulitis can be classified as **uncomplicated**, which usually responds to conservative measures and antibiotics, or **complicated**, such as perforation, abscess, fistula or obstruction. These usually require intervention in addition to antibiotics.

14.7.1 History, Examination and Investigations

- Left iliac fossa pain, tenderness, fever, past history of diverticular disease.
- CBC + diff, CRP, Na, K, creatinine, amylase, urinalysis.
- Erect CXR ± abdominal X-ray (if bowel obstruction suspected)
- CT abdomen/pelvis if:
  - No previous documented diverticulosis
  - Moderate/severe clinical presentation (severe sepsis, generalized peritonitis)
  - Not responding to IV antibiotics after 48-72 hours

14.7.2 Management

Uncomplicated diverticulitis:

- IV antibiotics: IV amoxicillin 1 g q6h, IV gentamicin (refer to the gentamicin/tobramycin dosing guidelines in the Pink Book), and IV metronidazole 500 mg q8h.
- IV fluids as clinically indicated.
- Clear fluids PO increasing to light diet as tolerated.
- Monitor temp, pulse, BP, creatinine, and urine output.
- **Deterioration or no improvement after 72 hours:**
  - Reassess and consider CT abdo/pelvis looking for complications.
- **Improvement:**
  - Change to oral antibiotics when afebrile for 24 hours, normal pulse and falling WBC. Give amoxicillin/clavulanate 625 mg TDS PO for 7-10 days.
  - GP follow up in 2 weeks.
  - Colonoscopy at 6 weeks unless:
    - CT proven uncomplicated diverticulitis and no other clinical features mandating colonoscopy.
    - Recent complete colonoscopy (<24 months).

Complicated Diverticulitis (discuss with admitting Consultant Surgeon):

- Free perforation - discuss with Surgeon and prepare for theatre.
- Abscess:
  - <5 cm - manage as for uncomplicated diverticulitis
  - >5 cm - radiological guided percutaneous drainage where possible
- Fistula/obstruction - discuss with Surgeon, management usually operative but will require preoperative investigation and management on case by case basis.
- Complicated cases will generally require surgical outpatient follow up at 6 weeks.

14.8 Small Bowel Obstruction

This is an emergency - the main danger is bowel strangulation and ischaemia.

14.8.1 Causes

- Adhesions, hernias containing bowel, Crohn's disease, volvulus, neoplasms, foreign bodies, ischaemia.

14.8.2 History and Examination

The findings largely depend on the level of the obstruction. Seek evidence of prior abdominal surgery. Typically there is severe, often sudden, abdominal pain and distension, vomiting and constipation. The abdominal pain is often central and colicky.

14.8.3 Initial Management

- CBC + diff, Na, K, creatinine, LFTs, amylase and lactate. ABGs can be useful if ischaemic bowel is suspected.
- IV fluid resuscitation with sodium chloride 0.9%. Potassium may be required.
- Chest and abdominal X-ray (supine and erect).
- Nasogastric tube.
- Indwelling urinary catheter. Monitor urine output and fluid balance.
- ECG if age >50 yrs.

14.8.4 Management

- **Suspected ischaemic bowel:** patients with small bowel obstruction may need urgent surgery if bowel ischaemia is suspected. The following clinical presentations may indicate ischaemia:
  - Peritonitis.
  - Fever.
  - Constant severe pain.
  - Raised WBC or CRP.
  - Incarcerated hernia.
  - Metabolic acidosis (pH <7.2 and base excess <-6).
- **Patients with a presumed adhesive bowel obstruction with no ischaemic bowel features**
  - The majority of patients with an adhesive small bowel obstruction will resolve with conservative management.
  - To decide early which patients will be suitable for conservative management, a water soluble contrast agent is given orally.
    - Give Gastrografin 100 mL PO or NG after the stomach is aspirated. Spigot nasogastric tube for a maximum of 4 hours.
    - Obtain an abdominal X-ray 8 hours following administration of Gastrografin.
    - If contrast is present in the colon, obstruction will resolve in 99% of patients without surgery, therefore conservative management should continue if no signs of ischaemia. Patients can be started on a light diet.
    - Patients with no contrast in colon are unlikely to resolve with conservative management and surgery is likely to be necessary.
- **Patients with non-adhesive small bowel obstruction with no signs of ischaemia:** these patients should have an abdominal CT scan performed to elucidate cause of the obstruction. This includes patients with:
  - No previous abdominal surgery.
  - Multiple recurrent episodes of small bowel obstruction with no previous CT abdomen.
  - Abdominal malignancy suspected.

14.9 Acute Pancreatitis

See *Acute Pancreatitis* (see page 107).

14.10 Haematemesis

See *Haematemesis* (see page 101).

15. Haematology

15.1 Haematology Department Information

The Canterbury Regional Cancer and Haematology Service comprises the departments of Haematology, Oncology, and Palliative Care.

Main Offices

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

Haematologists

Dr Andrew Butler, Dr Peter Ganly, Dr Steve Gibbons, Dr Emma Jane MacDonald, Dr Sean McPherson, Dr Mark Smith, Dr Ruth Spearing.

Inpatient Services

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

Consultation and On-call Services

- **Haematology** (Monday to Friday). Fax referrals to ☎ 81432. Please also send referral via internal mail to the Department of Haematology. The Registrar or Consultant taking referrals can be contacted on pager 7031.
- **Haemostasis Service**: ☎ 81246. This service comprises a Haematologist (Dr Mark Smith) and Nurse Specialists (pager 8527 or ☎ 81246) 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.
- **Laboratory Haematology**. 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.

Consultation Guidelines

Severe and/or unexplained anaemia, neutropaenia, and thrombocytopaenia. Leukaemia, lymphoma, myeloma and other haemopoietic malignancies. Bone marrow transplantation both autologous and allogeneic. Diagnosis and management of thrombotic disorders. Haemorrhagic diseases including inherited conditions such as haemophilia. Laboratory haematology.

Departmental Guidelines

Haematology Department Protocols and Guidelines (online at http://redbook.streamliners.co.nz).

15.2 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.

15.2.1 Investigation of a patient presenting with a possible haemorrhagic disorder

- Family history, history of pattern of bleeding, recent drugs, dietary history, possibility of HIV.
- CBC + diff, ESR or CRP, blood film examination.
- Prothrombin time, partial thromboplastin time, thrombin time and fibrinogen level. Use citrate tubes. Take care to add the correct amount of blood to these tubes and avoid heparin contamination from heparin containing IV lines, blood tubes, etc. Take blood samples **before** any transfusions are given.

  Note: *These are only screening tests and do not necessarily exclude defects which may result in abnormal bleeding. Consultation with the Coagulation Laboratory is strongly recommended (☎ 80374).*

15.2.2 Treatment

- This is entirely dependent on the results of the initial tests obtained. If a severe **thrombocytopaenia** (platelets $<10 \times 10^9$/L) is present then this constitutes a medical emergency. An accurate diagnosis is necessary and this will often require bone marrow examination. These patients may need platelet transfusions.
- Patients with known **coagulation defects** (Haemophilia A, Haemophilia B, Von Willebrand's 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** (online at http://redbook.streamliners.co.nz) 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 infusion, with concentrated freeze-dried preparations, e.g., CSL Factor VIII Biostate or recombinant factor VIII. A rough guide is given by the following formula.

Table 25 Factor VIII Infusion

Units of Factor VIII required = (weight in kilograms x % rise desired) ÷ 2
1. Currently each Biostate ampoule contains 500 or 1000 units.
2. Recombinant Factor VIII (Kogenate, Xyntha or Advate) 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 (Biostate) is used. Desmopressin may be given in a dose of 0.3 microgram/kg in 50 mL sodium chloride 0.9% IV over 30 minutes (starting 60 minutes pre-op, if requiring surgery). Desmopressin can be given undiluted subcut. Mild haemophilia A patients who infrequently use coagulation factor concentrates should receive recombinant products.
- In **Haemophilia B** (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.

15.3 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.

15.3.1 Investigations

- CBC + diff, film, and reticulocyte count along with standard biochemistry and LDH. Review previous results from Community Laboratory/GP to ascertain duration of anaemia.
- Mean cell volume (MCV) <80 femtolitre - probable iron deficiency or an inflammatory anaemia. Consider thalassaemia. Request iron studies, ferritin and CRP.
- MCV >100 femtolitre - could merely reflect an increased reticulocyte count (haemolysis/blood loss). If retics normal do plasma 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 pmol/L). Plasma methylmalonic acid measurement may be helpful but is falsely raised in renal impairment.
- MCV 80-100 femtolitre - 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.*

15.3.2 Treatment

- Once blood samples have been taken, and a bone marrow has either been performed or been deemed unnecessary, treatment may be started with oral iron and/or oral folic acid and/or IM hydroxocobalamin if one of these haematinic deficiencies seem likely. Recommended preparations are ferrous fumarate 200 mg PO BD, folic acid 5 mg PO daily and hydroxocobalamin 1 mg IM every other day for 6 doses, followed by maintenance treatment, usually 1 mg every 3 months.
- Transfusion should be given with extreme caution if a severe deficiency state is present. 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).

15.4 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.

15.4.1 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°C or history of fever >38°C 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 (search for "C240085" on the CDHB intranet).
- 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 4.5 g IV q8h plus gentamicin 5-7 mg/kg IV in 100 mL sodium chloride 0.9% over 30 min q24h

  or, if there is a history of penicillin allergy,
  - Meropenem 1 g IV q8h plus gentamicin 5-7 mg/kg IV in 100 mL sodium chloride 0.9% over 30 min q24h.

  ***Note**: See the gentamicin/tobramycin dosing guidelines in the Pink Book.*
- **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.

15.5 Nausea and Vomiting

Refer to *Management of Nausea and Vomiting in the Oncology section* (see page 205).

15.6 Venous Thromboembolism (VTE)

15.6.1 Prophylaxis of VTE

VTE Prophylaxis in Medical Patients

This remains controversial. The administration of LMWH to the majority of medical patients admitted to hospital may reduce the frequency of VTE, but at the same time will lead to more bleeding events, and has not proven to be cost-effective (ACCP 2012).

We recommend a more selective approach which identifies those medical patients at high risk for VTE using the Padua Prediction Score. These patients should then be given LMWH prophylaxis as outlined below.

Acutely ill hospitalized medical patients at low risk of VTE should not be given pharmacologic prophylaxis.

Table 26 The Padua Risk Assessment Model

Baseline Feature	Score
Active cancer	3
Previous VTE	3
Reduced mobility (>3 days)	3
Known thrombophilia	3
Recent (≤1 month) trauma or surgery	2
Elderly (age ≥70)	1
Heart and/or respiratory failure	1
Acute myocardial infarction or ischaemic stroke (1)	1
Acute infection and/or rheumatic disease	1
Obesity (BMI ≥30)	1
Ongoing hormonal treatment	1
Risk Groups	
High risk of VTE	≥4
Low risk of VTE	<4

1. For the recommended VTE prophylaxis of ischaemic stroke, see below.

Reference: Barbar et al, J Thromb Haemost, 2010 Nov;8(11):2450-7

- **Recommended prophylaxis schedule for patients at high risk, ≥4:**
  - Enoxaparin 40 mg subcut daily, provided there are no contraindications (e.g., active bleeding, thrombocytopaenia - platelets <100 x 10^9/L). The dose may need to be reduced in renal impairment - discuss with Consultant.
  - The duration of prophylactic treatment with heparins must be individualized. It should cover the obvious risk period such as immobilization, but must be stopped as soon as the perceived increased risk has passed.
- **For patients at low risk of VTE, <4**, thromboprophylaxis with LMWH should not be given.
- Compression stockings are not recommended for either group.

VTE Prophylaxis in Stroke Patients

- Early mobilization and optimal hydration should be maintained from the outset.
- The cause, ischaemic or haemorrhagic, needs to be established.

For patients with ischaemic stroke only, see CDHB Stroke Guidelines, pp55-56 (search for "stroke guidelines" on the CDHB intranet):

- 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 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, those with past history of DVT/PE or known thrombophilia.

- Give enoxaparin 40 mg subcut daily, less in renal impairment, e.g., 20 mg daily if eGFR <30 mL/min. The best timing for initiation of LMWH after stroke is not known. LMWH use is associated with an increased risk of intracerebral haemorrhage 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. Hydration and mobilization remain cornerstones of VTE prophylaxis.

  Note: *If LMWH prophylaxis is started, it is important to continue antiplatelet therapy.*

VTE Prophylaxis in Spinal Injury

Refer to *Traumatic Spinal Cord Injury - Management* (see page 275).

VTE Prophylaxis in Surgical Patients

Surgery is a significant cause of deep vein thrombosis and post-operative death from pulmonary embolism may occur from an unrecognized DVT. The risk of venous thromboembolism depends on the type of surgery, patient characteristics and the underlying disease.

- The various ways to reduce the risk of VTE post-surgery include drugs such as LMWH and oral anticoagulants, compression stockings, and intermittent pneumatic compression.
- There are many guidelines available and all aim to reduce the VTE risk while minimizing the risk of abnormal bleeding. We recommend the Guidelines for the Prevention of Venous Thromboembolism produced by the Department of General Surgery (search for "stroke guidelines" on the CDHB intranet). The following tables are taken from these guidelines, and have been adapted from the CDHB form C240158.

Note: *The recommendations below do* ***not*** *apply to patients undergoing thyroid or breast surgery at Christchurch Hospital, and they may not apply to other surgical disciplines. In these situations it is essential to discuss with the Surgeon/Anaesthetist involved what type of VTE prophylaxis (if any), is to be given.*

Table 27 VTE risk assessment before Elective General Surgery

Assess for contraindications for prophylaxis and VTE medical risk factors

Contraindications for use of pharmacological thromboprophylaxis	▸ Breast/thyroid surgery - individual Consultant will take personal responsibility to prescribe prophylaxis. ▸ Allergy to heparin. ▸ On warfarin. ▸ Haemorrhagic stroke (recent). ▸ Severe liver or kidney impairment. ▸ High risk of bleeding, e.g., haemophilia, thrombocytopaenia, brain metastases, oesophageal varices, recent (<3 months) GI or intracranial bleed. ▸ Other.	
Contraindications for use of mechanical thromboprophylaxis	▸ Lower limb condition which by application of TED stockings and/or intermittent pneumatic compression could result in vascular or neurological compromise. Examples may include severe peripheral neuropathy, peripheral vascular disease, gross leg oedema, recent skin graft.	
VTE medical risk factors	▸ History of DVT/PE. ▸ Active malignancy (<6 months). ▸ Obesity (BMI >30). ▸ Moderate to severe heart failure. ▸ Severe airways disease. ▸ HRT or oral contraception.	▸ Other thrombogenic drugs. ▸ Thrombophilia. ▸ Pregnancy or puerperium. ▸ Active inflammation. ▸ Immobility. ▸ Other.

Table 28 Treatment recommended related to risk and type of General Surgery			
Risk Category	**Surgery Type**	**Pharmacological prophylaxis**	**and Mechanical prophylaxis**
High	▸ Major[1] surgery and age >60 years. ▸ Major[1] surgery and age 40-60 years with any medical risk factors (see table above). ▸ Major[1] surgery, any age and previous VTE or active malignancy.	▸ Enoxaparin 40 mg subcut daily at 2100 hours for 5 days or until fully ambulatory.[2] **Unless** Weight is <45 kg or eGFR <30 mL/min then ▸ Enoxaparin 20 mg subcut daily at 2100 hours for 5 days or until fully ambulatory.[2]	▸ Pre-op TED stockings until fully ambulatory. and/or ▸ Intermittent pneumatic compression (IPC).[3]
Moderate	▸ Major[1] surgery and age 40-60 years without any medical risk factors (see table above). ▸ Major[1] surgery and age <40 years with any medical risk factors. ▸ Minor surgery and age >60 years. ▸ Minor surgery and age 40-60 years with any medical risk factors.	▸ Enoxaparin 20 mg subcut daily at 2100 hours for 5 days or until fully ambulatory.[2]	
Low	▸ Major[1] surgery and age <40 with no risk factors. ▸ Minor surgery <60 years with no risk factors.	▸ Not indicated.	▸ Pre-op TED stockings until fully ambulatory.

1. Major surgery: any intra-abdominal surgery and all other operations >45 minutes duration.
2. The first dose is given on the evening of surgery.
3. Refer to the Guidelines for the Prevention of Venous Thromboembolism produced by the Department of General Surgery (search for "stroke guidelines" on the CDHB intranet).

Note: *We emphasize that any perioperative anti-thrombotic drug or any of the other treatments listed above,* ***must be confirmed with the Surgeon and/or Anaesthetist involved****. They will make the final decision.*

VTE Prophylaxis for Orthopaedic Surgery

- Orthopaedic acute patients with fractures of the lower limbs are at high risk for VTE.
- All such patients should receive enoxaparin 40 mg subcut daily while immobile unless contraindicated (see above). A reduced dose may be appropriate if impaired renal function, e.g., 20 mg daily if eGFR <30 mL/min. The duration of LMWH will depend on the clinical circumstances but VTE risk may be high for 2-4 weeks following surgery.
- If anticoagulation is contraindicated then TED stockings or intermittent pneumatic compression should be used.
- Warfarin may be indicated in some patients - discuss with Consultant.

Note*: Dabigatran is licensed for prophylaxis of venous thromboembolism following total hip or knee replacement - see below.*

Guidelines for Prophylaxis using new Oral Anticoagulants

- Oral thrombin (dabigatran) and factor Xa (rivaroxaban and edoxaban) inhibitors have demonstrated rapid onset of action and predictable pharmacokinetics. At the present time, only dabigatran is funded. None of these three agents can be reversed if excessive bleeding occurs. See ***Anticoagulant Overdosage*** (see page 137).
- Dabigatran is funded by PHARMAC for the prevention of VTE post major orthopaedic surgery, specifically post hip and knee replacements.
- The recommended dose of dabigatran is 110 mg PO within 1-4 hours of the completion of surgery, and then 220 mg PO daily for 10 days after knee replacement, and between 28 to 35 days after hip replacement.
- Dabigatran is excreted mainly by the kidney (fu 0.8), so dosing appropriate to renal function is important.

Note: *Dabigatran may not be suitable for patients with renal impairment, may interact with other medications, and should be used with caution and in lower dose for patients over 75-80 years of age.*

- **Before using dabigatran, refer to:**
  - The PHARMAC website Guidelines for testing and perioperative management of dabigatran and Guidelines for management of bleeding with dabigatran.
  - The Medsafe datasheet on dabigatran.
  - The CDHB Clinical Pharmacology bulletin on dabigatran etexilate (July 2011).
- **Before using rivaroxaban, refer to** the CDHB Clinical Pharmacology bulletin on rivaroxaban (May 2013).

15.6.2 Guidelines for use of Anticoagulants if Spinal/Epidural Anaesthesia is used

LMWH, e.g., enoxaparin:

- **The following comments apply when a prophylactic dose of LMWH is being given. If the patient is on a therapeutic dose of LMWH, contact the Anaesthetist for advice.**
- 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 (e.g., 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, e.g., 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.
- When removing the epidural catheter, ensure that no LMWH has been given in the previous 12-16 hours. Once catheter is removed, any subsequent dose of LMWH should **not be given for 6-8 hours**. If any concerns contact the Duty Anaesthetist (pager 8120).

Dabigatran/rivaroxaban:

- Dabigatran should not be started until at least **6 hours AFTER** the procedure.
- Rivaroxaban: discuss with Anaesthetist (or Duty Anaesthetist pager 8120) if it is to be started within 24 hours of procedure.

15.6.3 Diagnosis of Deep Vein Thrombosis (DVT)

Clinical Features and Causes of DVT

- Suspect DVT if there is swelling, pain, tenderness, and dilated superficial veins.

Causes

- Surgery, immobilization including travel, oestrogen therapy (combined oral contraceptive pill, hormone replacement therapy), malignancy, pregnancy and puerperium, polycythaemia, thrombocytosis.
- Check for family history of VTE.
- **Thrombophilia testing is not required for the majority of patients with DVT/PE**. If the patient is under 45 years, **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.

Risk Assessment for DVT

Table 29 Clinical Model for Predicting the Pre-test Probability of Deep-Vein Thrombosis[1]

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 immobilization 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
Localized 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 (non-varicose)	1
Previously documented deep-vein thrombosis	1
Alternative diagnosis at least as likely as deep-vein thrombosis	-2

1. A score of 2 or higher indicates that the probability of deep-vein thrombosis is **likely**; a score of <2 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 pre-test probability for DVT and write this in the patient's notes.

Investigations for Suspected DVT

Assess the clinical probability of DVT (refer to *Risk Assessment for DVT* above), and get D-dimer result. Then proceed as follows:

- **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 "likely", 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%.
- D-dimer levels may be above the quoted upper limit in otherwise normal elderly patients (>60 years).
- **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 (see below).
- 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 visualized by ultrasound.

Calf Vein Thrombosis

- About 50% of symptomatic DVTs will be limited to the calf veins. 10% of these will extend into the proximal veins putting the patient at risk of PE.
- The management of calf vein thrombosis is controversial and requires balancing the risk of DVT extension against the risks of anticoagulation.
- 4-6 weeks anticoagulation (LMWH and warfarin), or longer if clinically indicated, does reduce the risk of extension and PE.
- If anticoagulation is not offered, then close monitoring with repeat USS at 48-72 hours is recommended to check whether clot extension has occurred. Consider a further repeat at 10-14 days if signs persist.

Note: *When management of calf vein thrombosis is uncertain, discuss with the Haemostasis Service.*

DVT in Patients with Cancer

The decision to give anticoagulants in this situation needs to be individualized. It may be inappropriate to initiate such treatment in patients with extensive metastatic disease with limited life expectation. In most cases, patients with VTE and active cancer require immediate treatment with therapeutic doses of LMWH and this should be given for 3-6 months.

At that time a decision to continue LMWH, change to warfarin, or to stop anticoagulants must be taken. Factors to consider are: is the cancer in remission or active, is any ongoing cancer treatment prothrombotic, and what treatment (if any) would the patient prefer?

Superficial Venous Thrombosis of the Lower Limb

Superficial venous thrombosis has replaced the term 'thrombophlebitis' as it is more descriptive and implies the recently established link to DVT.

- This remains primarily a clinical diagnosis, with pain, tenderness, erythema, and induration along the course of a vein.
- Risk factors include: venous stasis, e.g., varicose veins, pregnancy, infection, thrombocytosis, polycythaemia and cancer.
- 5% of patients presenting with superficial venous thrombosis will also have a DVT. For the remainder there is a 10% risk of extension to the deep veins over the next 10 days.

Management

- An ultrasound to exclude DVT should be done if there is an involved segment of vein of >5 cm, the saphenous vein is involved, and/or there is significant leg swelling. If DVT is present ***treat accordingly*** (see page 130).
- If there is no DVT, but superficial venous thrombosis is confirmed and:
  - The superficial venous thrombosis is within 3 cm of the sapheno-femoral junction, consider treatment as for DVT.
  - There are one or more of the above risk factors, consider prophylactic doses of LMWH for 30 days.
  - None of the above risk factors are present, consider conservative treatment with elevation of the limb, compression stockings, and pain relief. If evidence of infection, treat as appropriate.
- Follow up at 7-10 days or earlier if there is deterioration. If no resolution at this time, repeat the ultrasound.
- If superficial venous thrombosis is diagnosed clinically, give conservative treatment and follow-up as above.

Reference: Tait et al, BJH, Guidelines on the investigation and management of venous thrombosis at unusual sites. BJH 2012.159 pp 28-38.

Upper Extremity Deep Vein Thrombosis

- Accounts for up to 10% of all DVTs, but primary causes are rare.
- May affect axillary, subclavian, or brachial veins.
- Are considered primary if idiopathic or associated with the thoracic outlet syndrome or effort. If there is a definite cause they are regarded as secondary.
- Primary upper limb DVT is rare. The average age at presentation is the early thirties. It is associated with strenuous activity, especially involving repetitive arm movements. A thrombophilic defect may also be present.
- Secondary causes include: central venous catheters (CVC), active malignancies, inherited and acquired thrombophilia.

Investigations

- Venography or compression ultrasound to establish the diagnosis. CXR.
- If the condition is primary, a thrombophilia screen should be taken before treatment is given.

Management

This depends on the cause and the severity of the venous occlusion. Search for risk factors.

- Secondary:
  - Give 4-5 days LMWH and warfarin for 3 to 6 months. See *Treatment of VTE* (see page 130).
  - If associated with the presence of a CVC, then this will usually, but not always, need to be removed.
  - If associated with active cancer, consider LMWH for 3 months or for as long as clinically appropriate. See *DVT in Patients with Cancer* (see page 127).
- Primary:
  - Start *LMWH and warfarin as for lower limb DVT* on page 131. Consult a Haematologist re ongoing management.
  - If severely affected and/or associated with thoracic outlet obstruction or effort, such patients are likely to have long term problems with persisting venous obstruction. In this situation, consultation with Vascular Surgery is also recommended as thrombolysis with or without surgery may be indicated; thrombolysis is most effective if carried out within 7-10 days of onset.

15.6.4 Diagnosis of Pulmonary Embolism (PE)

If the patient is pregnant, see *Investigations for Suspected PE in Pregnancy* (see page 129).

Clinical Features and Causes of PE

- 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.

Causes

- Surgery, immobilization including travel, oestrogen therapy (combined oral contraceptive pill, hormone replacement therapy), malignancy, pregnancy and puerperium, polycythaemia, thrombocytosis.
- Check for family history of VTE.
- **Thrombophilia testing is not required for the majority of patients with DVT/PE**. If the patient is under 45 years, **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.

Risk Assessment for PE

Table 30 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		**Score**
Low		0-3 total
Intermediate		4-10 total
High		≥11 total
***Note**: Prevalence of PE: low 8%, intermediate 28%, high 74%.*		

Reference: Le Gal et al. Ann. Int. Med. 2006; 144: 165-171.

Note: It is essential to work out the pre-test probability for PE and write this in the patient's notes.

Investigations for Suspected PE

- All patients with suspected PE should have their pre-test probability assessed using the ***Revised Geneva Scoring system*** (see page 128) and have blood taken for a D-dimer assessment.
- Patients with signs of right ventricular dysfunction should have urgent investigations with a CTPA and/or echo, as they may benefit from thrombolytic therapy using the ***standard tPA protocol*** on page 134.
- Out of normal working hours, and if it is clinically safe to delay establishing the diagnosis, consider anticoagulation overnight and investigate the following morning.

Action to take when results are available

Note: *D-dimer levels may be above the quoted upper limit in otherwise normal elderly patients (>60 years).*

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.

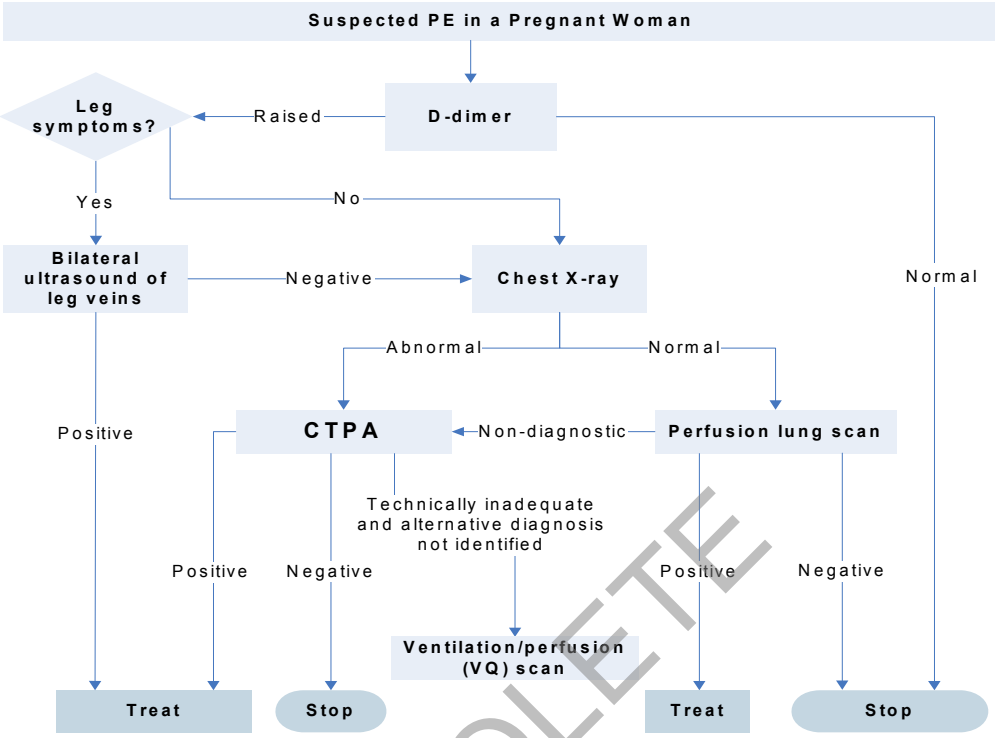
If the D-dimer is positive:

- 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**, see ***Investigations for Suspected PE in Pregnancy*** (see below).
- **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, 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.

ACCP Antithrombotic Guidelines 9th edition. Chest 2012; 141:7S-47S.

Investigations for Suspected PE in Pregnancy

- When a pregnant woman presents with chest pain and/or dyspnoea, the possibility of pulmonary embolism (PE) is always considered. The established risk equations such as the Wells score and the Revised Geneva score have not been validated for use in pregnancy.
- The American Thoracic Society (Leung et al) and an Australasian group representing the Society of Obstetric Medicine of Australia and New Zealand and the Australasian Society of Thrombosis and Haemostasis (McLintock et al) have published recommendations which we have reviewed in producing this CDHB guideline.
- The use of CTPA is associated with a significant radiation dose to the maternal breasts and, because of the altered haemodynamic environment in pregnancy, the rate of technically inadequate or non-diagnostic scans is increased. It is therefore desirable to make use of other diagnostic modalities where this is possible. The attached flow diagram has been developed in discussion with Obstetric Physicians, the Radiology Service, and the Department of Nuclear Medicine.
- In contrast to the above guidelines, we have chosen to use the D-dimer assay in the work-up. The rationale for avoiding its use is the fact that D-dimer levels go up in normal pregnancy. However, it is not always significantly elevated, and despite occasional reports of false negative results, **a normal D-dimer level makes the diagnosis of PE very unlikely**.
- A chest X-ray is often required to look for other possible diagnoses such as pneumonia or pneumothorax, and involves minimal radiation to both mother and fetus.
- While a perfusion-only lung scan is often all that is required, a ventilation/perfusion (VQ) scan minimally increases the radiation dose and may be opted for at the discretion of the Nuclear Medicine Department.



References:

Leung AN, Bull TM, Jaeschke R et al. An Official American Thoracic Society/Society of Thoracic Radiology Clinical Practice Guideline: Evaluation of Suspected Pulmonary Embolism In Pregnancy. American Journal of Respiratory and Critical Care Medicine 2011;184(10):1200-1208.

McLintock C, Brighton T, Chunilal S et al. Recommendations for the diagnosis and treatment of deep venous thrombosis and pulmonary embolism in pregnancy and the postpartum period. Aust NZ J Obstet Gynaecol 2012;52(1):14-22.

15.6.5 Treatment of VTE (DVT/PE)

Initial Management of VTE (DVT/PE)

The initial management of **DVT** consists of LMWH and warfarin (see *Initial Dosage of Heparin and Warfarin* on page 131).

The initial management of **PE** is:

- Anticoagulate (see *Initial Dosage of Heparin and Warfarin* on page 131).
- 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 134.

Heparin and Warfarin Therapy

Note: The following information needs to be considered before starting heparin/warfarin therapy.

- CBC + diff.
- Na, K, creatinine, alb, bili, ALP, GGT, ALT, AST.
- **INR/APTT in all patients**. In patients under 45, consider taking 2 extra 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.

Initial Dosage of Heparin and Warfarin

Table 31 Initial Dosage of Heparin and Warfarin

- **Low molecular weight heparin (LMWH)**, e.g., enoxaparin 1 mg/kg q12h subcut. On discharge, if LMWH is still required, change to 1.5 mg/kg subcut q24h. If necessary, adjust dosage according to the anti-Xa levels and/or renal function. **See below for dosage modifications in renal impairment and for extremes of weight.** For information about anti-Xa monitoring, see below.
  - **Duration:** until INR >2 for 2 consecutive days (normally given for at least 5 days).
  - **Monitoring:** Not usually required for LMWH but may be needed. See *LMWH Dosage and Monitoring section*, below.

and

- **Commence warfarin 5 mg or 10 mg PO** on Day 1 - see below and the *nomogram* on page 133 for essential information concerning both this initial dose and subsequent doses.
- **Duration:** refer to *Recommended INR Levels for Warfarin Treatment* on page 133.
- **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 133).

Treatment of DVT/PE in pregnancy

- Warfarin is teratogenic. Start the patient on LMWH in therapeutic dosage and seek advice from an Obstetric Physician.
- Anti-Xa monitoring should be used when LMWH is given for the treatment of DVT/PE in pregnancy.

ACCP Antithrombotic Guidelines 9th edition. Chest 2012; 141:7S-47S.

Low Molecular Weight Heparin (LMWH) Dosage and Monitoring

- 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 compared with unfractionated heparin, but it is still a significant problem and fatal haemorrhage may occur.
- A number of low molecular weight heparins are currently available for the treatment of DVT. CDHB recommends enoxaparin.
- Dose reduction of LMWH is required in certain situations.
- 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.

Dosage in renal impairment

- If the creatinine clearance (CrCl) is <60 mL/minute, then **treat as follows**:
  - Enoxaparin dose: q12h subcut (avoid q24h dosing while an inpatient)
  - The first dose is based on **actual weight**, 1 mg/kg.
  - Further doses are adjusted **as shown in the table below**.

These recommendations are based on the creatinine clearance and the fu, the fu for enoxaparin being 0.7.

Table 32 LMWH dosage in renal impairment

CrCl (mL/min)	Dosage of LMWH recommended
>60	full dose
50-60	70% of the weight-based dose
40-50	60% of the weight-based dose
30-40	50% of the weight-based dose
<30	Use unfractionated heparin[1]

1. If the creatinine clearance is <30 mL/minute, discuss with Consultant. Unfractionated heparin should be given in this situation with close monitoring (see below). Unfractionated heparin can be reliably reversed by protamine sulphate if abnormal bleeding occurs. See *Heparin Overdosage* on page 137.

Dosage in extremes of weight

- This is controversial with conflicting evidence in the medical literature. We have adopted a pragmatic approach and tried to avoid over- or under-dosage and to avoid giving too large a single dose.
- **For patients at extremes of weight, we recommend using anti-Xa levels to guide dosage after the third dose.**

Table 33 LMWH dosage in extremes of weight

- Weight <45 kg or >130 kg.
- 1 mg/kg q12h subcut for 3 doses based on actual weight. The second and third doses will need to be modified if there is renal impairment as above.
- An anti-Xa peak level should be taken three hours after the 3rd dose. The urgent testing required on this sample needs to be arranged with the laboratory.
- Subsequent dosage, i.e., from the 4th dose onwards, may need to be modified when the anti-Xa result is known.
- If still on LMWH at discharge it may be impractical to continue with q12h doses. Change to q24h dosage. Continue to monitor anti-Xa levels to guide dosage.

LMWH monitoring

- This is done using anti factor-Xa (anti-Xa) levels.
- Indications: LMWH therapy for >7 days; extremes of weight; pregnancy.
- Use a citrate (blue) tube for blood samples. Samples for anti-Xa levels should be taken 3 hours after enoxaparin administration (i.e., peak) and at steady state, e.g., after the first 3 doses when initiating enoxaparin therapy.
- Turnaround at the laboratory can be up to 4 days. Urgent testing (as little as 2 hours' turnaround) may be arranged by calling the laboratory.

***Note**: 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 0.3 and 1 unit/mL. Consult Haematology for advice if anti-Xa levels and the clinical findings appear inconsistent.*

Unfractionated Heparin Dosage

- Use the Unfractionated Heparin Intravenous Infusion Via Syringe Pump chart (search for "C160010" on the CDHB intranet). This gives full guidelines for prescribing IV unfractionated heparin.
- Check INR and APTT before starting treatment.
- Give loading dose (IV bolus) of 80 units/kg (max 10,000 units) then an initial maintenance infusion of 18 units/kg/hr (max 1,800 units/hr).
- Check APTT 6 hourly until in the "therapeutic range". This is approximately 2 - 2.5 times the upper normal range for APTT. The laboratory will indicate their recommended therapeutic range on the report if you state that the patient is on unfractionated heparin.
- Unfractionated heparin dosage should not be altered in relation to renal function and should be monitored with APTT testing.
- Unfractionated heparin effects can be reversed by *protamine sulphate* (see page 137).

Warfarin Dosage - the First 5 Days, Recommended INR Levels

- **Check INR and APTT before starting treatment**.
- Anticoagulant action begins in hours to days relative 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 can 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 34 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	seek advice
	1.5-1.9	8 mg
	2.0-3.0	3 mg
	>3.0	seek advice

1. 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. **The CDHB uses Marevan**.
2. We suggest for inpatients and at discharge only 1 mg tablets of warfarin are prescribed, to minimize confusion over dosage and tablet size.

Table 35 Recommended INR Levels for Warfarin Treatment

	Prothrombin Ratio (INR)	Duration
Pre and perioperative anticoagulation	1.5-2	Days
Treatment of calf DVT	2-3	4-6 weeks
Treatment of provoked DVT	2-3	12-26 weeks(1)
Treatment of provoked PE or massive DVT	2-3	26-52 weeks(1)
Treatment of unprovoked PE or DVT	2-3	life long(2)
Treatment of recurrent PE or DVT(3)	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. This assumes that any transient cause for DVT/PE has resolved. The presence of an inherited prothrombotic defect does not, of itself, influence the duration of anticoagulation in this instance.
2. If there is a low risk of bleeding and if this is consistent with the patient's preference. The decision to give life long oral anticoagulants should be taken at a formal assessment of the patient at 6-12 months after the initial thrombosis.
3. Recurrence despite prothrombin ratio between 2 and 3.

Warfarin - Drug Interactions

Many factors influence the individual's response to warfarin. Other drugs may inhibit or induce the enzymes that metabolize warfarin, and these enzymes may show inherited variation in activity. The anticoagulant effect of warfarin may also be influenced by diet, various disease states, and vitamin K metabolism.

Some drugs expected to potentiate warfarin effect

- **Antimicrobials**: azole antifungal drugs, cephalosporins, cotrimoxazole, isoniazid, macrolides, metronidazole, penicillins, quinolones, tetracycline
- **Cardiovascular**: amiodarone, diltiazem, fibrates, verapamil.
- **Central Nervous**: antidepressants (tricyclics, SSRIs, SNRIs).
- **Gastrointestinal**: omeprazole.

Some drugs expected to decrease warfarin effect

- **Antibacterials**: rifampicin.
- **Cardiovascular**: cholestyramine.
- **Central Nervous**: barbiturates, carbamazepine, phenytoin.

Other factors that may influence warfarin effect:

- Vitamin K, vitamin K rich foods, e.g., avocados, broccoli, will decrease the effect of warfarin.
- Herbal medicines may interact with warfarin. **Increased** effect - gingko, fish oils; **decreased** effect - St. John's wort, ginseng.

***Note**: These lists are **not** exhaustive. If an unfamiliar drug or complementary medicine is being combined with warfarin, look it up (e.g., UpToDate online), or seek advice from the ward pharmacist or the Drug Information Service, ☎ 80900.*

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 r-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 131.

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 3 months, except if ischaemic stroke is being treated by thrombolysis.

- 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 (e.g., warfarin 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.*

ACCP Antithrombotic Guidelines 9th edition. Chest 2012; 141:7S-47S.

15.7 Patients on Oral Anticoagulants 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.

15.7.1 Management of Patients on Warfarin Undergoing Surgery

Table 36 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 24-36 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 LMWH or unfractionated 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 subcut daily. Last dose given on the day BEFORE surgery. ▸ Test INR on day of surgery. If INR ≥1.5 discuss with Surgeon and Anaesthetist.

Continued over…

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 LMWH 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.

- **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
- **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.

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 antiplatelet therapy is also an important part of the prevention of further episodes - cessation of any antiplatelet therapy as well as oral anticoagulants needs to be considered in light of the risk of bleeding versus emboli. Consult Cardiac Surgeon.
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 congestive heart failure, previous thromboembolism, hypercoagulable conditions.

15.7.2 Management of Patients on Dabigatran Undergoing Surgery

Table 37 Management of Patients on Dabigatran Undergoing Surgery

- Assess the risk of bleeding against the risk of thrombosis when considering discontinuing anticoagulation.
- For minor procedures, dabigatran may not need to be discontinued.
- **If dabigatran does need to be stopped, it is important to plan ahead as there is no treatment available to immediately reverse dabigatran.**
- For detailed information see the PHARMAC website Guidelines for testing and perioperative management of dabigatran.
- If further advice is required, contact the Haematologist on call.

Note*: Refer to* **Guidelines for use of anticoagulants if spinal/epidural anaesthesia is used** *(see page 125).*

15.8 Patients on Antiplatelet Drugs Undergoing Surgery

15.8.1 Management of Patients on Antiplatelet Drugs Undergoing Surgery

This is a complex issue and management needs to be individualized. **The surgeon responsible for the surgical procedure will make the final decision with regard to antiplatelet drug dosage perioperatively**.

Some key factors to consider:

- Is the patient at high risk of bleeding complications, e.g., is there severely impaired renal function (CrCl <30 mL/min), or severe liver dysfunction?
- If the drug is stopped, what is the risk of re-thrombosis with the antiplatelet drug concerned, and the patient's clinical condition?
- Does the antiplatelet drug itself carry a high risk of bleeding complications?
- Does the proposed surgical procedure carry a high risk of significant morbidity/mortality in the event of bleeding, e.g., cardiac or intra-cranial operations?
- Can the antiplatelet drug be safely withheld and the procedure be delayed (e.g., for 3-4 days), to allow for some recovery of platelet function?

Some management considerations:

- **Aspirin** may increase operative bleeding but this is usually not significant, especially at doses of 100 - 150 mg /day, so surgery rarely needs to be delayed.
- **Clopidogrel**. If this drug is being given as antiplatelet monotherapy, it is reasonable to continue it during surgery unless it is cardiac or intracranial surgery. If the patient is considered a high bleeding risk or cardiac or intracranial surgery is to be done, stopping clopidogrel for 3-4 days before surgery should be considered. However in patients who have had a stent placed in the past 12 months, stopping clopidogrel carries a significant risk of re-thrombosis and consultation with a Cardiologist is essential before altering the dose of this drug.
- **Ticagrelor**, compared to aspirin and clopidogrel, carries the highest risk of causing increased bleeding at surgery, probably because it causes a greater degree of inhibition of platelet function. Where possible it should be stopped 3-4 days before surgery. However in patients who have had a stent placed in the past 12 months, stopping ticagrelor also carries a significant risk of re-thrombosis and **consultation with a Cardiologist is essential** before altering the dose of this drug.
- Consider restarting an antiplatelet drug 6 to 24 hours post-op, unless the result of any bleeding (e.g., intracranial) would be critical. In the latter situation, consultation with a Cardiologist and relevant Surgeon is recommended.

Reference: Guideline on the use of antiplatelet drugs in patients having cardiac and noncardiac operations. (American Society of Thoracic Surgeons). Ferraris et al. 2012;94:1761-1781.

15.9 Anticoagulant Overdosage

15.9.1 Heparin Overdosage

- Protamine sulphate is used to reverse overdosage with unfractionated heparin (UFH). Reversal is only necessary if there is serious bleeding. The dose of protamine is 1 mg IV per 100 units of UFH estimated to be remaining in the circulation - the half-life is about 1 hour. The maximum single dose of protamine is 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 the ability of protamine to neutralize LMWHs. Studies in healthy volunteers indicate that 65-80% of the anti-Xa activity is neutralized by protamine sulphate. 1 mg enoxaparin may be partially neutralized 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 neutralization 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.*

15.9.2 Warfarin Overdosage

Consider why the patient is on warfarin. For those patients with artificial heart valves, discuss with Cardiologist or Cardiothoracic Surgeon.

Note: *The following table is derived from the guidelines published by the Australian Society of Thrombosis and Haemostasis (Med J Aust 2013; 198 (4): 1-7 and Med J Aust 2004; 181(9):492-7). There are, however, significant differences, and this table reflects current practice within the CDHB. Our recommendations therefore differ from these two references and from the NZBS pocket guide and related iPhone app on this topic.*

Table 38 Guidelines for the management of an elevated international normalized 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 5-10[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 approaches the therapeutic range.
INR >10 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 approaches the therapeutic range. ▸ Where there is a high risk of bleeding,[3] cease warfarin therapy, give 2 mg vitamin K intravenously. Consider Prothrombinex-VF (25-50 unit/kg) and fresh frozen plasma (150-300 mL), measure INR in 6-12 hours, resume warfarin therapy at a reduced dose once INR approaches the therapeutic range.
Any clinically significant bleeding where warfarin-induced coagulopathy is considered a contributing factor	▸ Cease warfarin therapy. **Give the following treatment urgently:** 5-10 mg vitamin K intravenously, as well as Prothrombinex-VF (50 unit/kg) and fresh frozen plasma (150-300 mL). ▸ Monitor the patient continuously until the bleeding stops. For details, see [4] OR ▸ If fresh frozen plasma is unavailable, cease warfarin therapy, give 5-10 mg vitamin K intravenously, and Prothrombinex-VF (50 unit/kg). ▸ Monitor the patient continuously until the bleeding stops. For details, see [4] OR ▸ If Prothrombinex-VF is unavailable, cease warfarin therapy, give 5-10 mg vitamin K intravenously, and 15-30 mL/kg of fresh frozen plasma. ▸ Monitor the patient continuously until the bleeding stops. For details, see [4]
Any INR with minor bleeding	▸ Omit warfarin, repeat INR the following day and adjust warfarin dose to maintain INR in the target therapeutic range. ▸ If bleeding risk is high [3] or INR >5, consider vitamin K_1, 1 - 2 mg orally or 0.5 - 1 mg IV.

1. Bleeding risk increases exponentially from INR 5 to 10; 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 the following: active gastrointestinal disorders (such as peptic ulcer or inflammatory bowel disease); receiving concomitant antiplatelet therapy; major surgical procedure within the preceding two weeks; known liver disease; and those with a low platelet count (<50 x 10^9/L).
4. 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.
   - In all situations carefully reassess the need for ongoing warfarin therapy.

15.9.3 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 171. See also *Warfarin Overdosage* on page 137.

15.9.4 Bleeding Following Thrombolytic Agents

Abnormal haemorrhage may be very difficult to correct for some hours. If fibrinogen level is low, cryoprecipitate may help. Discuss with Haematologist.

15.9.5 Bleeding Following Dabigatran

- STOP dabigatran.
- Attempt to control the bleeding and give all general supportive measures.
- There is currently no specific reversal agent for dabigatran and its anticoagulant effect will not be reversed by the administration of vitamin K or plasma infusions such as FFP.
- For the latest advice on the control of haemorrhage associated with dabigatran see the PHARMAC website Guidelines for management of bleeding with dabigatran.

15.10 Prevention of Stroke and Systemic Embolism in Atrial Fibrillation (AF)

- The prevention of arterial embolism in chronic AF has until recently relied upon aspirin or warfarin.
- Dabigatran, a direct inhibitor of thrombin, has been funded (July 2011) to prevent embolism in AF.
- The choice between warfarin and dabigatran will need careful consideration.
- Warfarin has many advantages; it is inexpensive, has no known late effects, the anticoagulation effect can be reversed, and many patients are easily stabilized on the drug.
- Dabigatran and similar drugs may be useful for patients who dislike venepuncture, are difficult to stabilize on warfarin, live away from blood testing centres, or travel frequently. Dabigatran has fewer known drug interactions than warfarin, but caution is needed with the concurrent use of P-gp inhibitors such as verapamil, amiodarone and ketoconazole, and with P-gp inducers, e.g., rifampicin. Dabigatran is not affected by diet. Its half-life is short (approximately one third that of warfarin in the setting of normal renal and hepatic function) but its anticoagulant effect cannot be reversed. Currently dabigatran is considerably more expensive than warfarin.
- There is an increased risk of bleeding if either warfarin or dabigatran are given with antiplatelet drugs.

15.10.1 Management

- Warfarin should be given with the usual precautions. See *Treatment of VTE* (see page 130).
- Dabigatran is registered and funded for clot prevention in AF. This drug is excreted mainly by the kidney, so dosing appropriate to renal function is important. Dabigatran has a fraction excreted unchanged in the urine (fu) of 0.8.
  - Dabigatran when used to prevent thrombosis in atrial fibrillation: the standard recommended dose is 150 mg BD PO. This drug should be used with **caution in the elderly**, and a lower dose of 110 mg BD PO is recommended for patients over 80 years old.
  - **Dosage in renal impairment:**
    - The first dose is the same as the standard recommended dose for the indication.
    - Subsequent doses are adjusted if the creatinine clearance (CrCl) is <80 mL/minute (Cockcroft and Gault), as shown in the table below, based on the fu of 0.8.
    - Doses may need to be rounded to accommodate the available capsule sizes (75 mg, 110 mg, 150 mg).

Table 39 Dabigatran dosage in renal impairment

CrCl (mL/min)	Dosage of dabigatran recommended	Suggestions for AF dosage (1)
>80	standard dose	150 mg BD
51-80	60-85% of the standard dose	110 mg BD
30-50	45-60% of the standard dose	75 mg BD
<30	Dabigatran not recommended	

1. Higher or lower doses than these suggestions may be chosen according to the individual patient's thrombotic and haemorrhagic risks.

- **Before using dabigatran, refer to:**
  - The PHARMAC website Guidelines for testing and perioperative management of dabigatran and Guidelines for management of bleeding with dabigatran.
  - The Medsafe datasheet on dabigatran.
  - The CDHB Clinical Pharmacology bulletin on dabigatran etexilate (July 2011).

15.11 Acute Limb Ischaemia

15.11.1 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).

15.11.2 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.

15.11.3 Actions

1) Assess severity:
   - **Rutherford I**: Pain resolves at rest 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 an 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, urea and coagulation profile.
3) Contact Vascular Registrar/consultant on call.
4) Analgesia.
5) Give oxygen.
6) If no major contraindications, heparinize (5000 unit bolus of unfractionated heparin IV) to minimize 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.

16. Hyperbaric Medicine

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).

See also Hyperbaric Medicine (online at http://cdhb.health.nz under Specialist Care & Treatment).

16.1 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.*

16.2 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 any patients with unexplained symptoms after scuba diving for an opinion. If decompression sickness is suspected, prescribe 100% oxygen by mask.
  - Patients are admitted via ED under General Medicine. The first hyperbaric oxygen treatment is completed before arriving on the ward.
  - These patients have neurological symptoms and need daily full neurological examination recorded. They do not usually require further oxygen and can be up and around on the ward. There is a small risk of deterioration in their symptoms and, following full examination, require consideration of early re-treatment by the Hyperbaric Duty Doctor.
- Arterial gas embolism (from any cause, including iatrogenic, e.g., cardiopulmonary bypass).
- Anaerobic necrotizing soft tissue infections, irrespective of the suspected causative organism (e.g., clostridial myonecrosis, streptococcal necrotizing fasciitis); consider hyperbaric oxygen therapy combined in a planned way with surgery and antibiotics.
- *Carbon monoxide poisoning* (see page 232) (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.

16.3 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.health.nz
- **Mail:** Duty Medical Officer, Hyperbaric Medicine Unit, Christchurch Hospital, Private Bag 4710, CHRISTCHURCH.

16.4 Non-Acute Indications for Hyperbaric Oxygen

Refer to the HMU section of the CDHB intranet. Think hyperbaric for people with diabetes or post-radiation injury.

- Osteo- and soft-tissue radionecrosis, including planned surgery in a previously irradiated area. Consider hyperbaric oxygen therapy in any patient with a wound, surgical or not, after radiation treatment. Patients with proctitis, colitis or cystitis gain marked symptom relief from treatment.
- Diabetes-related ulcer and other selected "problem" wounds (including preparation for grafting). Refer any patient with a lower leg wound to the ***Diabetes Centre*** (see page 79) and copy to the Hyperbaric Medicine Unit.
- Refractory osteomyelitis.
- Non-healing chronic wounds. The nursing staff in HMU are wound care experts and many chronic wounds heal more quickly with a combined approach with hyperbaric oxygen therapy. Refer any patient with a problem wound not healing with usual measures.

These conditions require extended treatment courses (4-8 weeks, 2 hours daily). Referrals will be considered from both General Practitioners and Hospital Specialists. It is a big time commitment for patients and their families. The cost of treatment to the health system of additional referrals is very low, as treatment uses few consumables and the staffing and equipment is the same irrespective of the number of patients treated.

Some other conditions may be considered on an individual patient basis.

Risks

Hyperbaric oxygen therapy is usually safe and well tolerated. Risks include claustrophobia and anxiety about closed spaces; injured ear drums, sinuses and teeth (painful but usually heal); reduced eyesight (temporary); blood pressure rise (temporary); cough and sore chest with long treatments; oxygen convulsions (uncommon and rarely lethal); fire (very rare but lethal); collapsed lung (uncommon and rarely lethal).

Reference: Cochrane Database (search term 'hyperbaric oxygen')

17. Infectious Diseases and Tuberculosis

17.1 Infectious Diseases Department Information

Main Office

5th Floor, Riverside, ☎ 80951, fax 80952

Inpatient Services Ward 23 (temporary)

- Dr Mark Birch, Prof Steve Chambers, Dr Sarah Metcalf, Dr Alan Pithie
- Kate Gallagher - Home Intravenous Antibiotic Service (IVAS) Specialist Nurse
- Viki Robinson - Viral Hepatitis and HIV 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.
- Home 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.

See the CDHB Infection Prevention and Control Manual (search for "infection prevention" at http://cdhb.health.nz).

17.2 Acute Bacterial Meningitis

17.2.1 Clinical Features

Fever, headache, photophobia, neck stiffness and impaired sensorium. The latter may be the only sign in the elderly. Typically short duration of symptoms (<24 hrs) prior to admission.

17.2.2 Causes

- *S. pneumoniae*, *N. meningitidis*, *H. influenzae* (usually paediatric - rare since HIB vaccine). Listeria monocytogenes (immunosuppressed, elderly or pregnant).
- Leptospirosis, Gram negative bacilli (rare overall but important causes in neonates, the elderly, immunosuppressed, and post trauma), syphilis. Staphylococci (post-neurosurgical).
- Cryptococcus neoformans.

17.2.3 Pathogenesis

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

17.2.4 Investigations

- Blood cultures - 2 sets before antibiotics given.
- Lumbar puncture. **Caution - see *Management* on page 144 and *lumbar puncture technique* on page 57.**
- **Obtain a CT/MRI head scan urgently before doing a lumbar puncture if:**
  - There is clinical evidence of **raised intracranial pressure/space occupying lesion** (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.*

- Lumbar puncture: collect 2 mL of CSF into each of three numbered sterile vials. Send to Microbiology. Request 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 is available, but not routine.
- CBC + diff.
- Na, K, glucose, creatinine, AST, GGT, ALP, bili.
- Chest and sinus X-rays (not all cases).
- Coagulation profile.
- Special tests needed for cryptococcus, TB, viruses, amoeba - consult Microbiologists, if indicated.

Table 40 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

17.2.5 Initial Management

- **When to lumbar puncture (see page 57):**
  - If no contraindications to lumbar puncture, perform this as soon as possible and prior to administering antimicrobial therapy.
  - If patient is stable and needs a CT brain, do not administer antibiotics but arrange an urgent CT scan of brain.
  - If patient unstable or deteriorating rapidly, then administer antibiotics prior to CT scan. **Give dexamethasone and antibiotics.** Lumbar puncture promptly following CT scan if no radiological or clinical contraindications.
- **Start dexamethasone 10 mg q6h IV.**
- **When to start antibiotics:**
  - After dexamethasone has been given.
  - If patient unstable, reduced level of consciousness or unconscious, start antibiotics immediately.
  - If patient stable, delay antibiotics until after LP has been performed.
  - If there will be a delay while waiting for CT brain.
- **Which antibiotics to start:**
  - If between 15 and 60 years, and no evidence of immunosuppression, chronic sinus disease, or long term administration of antibiotics: give IV ceftriaxone 2 g q12h.
  - If over 60 years or evidence of immunosuppression, alcoholism or diabetes: give IV ceftriaxone 2 g q12h and IV amoxicillin 2 g q4h - q6h to cover potential Listeria monocytogenes infection.
  - If sinus disease or recent long term administration of antibiotics: give IV ceftriaxone 2 g q12h and IV vancomycin 30-40 mg/kg/day in 2 divided doses (see the Antimicrobial Guidelines in the Pink Book for dosing).
- **Early treatment modification:**
  - **If CSF suggests viral meningitis or shows *N. meningitidis*, stop steroids.**
  - If CSF shows *N. meningitidis*, use either IV benzylpenicillin or IV ceftriaxone.
  - If evidence of pneumococci in CSF either on Gram strain or by antigen test, continue treatment with both IV ceftriaxone 2 g q12h and vancomycin as per Pink Book dosing guidelines until sensitivities known and continue IV dexamethasone 10 mg q6h for 4 days. If there is renal impairment, monitor vancomycin levels and seek advice. See also the Pink Book. **Consult Infectious Diseases Service.**
- If in doubt, phone the Infectious Diseases Physician on call.

17.2.6 Subsequent Management

The spread of pneumococcal strains which are resistant to penicillin and ceftriaxone has led 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:
  - The following recommendations apply to all patients over 15 years. This includes previously well patients, and those with complicating pre-existing illness such as ear or sinus disease, immunosuppression, or recent pregnancy.
  - 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.
  - 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. Check with ID Consultant first.
  - Meningococci must be cleared from the nasopharynx before the end of the treatment period. Give ciprofloxacin 500 mg as a single oral dose or rifampicin 600 mg BD PO for 2 days. This is not necessary if the patient has received ceftriaxone therapy.
  - Close household contacts of patients with meningococcal meningitis should be given ciprofloxacin 500 mg stat as prophylaxis as above as soon as diagnosis made. Throat swab not necessary. If pregnant give ceftriaxone 250 mg single dose IM or IV. Public Health Unit staff will trace contacts and arrange for further appropriate prophylactic antibiotics to be given.
  - Notify MOH if applicable.
  - Under 15 years seek paediatric advice.

17.3 Aseptic Meningitis

- Clinical and laboratory evidence for meningeal inflammation with negative investigations for bacteria.
- Often has similar presentation to acute bacterial meningitis with fever, headache, neck stiffness and altered sensorium.
- Lumbar puncture should be performed in all patients with suspected meningitis, unless contraindication. See *Lumbar Puncture* on page 57 and *Usual CSF Patterns in Meningitis* on page 144.
- Most often due to viruses - enteroviruses, *herpes simplex* (see page 151), VZV (PCR tests available) and mumps. 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, syphilis, leptospirosis, neoplasia, drugs, cyst related, Mollaret's, SLE, Behçet's, sarcoidosis, amoeba, and others. Accurate cytology essential.
- Approach to the patient:
  - Obtain careful history including travel, exposure to other individuals with similar illness, animals, tuberculosis, sexual activity, drugs.
  - Consider non-viral / non-infectious aetiologies.
  - Consider clues on physical examination – exanthems (enterovirus, HIV, syphilis), vesicular lesions (HSV, VZV), cervical lymphadenopathy (HIV), parotitis (mumps).
- Management:
  - For patients with suspected bacterial meningitis based on CSF findings, antibiotics should be initiated promptly.
  - Patients with probable viral meningitis (CSF WBC count <500, >50% mononuclear cells, protein <0.8 g/L, normal glucose and negative Gram stain) may be observed without antibiotic therapy.
  - However if patients are elderly, immunocompromised or have received prior antibiotics, they should be given antibiotics.
  - When it is not clear if it is a viral or bacterial process, patients may be observed without antibiotics or given antibiotics. Repeat lumbar puncture may be useful.
  - Specific treatment is necessary for meningitis due to primary HSV, VZV, leptospirosis, syphilis, fungi and TB.
- Consult Infectious Diseases Service.

17.4 Tuberculous Meningitis

See the *Tuberculosis section* on page 154.

17.5 Encephalitis

See *Encephalitis in the Neurology section* on page 179.

17.6 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 18e Edition. The **systemic inflammatory response syndrome (SIRS) is the consequence of a dysregulated host inflammatory response which causes multiple organ dysfunction syndrome (MODS). SIRS may be a response to both infectious and non-infectious disorders**. It is defined as being present when two or more of the following criteria are met:

- Temperature >38°C or <36°C.
- Heart rate >90/min.
- Respiratory rate >20/min or $PaCO_2$ <32.
- 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.

17.6.1 Clinical Situations which may predispose to Septicaemia

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

17.6.2 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 54).

- 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, ALP, bili.
  - CXR.
  - Arterial blood gases.
  - Serum lactate.
- Review recent microbiology culture results if available.

17.6.3 Management

Fluids:

- Resuscitate with sodium chloride 0.9%. 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 65). Consult ICU.
- If patient remains hypotensive (systolic <80 mm Hg) despite adequate hydration (approximately 2 L over 1 hour) then inotropic support will be needed. Consult ICU.
- Fluid management in septic shock can be difficult.

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 respiratory support.
- Severe *acidosis* (see page 96) 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 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 (the Pink Book) will give guidance as to which drugs to use initially.
- In hospitalized patients organisms may have been previously isolated and the sensitivities available.
- If infection is cryptogenic (no primary site identifiable) then IV 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:
    - Amoxicillin + gentamicin + metronidazole.
    - Cefuroxime + metronidazole.

- *Cellulitis* (see page 149): Flucloxacillin.
- Pseudomonas sepsis:
  - Piperacillin/tazobactam + tobramycin.
  - Ceftazidime + tobramycin.
- Urinary tract:
  - Gentamicin.
  - Ceftriaxone.
- Neutropaenic fever/sepsis:
  - Piperacillin/tazobactam 4.5 g IV q8h plus gentamicin 5-7 mg/kg IV in 100 mL sodium chloride 0.9% over 30 min q24h

  or, if there is a history of penicillin allergy,
  - Meropenem 1 g IV q8h plus gentamicin 5-7 mg/kg IV in 100 mL sodium chloride 0.9% over 30 min q24h.

***Note**: See the gentamicin/tobramycin dosing guidelines in the Pink Book.*

- Oxygen therapy:
  - *Give oxygen if patient hypoxic* (see page 255).

17.7 Rheumatic Fever

- Rheumatic fever (RF) remains a major problem in New Zealand. The New Zealand Heart Foundation Guideline gives detailed advice on diagnosis, management, and secondary prophylaxis.
- There is also a CDHB Rheumatic Fever Prophylaxis Register of patients requiring secondary prophylaxis within the CDHB area - see below. Please contact Paediatrics for patients aged 15 or under. See HealthPathways for RF management in primary care.

Secondary Prophylaxis

- There is reasonable evidence that regular administration of **benzathine benzylpenicillin** prevents group A streptococcal pharyngitis and recurrent acute rheumatic fever. Note that this is intramuscular benzathine benzylpenicillin, **not** benzylpenicillin sodium (penicillin G).
- Duration of secondary prophylaxis:
  - Persons with a history of acute rheumatic fever with no or mild carditis: minimum of 10 years after most recent episode or until 21 years of age, whichever is longer.
  - All persons with history of acute rheumatic fever with moderate carditis: minimum of 10 years after most recent episode or until age 30, whichever is longer.
  - All persons with history of acute rheumatic fever with severe carditis: minimum of 10 years after most recent episode until age 30, with specialist review for consideration of longer prophylaxis.
- Dosage:
  - In New Zealand it is recommended that **900 mg (1.2 mega units) of benzathine benzylpenicillin IM** should be used for secondary prophylaxis for all persons weighing 20 kg or more. Dosing is by intramuscular injection every 28 days.
  - For patients who are penicillin allergic or if intramuscular is declined, please see the New Zealand Guideline for rheumatic fever.
- **CDHB Rheumatic Fever Prophylaxis Register:** All patients in Canterbury, West Coast, and South Canterbury who qualify for rheumatic fever prophylaxis should be notified to Community and Public Health, phone 03 364 1777 or fax 03 379 6484. Prophylaxis is usually administered in a primary care setting. Community and Public Health will liaise with the general practice.

17.8 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 desensitization 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.

17.9 Cellulitis and Soft Tissue Infections

- 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 146), 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.
  - In patients who are immunocompromised or who are not responding to standard treatment, consider subcutaneous aspirate or skin biopsy.
- Antibiotics:
  - **Uncomplicated cellulitis of uncertain aetiology:**
    - Use flucloxacillin 1-2 g IV q6h, until defervescence of fever, patient improved symptomatically or improved appearance of cellulitis, 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 clarithromycin IV with early switch to roxithromycin, or clindamycin.

Note: *Cellulitis commonly appears to worsen in the first 72 hours but this should not automatically lead to a change in antibiotics unless the patient's overall condition (e.g., fever, pulse, BP) deteriorates. Typically over this time the patient improves symptomatically and starts to defervesce. Community acquired MRSA infections are increasingly recognized. Consider these in all patients with cellulitis which is not responding to beta-lactam antibiotics.*

  - **Complicated cellulitis:**
    - Cellulitis associated with burns, chronic venous 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: Amoxicillin/clavulanate 1.2 g IV q8h with or without gentamicin **or** cefuroxime + metronidazole **or** flucloxacillin + gentamicin + metronidazole.
  - **Erysipelas and cellulitis** thought to be caused by Group A Streptococci can be treated by penicillin alone. Give benzylpenicillin 1.2g q4h IV.
  - **Necrotizing skin and soft tissue infections**:
    - **Early on these are very difficult to distinguish from cellulitis / soft tissue infections and diagnosis is frequently delayed leading to high morbidity and mortality.** Changes of skin necrosis and anaesthesia are late signs and require urgent management.

- Suggestive clinical features include severe / disproportionate pain, tenderness beyond visible erythema, bullae formation, haemorrhagic bullae, necrosis and anaesthesia.
- The Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score can help distinguish necrotizing fasciitis from other soft tissue infections.

Table 41 Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC)

Variable		Score
C-reactive protein (mg/L)	<150	0
	≥150	4
White Blood Cell Count (10^9/L)	<15	0
	15-25	1
	>25	2
Haemoglobin (g/L)	>135	0
	110-135	1
	<110	2
Sodium (mmol/L)	135 or more	0
	<135	2
Creatinine (micromol/L)	141 or less	0
	>141	2
Glucose (mmol/L)	10 or less	0
	>10	1

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- A score of ≥6 should raise suspicion of necrotizing fasciitis and a score of ≥8 is strongly predictive of the disease.
- Rescoring of patients during admission who aren't responding to treatment is also useful.
- **Management: IV flucloxacillin 2 g q4h-q6h + IV clindamycin 600 mg q6h + IV gentamicin**. If penicillin allergy, give **IV 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.

17.10 Infection with Antibiotic Resistant Organisms

- Infection with antibiotic resistant organisms 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 hospitalized within two years, either in New Zealand or overseas. Community acquired MRSA infections are of increasing concern. 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 colonized 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 immunosuppressed, 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 IV vancomycin to IV ceftriaxone until sensitivities available. See *Meningitis* on page 143.
- **Extended spectrum beta-lactamase inhibitor (ESBL) producing Gram negative bacilli.** These are being isolated increasingly commonly, including in the community as a cause of urinary tract infections and in the hospital setting, especially in patients with prior antibiotic exposure or who have recently travelled overseas. Consult Infectious Diseases regarding treatment of infections with these organisms.

17.11 Herpes Simplex

17.11.1 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 generalized 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 immunofluorescence with a cotton swab. Place in viral transport medium to send to the laboratory.
- **Treatment**:
  - **Primary herpes infection:**
    - Aciclovir 400 mg PO 3 times daily for 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 400 mg PO 3 times daily only of benefit if started early.
    - Immunosuppressed - seek advice.
  - **Prophylaxis if indicated:**
    - Aciclovir 400 mg PO BD.

17.11.2 Herpes Simplex Meningitis

- **Primary attack**:
  - Usually occurs with systemic illness and ulceration.
  - Aciclovir 10 mg/kg IV q8h which can be switched to PO aciclovir on discharge for 10 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.
  - Immunosuppressed - aciclovir 5 mg/kg IV q8h for 5 days.

17.12 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.
  - 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.

17.13 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.

17.13.1 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 the on-call Infectious Diseases Physician (not the Registrar) **immediately** for advice. 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.

17.13.2 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 letter of first name.
  - M or F (Sex).
  - Date of birth (DDMMYY).

17.13.3 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.

17.13.4 Clinical Presentation

- Acute infection:
  - "Mononucleosis-like" fever, lymphadenopathy, sore throat, truncal rash (maculopapular), diarrhoea.
  - Aseptic meningitis.
  - These patients are **infectious.** Current generation HIV antigen/antibody EIA tests are likely to be positive early in the course of the infection from as early as 10 days onward. If negative and high clinical suspicion this should be repeated and consider viral RNA testing. Seek advice since treatment in the acute phase may be indicated.
  - If diagnosis suspected ensure a sexual, drug and blood transfusion history taken.
- Persistent generalized 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.

17.13.5 Some Specific Complications of Late Stage HIV Disease

May be presenting feature.

Pneumonia - *P. 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 jirovecii pneumonia.

- Pneumocystis jirovecii 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 immunofluorescence 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. 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 and Ophthalmology urgently.

GI Disease

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

Constitutional Disease

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

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

17.14 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. A specialist TB nurse can be contacted through Cardio-Respiratory Integrated Specialist Services (CRISS). The TB nurse works both in the hospital and in the community, can coordinate TB treatment including directly observed treatment (DOTS) in the community, and educates patients and staff on isolation procedures, TB disease and TB medications. Remember that TB is a ***notifiable disease*** (see page 284).

17.14.1 Incidence

The annual incidence of TB in New Zealand has fallen to 7-10 per 100,000 with 20 to 30 cases annually in Canterbury. There are marked differences between ethnic groups with higher incidences of TB in Maori (x5), Pacific Islanders (x10), and Asian ethnicity (x25) compared to Pakeha. 70% of cases of TB in NZ occur in people born outside NZ. In NZ 1% of patients diagnosed with TB also have HIV infection.

17.14.2 Diagnosis

A delay in diagnosis is often due to TB not being considered in the differential. This leads to prolonged infection risk as well as increased morbidity and mortality.

- **Risk situations for TB include:**
  - Previous residence in high-incidence TB country - especially if recent arrival in NZ.
  - Prolonged close contact with an infectious case, usually domestic contact over weeks or months. Consider possibility of institutional contact, i.e., refugee camps, prisons, rest homes.
  - Previous TB, especially inadequate treatment of previously active TB, e.g., a drug regimen that did not include both isoniazid (available since 1950) and rifampicin (available since 1965), noncompliance (treatment not directly observed), or an unknown treatment regimen.

- Radiological or pathological evidence of previously unrecognized, and therefore untreated, naturally remitting TB, such as abnormal calcification on radiograph, or granulomatous inflammation on surgically resected tissue.
- Immunosuppression by disease or medication - including TNF-alpha inhibitor treatment.

- **Clinical scenarios:**
  - Pulmonary TB:
    - Approximately 2/3 of TB cases are pulmonary TB.
    - May be asymptomatic initially but with radiological change and still have active disease. Will eventually progress to becoming symptomatic.
    - Cough - dry or productive, haemoptysis.
    - Chronic respiratory symptoms with chest X-ray abnormality, particularly bilateral upper zone pleuropulmonary fibrosis with cavitation, calcified pulmonary nodules, or calcified intrathoracic lymph nodes.
    - Persistent or recurrent pleural effusion - usually lymphocytic exudate.
  - Lymphadenopathy or lymph node suppuration.
    - Patients typically have chronic unilateral non-tender lymphadenopathy without systemic symptoms. This usually involves the cervical nodes, but other node groups may be affected. Rarely nodes become fluctuant or develop a draining sinus.
    - TB lymphadenitis should be considered in patients from high endemic countries with chronic lymphadenopathy and any lymph node aspirates or biopsies taken from such patients should be sent for TB culture as well as cytology / histology.
  - Chronic spinal osteomyelitis, persistent septic arthritis.
    - TB causing osteomyelitis (typically spinal) or septic arthritis usually presents with local pain which becomes increasingly severe over weeks to months. Joint disease may also present with swelling and progressive loss of joint function. In patients with subacute presentation TB should be considered and diagnostic samples should be sent for TB culture as well as routine bacteriology, especially if the patient is from a high risk country or at risk of TB exposure in the past.
  - Chronic diarrhoea with/without ascites. Often with systemic symptoms of weight loss, fever and malaise.
  - Sterile pyuria - if other common causes excluded.
  - Pyrexia of unknown origin.
  - Chronic meningitis.
    - Patients present with a subacute febrile illness associated with headache and often progressing through personality change, confusion, cranial neuropathies and long tract signs to coma and paralysis. They may have features of meningism as well as vomiting, lethargy and seizures. Approximately 1/3 of patients will have miliary tuberculosis.
    - The CSF typically shows a mononuclear pleocytosis, low glucose and high protein. Demonstration of acid fast bacilli (AFB) in the CSF is the most effective way to make the diagnosis and diagnostic yield of both AFB stain and TB culture increases with repeated (up to 4) LPs.
    - CSF PCR for TB is available but has poor sensitivity despite high specificity. A large volume (e.g., 10 mL) of CSF is needed for TB testing.
- Disseminated or miliary TB is rare but has a high associated mortality.
- All patients diagnosed with extrapulmonary TB should have CXR and consideration as to whether they have concomitant pulmonary involvement as this frequently co-exists and requires respiratory isolation.

17.14.3 Respiratory Isolation

- Not all patients with TB need admission and some are treated in the community. Home isolation is often possible and the TB nurse from CRISS may be available to help assess the feasibility and safety of this and educate the patient and their family.
- If a case of pulmonary TB is suspected in the community but sputum results are still awaited, home isolation procedures should be put in place pending results.
- All suspected inpatient cases of pulmonary or laryngeal TB should be placed in respiratory isolation, ideally in an airborne isolation (negative pressure) room, pending confirmation (or exclusion) of the diagnosis.
- Staff must wear a fitted particulate respirator / N95 mask.
- Patients must wear a surgical mask if they need to leave the isolation room e.g., for investigations, transfer etc.
- See the CDHB Infection Prevention and Control Manual (search for "infection prevention" at http://cdhb.health.nz).

- Extrathoracic TB and pleural TB without pulmonary involvement are **not** infectious. However pulmonary TB commonly coexists with extrathoracic TB and infection risk should be investigated. For example 70% of patients with cervical or supraclavicular nodal TB will also have active pulmonary TB.

17.14.4 Multi Drug Resistant TB

Multi Drug Resistant TB (MDR TB) is diagnosed in NZ however <1% of cases are MDR TB. Suspect MDR TB in:

- Cases from high-incidence areas.
- Previously treated but relapsed disease.
- Contacts of a MDR case.

All suspected MDR TB cases must be strictly isolated in a negative pressure room.

- If MDR TB suspected and TB confirmed on initial samples, i.e., AFB-positive sputum, discuss with microbiology proceeding to rapid drug sensitivity testing. The geneXpert system™ can detect rifampicin resistance on AFB positive sputum within hours.
- All MDR TB must be treated in consultation with a specialist centre and must involve Respiratory or Infectious Disease team input.

17.14.5 Collection of Specimens

In cases of suspected TB an early and vigorous attempt should be made to collect relevant specimens for mycobacterial culture. In most cases treatment should not commence until the diagnosis is confirmed.

- Three spontaneous or induced sputum samples (contact Physiotherapy department) 8-24 hours apart, or bronchoalveolar lavage at bronchoscopy. Induced sputum has a higher yield than bronchoscopy. Bronchoscopy may be preferred if other differentials such as malignancy are being considered.
- Pleural fluid aspirate - positive culture in only 10-35% cases. Pleural biopsy increases yield to 60-80% and should be pursued if diagnosis uncertain.
- Joint fluid aspiration.
- Lymph node aspirate (positive in only 33% cases) or excisional biopsy of node.
- Early morning urine x3 for TB culture if sterile pyuria present.

Note: *Acid fast bacilli or TB culture must specifically be requested when samples are sent for microbiology. Tissue should be sent in saline for TB culture.*

17.14.6 Interferon Gamma Release Assay (IGRA) – Quantiferon-TB Gold

- The Mantoux test and IGRA both test for TB exposure/infection but do not distinguish latent TB infection from active disease.
- Latent TB is 'dormant' TB infection in the absence of active disease.
- These tests are never diagnostic for active TB and should not be performed routinely when investigating for active disease. Rather, positive cultures should be sought.
- A positive result may help increase clinical suspicion in occasional difficult cases. The sensitivity of IGRA in active disease may only be 80% and therefore cannot be used to exclude active disease.
- IGRA is not affected by past BCG vaccine (unlike the Mantoux test), is unaffected by most non-TB mycobacterial infection and requires one blood test.
- IGRA may be used in place of a Mantoux for the diagnosis of latent TB especially in BCG vaccinated individuals or the immunocompromised. In TB contact cases an IGRA may better predict those at risk of developing active disease.
- Active disease must be excluded before a diagnosis of latent TB can be made. If latent TB is diagnosed a decision must be made whether to treat. This should involve specialist input. Recent contacts of TB cases and those with impaired immunity due to illness or medications may require treatment to prevent progression to active disease.

17.14.7 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 amoxicillin/clavulanate.

- All patients diagnosed with active TB should be offered HIV testing.
- The NZ Guidelines for TB (see below) have recently been updated and are available online. They are an excellent resource.

Reference: Guidelines for Tuberculosis Control in New Zealand 2010 www.moh.govt.nz.

17.15 Malaria

17.15.1 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.**

17.15.2 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.*

17.15.3 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.
- Na, K, creatinine, AST, ALT, GGT, ALP, bili, glucose.
- CXR.
- Blood culture.
- Urinalysis.

17.15.4 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***.

  Note: *All pregnant women with malaria should be admitted.*

  Note: If the patient has acquired P. vivax in Papua New Guinea then admission and close observation may be needed. *These patients should be treated with atovoquane / proguanil (see below).*

  Otherwise, treatment is:

  - Chloroquine: 1000 mg stat PO, 500 mg 6 hours later then 500 mg daily for 3 days or hydrochloroquine 800 mg stat, then 400 mg 6 hours later then 400 mg daily for 3 days.
    - If neither are available, 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. Discuss with the Infectious Disease Service if the patient is not already under their care.

      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% glucose 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. 8 hours from the start of the loading dose give quinine 10 mg/kg over 4 hrs by IV infusion then 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.*

17.15.5 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.

17.16 Immunization Information for the International Traveller

Information on immunization for overseas travel is available from specialized travel clinics or from the following internet web page: www.fit-for-travel.de.

17.17 Causes of Fever in the Returning Traveller

These include malaria as well as typhoid and paratyphoid fevers, dengue fever, typhus, Legionnaires' disease, tuberculosis, amoebic liver abscess, hepatitis (viral), respiratory viruses including influenza and viral haemorrhagic fever.

17.17.1 Investigations

- Blood cultures - 3 sets.
- Urine culture.
- Stool culture.
- CBC + diff, blood film and eosinophil count.
- Coagulation profile for DIC.
- Na, K, creatinine, AST, ALT, GGT, ALP, 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 unless patient has known severe adverse effects from ceftriaxone. In that case, use ciprofloxacin 400 mg q12h IV, or 500 mg BD PO, until sensitivities are known. Aminoglycosides are ineffective.

18. Nephrology

18.1 Nephrology Department Information

Main Office

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

Inpatient Care Ward 14

- Dr Nick Cross, 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 kidney injury, chronic renal failure, drug-induced renal disease, urinary tract infections, renal hypertension, systemic diseases involving the kidney (including diabetic nephropathy), electrolyte disturbances, dialysis or kidney transplant patients under other services.

Acute Dialysis Unit

- 3rd Floor, Parkside West, ☎ 89108, fax 89109

Home Dialysis Training Unit

- 550 Hagley Avenue, ☎ 80610

18.2 Acute Kidney Injury

Acute kidney injury (AKI) is defined by a recent elevation of plasma creatinine. 30% of patients are not oliguric and some may be polyuric. The following are important aspects of the management of acute kidney injury.

- 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 kidney injury 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!

18.2.1 Causes

- Pre-renal:
  - Hypovolaemia/hypotension related to volume depletion, pump failure or vasodilatation.
- Renal:
  - Nephrotoxins including drugs and chemicals (including 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.

18.2.2 Investigations

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

- Abdominal and rectal 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. 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.
- 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 274). These tests should not usually be ordered out of normal working hours and the clinical problem should be discussed with a Physician (call Lab if wanted urgently).
- Urgent renal biopsy may be indicated, particularly when the urine sediment is active, i.e., red cells, casts, suggesting glomerulonephritis.

18.2.3 Management

- Stop any potentially nephrotoxic drugs.
- Ensure optimal hydration with appropriate fluid - blood or sodium chloride 0.9%. When the patient has been rehydrated give 600 mL plus urine output and other losses per 24 hours, either as oral fluid or 5% glucose. Replace sodium losses as sodium chloride 0.9% within this volume.
- Do not give diuretics unless the patient is volume overloaded.
- Ureteric obstruction - consult Urology team urgently.
- Bladder outlet obstruction - catheterize. Refer to Urology urgently if unable to do this - beware high sodium diuresis.
- Hyperkalaemia. This may be immediately life threatening and should be treated according to its severity.
  - Haemolysis or delay in sample process may cause spurious hyperkalaemia. Consider a repeat sample to confirm.
  - The response to hyperkalaemia should reflect the clinical situation, and the following is a guide only. Monitor any therapy given with repeat potassium measurements.

Table 42 Treatment of Hyperkalaemia

Plasma Potassium	Treatment
5 - 6 mmol/L	Rarely an issue unless acute rise from very low concentration. Consider dietary advice if chronically elevated. Consider risk/benefit of drugs that increase potassium.
6 - 7.5 mmol/L	Check ECG. Withhold ACE inhibitors, angiotensin receptor inhibitors, and potassium sparing diuretics. Stop any NSAIDs/potassium supplements. Rehydrate if dehydrated (sodium chloride 0.9% is best). Consult Nephrology urgently if not rapidly reversible or if patient is oliguric. To lower extracellular potassium acutely by diverting into cells use: ▸ 100 mL of 50% glucose (dextrose) IV over 15-30 minutes plus 10 units actrapid insulin IV, or ▸ 100 mL of 8.4% sodium bicarbonate (100 mmol) over 4 hours if metabolic acidosis and 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 urgent dialysis.

Note: *Treatment of hyperkalaemia with insulin and glucose or with bicarbonate produces only temporary reduction in potassium. Salbutamol (usually given as continuous nebulizer) has a similar effect but may cause marked tachycardia and/or tremor. Routine use is not recommended.*

Note: *Potassium elimination occurs via kidneys, or bowel. Bowel elimination may be increased by use of sodium resonium but there are significant potential side effects. Routine use is not recommended - consult Nephrology to discuss.*

- **Indications for urgent dialysis** - discuss with Nephrologist:
  - K >7.5 mmol/L unresponsive to above therapy - discuss.
  - Pericarditis.
  - Cardiac failure or fluid overload not responding to diuretics.
  - pH <7.1 mmol/L causing clinical consequences and unresponsive to appropriate therapy.

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

Urine Chemistry in Oliguria

A high urinary sodium >20 mmol/L, in the absence of diuretic therapy, may indicate established acute tubular necrosis and predict a poor response to appropriate pressure and volume resuscitation. Similarly a urine to plasma osmolality of <1.1 may predict poor response.

18.3 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 52). This is particularly important for drugs such as low molecular weight heparin, dabigatran, the aminoglycosides, cephalosporins, ranitidine, digoxin, ACE inhibitors and some beta-blockers. Drugs which are metabolized 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 (including COX-2 inhibitors).

ACE inhibitors are used widely for the management of hypertension, chronic kidney disease and cardiac failure. However, patients may develop AKI due to an excessive dosage related to the renal clearance of the drug or in the presence of renovascular disease. This is more likely to occur if the patients are also taking a diuretic or NSAID, or are dehydrated from any cause. ACE inhibitors should be stopped before surgery and should not generally be used in combination with potassium-sparing diuretics, potassium supplements, or NSAIDs. Refer to the recommendations for the doses of quinapril and enalapril in renal impairment.

18.4 Renal Function - Assessment

Glomerular filtration rate (GFR) is the best measure of renal function. The plasma creatinine concentration alone is not a sufficiently accurate predictor of glomerular filtration rate, particularly for small or elderly patients.

- In practice GFR is estimated either by the Cockroft and Gault or eGFR formulae. Both provide a guide to GFR which is adequate for most clinical situations. These formulae are unreliable at extremes of weight and/or when the creatinine is changing and for a patient with a near normal GFR although the new CKD-EPI equation performs better in this regard.
- Note that GFR falls by approximately 0.75 - 1 mL/min per year over the age of 40.
- If renal function is impaired:
  - Modify the dose of drugs that are renally excreted.
  - Try to use drugs that are not nephrotoxic.
  - If a critical dose drug known to be nephrotoxic must be used, consider getting a more accurate guide to GFR such as a radionuclide GFR measurement.

Cockcroft and Gault Formula

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 modified formula is as follows:

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

Note: Use actual body weight if this is less than the ideal body weight.

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

CKD-EPI

This formula is used by Canterbury Health Laboratories to estimate GFR from measured serum creatinine. It takes into account serum creatinine, age and gender. It has now superseded the MDRD formula for estimating GFR based on serum creatinine at Christchurch Hospital. The prime reason for its adoption was that at near normal GFR, i.e., >60 mL/min, the CKD-EPI formula has less bias and greater accuracy.

Note: *All estimates of GFR from serum creatinine in patients with GFR >60 mL/min are still relatively approximate.*

18.5 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*.

18.5.1 Diagnosis

- The diagnosis is confirmed by culturing a mid-stream urine (MSU) specimen.
- When interpreting the number of bacterial colony forming units/L of urine (cfu/L) of any uropathogen, you will need to take into account the presence/absence of pyuria (epithelial cells indicate contamination) and symptoms, regardless of the patient's gender.

18.5.2 Investigations

- Always consider the question - "**Is this infection a pointer to some underlying abnormality in the urinary tract?"**
- In general the indications for investigations 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 or pyuria persists, or
  - Therapy has failed.
  - They have a history of kidney stones or symptoms suggesting renal colic.

18.5.3 Management of Lower Urinary Tract Infections

For treatment of a lower urinary tract infection 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 43 Drug Guidelines for Cystitis

Single dose
Norfloxacin 800 mg
Trimethoprim 600 mg[1]
3 day course
Norfloxacin 400 mg BD
Trimethoprim 300 mg daily
Nitrofurantoin 50 mg TDS (ineffective for Proteus)
Amoxicillin 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 prophylactic antimicrobial therapy.
- Try simple measures - increase fluid intake, increase frequency of micturition. In women, also consider treatment of cervical erosion or vaginitis, post coital voiding, application of an antiseptic cream to the periurethral area prior to intercourse.
- 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.

18.6 Acute Pyelonephritis

- A syndrome of fever (>37.8°C) ± rigors, loin pain or tenderness together with infected urine. If no fever or pyuria, 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 hospitalization.
- 10-15% will have a bacteraemia.

18.6.1 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**).

18.6.2 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 L (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 146); 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.

18.6.3 Management

- If the patient is dehydrated and/or vomiting, give IV sodium chloride 0.9%.
- Parenteral antimicrobial therapy usually consists of a single intravenous dose of antibiotic (e.g., gentamicin). The choices of **parenteral** agents are gentamicin - initial dose 3 mg/kg, ciprofloxacin 200 mg q12h, ceftriaxone 2 g q24h.
- Oral therapy (e.g., ciprofloxacin 250 mg BD) starts on the second day of treatment and is given for 5 days.

Notes:

- The aminoglycosides and quinolones are the drugs of choice.
- Check local sensitivity patterns for trimethoprim.
- Ampicillin or amoxicillin 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. Amoxicillin/clavulanate 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.

19. Neurology

19.1 Neurology Department Information

Main Office

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

Consultant Staff

- Prof Tim Anderson, Dr Roderick Duncan, Dr John Fink, Dr Deborah Mason, Dr Philip Parkin, Dr Jon Reimers.

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 Neurology Consult Registrar (pager 7175), 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.

19.2 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.

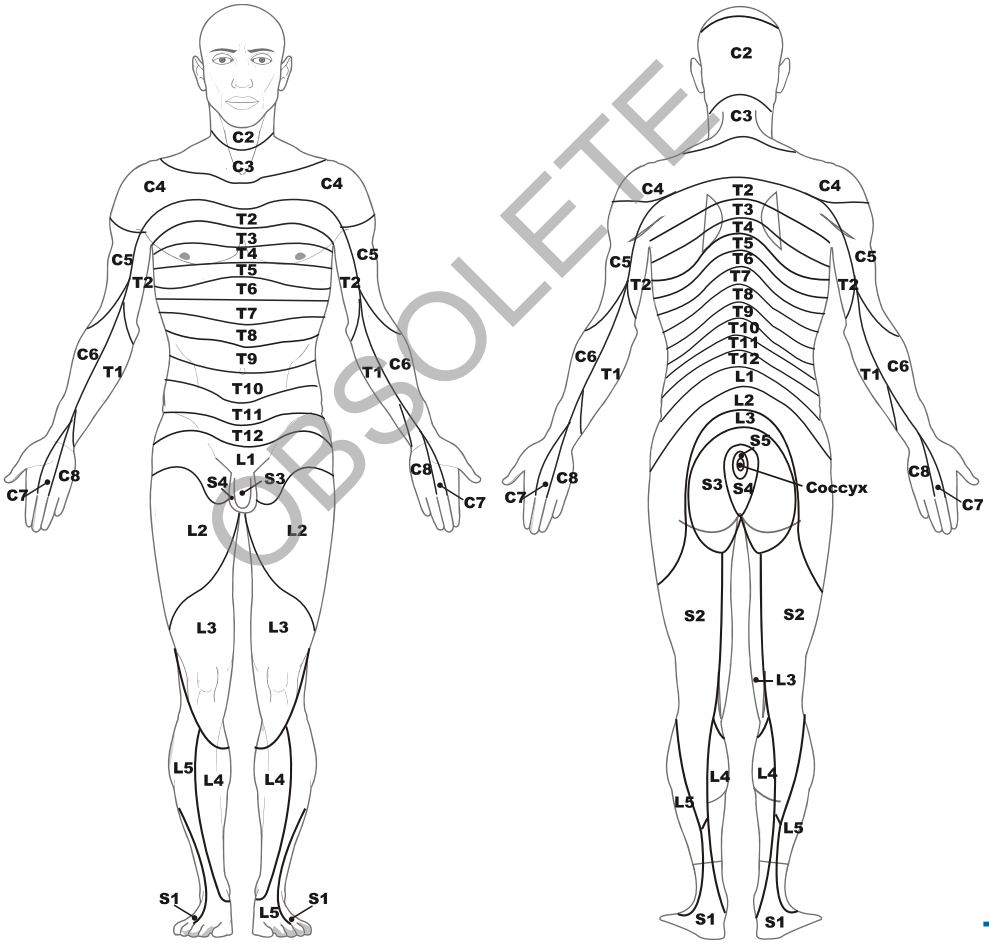
Reflexes

- 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

19.2.1 Dermatome Distribution



19.3 Stroke

19.3.1 Acute Stroke Unit (ASU)

Admission policies:

- The ASU will be transferred to Ward 24 Christchurch Hospital in December 2013.
- Patients with stroke may either be admitted to the ASU under the care of Neurology or under General Medicine (GM).
- General guidelines for the division of patients with stroke between GM and Neurology are as follows:
  - **Admission under Neurology** is indicated if specialized neurological assessment, monitoring, or management may be required. For example:
    - Patients considered for thrombolysis. This will be administered on Ward 24.
    - 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.
  - **Admission under General Medicine** is indicated for patients who present with stroke as a manifestation of systemic cardiovascular disease without other acute neurological issues.
    - These patients will be admitted to the Acute Medical Assessment Unit (AMAU), also in Ward 24, under one of the GM Teams on call.
    - When the diagnosis of stroke has been confirmed, the patient will be transferred to the ASU under the Acute Stroke Team.

Patients with stroke on other hospital wards:

- The Acute Stroke Team should be notified of all patients in the hospital with stroke and will visit them on their ward. Contact the Stroke Clinical Nurse Specialist on pager 8978 (0730-1600 Mon-Fri), or the designated Stroke Nurse on Ward 24, morning or afternoon shifts daily including weekends.

19.3.2 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.
  - Small vessel (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 (including warfarin, dabigatran, and LMWH use).
  - 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 175.

Cerebral Venous Sinus Thrombosis (CVT)

- Uncommon but important to recognize 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**.

19.3.3 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 174.
  - 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.
  - Thrombophilia screen, lupus anticoagulant, anticardiolipin antibodies, homocysteine (generally younger patients only).
  - Echocardiogram (transthoracic or transoesophageal; particularly if recent myocardial infarction, dilated cardiomyopathy, or if mitral valve disease or LV aneurysm suspected, or for younger patients (usually <50 years old) with no other cause identified (?patent foramen ovale - transoesophageal echo 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 CTV for suspected cerebral venous sinus thrombosis.
- CXR is **not** a routine investigation for stroke. Request as clinically indicated.

19.3.4 Acute Management of Ischaemic Stroke

- **Thrombolysis** with tissue plasminogen activator for acute ischaemic stroke.
  - May be considered for 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 cellphone via the hospital operator.
  - A detailed thrombolysis protocol is available in the Acute Stroke Unit and on ward 28 (search for "stroke guidelines" on the CDHB intranet).
- **Aspirin 150-300 mg daily** should be started once ICH excluded by CT (withhold 24 hours for patients receiving thrombolysis). After 48 hours, consider changing to clopidogrel 75 mg PO daily. See ***Secondary Prevention of Ischaemic Stroke*** (see page 172) for the range of antiplatelet drugs available.
- **Maintain patient 'homeostasis':**
  - Avoid aspiration pneumonia. Document bedside swallowing assessment for all patients.
    - Keep 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.
    - Subcutaneous route may be a good option if supplementation to poor oral intake is required.
  - Maintain euglycaemia.
    - Detailed guidelines are available in the ASU (search for "stroke guidelines" on the CDHB intranet). Particular care is needed for insulin-dependent diabetics who are made NBM following stroke.
  - It is reasonable to continue usual antihypertensives *(Lancet Neurol 2010;9:767-775)* but hypotension should also be avoided.
  - 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.

Table 44 Intravenous Labetalol Protocol

If systolic BP is 180-230 mm Hg or diastolic BP is 105-120 mm Hg (≥2 recordings, 5-10 minutes apart):

- Give IV labetalol 10 mg over 1-2 minutes.
- The dose may be repeated or doubled every 10-20 minutes up to a total dose of 150 mg.
- Monitor BP every 15 minutes during labetalol treatment and observe for development of hypotension.
- If BP is higher than the above limits, seek Specialist advice.

    - Detailed protocols are available in the Acute Stroke Unit. **Convert antihypertensive treatment to oral or NGT administration if continued IV boluses are required >24 hours.**
  - 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 >48 hours. For some patients, NGT feeding can be delayed safely up to one week from stroke onset. These decisions should be made in conjunction with the multidisciplinary team.
  - DVT prophylaxis: see the Thrombosis section - ***VTE prophylaxis for patients with ischaemic stroke*** on page 122.
  - Early mobilization out of bed within 24h should be expected.
    - Even short periods are beneficial, e.g., up to commode for toilet or sitting in a chair.
- **Heparin:** intravenous unfractionated heparin, subcutaneous unfractionated heparin, LMWH or heparinoids are not routinely recommended for treatment of patients with acute ischaemic stroke.
  - IV unfractionated 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.

19.3.5 Acute Management of Intracerebral Haemorrhage (ICH)

General ICH Management Guidelines

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 170). **There are some important differences, however:**

- Avoid aspirin, heparins, thrombolytic agents.
- Full coagulation screen is required urgently.
- If taking oral anticoagulants, this is a **life threatening emergency and immediate action to reverse the anticoagulant effect is required (if this is possible)**. See *Reversal of the Warfarin-related Coagulopathy* below, *Warfarin Overdosage* on page 137, or *Bleeding following Dabigatran* on page 139, as appropriate.
- 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 170.

  ***Note**: There is evidence a lower target of 140 mm Hg systolic is safe in acute ICH, but that this confers only marginal 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.
- 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.

  ***Note:** 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

- Intracerebral haemorrhage in a patient taking warfarin is a **medical emergency** with a mortality of between 43-70% at 30 days.
- 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, ongoing bleeding can continue for 24-48 hours. ICH volume and further expansion of ICH are both independent predictors of mortality.
- 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

- **Any** patient with both an acute intracerebral haemorrhage and taking warfarin should have immediate intravenous reversal of the coagulopathy. This includes:
  - **Stop warfarin.**
  - **Give vitamin K 5-10 mg IV immediately.**
  - **Give Prothrombinex VF 50 units/kg IV immediately.**
  - **Give fresh frozen plasma (FFP) 150-300 mL.**

Notes:

- Vitamin K takes 6-24 hours to be effective.
- Prothrombinex VF rapidly reverses the coagulopathy within 15 minutes. It is accessed by either:
  - Following Life-Threatening Bleeding on Warfarin protocol in the Emergency Department (search for "intracerebral haemorrhage" on the CDHB intranet) - warfarin reversal pack to accompany patient to CT scanner from ED, or
  - Contacting the New Zealand Blood Service doctor on call.
- If Prothrombinex VF is **not** available, vitamin K (as above) and larger doses of fresh frozen plasma (FFP) can be given (at a dose of 15-30 mL/kg) but produces suboptimal anticoagulation reversal.
- 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.

Oral thrombin inhibitors (Dabigatran) related ICH

- Like warfarin-related ICH, these patients need urgent assessment in an attempt to minimize the extent of the ICH. No specific method of reversal is available. See *Bleeding Following Dabigatran* on page 139.

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 for anticoagulation, location and severity of bleed, comorbidities, age and concurrent medications.

19.3.6 Secondary Prevention of Ischaemic Stroke

- **Clopidogrel 75 mg daily** is recommended as first-line treatment for all patients with ischaemic stroke not treated with oral anticoagulants, unless contraindicated.
  - Clopidogrel *monotherapy* is indicated for long-term stroke secondary prevention – it is an inexpensive and fully subsidized medication with a modest additional benefit when compared with aspirin.
  - Combination therapy of aspirin + clopidogrel is not indicated for long-term vascular secondary prevention due to increased bleeding risk.
- **Aspirin 75-100 mg daily + dipyridamole 150 mg BD** is an alternative treatment option, equally effective as clopidogrel.
- **Aspirin 75-100 mg daily** monotherapy may be a reasonable treatment for some patients.
- **Statin lipid-lowering therapy.** Atorvastatin 80 mg daily is proven to reduce the risk of recurrent stroke in patients with fasting LDL 2.6 or greater. Simvastatin 40 mg is proven to reduce the risk of recurrent vascular disease events in patients with random total cholesterol greater than 3.5 mmol/L.
  - Lower doses of atorvastatin or simvastatin may be required depending on patient tolerability.
  - At least two-year life-expectancy is required for patients to gain significant stroke-prevention benefit from statin treatment.
  - 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 the 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, which should be continued.
- **Warfarin** is recommended for cardioembolic stroke.
  - 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 had a stroke or TIA** the benefits of oral anticoagulants usually **far outweigh** the risk of haemorrhage (including risk of subdural haematoma due to falls) due to oral anticoagulants.
- In atrial fibrillation, **dabigatran** is available as an alternative to warfarin. The usual dose is 150 mg BD PO. This drug should be used with caution in the elderly. If it is given, a lower dose of 110 mg BD PO is recommended for patients over 80 years of age. Dabigatran should not be used for patients with severe renal impairment (CrCl <30 L/min). For full details of dabigatran therapy, see the *Thrombosis section* on page 139.
- **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 174.
  - 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 186).

19.3.7 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 179). 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 commenced. 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 170).

19.3.8 Rehabilitation

- Rehabilitation efforts should commence as soon as possible after stroke, e.g., mobilization 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. If not, early referral to The Princess Margaret Hospital Stroke Rehabilitation Unit (>65 y), or Burwood Hospital (Brain Rehabilitation Service for <65 y) should be made.

19.3.9 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 45 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 24 hours.

Note: *Urgent (same/next day) outpatient ultrasound is now available for high-risk patients (ABCD2 4-7).*

- One outpatient Carotid USS slot is held daily (Mon-Fri) at 3.00 pm for this purpose.
- The Acute Neurology Clinic and Neurology TIA Clinic are available to facilitate ambulatory assessment and management of TIA patients in order to avoid otherwise unnecessary admission (call the Acute Neurology Registrar). However, Neurology clinics are not intended to be used for follow-up of TIA patients who have already been admitted overnight to hospital under a general medical team, unless a Neurology Consultant opinion is specifically required.

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

Note: *Patients who have definite posterior circulation symptoms only or who would not be considered surgical candidates under any circumstances do* ***not*** *require carotid ultrasound.*

- **Commence secondary prevention measures before discharge** (see below)**.**
- Lifestyle advice including *smoking cessation advice* (see page 186).

Patients at low risk:

- 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.
- Specialist assessment and investigations within 7 days (NZ TIA guidelines).
  - CT head prior to discharge 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:
  - Antiplatelet agent(s) - clopidogrel, aspirin plus dipyridamole, or aspirin alone.
  - Blood pressure lowering therapy, e.g., ACE inhibitor + diuretic.
  - Statin - usually atorvastatin 80 mg/day or simvastatin 40 mg/day.
  - Oral anticoagulants - if atrial fibrillation or other cardiac source of emboli.
  - *Nicotine replacement therapy or other smoking cessation aid* (see page 186).
- 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.*

19.4 Subarachnoid Haemorrhage (SAH)

19.4.1 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%.

19.4.2 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.

19.4.3 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 provided there is no contraindication.

  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 could prove fatal. See* **lumbar puncture** *on page 57 for a full list of contraindications.*

  Remember: *There are only three ways to diagnose a subarachnoid haemorrhage:* **lumbar puncture** *(see page 57), 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. Neurosurgical intervention to clip an aneurysm or excise an AVM (craniotomy) or neuro-interventional treatment to coil an aneurysm or embolize an AVM, will then be undertaken as appropriate.

19.4.4 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 L of IV fluids per day assuming that there is also a normal oral intake, and no increased risk of fluid overload.
- The patient should be admitted under the care of a Neurosurgeon or if admitted to another team, a neurosurgical referral is required in all cases.
- Nimodopine (IV or oral) as per Neurosurgery protocol 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: ondansetron is the drug of choice with metoclopramide second choice; avoid prochlorperazine and cyclizine, the latter can cause sedation. Avoid straining. Do not prescribe codeine phosphate. 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.
  - If the patient was already on anti-hypertensive drugs before the haemorrhage, continue the current therapy. Beware of hypotension which may occur in conjunction with the intravenous nimodipine - such hypotension may seriously impair cerebral blood flow.

19.4.5 Christchurch Hospital Admission Arrangements

- Patients who have sustained a subarachnoid haemorrhage should be admitted under the care of Neurosurgery.

19.5 Generalized Convulsive Status Epilepticus

19.5.1 Definition

- Continuous generalized motor seizure activity, or intermittent generalized motor activity with no recovery of consciousness, lasting 30 minutes or more.

19.5.2 Pre-status

Pre-status is a phase of accelerating seizures that may last hours or days, and that heralds status in many patients. Prompt treatment at this stage can prevent status. Consider rectal diazepam or buccal midazolam. Address potential causes as below.

19.5.3 Pseudostatus

Suspect this if convulsive movements are tremorous or thrashing, rather than jerking, and if the patient's sat.O_2 remains high despite apparent prolonged seizure activity.

19.5.4 Common causes of convulsive status epilepticus

- Patients on anticonvulsant treatment for epilepsy:
  - Non-compliance, withdrawal or change of anticonvulsants.
  - Non-CNS infection.
- New presentation of seizures:
  - Acute cerebral insult (head injury, stroke, hypoxia)
  - CNS infection (encephalitis, meningitis)
  - Brain tumour
  - Benzodiazepine or alcohol abuse/withdrawal
  - Drug toxicity
  - Metabolic disturbance (low blood sugar, calcium)

19.5.5 Immediate treatment and investigation

- Support:
  - Check pulse, BP and airway.
  - 100% oxygen.
  - IV line.
  - Monitor pulse, BP, respiration and sat.O_2.
  - Bag patient between convulsive movements if sat.O_2 drops.
- Contact ICU informing them that the patient may require urgent transfer in 10-15 minutes if initial treatment does not control the seizures.
- Drugs:
  - Give lorazepam 4 mg IV over 2 minutes.
  - If seizures continue after a further 2 minutes, give:
    - **Either** phenytoin 15 mg/kg IV in 100 mL sodium chloride 0.9% at a rate of 50 mg/minute, max 25 mg/minute in the elderly. Can cause hypotension, bradycardia, and arrhythmias. Check pulse and respiratory rates and BP every 5-10 minutes. Do a continuous ECG recording for at least an hour post infusion.
    - **Or** sodium valproate 30 mg/kg IV over 5 minutes. Check pulse and respiratory rates and BP every 5-10 minutes.
- Contact ICU re transfer if seizures continue, and/or if there are concerns about the patient's airway and/or breathing.
- Once there, management will be in conjunction with ICU staff, but will include intubation, ventilation and further drugs to stop the seizures such as midazolam, phenobarbitone, and propofol.
- Tests:
  - Capillary blood glucose test - if below normal range give thiamine 100 mg IV and then 50 mL of 50% glucose (dextrose).

Note: *The choice between phenytoin and valproate is dictated by the Consultant responsible for the patient. At the present time both are probably equally effective in controlling status. Most senior staff are well acquainted with giving phenytoin, but valproate may be easier to give with fewer complications. Data supporting the use of valproate at this dose rate is relatively small, and an international RCT is underway to compare these two drugs, and levetiracetam, for the control of status (Epilepsia 2013;54: S6 89-92).*

19.5.6 Measures to take next

- If the seizures have stopped, seek Neurology advice on further management.
- CBC, Na, K, Ca, Mg, blood glucose.
- Give thiamine 100 mg IM or IV.

- If the patient is on anticonvulsants for epilepsy, give their total daily dose of all their drugs as soon as practicably possible, IV or NG if IV not available. Check levels pretreatment. If a drug has recently been withdrawn, reload, and reinstate the former dose. Chart usual daily anticonvulsant drug dosages.
- The Neurology consultation will provide advice on imaging, CSF examination, aciclovir and further anticonvulsant therapy.

19.6 Epilepsy: Patients Presenting with New Possible or Definite Seizures

19.6.1 Diagnosis

The common differential diagnoses are convulsive syncope, cardiac syncope and psychogenic seizure. The diagnosis is based on the patient and eyewitness descriptions of the attack. Attack descriptions must be documented in detail, as they may not be available later and they are the main information on which the diagnosis is based. Do not simply document your own (or someone else's) opinion of what the attack is.

19.6.2 Potential Causes

- Document any history of potential causative factors.
- Document the results of neurological examination.
- If definite or probable seizure(s), request outpatient CT.

19.6.3 Management

- If the patient is alert and well:
  - No acute medical management is necessary.
  - Advise the patient that NZTA requires them not to drive for one year. Document this advice in the notes and in any referral.

  Note: *On receipt of a report from a neurologist, NZTA may allow driving in some low risk patients with single seizure if there is no recurrence after 6 months.*
  - Advise against high risk activities (swimming alone, working at heights etc.).
  - Give first aid seizure management advice.
  - Discharge:
    - If the patient has had a single seizure, refer to Neurology outpatients only if the diagnosis is uncertain. Advise patient and GP that referral to Neurology will be necessary if the seizure recurs. Do not commence anticonvulsant treatment.
    - If the patient has had more than one seizure, refer to Neurology. Do not commence anticonvulsant treatment unless you feel the patient is at risk, in which case contact the Neurology Registrar.
    - If seizure(s) are due to alcohol, do not refer to Neurology. Request CT and advise not to drive for 1 year.
- If recovery is delayed, with prolonged drowsiness, or coma, or there is an acute or subacute onset neurological deficit:
  - Request urgent CT.
  - Contact the Neurology Registrar for consideration of admission and/or further assessment.

19.7 Raised Intracranial Pressure

19.7.1 Clinical Features

These include a deteriorating level of consciousness, increasing drowsiness, stupor, coma/deteriorating GCS; lateralizing/focal neurological signs which may be progressive. Raised intracranial pressure can cause papilloedema in a proportion of patients - probably less than 50%, the remaining 50% or more will have raised intracranial pressure and will not develop papilloedema due to variations in the anatomy of the optic nerve sleeve.

19.7.2 Causes

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

19.7.3 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.

19.7.4 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 1 g/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 - lateralizing (focal) features, focal seizures, drowsiness or papilloedema. When bacterial meningitis is strongly suspected, but features consistent with raised intracranial pressure are present, administer intravenous antibiotics immediately. Then arrange for urgent CT scan. If the CT shows no mass lesion nor any evidence of raised intracranial pressure, perform a lumbar puncture for CSF examination. Refer to* **Meningitis - Management** *on page 144 and* **Lumbar Puncture** *on page 57.*

19.8 Encephalitis

19.8.1 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.

19.8.2 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.

19.8.3 Investigations

Important differential diagnoses include meningitis, severe sepsis, cerebral neoplasia, SLE, toxic metabolic encephalopathy (see *Stupor and Coma* on page 181).

- 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, ALT, GGT, ALP, bili.
- Blood cultures, throat swabs (bacteria and viruses), stool culture for viruses, serum for storage, serology for EBV, CMV, HIV.

- 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.

19.8.4 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.

19.9 Spinal Cord Compression

19.9.1 Causes

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

19.9.2 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 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 of the whole spine.
- Catheterize if urinary retention present and record residual volume.
- CBC + diff, ESR, Na, glucose, K, Ca, creatinine, AST, ALT, GGT, ALP, bili, albumin, CXR. Serum protein electrophoresis and serum free light chains. 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 essential 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.

19.10 Subdural Haematoma

- Subdural haematomas generally occur following trauma to the head. The rate of volume increase of the subdural haematoma will determine its timing of presentation as follows:
  - Acute: presentation within 24 hours of injury. These haematomas always comprise clotted blood and have high density on CT.
  - Subacute: presentation from 24 hours to 10 days from injury. The original clotted haematoma undergoes lysis over this period of 10-14 days with an increase in liquid and a decrease in the clotted component. There is therefore a reducing radiodensity, often with mixed densities on CT.
  - Chronic: presenting after 10 days from head injury. As clot lysis continues these become progressively more liquid as time proceeds and the appearances on CT are of reducing radiodensity to hypodense, often with low density/higher density "fluid level". The average duration from head injury to a patient presenting with a

chronic subdural haematoma is generally 4-6 weeks. In approximately one third of patients with chronic subdural haematomas, no definite history of head injury/trauma can be elicited.

- 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, localizing 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.

19.10.1 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* (see page 171) and *warfarin overdosage* (see page 137).**

19.11 Stupor and Coma

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

1) Vital functions are stabilized.
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.

19.11.1 Emergency Management / Resuscitation

Refer to *Emergency Management/Resuscitation* (see page 65).

19.11.2 Causes

There are 5 main causes of coma:

- Drug overdose
- Head injury
- Intracranial lesions (haemorrhage, infarction, tumour)
- Toxic/metabolic
- Infection

Distinguish between 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, cyst or tumour, hydrocephalus.
  - Subtentorial
    - Brainstem / cerebellar - infarction, haemorrhage, tumour, abscess or cyst.

- **Metabolic**
  - Drugs - alcohol, hypnotics, psychotropics.
  - Hypoglycaemia / hyperglycaemia.
  - Hypoxia - shock, cardiac arrest, carbon monoxide.
  - Electrolyte or acid / base disturbance - acidosis, alkalosis, hyponatraemia, hypernatraemia, hypercapnia, hyperosmolar coma.
  - Encephalopathies - toxic, hepatic or renal failure.
  - Endocrine - hypopituitarism, hypothyroidism, hypoadrenalism.
  - Thiamine deficiency.
  - Hypothermia/hyperthermia.
- **Other**
  - Head injury.
  - Epilepsy / post-ictal.
  - Hysteria / hypnosis.
  - Infection - encephalitis, meningitis, septicaemia.

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

19.11.3 Examination

General

- Look for evidence of head injury, IV drug abuse, signs of chronic illness.
- 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/lateralizing 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). Kernig's sign (resistance to hip flexion) is usually positive in association with neck stiffness in diffuse meningeal irritation from meningitis or subarachnoid haemorrhage.

The Level of Consciousness

The *Glasgow Coma Scale* (see page 63) 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 vocalization. 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. Assessment of the level of coma should be made serially. 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. Note:
  - Opiates can produce pinpoint pupils with constriction to light too small to see.
  - Atropine and tricyclic poisoning can produce dilated and fixed pupils.

- Enlarged (>5 mm) 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 (<1 mm) suggest: bilateral pontine lesions, or opiate overdose, or miotic eyedrops for glaucoma.
- Midposition fixed pupils suggest midbrain lesion.
- 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 cotton wool. 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.

- **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 localizing 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 localizing.
  - **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.
  - **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 in caloric testing.
    - Oculocephalic (and caloric) responses are generally normal in hemisphere lesions.

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

- **Consider performing caloric testing** - seek advice.

Respiratory Pattern

- The pattern of respiration has less localizing 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.
  - **Apneustic breathing** (prolonged inspiratory gasp with end-inspiratory pause) generally accompanies lower pontine lesions.

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 generalized seizures raises possibility of metabolic or ischaemic-hypoxic aetiology.

19.11.4 Investigations

- CBC + diff.
- Glucose, Na, K, osmolality, Ca, AST, ALT, GGT, ALP, bili.
- Arterial blood gases.
- Blood cultures - 2 sets.
- Drug screen.
- CT brain unless metabolic cause definitely identified. MRI if CT inconclusive.
- If meningitis a possibility give antibiotics, do CT, then *lumbar puncture* (see page 57) if safe to do so.
- EEG may be considered to identify psychogenic unresponsiveness or partial complex status epilepticus.

19.12 Facial Nerve (VII) Palsy

19.12.1 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.**

19.12.2 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.

19.12.3 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.

19.12.4 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 5 times a day PO for 7-10 days. Check renal function.
- If facial palsy also treat with prednisone as for Bell's palsy (above).

19.12.5 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.

20. Nicotine Dependent Patients

20.1 ABC(+D) Strategy for Smoking Cessation - all health professionals to implement

A: **A**sk all patients for their smoking status.
B: provide **B**rief advice to quit and offer support.
C: consider nicotine replacement therapy (NRT)and refer to **C**essation support.
D: Document smoking status and intervention.

20.2 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 more comfortably 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.
- Provide advice to quit - be supportive and non-judgmental.
- 24% of people who smoke will attempt to quit after receiving brief advice from a health professional - this increases to 35% when an offer of support (referral) is made.
- 1/40 people who smoke will successfully quit on brief advice from a doctor.
- 80% of smokers want to quit and nearly 50% of smokers try to quit each year.
- NRT doubles the chances of success (NNT = 23). Other cessation medications are equally effective.

20.3 Nicotine Replacement Therapy 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 and lozenges have faster onset of action and achieve high levels of nicotine concentration at around 15-30 minutes. The nicotine inhalator and the QuickMist mouth spray deliver 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 subsidized in the community.
- Inhalator (15 mg) is available for mental health service users but is not subsidized in the community.
- Many people who smoke will 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 from waking to first cigarette.
- NRT should be used for at least 8-12 weeks, although many smokers may require longer treatment courses. Underdosing is often the cause of NRT failure. Dosage depends on individual level of addiction.

Table 46 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

Other pharmacological management options

- Varenicline/Champix® (NNT=10) - fully subsidized under Special Authority
- Bupropion/Zyban® (NNT=18) - fully subsidized
- Nortriptyline (NNT=18) - fully subsidized

Practitioners should refer to the product information for contraindications, precautions, and potential drug interactions. Further advice on these can be obtained from the clinical ward pharmacists or Drug Information ☎ 80900.

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, and restlessness.

Notes regarding NRT dosage

- Cigarette consumption on its own 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.
- 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.
- **Contraindications:** same as for smoking, i.e., acute myocardial infarction, unstable angina pectoris, severe arrhythmias, recent CVA, Buerger's disease. 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 metabolizing enzymes, notably CYP1A2. Smoking cessation may result in elevated concentrations of drugs that are metabolized by this pathway such as theophylline, caffeine, and clozapine.*
- **Pregnant or breastfeeding:** Whether the mother smokes or uses NRT, nicotine passes through the placenta to the fetus, 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.

20.4 Discharge - Electronic Discharge Summary (EDS)

Complete all smokefree fields in the EDS. If no evidence of provision of an intervention is found, deliver an intervention by providing a Quitpack/Quitcard.

Refer to a cessation programme:

- 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 03 379 9947.
- QUITLINE - provides a range of telephone and online cessation services - phone 0800 778 778.
- Pacific Trust Canterbury - for Pacific patients and families - phone 03 366 3900.

Quitcards enable access to subsidized NRT at a community pharmacy for $5 per product (two months' supply). Parkside Pharmacy in the hospital provides NRT free to the patient. NRT products can also be provided to patients using a standard prescription.

21. Nutrition

21.1 Nutrition Support

21.1.1 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 individualized, 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 2.6 L 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.

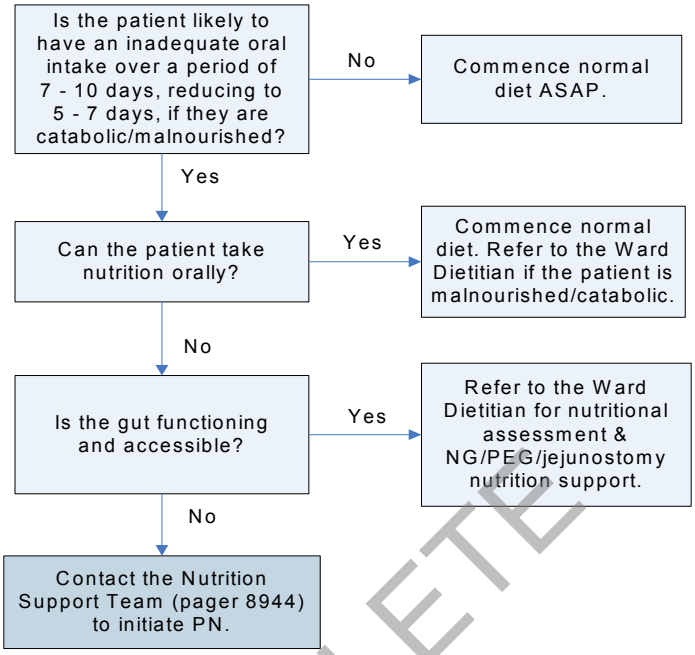
PN can be initiated once a peripherally inserted central catheter (PICC) or other central venous catheter has been placed and the Nutrition Support Team (pager 8944) or the ward dietitian has been consulted. PN is initiated at half rate for the first 6 hours. If blood glucose levels are satisfactory, and the patient is not at risk of ***refeeding syndrome*** (see page 189), 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 10:00 am.

Further useful information on nutrition support is available on general surgical wards and in ICU, CHOC, BMTU, Ward 26, and on the CDHB intranet (search for "TPN prescribing").

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

Table 47 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, ALP, AST, ALT, GGT, zinc, triglycerides, CBC + diff, prothrombin time.
- Vitamin K (phytomenadione) must be provided via IV line 2 mg once weekly - separately.
- Patients referred after 10:00 Friday and in the weekend will receive a standard PN (1.5 L bag). Early referral is encouraged.

21.2 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. Patients with a history of alcohol abuse, anorexia nervosa, or little or no nutritional intake for the last 5-7 days are at particular risk.

Awareness of refeeding syndrome and identification of patients at risk is crucial as the condition is preventable and the metabolic complications are avoidable. The triad of confusion, ataxia, and ophthalmoplegia in Wernicke's encephalopathy can present if refeeding syndrome is not suspected and managed appropriately.

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 potassium, phosphate, or magnesium **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, anorexia nervosa or drugs including insulin, chemotherapy, antacids, or diuretics.

21.2.1 Management

For patients who are **at risk of refeeding syndrome**:

- Check Na, K, Ca, PO_4, and Mg.
- **Before feeding starts, give:**
  - Thiamine 100 mg IV or PO, then thiamine 100 mg PO or IV TDS for 3 days.
  - Vitamin B Complex 2 tabs PO BD. This preparation contains vitamins B1, B2, B3, and B6.
  - A multivitamin preparation should also be given once daily.
  - Vitamin supplementation should be discontinued 5 days after feeding is established unless patient is severely malnourished.

For severely affected patients or if Wernicke's encephalopathy is suspected:

- Check Na, K, Ca, PO_4, and Mg.
- **Before feeding starts, give:**
  - Thiamine 400 mg IV or IM, **followed by** thiamine 400 mg IV or IM TDS for 2 days. Then give thiamine 200 mg IV or IM daily for a further 5 days.
  - Vitamin B Complex 2 tabs PO BD. This preparation contains vitamins B1, B2, B3, and B6.
  - Continue with thiamine 100 mg PO BD and Vitamin B Complex 2 tabs PO BD and multivitamin tablets until signs resolve or plateau.

Contact dietitian to start feeding.

- Rehydrate carefully. Avoid use of IV glucose (glucose load may induce refeeding syndrome). IV rehydration will need to be given via an infusion pump.
- Supplement and/or correct levels of the following unless prefeeding levels are high:
  - Potassium: suggest 2-4 mmol/kg/day PO or IV.
  - Phosphate: 0.3-0.6 mmol/kg/day PO (sodium acid phosphate) or IV (potassium dihydrogen phosphate).
  - Magnesium: 0.2 mmol/kg/day IV or 0.4 mmol/kg/day PO.
  - Calcium supplements may also 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 management of phosphate and magnesium replacement and PN in general, refer to the guidelines available in the Surgical Progressive Care Unit (SPCU) which is in Ward 15.*
- Monitor Na, K, PO_4, Ca, and Mg for the first 2 weeks and amend treatment as appropriate.

References:

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

K Sriram, W. Manzanares, K Joseph, Thiamine in Nutrition Therapy Nutrition in Clinical Practice 2012; Vol27(1)41-50

21.3 Eating Disorders

General comments

When patients with eating disorders (anorexia nervosa, bulimia nervosa, and eating disorder not otherwise specified) are medically unstable they may require medical admission. The most common causes of admission will be complications of malnutrition or the ***refeeding syndrome*** (see page 189).

Common presenting complaints include:

- Acute medical complications of malnutrition: syncope, seizures/confusion, pancreatitis, cardiac failure, gastric dilatation.

- Dehydration.
- Symptomatic hypoglycaemia (<2.5 mmol/L).
- Physiological instability:
  - Bradycardia - heart rate <50/min.
  - Hypotension - systolic BP <80 mm Hg.
  - Hypothermia - temp <35.5°C.
  - Significant postural drop in BP (>20 mm Hg) or rise in heart rate (increase by > 30 bpm).
- Electrolyte imbalance:
  - Hypokalaemia (<2.5 mmol/L).
  - Hypophosphataemia (<0.5 mmol/L).
- Haematology:
  - Symptomatic anaemia.
  - Pancytopaenia.
- Abnormal ECG:
  - Arrhythmia.
  - Diminished amplitude of QRS complex and T waves.
  - Prolonged QTc (>450).
- Arranged admission for refeeding that requires medical management (in conjunction with Eating Disorders Team Physician).

21.3.1 Management

- All patients with heart rate less than 40, QTc >500, arrhythmia, or potassium <2.5 should be on bed rest, and telemetry.
- For all patients requiring admission, give:
  - Thiamine 200 mg BD, IV, IM or PO.
  - Vitamin B Complex 2 tabs PO BD. This preparation contains vitamins B1, B2, B3, and B6.
  - A multivitamin preparation once daily.
- If confused, drowsy or signs of Wernicke's encephalopathy, give:
  - Thiamine 400 mg IV or IM TDS for 2 days and then 200 mg daily IV or IM for a further 5 days.
  - Vitamin B Complex 2 tabs PO BD. This preparation contains vitamins B1, B2, B3, and B6.
  - A multivitamin preparation once daily.
- Avoid over-treating hypoglycaemia, as any glucose level above physiological will worsen the refeeding syndrome by stimulating insulin release. This will drive consumptive loss of phosphate and thiamine and glucose, and intracellular shifts of potassium and magnesium.
- Rehydrate and correct any electrolyte imbalance. Avoid IV glucose pre thiamine.
- Do not use a Bair Hugger for hypothermia as this will increase metabolic rate and cause peripheral vasodilation, and may worsen physiological instability.
- Contact the Eating Disorders Team (Physician and Eating Disorders Liaison Nurse) and consult the ward Dietitian to establish a feeding plan as soon as possible, to avoid delays in establishing adequate nutrition.
- Patients may require nasogastric feeding in the initial phase of treatment if unstable with the refeeding syndrome. General principles of nasogastric feeding include: starting slowly and gradually increasing feed rates to target over 2-3 days to control the refeeding syndrome. When on nasogastric feeding all nutrition should be through the nasogastric tube and the patient should not eat extra food (unless symptomatic hypoglycaemia occurs). Once stable at target caloric intake, the nasogastric tube can be spigoted and transition to oral nutrition should occur. This may require the Eating Disorders Team support if the patient is struggling.
- Routine daily cares in addition to standard care should include daily electrolytes (request Na, K, Ca, PO_4, Mg), ECG, and lying and standing blood pressure and pulse.
- On admission also consider:
  - Creatine kinase, looking for evidence of excessive exercise.
  - LH/FSH, oestradiol, TSH and T4, looking for evidence of hypothalamic dysfunction.
  - Beta-hydroxybutyrate, looking for evidence of starvation related ketoacidosis (increased risk with co-existent alcohol abuse).
  - LFTs are commonly deranged.

Reference: CDHB Department of General Medicine - Green Book.

22. Obstetrics and Gynaecology

22.1 Obstetrics and Gynaecology Department Information

- Delivery Suite, third floor, ☎ 85711 / 85715
- 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 Registrar - Monday to Friday 0800-1600 pager number available via GAU ☎ 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. There is a more comprehensive set of pathways on the HealthPathways website before considering referral to the Gynaecology department.

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

22.2 Gestational Proteinuric Hypertension (Pre-eclampsia)

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

22.2.1 Clinical signs

- Hypertension.
- Proteinuria.
- Rapid development of oedema.
- Headache.
- Visual disturbance.
- Hyperreflexia/clonus.
- Epigastric pain and tenderness.
- Nausea and vomiting.
- Chest pain or dyspnoea.
- Altered level of consciousness; seizures.
- Placental abruption.

22.2.2 Investigations

- CBC + diff, Na, K, creatinine, urea, urate, alb, bili, ALP, AST, GGT, ALT, coagulation profile (if platelet count reduced), and urinary protein/creatinine ratio.
- Pulse oximetry for oxygen saturation.
- Group and hold if delivery imminent.
- Ultrasound scan of fetus including doppler studies.
- Cardiotocograph (CTG) if >24 weeks gestation.

22.2.3 Management

- **Call Obstetric Registrar**.
- If BP ≥160 mm Hg systolic or ≥100 mm Hg diastolic, institute antihypertensive therapy:
  - Loading dose of methyldopa 1 g 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 immediate release 10 mg orally and repeated as required, **or**
  - Hydralazine 5 mg IV **slowly** repeated as required (beware of precipitating hypotension). Total dose 30 mg.

  Whichever option is used, the fetus 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 2 or more beats of 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.

22.3 Ovarian Hyperstimulation Syndrome

22.3.1 Definition and Pathophysiology

This is an iatrogenic potentially life threatening condition that can occur after IVF treatment or ovulation induction resulting from vasoactive products being released from hyperstimulated ovaries. It is worsened and prolonged when pregnancy occurs due to ongoing endogenous HCG stimulation of ovaries.

Refer to CWH Gynaecology Assessment Unit guideline for classification of severity of condition, and consult with on-call Gynaecologist.

22.3.2 Possible Symptoms

- Abdominal bloating /distension and pain
- Nausea and vomiting
- Breathlessness
- Reduced urine output

22.3.3 Assessment

Examine the patient for signs of:

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

Do **not** perform bimanual examination.

22.3.4 Investigations

- CBC. Check beta-HCG if pregnancy state unknown.
- Urea, Na, K, creatinine, alb and LFTs.
- Coagulation profile.
- Arterial blood gases if dyspnoeic
- Ultrasound scan of ovaries (dimensions) and abdomen (ascites).

CXR rarely gives additional information to clinical findings in initial workup.

22.3.5 Management

- Rehydration with crystalloid if dehydrated.

- Fluid balance chart, serial girth measurement and daily weight.
- Initially 4 hourly observations.
- Analgesia (paracetamol or codeine - **avoid** NSAIDs).
- Antiemetics as required.
- Full length TED stockings and prophylactic LMWH daily (at least until discharge).
- Patients should continue their progesterone luteal support (usually pessaries).

22.4 Ectopic Pregnancy

- Still a cause of maternal death.
- Should be considered in any women of reproductive age who present with abdominal pain/discomfort and/or vaginal bleeding.
- Other presentations can include non-specific gastrointestinal symptoms (vomiting, diarrhoea, painful defaecation).
- A pregnancy test (urine initially and subsequent quantification with serum beta-HCG) is therefore essential in the early phase of assessment.

22.4.1 Clinical symptoms and signs

- Pelvic pain (can be localized, non-specific, of variable intensity and duration, or absent).
- Vaginal bleeding (spotting, heavy or none).
- Asymptomatic mass found at USS or empty uterus on ultrasound (no pregnancy sac) with a beta-HCG level of >1500 unit/L.
- Occasionally may present with maternal collapse ± other features.
- A history of amenorrhoea may not be obtained.
- Differential diagnoses include ruptured corpus luteum of pregnancy, torsion of ovarian cyst, miscarriage, appendicitis, renal colic, pelvic inflammatory disease or endometriosis.

22.4.2 Management

- **If collapse, hypotension or hypovolaemia:**
  - Transfer the patient to Resus.
  - Call for the ED Consultant, Gynaecology Registrar or other available senior help.
  - Insert two large bore IV cannulae and infuse warm crystalloid.
  - CBC, beta-HCG.
  - Cross-match 4 units of blood.
  - Catheterize and test urine urgently for pregnancy test.
  - If pregnancy confirmed once the patient is adequately resuscitated, arrange transfer to theatre.
- **Non-acute presentation:**
  - Urine pregnancy test: if negative, ectopic gestation is virtually excluded and other causes need to be considered.
  - CBC, group and hold, and beta-HCG level.
  - If patient stable arrange for a pelvic ultrasound scan.
  - Discuss with the on-call Gynaecology Registrar.
  - If ultrasound not readily available/out of hours admit to hospital until ultrasound can be performed.
  - Further guidelines are available in the Gynaecology Assessment Unit, or search for "ectopic" on the CDHB intranet.
- **Non-sensitized rhesus (Rh) negative women:**
  - Non-sensitized rhesus (Rh) negative women should receive anti-D globulin in the situation of a confirmed ectopic pregnancy.
  - Discharge documentation in the form of a standard letter to GP must state whether or not anti-D has been given.

22.5 Miscarriage

- Defined as loss of a pregnancy before 24 completed weeks of pregnancy.
- Most present with vaginal bleeding but diagnosis may not be simple: also consider ectopic pregnancy.

22.5.1 Management

If history of heavy bleeding/hypotension/hypovolaemia:

- IV access and fluid resuscitation.
- Call Gynaecology Registrar.
- CBC, group and hold.
- Consider possible ectopic pregnancy.

If symptoms are mild, the cervical os is closed, and bleeding light then it may settle.

If the os is open then it is an inevitable miscarriage with products within the uterus still to be passed. Bleeding will be heavy and tissue will usually be passed spontaneously. If bleeding is profuse and **the os is open** then consider egometrine 0.5 mg IM to produce a sustained contraction.

If the uterus is small for dates then it is possible a miscarriage has occurred where the fetus dies but remains.

- If Rhesus negative, give anti D if over 12 weeks amenorrhoea.
- Arrange an ultrasound scan of the pelvis.
- Check cervical smear result.

22.6 Menorrhagia

Heavy vaginal bleeding with negative urinary beta-HCG.

22.6.1 Aetiology

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

22.6.2 Investigations

- Urinary beta-HCG.
- Speculum examination and check smear status.
- Cervical and vaginal swabs (Chlamydia, Neisseria, high vaginal swab for bacterial vaginosis).
- CBC + diff and iron studies - consider group and hold if bleeding heavy.
- Ultrasound scan of pelvis.
- INR, APTT, fibrinogen. Consider platelet dysfunction, inherited and acquired.
- TFTs if history suggestive.

22.6.3 Management

- IV fluid replacement and resuscitation if necessary.
- Call Gynaecology Registrar.
- Heavy cervical bleeding (e.g., neoplasia, post cervical surgery): consider silver nitrate or Monsel's solution (ferric subsulphate) application. If this fails then tamponade with a vaginal pack pressed closely to cervix and insert a Foley urinary catheter into the uterus. Seek advice from the on-call Gynaecology Registrar before doing this.
- Tranexamic acid 1 g QID PO.
- Norethisterone or medroxyprogesterone 30 mg stat then norethisterone 5 mg TDS or medroxyprogesterone 10 mg BD. All PO.

  ***Note**: Discuss with Gynaecology Registrar **before** giving high dose progestogens if uterine bleeding and possible risk for neoplasia (hyperplasia can be temporarily histologically corrected by progestogens).*
- Infection: treat with broad spectrum antibiotics.
- Polyps require removal and histology.

22.7 Genital Tract Infection

This section refers primarily to infections of the lower female genital tract. The following section, on pelvic inflammatory disease (PID), relates to the upper female genital tract.

Table 48 Genital Tract Infection

	Diagnosis	Treatment	Contact trace
Candida	High vaginal swab Microscopy & culture Identify 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 and culture Identify on cervical smear	Metronidazole 2 g PO stat	Treat partner Screen for other STI
Chlamydia	**Females:** Vulvovaginal swab (Chlamydia swab rubbed around external urethra a couple of times then sliding about 5 cm into the vagina and rotating around the vaginal walls for 5-10 seconds (send for Chlamydia Nucleic Acid Amplification Test (NAAT)) **Males:** PCR from first void urine	Azithromycin 1 g PO stat **or** Doxycycline 100 mg BD 7 days **In pregnancy**, give azithromycin 1 g PO stat. Give amoxicillin 500 mg TDS PO for 7 days if there are problems with azithromycin	Screen and treat partners Screen for other STI
Gonorrhoea	Endocervical swab for culture Urethral/anal swab Throat swab	Ceftriaxone 500 mg IM and azithromycin 1 g PO stat	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 Catheterize if needed	Screen for other STI

22.8 Pelvic Inflammatory Disease

- Pelvic inflammatory disease (PID) is an acute infection of the upper genital tracts in women, which may include the endometrium, fallopian tubes, ovaries, peritoneum and adjacent pelvic organs.
  - Often a sexually transmitted disease.
  - May occur post-operatively/post instrumentation, e.g., IUCD insertion, termination or spontaneous abortion.
  - Causative organisms include *N. gonorrhoeae*, *C. trachomatis*, mycoplasmas, mixed anaerobes.
  - Risk factors include age <25 years, new or multiple sexual partners, previous sexually transmitted disease.
- Presenting symptoms include:
  - Up to 60% of cases are sub-clinical with minimal signs or symptoms.
  - Lower abdominal pain usually bilateral and of recent onset.
  - Deep dyspareunia.
  - Abnormal uterine bleeding (post-coital, intermenstrual).
  - Vaginal or cervical discharge.
- Clinical signs:
  - Uterine or adnexal tenderness.
  - Cervical motion tenderness.
  - Mucopurulent cervical discharge.
  - Fever.

22.8.1 Investigations

- All women with suspected PID should have a bimanual pelvic examination and speculum examination with endocervical swabs taken for PCR (*N. gonorrhoeae* and *Chlamydia*) and culture (*N. gonorrhoeae*) as well as high vaginal swab for culture.
- MSU, urine pregnancy test.
- CBC, CRP.
- Serology for syphilis, hepatitis B and HIV recommended.
- No single symptom, sign or investigation is diagnostic therefore a low threshold for treatment is recommended. The current syndromic criteria used to initiate antibiotics is a woman at risk for STIs with lower abdominal pain and **one** or more of:
  - Cervical motion tenderness or
  - Adnexal tenderness or
  - Uterine tenderness.

22.8.2 Management

- Consult with Sexual Health or Gynaecology.
- Patients who are **pregnant or with severe PID** or who have failed previous therapy should be referred to Gynaecology for management. Definition of treatment failure is based on 72 hour review.

See also: NZ Sexual Health Guideline http://www.nzshs.org/treatment_guidelines/Pelvic_Inflammatory_Disease_2009.pdf

22.8.3 Antibiotics

Mild to Moderate PID

- Ceftriaxone 500 mg IM single dose, **plus**
- Doxycycline 100 mg PO BD for 14 days, **plus**
- Metronidazole 400 mg PO BD for 14 days (may be discontinued if not tolerated).

In cases where compliance may be an issue, substitute azithromycin 1 g PO stat and on day 8 instead of doxycycline.

For patients with **severe penicillin allergy**, use gentamicin 5 mg/kg IV as a single dose instead of ceftriaxone.

Severe PID or failed therapy

- Ceftriaxone 1 g IV q24h, **plus**
- Metronidazole 500 mg IV q8h, **plus**
- Doxycycline 100 mg PO BD until afebrile for 24 hours, **then**
- Metronidazole 400 mg PO BD, **plus**
- Doxycycline 100 mg PO BD, to complete 14 days of therapy.

For patients with **severe penicillin allergy** replace ceftriaxone with gentamicin 5 mg/kg IV once daily. Adjust dose according to levels. See the gentamicin/tobramycin dosing guidelines in the Pink Book.

For patients with tubo-ovarian abscess or intolerance to metronidazole or doxycycline, clindamycin 450 mg PO QID may be used.

Notes:

- **PID in pregnancy can be difficult to treat as some of the antibiotics used may cause nausea and vomiting. Seek advice.**
- All patients should be given a PID information sheet, encouraged to notify sexual partners and to advise them to present for testing and treatment regardless of negative test results.
- Patients should be followed up to confirm resolution of symptoms and signs.

See also Pelvic Inflammatory Disease on HealthPathways.

23. Older Persons Health Specialist Service

23.1 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-focused 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 John Elliot, Dr Val Fletcher, Dr John Geddes, Dr Nigel Gilchrist, Dr Carl Hanger, Dr Sarah Hurring, Dr Hamish Jamieson, Dr Julie Kidd, Dr Anne Roche, Dr Andrew Sidwell, Dr John Thwaites, Dr Joyce Wan, Prof Tim Wilkinson

Consultant Psychiatrists

- Dr Chris Collins, Dr Matthew Croucher, Dr Brian Deavoll, Dr Dominic Lim, Dr Colin Peebles, Dr Jo Reeves

Medical Officers

- Dr Michele Dhanak, Dr Angela Harding, Dr Gerald Johnstone

Primary Care Liaison Team

- Dr Michael Thwaites (GP), Robyn Bayly (Physiotherapist), Ginny Brailsford (Pharmacist), Donna Rados (Registered Nurse)

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 clinician 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: single point of entry (SPOE) for OPHSS ☎ 66371, fax 67998.
  - 0830-1630 weekends: ☎ 337 7899 and ask for the PSE Duty Clinician.
  - 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). Search for "OPHSS" on the CDHB intranet.

Points to Remember

- The best value is obtained by referring for a consultative service rather than a "takeaway" service, and by referring early, outlining the specific clinical questions to be addressed.
- 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 specializes in the recognition and management of these issues, both before and as they arise, to maximize the health and independence of elderly people.

Department Guidelines

Refer to the OPHSS Guidelines - search for "OPHSS" on the CDHB intranet.

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 older patients.

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 as they 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. The reasons for threatened independence must be addressed.

Do **not** 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.

23.2 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. is essential.
- **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 recognized.
- **Medications**: 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 rationalization 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: Test short and long distance vision.
  - Hearing: Impacted wax in ears.
  - Nutrition: e.g., evidence of weight loss, angular stomatitis, myopathy, cramps, etc.
  - Evidence of poorly fitting dentures.
  - Cognitive function. Perform Mini Mental State Examination (MMSE) or Montreal Cognitive Assessment Test (MOCA) if cognitive impairment is suspected, and consult *Confusion Assessment Method (CAM)* (see page 237) 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.

23.3 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.

23.4 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 236) 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 MMSE or MOCA routinely (all inpatients should be screened on admission unless it is clear that they are not cognitively impaired) and the CAM Screen to help identify delirium.
- Try to make an accurate diagnosis.
- Treat any underlying cause (infection, dehydration, faecal impaction, polypharmacy etc), but remember that the absence of a clear medical trigger for delirium does not mean delirium is not present.
- Consider stopping or reducing medication that may be contributing but do not suddenly cease longstanding medications except in an emergency, and be careful about permanently stopping longstanding medications that were being used to prevent relapse of a recurring condition.
- 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 (**not in patients with cerebral Lewy body disease or Parkinson's disease** - see below). Regular dosing is preferred to PRN. Titrate up or down according to the response and withdraw as soon as possible.
- Use lorazepam 0.25-0.5 mg BD PO for patients with Parkinson's disease or dementia with Lewy bodies; quetiapine 12.5 - 25 mg BD PO is another option (Consultant endorsement recommended).
- Avoid using intramuscular injections, except in emergency situations (refer to *Psychotropic medication* on page 238).
- 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 if agitated or mobile. Consider nursing on a mattress near to or on the floor if there is a risk of falling.

- Use a soft night-light.
- Minimize 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).

23.5 Continence Problems

Urinary Incontinence

- Attempt to ascertain whether the patient has stress or urge incontinence.
- Consider medication as possible contributor, especially cholinergic agents.
- 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.

23.6 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 optimize 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.

23.7 Falls in Older People

Falls are common, have serious health causes, and are a major cause of injury and subsequent disability.

Falls in hospital

In hospital, major risk factors for falls are the effects of **acute illness** (delirium, muscle weakness, dependency on staff for basic ADLs), **medications** affecting blood pressure or alertness, and **unfamiliar environment** (e.g., slippery floors, toilet is far away, unstable furniture, usual walking aid is not available). These often manifest themselves when the older person wants to get up to go to the toilet.

Risk factors for falling

Intrinsic risk factors (pertaining to the individual) include:

- Musculoskeletal: leg muscle weakness, osteoarthritic knees, impaired balance (reactions)
- Poor vision
- Impaired cognition (poor insight, judgement, and decision making)
- Medications: benzodiazepines and other psychotropics, diuretics, polypharmacy
- Postural hypotension (often via illness or medications)
- Acute illness causing muscle weakness, delirium, or dehydration

Extrinsic risk factors (relating to the environment) include:

- Slippery, unstable, or uneven surfaces (wet floor, loose rugs, ice)
- Poor lighting
- Unstable activities (climbing)

Most falls in older people are due to intrinsic factors, many of which can be modified.

Assessment

It is vital to obtain a clear history of each fall, including collateral history from family or another witness. Ask:

- What were you doing just before the fall, and then what happened?
- How had you been feeling over the last few days before the fall?
- Were you able to get up off the floor and how long before you could get up?
- Did you have any injuries? Did you feel back to normal?

Notes:

- Try to avoid the term "mechanical fall" - it is non-discriminatory. Instead describe what happened.
- Most patients have more than one cause for falls.
- If patients say "I must have tripped", seek alternative causes.
- Syncope and loss of consciousness raise possibilities of significant cardiac or neurological disorders and should be investigated accordingly.

Examine for orthostatic hypotension, gait abnormalities, muscle weakness, joint pain as well as injuries sustained in the fall.

Prevention or treatment options

- Strength and balance training reduces falls risk:
  - Programmes include the Otago Exercise Programme, Tai-Chi groups, or individualized strength and balance training with a physiotherapist.
- Medication review, with particular emphasis on reducing or stopping medications with sedative CNS activity (benzodiazepines, psychotropics, and antidepressants), or those with hypotensive actions.

  Note: *Medication review must take a holistic view, including ongoing indications, and not just risk of falls. Gradual planned withdrawal, together with discussion with the general practitioner, is usually needed, particularly for centrally acting medication such as benzodiazepines or antidepressants.*
- Environmental assessment, such as a home visit, is particularly important for those with visual impairment.
- Prevention of deconditioning with acute illness or immobility - it is important to maintain mobility and activity during acute illness or hospitalization.
- Hip protectors may be used to reduce the risk of hip fractures.

24. Oncology

24.1 Oncology Department Information

The Canterbury Regional Cancer and Blood Service comprises the Departments of Oncology, Haematology, and Palliative Care.

Main Office

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

Oncologists

Dr Brendon Anderson, Dr Scott Babington, Dr Jim Edwards, Dr Bernie Fitzharris, Dr Kate Gardner, Dr David Gibbs, Dr Chris Harrington, Dr Dean Harris, Dr Melissa James, Dr Mark Jeffery, Dr Lisa Johansson, Dr Lawrence Ko, Dr Avtar Raina, Prof Bridget Robinson, Dr Matthew Strother, Dr Iain Ward, Dr Michelle Vaughan, Dr Steve Williams

Inpatient Services

Oncology Ward, Ward 26, Riverside Block.

Consultation and On-call Service

- **Oncology** (Monday to Friday). We prefer to see most new referrals in outpatients so that patients can arrange for their families and support people to attend with them. We are always able to offer advice or to review inpatients if necessary. Phone the on-call Oncologist for advice if you are not sure about the urgency of a situation.
- 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 patients who are receiving radiation or chemotherapy under our 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
- Dr Rachel Wiseman, ☎ 89611
- Dr Amanda Landers, ☎ 027 271 4918
- Willem Vink, Nurse Practitioner, ☎ 81473
- Liz Bremen, Nurse Specialist, ☎ 81885
- Tammy Horton-Davey, Nurse Specialist, ☎ 81985

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, see the Palliative Care Guidelines (online at http://cdhb.palliativecare.org.nz).

24.2 Cancers Requiring Early Multidisciplinary Care

- The investigation and management of many cancers is complex, and for some cancers, the possibility of cure may be reduced by delays in investigation and treatment. Early discussion or referral to an Oncologist, Paediatric Oncologist, Haematologist, or other appropriate Specialist is recommended, especially for:
  - Testicular cancer.
  - Sarcomas.
  - Germ cell tumours - ovary, extragonadal, retroperitoneal and mediastinal.
  - Gestational trophoblastic disease.
  - Undifferentiated cancers, especially in younger patients.
  - Any cancer in children or teenagers.
  - Osteosarcoma and Ewing's sarcoma.
  - Leukaemias.
  - Lymphomas - Hodgkins and non-Hodgkins.
  - Head and neck cancer.

24.3 Think Before You Biopsy

A tissue diagnosis is important in managing many cancers. However, taking the wrong sample or sampling the wrong site can delay diagnosis and may even compromise the chances of successful treatment.

Before biopsy, think: should this be biopsied at all?

Situations where inappropriate biopsy may interfere with treatment include:

- Potentially resectable liver metastases.
- *Soft tissue sarcomas* (see below).
- Head and neck cancer.

Is the correct sample being taken?

Before biopsies are organized, 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 sodium chloride 0.9%, refrigerate, and deliver to the Laboratory next morning.

24.4 Soft Tissue Sarcomas

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.

24.5 Spinal Cord Compression

- Consider in all patients with cancer and back pain, especially if accompanied by resistance to analgesia, sensory loss, alteration in bladder or bowel function, limb weakness or lack of co-ordination.
- If cord compression is suspected, give dexamethasone 16 mg stat PO or IV and cover with omeprazole (prior to investigation) and refer **immediately** to on-call Radiation Oncologist.
- 30% of cases of spinal cord compression involve multiple levels, so MRI of the **whole spine** should be requested.

24.6 Superior Vena Cava Obstruction

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

24.7 Management of Severe Neutropaenia/Immunosuppression

Refer to *Severe Neutropaenia* (see page 121) in the Haematology section.

24.8 Hypercalcaemia

A high proportion of hypercalcaemic patients will have an associated underlying malignancy, the commonest being breast cancer, lung cancer and myeloma. Some 10-20% of patients with cancer will become hypercalcaemic at some time during their course. Notably some malignancies, which may be cured or have a prolonged remission, may re-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 94) 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.

24.9 Paracentesis and Pleural Aspiration

For guidelines for paracentesis and pleural aspiration for oncology/palliative care patients, refer to the Palliative Care Guidelines (online at http://cdhb.palliativecare.org.nz).

24.10 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 103).

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.

24.10.1 Chemotherapy - Associated Nausea and Vomiting

- **Pre-chemotherapy:** antiemetics are individually tailored to the emetogenicity of the regimen, and vary between domperidone10 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 sodium chloride 0.9% for highly emetogenic regimens.
- **Post-chemotherapy:** again treatment is tailored according to chemotherapy regimen.
  - Mildly emetogenic regimens - domperidone 10-20 mg PO QID or metoclopramide 10 mg PO QID.
  - Moderately emetogenic regimens - dexamethasone 4-8 mg PO 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 domperidone or metoclopramide, 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.*

24.10.2 Antiemetics for Oncology, Haematology, and Palliative Care Patients

- Domperidone: 10-20 mg PO QID before food and nocte
- Metoclopramide: 10 mg PO, IV, or subcut 4-6 hourly (commonly given QID before food)
- Haloperidol: 0.5-3 mg PO, subcut or IV 6-12 hourly, or single nocte dose (max 5 mg/day)
- Cyclizine: 25-50 mg PO or slow IV BD or TDS (max 150 mg/day)
- Prochlorperazine: 5-10 mg PO 6 hourly or 25 mg PR 8 hourly
- Dexamethasone: 2-4 mg PO daily
- Lorazepam: 0.5-1 mg PO 6-8 hourly

Notes:

- **Avoid** metoclopramide and domperidone in bowel obstruction; haloperidol or cyclizine are preferred and can be given subcutaneously.
- Domperidone is an alternative prokinetic agent (oral only) that can be used if dystonic reactions or other side-effects are encountered with metoclopramide.
- 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.
- **Avoid** ondansetron in patients with constipation, bowel obstruction, or in patients on opioids.
- Subcutaneous injections of metoclopramide and haloperidol can be effective given regularly or in varying combinations in a continuous infusion. Refer to the Syringe Driver Compatibility Chart in the Palliative Care Handbook (online at http://handbook.palliativecare.org.nz), which 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 Palliative Care Guidelines (online at http://cdhb.palliativecare.org.nz), or refer to the Palliative Care service.
- If nausea and vomiting remain a problem, consult Oncology or the Palliative Care Service ☎ 81473 for further advice.

24.11 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.

- The side effects from radiation can be divided into acute and late effects. Acute side effects are those which occur during treatment and immediately after. Late side effects occur at least 6 months after treatment.
- 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.
- 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 include thinning of the skin, scar formation and telangiectasia.
- More slowly proliferating tissues are more likely to develop late side effects so caution is taken to limit the dose of radiation treatment to these tissues. Examples of these late effects include:
  - Spinal cord: myelitis and subsequent development of neurological signs and symptoms.
  - Kidney: hypertension.
  - Optic nerve: 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.
- In some instances the combination of radiation and cytotoxic chemotherapy may increase the risk of acute or late tissue reactions.
- Rarely, any tissue may undergo premalignant or malignant change years after exposure to ionizing radiation, e.g.:
  - Myelodysplasia and acute myeloid leukaemia.
  - Breast cancer after radiotherapy for Hodgkin's lymphoma.
- Patient information leaflets, which summarize important side effects of radiotherapy to different body sites, are available on HealthInfo (the public site) and HealthPathways.
- The challenge in radiation is to give the maximum dose to the tumour to optimize the chance of cure while sparing as much as possible the normal tissues to decrease the risk of acute and late side effects.

24.12 Palliative Care

For guidelines for referring patients to Palliative Care, see the Palliative Care Guidelines (online at http://cdhb.palliativecare.org.nz).

For management of pain in the cancer/palliative setting, refer to *Chronic/Persistent Pain in the Cancer/Palliative Setting* (see page 217).

25. Ophthalmology

25.1 Ophthalmology Department Information

Main Office

- Ground Floor, Pathology Building, 19 St Asaph Street

Inpatient Services

Inpatient teams are by Consultant and are currently in Ward 20. The Consultants are:

- Dr Antony Bedggood, Dr Jim Borthwick, Dr Geoff Duff, Assoc Prof Mark Elder, Dr Sean Every, Dr Russell Lienert, Dr Ainsley Morris, Dr Jo-Ann Pon, Dr John Rawstron, Dr Allan Simpson, Dr Rebecca Stack, 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, optical coherence tomography scanning, and retinal photography.

Referral Guidelines

All referrals require a visual acuity. If the visual acuity is not normal, then the test must 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.

Further referral and clinical information is also available on HealthPathways.

25.2 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), retained foreign body in the eyes, infection including intraocular pre-septal and orbital cellulitis and conjunctivitis.

25.2.1 Common Causes for Decreased Vision

- **Sudden painless:**
  - Retinal artery occlusion (pale retina), retinal vein occlusion, ischaemic optic neuropathy including 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.

25.2.2 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.

25.3 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, consider treating with hyperbaric oxygen. 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 273) for more details.

26. Orthopaedic Medicine

26.1 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 John Geddes, 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) or Christchurch Hospital.

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.

26.2 Identification, Treatment, and Management

26.2.1 At Risk Patients

The patients who are most at risk of complications are elderly males, patients with dementia, institutionalized 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.

26.2.2 Fall Aetiology

A detailed history must be obtained to ascertain the contributing factors to the fall, and in particular whether there was a secondary cause such as arrhythmia, silent myocardial infarction, postural hypotension, drug effect, neurological event.

26.2.3 Pre-morbid Function

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

26.2.4 Drugs and Hip Fractures

Many drugs are recognized 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 optimized.

26.2.5 Analgesia

Refer to the *guidelines on analgesia* on page 214. 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.

26.2.6 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.

26.2.7 Thromboprophylaxis

Deep vein thrombosis occurs commonly in patients with fractures of the lower limbs. Refer to *Surgical VTE Prophylaxis* on page 123.

26.2.8 Management of Patients on Anticoagulants

Refer to the Thrombosis section for management of *patients on oral anticoagulant therapy undergoing surgery* on page 135.

26.2.9 Antibiotic Prophylaxis for Fracture Surgery

Reduces deep and superficial wound infections.

All patients undergoing surgery for fracture fixation should receive antibiotic prophylaxis perioperatively.

26.2.10 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 institutionalization. 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 236). The delirium service is available (contact through the operator at TPMH) for consultation and advice.

26.2.11 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 188).

26.2.12 Constipation

Constipation is very common in these patients. Please ensure early and optimal use of laxatives as outlined in *Constipation* (see page 105). When prescribing regular opioid analgesia, regular laxative therapy should generally be prescribed at the same time.

26.2.13 Osteoporosis Treatment

Refer to *management of osteoporosis* (see page 211).

26.2.14 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 at Burwood Hospital is recommended. Those with ongoing medical problems are best rehabilitated on the medical wards at TPMH.

27. Osteoporosis

27.1 Bone Clinic

Main Office

- TPMH 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.

27.2 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 Garvan (http://garvan.org.au/promotions/bone-fracture-risk/calculator/) calculation tools. These calculations can be made without BMD to get an estimate of fracture risk.

These tools are guides to fracture risk. FRAX takes into account a number of important risk factors for fracture but does not include the severity of these risk factors. For instance it does not adjust the risk according to steroid dose or cigarette pack year history. The Garvan tool takes into account the number of fractures and fall history. If these factors are present, the Garvan tool can more than double the fracture risk as assessed by FRAX. These tools are mainly geared to "normal people" not those who might be in hospital clinics or beds. They use femoral neck or total hip BMD and ignore spinal BMD.

- **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 Depo-Provera™), coeliac disease, anticonvulsant use, COPD, alcoholism, hyperthyroidism, 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 Organization 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 for PHARMAC medication access.*

Guidelines for treatment based on BMD results:

- **T score < -2.5**: treat.
- **T score -1 to -2.5**: treat if fragility fracture or 10 year fracture risk calculation is ≥20% for overall fracture risk or ≥3% for hip fracture risk. Otherwise correct risk factors and consider calcium and vitamin D.
- **T score > -1**: don't treat.
- **If on supra-physiological steroid therapy, treat if T score < -1.5**.

27.2.1 Investigations

All patients should have Ca, PO_4, alb, ALP, creatinine, and CBC + diff. If BMD for age is low (i.e., Z < -2), consider secondary causes of osteoporosis. Possible tests include: vitamin D, PTH, testosterone & SHBG (in males), LH, FSH, coeliac antibodies, TFTs, SPE and serum free light chains, fasting urine calcium/creatinine ratio.

27.2.2 Treatment

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

Calcium

- By diet or supplement to approximately 1000 mg per day. Calcium carbonate 1.25 g (500 mg Ca per tablet) one BD with food or calcium carbonate effervescent 1.75 g (1000 mg Ca) one nocte.

  It remains controversial whether calcium supplements increase the risk of vascular complications, particularly in women over 70. Calcium by diet is preferred or alternatively take smaller doses with food to diminish peak calcium levels. A number of large controlled trials have failed to show any increased cardiovascular risk if using both calcium and vitamin D supplements together.

Vitamin D

- Consider loading dose, i.e., cholecalciferol 1.25 mg (50,000 units) one daily for 3 days if likely to be low, for example older post hip fracture patients, institutional care, anticonvulsant medication. Maintenance dose is recommended for most patients on bisphosphonates: cholecalciferol 1.25 mg (50,000 units) monthly. If taken daily, a dose of at least 800 units is required.

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

Bisphosphonates

- **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. Alendronate leads to a 50% reduction in all fractures, but **special authority is required**. Fosamax Plus™ contains 800 units per day of vitamin D. Initial loading with cholecalciferol however is still required in those likely to have significant vitamin D deficiency (see above). Some patients may require a higher maintenance dose of vitamin D 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, particularly at the femoral neck, and there have been no further fractures, particularly vertebral, for 2 years. This could be most relevant in younger patients with potential long term exposure to bisphosphonates.

- **IV zoledronic acid** (IV pamidronate is now rarely used for osteoporosis):
  - Zoledronic acid has the same indications as alendronate and requires **special authority**.
  - It has similar efficacy to alendronate on fracture risk.
  - It is the first choice agent for patients who have GI intolerance to oral bisphosphonates or a high likelihood of this, or potential compliance issues.

  To avoid possible hypocalcaemia it is important to ensure that patients are vitamin D replete before administering an IV bisphosphonate. If in doubt give 2 cholecalciferol tablets (2.5 mg 100,000 units) in the week before the infusion. Information packs for patients are available through the Bone Clinic.
  - Give zoledronic acid 5 mg IV every 1-3 years. The funded preparation is Aclasta™ and this contains zoledronic acid 5 mg in 100 mL for infusion over 15-30 minutes.
  - If calculated creatinine clearance is <35 mL/min zoledronic acid is 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 cause transient fever and flu-like symptoms following the first dose in up to 20% of patients. This can be reduced by taking paracetamol 1 g QID for 48 hours and ensuring good hydration. GI 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.

Hormone Replacement Therapy (HRT)

- It is important in young hypogonadal females, for example premature menopause, to preserve bone mass. HRT is likely to be beneficial. In postmenopausal women, HRT may be safe and effective in patients under 60 years or within 10 years of menopause and has similar fracture efficacy to bisphosphonates. 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.
- **Teriparatide (PTH) and raloxifene are approved for funding** and require special authority**:**
  - Raloxifene 60 mg tablet daily is an option for women who cannot take bisphosphonates.
  - Teriparatide 20 microgram subcut daily is a potent stimulator of bone formation and is funded as a rescue option for those who have a fracture after at least 1 year of bisphosphonate treatment.
  - Additional criteria include:
    - T score is ≤ -3.
    - Must have at least 2 fractures.

Consider Falls Risk

- Review medications (anti-hypertensives, hypnotics), safe environment, physiotherapy, hip protectors. See *Falls section in Older Persons Health* on page 201.

28. Pain Management

28.1 Pain Management Contact Information

Pain Management Centre (Burwood)

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

Acute Pain Management Service (APMS) (Christchurch and Christchurch Women's Hospitals)

- Dr Tim Chapman, Coordinator, APMS
- Richard Craig, Specialist Nurse, pager 8114
- Gayle Morgan, Specialist Nurse, pager 7015 (CWH)

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
- Dr Rachel Wiseman, ☎ 89611
- Dr Amanda Landers, ☎ 027 271 4918
- Willem Vink, Nurse Practitioner, ☎ 81473
- Liz Bremen, Nurse Specialist, ☎ 81885
- Tammy Horton-Davey, Nurse Specialist, ☎ 81985

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, see the Palliative Care Guidelines (online at http://cdhb.palliativecare.org.nz).

28.2 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.

Individualize 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 241 since the treatment of these patients is covered by legislation.*

28.3 Severe Acute Pain

Opioids are the most potent analgesics and should be used where there is a diagnosis of severe pain.

- Morphine remains the gold standard and is generally well tolerated, although nausea can be a problem along with constipation.
- Oxycodone is an alternative opioid to morphine. Oxycodone's bioavailability is about twice that of morphine so careful consideration should be given to appropriate dosing.

- Fentanyl, a synthetic opioid, can also be used, especially where severe pain is anticipated in certain procedures. Discuss with APMS. (Transdermal fentanyl is not appropriate for the management of acute pain.)
- 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 *Serotonin Toxicity* (see page 229).
- Pethidine is used infrequently. It does not have any specific benefit in smooth muscle spasm. It has a short 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.
- 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/catheters.
- 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 mobilization. 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 (Christchurch Hospital) or 7015 (Christchurch Women's) 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.

28.3.1 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/algorithms such as the IV Opioid policy for hospital wards.
- Reassess at regular intervals and adjust prescription accordingly.

28.3.2 Nurse's Responsibilities

- Assess opioid dosing levels at regular intervals.
- Administer opioid according to existing hospital protocols and patient's drug chart.
- 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 sat.O_2 before and at appropriate intervals after the administration of the opioid. See *Frequency of Observation during Acute Pain Medication* on page 216 and *Pain and Sedation Scores* on page 216.
- Request an urgent medical review if the pain protocol/prescription is not fully effective.

28.3.3 Management of Complications

Manage appropriately any untoward effects (see *Management of Severe Complications* on page 217 and *Management of Opioid Side Effects* on page 219).

Table 49 Dosage Guidelines for Systemic Opioids in Acute Pain

Drug	Route	Dose For Adults	Notes & Dose Intervals
Morphine	IM or subcut	0.15 mg/kg	3-6 hourly.
	IV over 1 minute	0.02 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 12.5, 25, 50, 75, 100 microgram/hr	Takes approximately 24 hours to reach steady state. **Not** recommended in acute pain unless under specialist supervision.

1. The oral and rectal routes are not usually recommended for severe breakthrough acute pain.
2. Doses for neonates and children vary. Refer to paediatric guidelines.
3. 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 subcut 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 subcut bolus. This may be appropriate for patients already taking high dose morphine, either orally or via continuous subcut infusion (e.g., via a subcutaneous syringe pump). Refer to the Christchurch Hospital Palliative Care Guidelines.
4. Parenteral oxycodone is not generally recommended in acute pain management.

Table 50 Frequency of Observation during Acute Pain Medication

Route	Frequency[1]
IM/subcut	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 51 Pain and Sedation Scores

Pain Scores		Sedation Scores	
0	No pain	0	Wide awake
1	Mild discomfort	1	Easy to rouse (1a = asleep but easy to rouse)
2	Moderate discomfort	2	Constantly drowsy, easy to rouse but unable to stay awake (e.g., falls asleep during conversation)
3	Painful	3	Severe sedation, somnolent, difficult to rouse
4	Severe pain		
5	Worst imaginable pain		

Table 52 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 (1) ▸ give naloxone (2)
▸ Non-life-threatening	▸ stop opioid administration (1) ▸ 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. In palliative care patients, consider delaying and/or reducing the dose of opioid rather than stopping.
2. 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.)

28.3.4 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.

28.4 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: up to 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. Codeine, 60 mg, 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, and 100 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 stabilized 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 subcut 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 1/6th of total daily morphine dose.

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

Incident Pain:

- Use morphine elixir, 1/6th 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. **This is available in four formulations:** Oxycodone CR tablets (controlled release), OxyNorm™ capsules and OxyNorm oral liquid™ (both immediate release), and oxycodone injection. For more details, refer to the Palliative Care Guidelines.
  - Oxycodone is an opioid analgesic and is approximately twice as strong as morphine when given orally.
  - There have recently been concerns relating to the inappropriate prescribing of oxycodone, its increasing availability in the community, and its high potential for abuse.
  - Oxycodone is similar to morphine in its action and has a similar side effect profile. This drug is more expensive than morphine and there is no clinical evidence to support its use first line. **Morphine therefore remains the drug of first choice for acute and chronic persistent pain.**
  - The main indications for the use of oxycodone are:
    - Persistent hallucinations or other signs of morphine neurotoxicity.
    - Evidence of morphine tolerance/intolerance.

  ***Note**: It is renally cleared and should be used with caution in renal impairment.*

  - See the *Opioid Conversion Guide* on page 219.
- Transdermal fentanyl.
- Methadone.

For advice regarding indications and prescribing, refer to the Palliative Care Guidelines (online at http://cdhb.palliativecare.org.nz). A referral to the Palliative Care Service is strongly recommended particularly if considering fentanyl or methadone - ☎ 81473 (or page via the operator).

***Note:** Pain is a physiological antagonist to morphine induced respiratory depression. Morphine doses can usually be increased until pain is controlled.*

28.4.1 Opioid Conversion

Table 53 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) to morphine or oxycodone, titrate with an initial dose of morphine elixir (or tablets) 10 mg q4h (or OxyNorm 5-10 mg q6h)

28.4.2 Management of Opioid Side Effects

Nausea and vomiting

- Domperidone 10 - 20 mg PO QID before food.
- Metoclopramide 10 mg PO/subcut q4-8h (or QID before food).
- Haloperidol 0.5 mg PO/subcut q8h (can be given as a single nocte dose of 1.5-3 mg).
- Cyclizine 25-50 mg PO q8-12h. Can be given IV q8h or subcut. If prescribed subcut it must be given by infusion with a maximum dose of 150 mg/24 hours.

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. **Domperidone** has fewer side effects, and extrapyramidal reactions are very rare. Can only be given orally.

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 subcut infusion can also be effective for intractable nausea.

Constipation

- Regular stool softeners (e.g., docusate) with stimulants (e.g., bisacodyl, senna) or a combination laxative such as docusate and senna should be used routinely when taking opioids.
- Pinorax™ or Pinorax Forte™ daily is also a combination laxative and can be used in conjunction with docusate and senna. It is licensed for use in opioid-induced constipation but only for patients with advanced incurable disease.
- 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 the Palliative Care Guidelines (online at http://cdhb.palliativecare.org.nz).

Opioid-induced Hyperalgesia

This is a clinical syndrome whereby a patient experiences increased pain, usually to touch, as a result of too high a dose of opioid (or where the opioid has been increased too rapidly) and which may improve on dose reduction.

Reference: Silverman SM. Opioid induced hyperalgesia: clinical implications for the pain practitioner. Pain Physician 2009; 12:679.

28.4.3 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.
- Tricyclic antidepressants, e.g., nortriptyline, and anticonvulsant agents, e.g., sodium valproate, gabapentin, 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.

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

28.5.1 Acute Pain

- Good management of acute pain reduces the chance of developing persistent pain.
- Frequent pain assessment is essential to good pain management and to quality of life.
- 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

Identify patients with high risk factors for developing persisting post surgical pain (see *Summary - Recommendations in the Prevention and Management of Acute Persistent and Chronic Pain* on page 222), and follow up after discharge.

Table 54 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

28.5.2 Chronic Pain

- Chronic pain is pain that persists and lasts beyond the usual healing period. One in six (16.9%) New Zealanders report chronic pain. Chronic pain remains a major public health problem.
- **The biopsychosocial model is helpful here.** Patients' understanding and interpretation of symptoms (beliefs and cognition) can modulate their pain experience. Patients with psychological risk factors (fear avoidance, catastrophizing, pain behaviour, depression), can be identified and early preventative measures instituted.
  - Assessing patients with chronic pain includes a full medical history and detailed examination according to a biopsychosocial approach and applying 'universal precautions' to make a misuse risk assessment.
  - A management plan should consider a range of non-opioid modalities, with a focus on active rather than passive strategies. Integrated multidisciplinary pain services have been shown to improve pain and function outcomes for patients with complex chronic pain issues, but access is often limited.
  - Time-limited opioid use is recommended with initial and regular monitoring, including pain and function scores, urine toxicology, compliance with regulatory surveillance systems and assessment for adverse reactions and drug-related aberrant behaviours.
  - When ceasing prescribing, opioids should be weaned slowly, except in response to violence or criminal activity. Seek advice.
- Refer the patient to the Pain Management Centre, Burwood Hospital.

Reference: Holliday S, Hayes C, Dunlop A. Opioid use in chronic non-cancer pain--part 2: prescribing issues and alternatives. Australian Family Physician 2013;42(3):104-111.

28.5.3 Pharmacological Treatment

- Note allergies, drug intolerances, contra-indications, and adverse effects.
- Analgesics should be individually tailored and monitored.
- 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 (TDS may be sufficient in smaller or frailer patients).
- Non-steroidal anti-inflammatory drugs (NSAIDs): ibuprofen (200 mg eight hourly), diclofenac (50 mg eight hourly), and naproxen (250 mg twelve hourly).
- Tramadol: start oral tramadol, immediate release 50 mg six hourly or tramadol slow release 50 mg twelve hourly. (The long-acting tramadol preparation of 50 mg is not funded in the community; higher long-acting tramadol doses are funded.)
- 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 and should not be used.*

Secondary Analgesics (or Co-Analgesics)

- Antidepressants: start with nortriptyline 5 mg to 10 mg nocte.
- Anti-epileptics (anti-convulsants): start with 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.

Table 55 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 factors	Genetic risk factors Female, younger age Pain before surgery Preoperative chronic pain Preoperative anxiety, fear, and depression Low income, low self-rated health, lack of education.
Intraoperative risk factors	Site e.g., thoracotomy, sternotomy, major limb amputation. Extent and duration of surgery. Incision type. Nerve damage.
Postoperative risk factors	Unrelieved pain Severe pain Surgery in a previously-injured area Amount of analgesics consumed (in the first 7 days) Re-operations
Chronic pain is a major **public health problem**. *Use the* **biopsychosocial rehabilitative** *approach in the patient with persistent or chronic pain* (see page 221).	
Use low dose **multimodal** pharmacological analgesia with a baseline of regular paracetamol. Continue analgesia well into the postoperative period.	
Use **secondary analgesics** in chronic pain.	Use antidepressants (tricyclics) and anticonvulsants (gabapentin and pregabalin) as first line co-analgesics.

29. Palliative Care

For guidelines for referring patients to Palliative Care, see the Palliative Care Guidelines (online at http://cdhb.palliativecare.org.nz)..

For management of pain in the cancer/palliative setting, refer to ***Chronic/Persistent Pain in the Cancer/Palliative Setting*** (see page 217).

30. Poisoning / Drug Overdose

30.1 Management Priorities

Poisonings are a common cause of Emergency Department presentation. Roughly half involve intentional self-harm overdoses or recreational drug use in adults. The other group is children under 5 years with accidental exposure to medications, household chemicals or plants.

Poisoning is a major cause of death in patients under 40 years of age. However if a patient survives to reach hospital then the overall mortality is much less than 1%. This outcome is due mostly to good supportive care. Clinical advice can be obtained from Emergency Medicine staff or the National Poisons Centre (0800POISON). The approach to a poisoned patient can be summarized as follows:

- Resuscitation
- Risk assessment
- Supportive care and monitoring
- Investigations
- Decontamination
- Enhanced elimination
- Antidotes
- Disposition

Appropriate reference material and advice:

- The Toxicology Handbook 2nd edition (Murray, Daly, Little, Cadogan Churchill Livingstone, Sydney) - a concise and practical guide appropriate for Australasian practice.
- TOXINZ.com - an on-line reference available in the Emergency Department and Clinical Pharmacology Drug Information Service.
- Clinical Pharmacology Drug Information Service ☎ 80900 or via the intranet.
- Emergency Medicine Physician, Clinical Pharmacologist or General Physician.

Telephone advice:

- **URGENT:** telephone 0800 POISON / 0800 764 766.
- Non-urgent: Monday to Friday 0900-1700 hours ☎ (03) 479 7227 and ask for Poisons Centre.

30.2 Resuscitation - Airway, Breathing, Circulation and Supportive Treatment

- The first priority in all unwell patients is attention to:
  - Airway, Breathing, Circulation: See *The ABCs* (see page 63)
- Many poisoning patients have potential A, B and C problems which should be anticipated, identified, then managed expediently.
- Central nervous system depression or convulsions can cause problems with airway and breathing. Arrhythmias or cardiovascular depression are serious consequences of some poisonings.
- Seizures should be anticipated by the type of poison ingested. Common causes are tricyclic antidepressants, venlafaxine and amphetamines (including 'legal' recreational drugs). Most toxic seizures will respond to benzodiazepines in sufficient doses. Phenobarbitone is second line. Level of consciousness and airway must be closely monitored if using this agent. Phenytoin is contraindicated for toxic seizures.
- Check for hypoglycaemia, hypothermia or hyperthermia. A core temperature above 38.5ºC from a toxic cause is associated with serious toxicity and requires urgent cooling.
- The majority of poisoned patients can be managed supportively and expectantly. A few require gastrointestinal decontamination. This should not be considered routine but an intervention with specific indications. A small number of drugs have specific antidotes and these should be used judiciously.

30.3 Risk Assessment

The key step to managing poisoned patients is a realistic estimation of the likely toxicity expected for a poisoning episode. This prediction should include the maximum possible effect on the patient and the likely time course of events.

This assessment will provide a quick guide to whether the effects are likely to improve or worsen. Toxicity can be estimated as nontoxic, mild, moderate or severe. The pros and cons of possible interventions are balanced against this careful assessment of risk.

Key information:

- Identify the poison correctly (careful with trade names and homonyms).
- Estimate dose taken and express as per kg body weight. If unknown, estimate from the maximum amount that could have been taken.
- Time of exposure (is peak absorption expected soon or has it passed?).
- Clinical features already observed: signs and toxidromes.
- Refer to current poisons reference database or text.
- Consider the patient's intent in relation to the poison exposure. A serious suicide attempt is usually associated with large doses, whereas exploratory ingestions by small children are almost always sub toxic.

Toxidromes: a number of toxins/poisons produce recognisable toxic syndromes which may be rapidly identified at the bedside. These indicate systemic toxicity.

- **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: D**efecation, **u**rination, **mi**osis, **b**radycardia, **e**mesis, **l**acrimation, **s**alivation (DUMBELS).
- **Nicotinic e.g., insecticides:** Tachycardia, hypertension, muscle fasciculations, weakness, paralysis.
- **Narcotic:** CNS depression, hypotension, hypoventilation, miosis.
- **Withdrawal e.g., from opioids, alcohol, benzodiazepines:** Diarrhoea, mydriasis, tachycardia, lacrimation, abdominal pain, hallucinations, piloerection.

30.4 Supportive Care

Good supportive care of vital functions is probably the most important aspect of managing the poisoned patient.

A good management plan involves documenting:

- The type of observations required, noting the expected side effects of poisoning.
- Triggers for notification of medical staff.
- Criteria for changing management.
- Plan for psychosocial risk assessment where suicidal intent is suspected.

30.5 Investigations

Vital signs, blood glucose and ECG are important basic investigations.

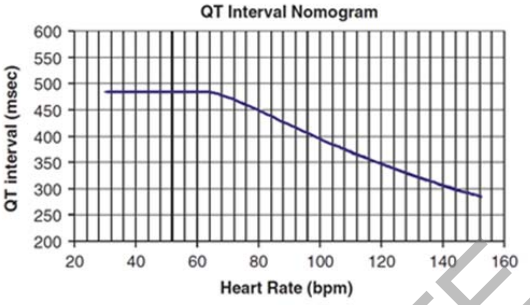
A screening paracetamol level is advisable in self-harm poisonings. Paracetamol toxicity has no early clinical signs and fulminant hepatic toxicity is preventable if treatment is started early. A venous blood gas may detect acidosis or severe hypercapnia. Specific drug levels may be of use with a limited number of drugs:

- Carboxyhaemoglobin
- Carbamazepine
- Salicylate
- Digoxin
- Methanol
- Iron
- Lithium
- Valproic acid

Toxicology screening of urine is available but is rarely used to guide management.

A number of drugs can cause QT prolongation with the associated risk of torsades de pointes (TdP), e.g., haloperidol, chlorpromazine, citalopram, etc.

ECGs can be taken and the QT interval plotted on the nomogram below to assess the risk of TdP. The nomogram is more reliable than the QTc as the QTc underestimates the risk of TdP at low heart rates. A QT interval measured above the nomogram line should be considered a risk for TdP. These patients should have a period of cardiac monitoring or serial ECGs until the QT normalizes.



The QT nomogram is a risk assessment tool that accurately predicts arrhythmogenic risk for drug-induced QT prolongation. The sensitivity and specificity of the QT nomogram is 97% and 99% respectively (Bazett QTc =500 ms is 94% and 97% respectively).

Reference: Chan A, Isbister GK, Kirkpatrick CM, Dufful SB. Drug-induced QT prolongation and torsades de pointes: evaluation of a QT nomogram. QJM. 2007 Oct;100(10):609-15.

30.6 Decontamination of the Gastrointestinal Tract

After attention to ABC and risk assessment, gastrointestinal decontamination measures may be considered. There is no strong evidence that decontamination improves major outcomes. Decontamination is therefore not routine, but considered if significant toxicity is likely. The method chosen must have the potential to be effective with minimal risk to the patient.

The options include:

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

Activated Charcoal

- First choice for gastrointestinal decontamination, if any is indicated.
- Give 1 g/kg body weight (max 50 g) as a single dose orally.
- Repeated doses are indicated with some poisonings to interrupt the enterohepatic circulation and possibly as enteral dialysis (e.g., theophylline slow-release).
- **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 (remember: alcohols, acids, alkalis, ions, metals).
- Charcoal should be given only if the perceived benefits outweigh the risks. Activated charcoal may cause vomiting, and if aspirated, will cause a lung injury. It should not be used for trivial or low risk overdoses. There are some overdoses where charcoal is effective greater than 1 hour after ingestion. Consult the TOXINZ internet database.
- **Charcoal should not be given to patients who may become unconscious or have seizures (unless they are already intubated).**

Gastric Lavage

- The indications for gastric lavage are very limited. Consider for a serious poisoning in which charcoal is ineffective and the airway is protected. Consult senior medical staff if you think it is indicated.

Whole Bowel Irrigation

- May be considered for serious poisonings with:
  - 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 polyethylene glycol (e.g., Klean-Prep™). Call the National Poisons Centre for the latest protocol.

There are many practical difficulties. Consultation with senior medical staff is essential.

30.7 Enhanced Elimination

These may include alkaline diuresis, haemodialysis or haemofiltration. Consult with senior medical staff. Haemoperfusion is not readily available at Christchurch Hospital.

30.8 Antidotes

- Logical antidotes antagonize at the level of cellular mechanisms, e.g., naloxone for narcotics and flumazenil for benzodiazepines, N-acetylcysteine for paracetamol.
- Empiric antidotes are drugs designed to achieve a physiological result, e.g., inotropes to restore blood pressure, atropine to raise heart rate etc.
- **There are few specific antidotes that have proven benefit without significant risk of adverse effects. N-acetylcysteine is a good example. Other agents should be used judiciously.**

30.9 Case Management

- Most patients will be assessed, observed, and discharged from ED.
- Patients who are critically unwell, unstable, or who require airway management or dialysis are admitted to ICU under a General Medicine Team.
- Patients who require prolonged antidotal treatment or who are expected to have prolonged toxicity (>12 hours) should be referred to General Medicine for admission.
- Those requiring prolonged cardiac monitoring who are alert and cooperative are admitted under General Medicine on telemetry (AMAU or Ward 12).
- Psychiatric Emergency Services are available if there is possible suicidal intent.

30.10 Specific Poisons

30.10.1 Paracetamol

- 140 mg/kg is the threshold dose at which an oral overdose can cause hepatotoxicity in adults. Hepatotoxicity is not uncommon but fatalities are rare. Charcoal may be useful up to 2 hours.

Follow the Australasian Consensus guidelines unless there are good reasons for deviating *(Daly FF, Fountain JS, et al. Med J Aust 2008; 188: 296-301).*

- Single dose, staggered dose and late presentations of paracetamol poisoning are described and management guidelines provided. Access at TOXINZ.com or from ED.
- Blood levels direct treatment. In adults, levels should be taken at 4 hours (or immediately if presentation is more than 4 hours after the ingestion). Use the green lithium heparin tube.
- Treatment is effective if begun within 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 has developed renal toxicity.

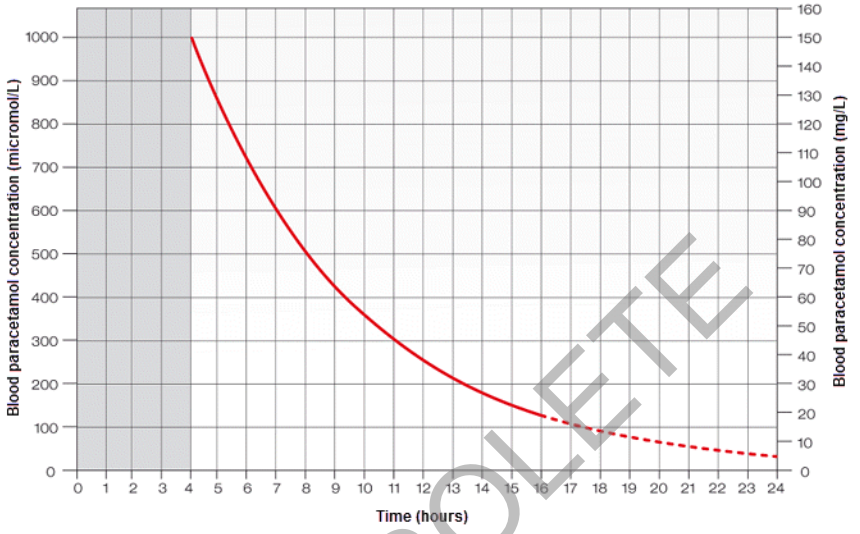
- 72 - 96 hours: peak of transaminase elevation, bilirubin and prothrombin time. Nausea and vomiting returns. Elevated creatinine. Fetal death if pregnant.
- 4 days to 2 weeks: resolution or hepatic failure (then either transplant or death).

Note: *Treatment is directed by the* **nomogram** *(overleaf).*

Note: *Treat* ***all*** *patients with plasma levels above the nomogram line with N-acetylcysteine.*

Note: *Ensure that the correct units are used (i.e. micromol/L or mg/L) when plotting on the nomogram.*

Table 56 Paracetamol Toxicity Treatment Nomogram



Reference: Daly FF, Fountain JS, et al. Med J Aust 2008; 188: 296-301. Adapted from Smilkstein MJ et al. Ann Emerg Med 1991; 20: 1058-63.

Table 57 N-acetylcysteine Dosage in Paracetamol Poisoning

N-acetylcysteine:

INITIALLY: 150 mg/kg in 200 mL 5% glucose over 60 minutes
THEN: 50 mg/kg in 500 mL 5% glucose over 4 hours
THEN: 100 mg/kg in 1000 mL 5% glucose over 16 hours

- N-acetylcysteine is most effective when given within 15 hours of ingestion but there is some 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.
- **Anaphylactoid reactions:**
  - 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 reactions, treat with adrenaline.** See *Anaphylaxis* (see page 72).
- The Australasian Consensus guidelines give specific advice in the event of late presentation, staggered overdose, therapeutic overdose and slow release preparations.

30.10.2 Tricyclic Antidepressants

- Cause multiple receptor effects (sodium, potassium, and calcium channels, muscarinic and alpha-receptor blockades, some sympathetic agonist effects). At higher doses sodium channel blockade predominates and can be lethal.
- Large volume of distribution, liver metabolism with enterohepatic circulation.
- The main toxic effects are usually apparent within 4 hours of ingestion:
  - CNS depression, irritation and convulsions.
  - Anticholinergic effects.
  - Tachycardia first, arrhythmias next, then progression to cardiovascular depression.

  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.

Table 58 Effects of tricyclic antidepressant overdosage

Dose	Effect
<5 mg/kg	Minimal symptoms
5-10 mg/kg	Drowsiness and mild anticholinergic symptoms
>10 mg/kg	Potential for coma, hypotension, seizures and arrhythmias within 2-4 hrs
>30 mg/kg	Life-threatening toxicity with pH dependent cardiotoxicity and coma.

- 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 that may reduce the cardiotoxicity caused by sodium channel blockade. Give 50-100 mmol sodium bicarbonate IV over 5-10 minutes. Improvement may be apparent within minutes.
- If intubated for airway management then hyperventilation will 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.
- Many arrhythmias will respond to bolus bicarbonate as above. All drugs that prolong depolarization are contraindicated.
- Lignocaine 1.5 mg/kg by IV bolus is considered the next line of treatment. Magnesium may be considered. Pacing, cardioversion and defibrillation are relatively safe but unlikely to be effective.
- Hypotension can be treated with IV fluid boluses and bicarbonate. If persistently hypotensive then vasopressors such as adrenaline or noradrenaline can be infused.
- 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 a mental health assessment.

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

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

- Overdoses with SSRIs **alone** cause sedation and usually have a good outcome with supportive care.
- Serious toxicity and death can occur when SSRIs are taken in combination with MAOIs or tricyclic antidepressants (see *Serotonin Toxicity*, below).
- Seizures have been reported and are significantly more common with SNRI overdosage.
- In most cases CNS depression will predominate.
- Treatment is supportive.

Serotonin Toxicity

- May occur with **combinations** of drugs such as MAOIs with SSRIs, SNRIs, clomipramine, other tricyclic antidepressants, lithium, pethidine, tramadol.

- The **Hunter Serotonin Toxicity Criteria** are used to diagnose serotonin toxicity. Diagnosis requires a combination of the above 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 (see TOXINZ for criteria).
- Serotonin toxicity is usually mild and transient. Treatment is supportive. Diazepam is first line for agitation.
- Hyperthermia **above 38.5°C** indicates serious toxicity and may herald the onset of multiorgan failure. Treatment for hyperthermia needs to be aggressive and will usually involve active cooling, intubation and paralysis.
- Seizures can occur with citalopram and escitalopram. Dose dependent QT prolongation can occur. Torsades de pointes (TdP) has been reported in rare cases. Those at higher risk of arrhythmia are patients taking a large overdose (>600 mg), or those with congenital QT prolongation, pre-existing heart conditions or >60 years old. Cardiac monitoring may be required. Consult a Specialist for advice if a patient has risk factors for serious toxicity. Use the *QT nomogram to assess risk* (see page 225).

30.10.4 Sodium Valproate

Valproic acid is an anticonvulsant that is commonly used as a mood stabilizer. In overdose, response may be variable and development of toxicity may be delayed up to 12 hours.

Elimination by haemodialysis may be considered in cases where severe toxicity is anticipated (dose >1000 mg/kg) and ideally before multiorgan dysfunction has occurred (consult ICU).

Table 59 Effects of sodium valproate overdosage

Dose	Effect
<200 mg/kg	Asymptomatic or mild drowsiness and/or ataxia
200-400 mg/kg	Variable CNS depression
400-1000 mg/kg	Significant CNS depression. Coma requiring intubation may occur up to 12 hours post ingestion. Multiorgan toxicity more likely in higher dose range.
>1000 mg/kg	Potentially lethal with profound coma and multi-organ failure (cerebral oedema, hypotension, lactic acidosis, hypoglycaemia, hyponatraemia, rarely hypernatraemia, etc)

30.10.5 Antipsychotics: Phenothiazines and Butyrophenones

- Dopamine receptor blockade can produce symptoms such as dystonia, akathisia, parkinsonism, tardive dyskinesia and neuroleptic malignant syndrome.
- Most commonly seen are dose dependent CNS depression, tachycardia, hypotension and anticholinergic effects.
- Treatment is generally supportive. Hypotension is common and responds to fluid loading. QT prolongation commonly occurs but arrhythmias are extremely uncommon - see *risk assessment* on page 225.
- Dystonic reactions can be treated with benztropine 2 mg IM or IV.

Neuroleptic Malignant Syndrome

- A life-threatening idiosyncratic reaction to older 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)
- Check CBC, CK, Na, K, creatinine.
- 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.

30.10.6 Antipsychotics: Quetiapine

- Quetiapine is a second generation atypical antipsychotic agent and is common in intentional overdoses. Symptoms of overdose are sedation, confusion, delirium, coma, tachycardia and hypotension. CNS depression is predictable and dose dependent. Seizures occur infrequently.
- Hypotension is common. Most patients are otherwise healthy adults and will tolerate mild hypotension. Excessive IV fluid loading is not required as long as patients appear clinically well perfused. Clinically significant QT prolongation is rare and torsades de pointes has not been reported.
- Risk assessment:
  - <3 g mild to moderate sedation and sinus tachycardia
  - >3 g Increasing risk of CNS depression progressing to seizures and coma.
- Onset of symptoms may occur within 2-4 hours and may last 24-72 hours. Sedation is common and therefore decontamination is contraindicated. For the majority observation and supportive treatment is sufficient. Coma and profound hypotension will require Intensive Care treatment with invasive fluid management and vasopressor support.

30.10.7 Benzodiazepines

- Rarely serious in isolation but may need prolonged observation.
- 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 as t½ of benzodiazepines is usually longer.*

***Note**: Do not use flumazenil in mixed overdoses (e.g., benzodiazepines and tricyclic antidepressants). It may precipitate toxic seizures.*

30.10.8 Opiates

- Altered level of consciousness, respiratory depression (typically a very slow respiratory rate with a maintained tidal volume), miosis, hypothermia.
- 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 naloxone 0.4 mg per hour may be required.
  - Naloxone will reverse all the actions of the narcotic including analgesia.
  - May bring about an agitated 'withdrawal' state in an addict so reverse in small aliquots to achieve spontaneous respiration without full reversal.

30.10.9 Lithium

Acute lithium overdosage is more benign than chronic toxicity

- Well absorbed orally. Eliminated solely by the kidneys.
- Activated charcoal doesn't bind lithium.
- In acute overdosage with normal renal function <25 g is benign (even if on therapeutic lithium already, i.e., acute on chronic overdose).
- Coma is very unusual with acute lithium overdose.
- Nausea, vomiting, pain and diarrhoea may occur due to GI irritation.
- Correct dehydration. Ensure renal function is normal.
- Patients with no neurotoxicity, good urine output and serum lithium of <2.5 mmol/L do not require further medical care.

Chronic lithium toxicity

- Lithium neurotoxicity usually due to impaired lithium excretion.
- Much higher risk of serious toxicity and permanent neurological damage.

- Lithium excretion can be impaired by dehydration, NSAIDs, ACE inhibitors, or diuretics.
- Consider chronic toxicity in anyone on therapeutic lithium who presents with neurological symptoms or decreased consciousness.
- Hansen and Amidsen Grading:
  - Grade 1 Tremor, hyperreflexia, weakness, ataxia.
  - Grade 2 Stupor, rigidity, hypotonia, hypotension.
  - Grade 3 Coma, convulsions.
- Serum levels may not correlate with CSF levels or clinical toxicity.
- Management requires careful attention to correct water and sodium deficits.
- Haemodialysis may be useful if deteriorating with significant symptoms and level >2.5 mmol/L or if urine output inadequate.
- Significant toxicity will require ICU care.

30.10.10 Digoxin and other Cardiac Glycosides

- Acute poisoning:
  - Digoxin plasma concentration can be measured and may be useful.
  - Oleander, foxglove, lily of the valley and rhododendron all contain toxic glycosides.
  - Plasma potassium rises in proportion to toxicity.
- Chronic poisoning:
  - Digoxin levels may not be very high (tissue distribution).
  - Life-threatening toxicity is more common in the elderly with acute renal impairment or supra-therapeutic dosing.
  - Plasma potassium may be normal or low.
- Treatment:
  - Bradyarrhythmias:
    - Atropine IV to a maximum of 2 mg. May need pacing.
  - Tachyarrhythmias.
    - Magnesium sulphate 2-4 g IV.

Note: If cardioversion is required, it may precipitate ventricular tachycardia/ventricular fibrillation, or asystole. Use low energy (10-25 joules). Seek Consultant advice.

  - Hyperkalaemia: ***Standard treatment*** (see page 161), but avoid calcium as it may potentiate digoxin cardiotoxicity.
- Fab fragments (Digibind™ - kept in the Emergency Drug Cupboard):
  - 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 sodium chloride 0.9% (10 amps if unstable).
    - If no response repeat dose.
    - If still no response in adults, see TOXINZ internet database and seek Consultant advice.
  - For dosage in less urgent clinical situations, see Digibind Drug insert.
  - Cautions 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.

30.10.11 Carbon Monoxide Poisoning

- Carbon monoxide (CO) is a common cause of non-medicinal poisoning. Most exposures happen secondary to suicidal intent but some may occur accidently at home or the workplace. An early carboxyhaemoglobin level may be of use in an occupational safety investigation.

- 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 randomized double-blind clinical trial suggests that where it is readily available (as in Christchurch), it should be offered to some patients (see indications below).
- Consider the diagnosis in all burns, smoke inhalation, coma or attempted suicide cases.

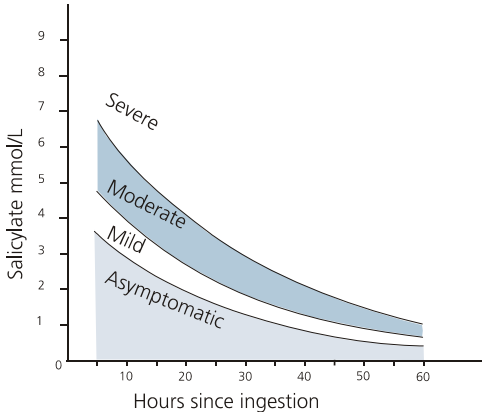
Treatment

- 100% oxygen for all patients via a breathing system with a tight-fitting mask, reservoir bag and high fresh gas flow for at least 6 hours. This hastens the elimination of CO.
- Indications for HBOT include presence of at least one of these high risk criteria:
  - Loss of consciousness (clear history of LOC or unconscious 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 >55 years
    - Metabolic acidosis
  - Pregnancy - fetal haemoglobin avidly binds CO making the fetus very susceptible to hypoxic injury.
- To arrange HBOT, or for advice, contact the Hyperbaric Medicine Unit duty doctor (see *Referral of Patients to Hyperbaric Unit* on page 141.)

30.10.12 Aspirin (and other salicylates)

- Acute poisoning: classical symptoms of vomiting, tinnitus, hyperventilation (initial respiratory alkalosis) followed by metabolic acidosis. Severe toxicity may result in coma and seizures.
- Chronic poisoning (over 12 hours) has a higher morbidity and mortality - 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).
- Risk assessment:
  - Approx 150 mg/kg causes vomiting.
  - 150-300 mg/kg mild/moderate toxicity.
  - >300 mg/kg moderate/severe toxicity.
  - >500 mg/kg potentially lethal.
- 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.
- Venous or arterial blood gas. An abnormal pH may be a better indication of toxicity than salicylate levels, particularly if the time since ingestion is uncertain.

Table 60 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 activated charcoal 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. Charcoal can be repeated if levels continue to rise after 4 hours.
- IV fluids - all patients with significant salicylate poisoning are dehydrated.
- Check blood glucose, Na, K, creatinine, INR and APTT.
- If metabolic acidosis is present or if significant symptoms, then **alkalinize urine** - sodium bicarbonate 1 mmol/kg boluses IV until pH greater than 7.5, then 1000 mL of 5% glucose, plus 100 mmol of sodium bicarbonate + 40 mmol potassium chloride and the rate adjusted according to regular measurements of pH, potassium, sodium 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 5 mmol/L or if very unwell, then **consider haemodialysis** (contact Nephrologist on call). Transfer to ICU.

30.10.13 Local Anaesthetics

- Local anaesthetic (LA) toxicity usually occurs in the setting of regional anaesthesia. Ropivicaine and lignocaine are the most common LAs used in the hospital.
- Toxicity occurs with inadvertent intravascular injection or rapid systemic redistribution.
- Herald signs are dizziness, tinnitus, unexpected agitation, confusion and tachycardia which may progress to seizures, coma and cardiovascular compromise.
- Management:
  - Stop procedure. Summon help immediately. Resuscitate if necessary.
  - Reinflate cuffs if performing Bier's block.
  - Treat seizures with benzodiazepines. Arrhythmias may be treated with sodium bicarbonate 8.4% 100 mL by IV bolus. Repeat if necessary. Cardiac arrest or pre-arrest states can be treated initially with lipid emulsion (Intralipid) 1.5 mL/kg of 20% IV push.

30.10.14 Recreational Stimulants and Party Pills

- Included in this class are amphetamines, methamphetamine (P), methylenedioxymethamphetamine (MDMA), 3,4-Methylenedioxyamphetamine (MDA), *para*-Methoxyamphetamine (PMA), 4-methylmethcathinone (Mephedrone, 4-MMC), 3,4-methylenedioxy-N-methylcathinone (Methylone, bk-MDMA), methylenedioxypyrovalerone (MDPV).
- Usual constituents of party pills/Herbal Highs are 1,3-dimethylamylamine (DMAA), butylone, benzylpiperazine (BZP) and 3-trifluoromethylphenylpiperazine (TFMPP).
- Hallucinogenic amphetamine-like actions.
- Absorption related to administration route.
- Variable 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 well established as the potency and purity vary. Severe toxicity can be due to cerebral haemorrhage, cerebral oedema, hyponatraemia or toxic hyperthermia. Deaths have occurred after ingestion of a single tablet.

- **Treatment** - mainly supportive care
  - Consider activated charcoal.
  - If mild toxicity, observe for 4 hours after resolution of symptoms.
  - Benzodiazepines for agitation and/or seizures.
  - For refractory seizures, use phenobarbital (phenytoin contraindicated).
  - ECG monitoring - treat tachyarrhythmias with beta-blockers.
  - Hypertension may respond to beta-blockers or may require treatment with short acting agents, e.g., GTN infusion titrated to response.
  - *Hyperthermia* (see page 78) - temperatures >38.5°C may herald multiorgan toxicity. Aggressive cooling is required.

30.10.15 Synthetic Cannabinoids

- Synonyms: Spice, K2, Kronic, endocannabinoids.
- Are a large number of compounds that are agonists of endo-cannabinoid receptors CB1 and CB2.
- Heat-stable and usually administered by smoking.
- Onset of effects rapid and include euphoria, disorientation, sedation progressing to agitation, and paranoia.
- More serious adverse effects include psychosis, seizures, toxic kidney injury and coronary vasospasm causing myocardial infarction.
- Chronic and habitual use are commonly reported.
- Withdrawal syndromes and cannabinoid hyperemesis have been observed.
- Treatment:
  - Mainly supportive care.
  - Remember to check renal function.
  - Large doses of benzodiazepine may be required for violent agitation/psychosis.
  - There are no specific antidotes for the treatment of withdrawal or cannabinoid hyperemesis.

30.10.16 Alcohol

See *Alcohol Related Problems* (see page 11).

31. Psychiatry

31.1 Psychiatric Services Contact Information

- **Psychiatric Emergency Service (PES)**: ☎ 80482 between 0830 - 1700 (after-hours, page via the Christchurch Hospital operator).
- **Psychiatric Consultation Service**: ☎ 86615 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 198 for contact details.

31.2 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, benzodiazepines).
- As a severe disagreement or misunderstanding in staff/patient relations, sometimes augmented by alcohol or other drug abuse.
- Occasionally as a factitious disorder.

31.3 Delirium

Refer also to the CDHB Guidelines for Care of Patients with Delirium, Ref. 0020 (search for "delirium guidelines" on the CDHB intranet).

31.3.1 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.
- **Disorganized 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 237) and a cognitive screening test, *Mental Status Quotient (MSQ)* (see page 166), Mini Mental State Examination (MMSE), or Montreal Cognitive Assessment Test (MOCA).

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 61 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: Disorganized thinking
This feature is shown by a positive response to the following question: ▸ Was the patient's thinking disorganized 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.

31.3.2 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.

31.3.3 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 62 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 ("serotonin toxicity").

31.3.4 Management of Delirium

- Prevention: vigilance in high risk patient, accurate medication/drug/alcohol history, optimize 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 (utilize support of friends/family) - includes provision of clock, calendar, familiar objects, view to outside.
  - Quiet, single room whenever possible; minimize room changes.
  - Make sure glasses and hearing aids are worn.
  - Minimize 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 0.25 - 0.5 mg PO or subcut, once or twice daily. In urgent situations, higher doses (0.5 - 2.5 mg) of haloperidol may be necessary (best given subcut or IM with additional doses every 30-60 minutes as required).
  - Younger patients may need higher doses (1 mg subcut or IM initially, for milder symptoms; up to 5 mg for severe), repeated every 30-60 minutes as required.

- If IV haloperidol is contemplated **discuss with the Consultant first** as there is a risk of prolongation of QT interval and torsades de pointes tachycardia with higher doses. IV treatment seldom causes extrapyramidal side effects.
- **Cerebral Lewy body disease (Parkinson's disease, dementia with Lewy bodies):** neuroleptics with dopamine-blocking action can cause serious neurotoxic reactions. If tranquillizer is required, use lorazepam 0.25 - 0.5 mg PO once or twice daily. In emergencies, use lorazepam 0.5 - 1 mg IM or clonazepam 0.25 - 0.5 mg IM. For distressing psychotic symptoms oral atypical neuroleptics in low dosage (e.g., quetiapine 12.5 - 25 mg PO) may be used (Consultant endorsement recommended).
- **Alcohol withdrawal** (see page 11) **or benzodiazepine withdrawal delirium** (see page 241): 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.
- Psychotropic drugs should be tapered gradually as target symptoms resolve - usually over 1-2 weeks. At discharge, every remaining psychotropic drug must have a withdrawal plan in the discharge summary, or a rationale for its continuation, especially any started during the admission.
- Resolution of delirium may be prolonged - sometimes many weeks.

31.4 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 when 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.

31.4.1 Management

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 uncooperativeness 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.

Preferred Antipsychotic Drugs

Table 63 Oral/parenteral doses of antipsychotic drugs

Drug	Acute		Max Daily Dose
	IM	PO	
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

Common side effects of antipsychotic drugs

- Agitation (initially).
- Metabolic syndrome (including weight gain).
- Extrapyramidal side effects: parkinsonism; acute dystonias; dyskinesias. These are less common with these medications than with typical (older) antipsychotics.

  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.
- The preferred benzodiazepine is lorazepam 1 - 2 mg IM or PO (max daily dose 8 mg).

31.5 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. Medications should be first line only for moderate or severe depression.

31.5.1 Management

- Selective serotonin re-uptake inhibitors (SSRIs) are first choice in patients over 25, 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 re-uptake inhibitors (SNRIs) are second choice in patients over 25. Give venlafaxine 75 mg initial daily dose, max daily 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.

  Note: Caution in patients who are under 25. *Fluoxetine is the first choice; paroxetine and venlafaxine should be avoided; citalopram should be used with caution and tricyclics have not been shown to be effective.*
- 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).

31.6 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. Although this is likely to be as a consequence of depression, 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.

31.6.1 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.
- Utilize 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 amended 2008 on the internet). You are able to detain a person against their will in this situation and will not be criticized or encounter legal difficulties for doing so.*

31.7 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 is used for withdrawal management. Convert average daily dose into "equivalent" dose of diazepam, and give in 3-4 divided doses up to a maximum of 40 mg/24 hours. Reduce dose by 5-10% daily.
  - A benzodiazepine reduction regime is available from the Community Alcohol and Drug Service.
- **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.
  - Use opioid withdrawal medications in the first instance. Clonidine is a useful adjunct.
  - If considering an opioid withdrawal/detox regimen (e.g., methadone), please consult with the doctors at the Community Alcohol and Drug Service.
  - Further information about drug withdrawal management is available from *Substance Withdrawal Management: Guidelines for Medical and Nursing Practitioners in Primary Health, Specialist Addiction, Custodial and General Hospital Settings* available on www.matuaraki.org.nz.

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).*

31.8 Opioid Substitution Programmes

Buprenorphine with naloxone (Suboxone) sublingual tablets are now subsidized for the treatment of opioid dependence, and patients on this medication are managed by the Christchurch Methadone Programme.

Methadone and buprenorphine/naloxone prescribing and supply

- The Christchurch Methadone Programme (CMP) is 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 authorized under the Misuse of Drugs Act 1975.
- The prescribing and administration of methadone or buprenorphine/naloxone to patients on an opioid substitution 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 publications, *Practice Guidelines for Opioid Substitution Treatment in New Zealand, 2008* and *New Zealand Clinical Guidelines for the Use of Buprenorphine (with or without Naloxone) in the Treatment*

of Opioid Dependence 2010. These are 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 an opioid substitution programme.*

- **Patients who are enrolled with an opioid substitution programme are not to receive methadone or buprenorphine/naloxone in any CDHB hospital until their daily dose (in milligrams) has been confirmed with the patient's CMP case manager:**
  - Weekdays between 0830 and 1700 hours, Christchurch Methadone Programme, ☎ 335 4350
  - After-hours, phone the Kennedy Detox Centre, ☎ 339 1139
- Some patients on buprenorphine/naloxone may only be prescribed a dose every 2-3 days due to its long half-life.
- No methadone or buprenorphine/naloxone is to be prescribed or dispensed until confirmation is obtained of the last consumed dose. This is to protect against accidental overdose. CMP/Kennedy will check with the dispensing pharmacy to determine whether the patient has had their dose on the day of admission and if takeaways were provided, and will suspend third party scripts. This is so that extra supplies of methadone or buprenorphine/naloxone cannot be collected by a third party while the patient is admitted.
- If the patient presents in the afternoon or evening they have probably already attended their pharmacy. If the time of the last consumed dose is unable to be confirmed, do not prescribe methadone or buprenorphine/naloxone until the following day.
- If the patient brings takeaway doses into hospital, these doses should only be used for the patient to consume after they are discharged from hospital.

  Note: *Takeaway doses of methadone are diluted.*
- Prescribers to patients receiving methadone or buprenorphine/naloxone need to ensure that:
  - The potential for overdose is minimized,
  - The patient is not unsafely intoxicated with other drugs, and
  - The potential for diversion is limited.
- Written authorization must be obtained from the CMP before hospital doctors can prescribe methadone or buprenorphine/naloxone to in-patients. The CMP has provided a form that can be faxed to obtain appropriate authorization. Each authority to prescribe lasts one week. The CMP can extend or cancel authorities on request.
- 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.
- For out-of-town patients receiving methadone or buprenorphine/naloxone:
  - Contact the originating programme to confirm dosage, find out when it was last dispensed, and determine if the patient is in possession of any takeaway doses not yet consumed. **This is the responsibility of the admitting team**. The patient will know some of these contacts but any details they provide must be checked.
  - 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 patients attempting to obtain further doses in the community resulting in overdose as has occurred in the past).
- 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. Due to the partial agonist activity of buprenorphine, the management of acute pain can be clinically challenging and advice should be sought from the Acute Pain Team.
- At discharge, the CMP case manager or (if after hours) the Kennedy Detox Centre must be called to arrange reinstatement of opioid supply in the community.
- Do **not** discharge any patient with a methadone or buprenorphine/naloxone supply or prescription.

Driving Considerations

- For all patients prescribed methadone or buprenorphine/naloxone, it is important to consider the implications of their 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.

32. Respiratory Medicine

32.1 Respiratory Medicine Department Information

Main Office

2nd Floor, Riverside, ☎ 80280, fax 80914

Inpatient Services

Two inpatient teams on Ward 25:

- Resp 1 - Dr Greg Frazer / Dr Richard Laing / Dr Christina McLachlan / Dr Bronwen Rhodes / Dr Rachel Wiseman
- Resp 2 - Dr Lutz Beckert / Dr Chris Drennan / Dr Mike Epton / Dr Michael Hlavac / Dr Libby King

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 Integrated Specialist Service (CRISS), ☎ 88303, fax 80849
  - Domiciliary oxygen service
  - Maori Respiratory Educator
- Respiratory Education Service
  - Respiratory Education Nurse, ☎ 81140, fax 81260
- Pulmonary Rehabilitation - referral details on HealthPathways
- Respiratory Outpatients
  - Enquiries and appointments, ☎ 80280, fax 80914
  - Clinic Nurse, ☎ 80463, fax 81260
- Respiratory Research Group
  - Dr Malina Storer, Research Manager, ☎ 89040, fax 86299
- Sleep Unit
  - Paul Kelly, Technical Director, ☎ 81089, fax 81089

Refer to the Respiratory Services Protocols and Guidelines (online at http://respiratory.streamliners.co.nz).

32.2 Respiratory Failure

32.2.1 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.*

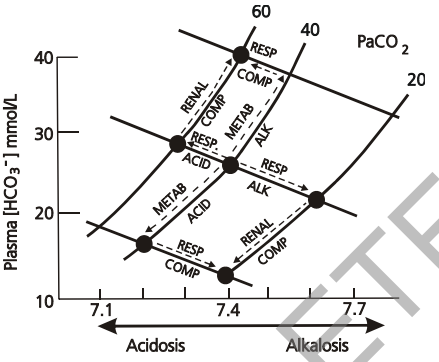
32.2.2 Classification

- 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 96):



32.2.3 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 64 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 65 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 from a 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.*

32.3 Obstructive Sleep Apnoea

32.3.1 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 characterized 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 recognized 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 below).
- Overnight oximetry to estimate a desaturation index (DI). This can be requested through the Sleep Unit using a Sleep Studies Request Form (search for "C270075" on the CDHB intranet).

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 66 Epworth Sleepiness Score

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently, try to work out how they would have affected you. Use the following scale to choose *the most appropriate number* for each situation:

0 = would *never* doze
1 = *slight* chance of dozing
2 = *moderate* chance of dozing
3 = *high* chance of dozing

	Chance of Dozing
Sitting and reading	
Watching television	
Sitting inactive in a public place (e.g., theatre, meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	

32.3.2 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 be 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.

32.4 Chronic Obstructive Pulmonary Disease (COPD)

32.4.1 Summary

- Smoking is the most important risk factor for COPD, and should be addressed at 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 which one should also consider, including higher risk of lung cancer, ischaemic heart disease, diabetes and osteoporosis.
- 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.

32.4.2 Definition

Chronic Obstructive Pulmonary Disease is characterized by airflow limitation that is **not fully reversible**. The airflow limitation is in most cases both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases. It is a progressive, disabling disease with serious complications and exacerbations that are major burdens for healthcare systems.

Small-airway narrowing (with or without chronic bronchitis) and emphysema caused by smoking are the common conditions resulting in COPD. Chronic bronchitis is daily sputum production for at least three months of two or more consecutive years. Emphysema is a pathological diagnosis, and consists of alveolar dilatation and destruction. Breathlessness with exertion, chest tightness and wheeze are the results of airway narrowing and impaired gas exchange. The loss of lung elastic tissue in emphysema may result in airway wall collapse during expiration, leading to dynamic hyperinflation and consequent increased work of breathing.

The clinical features and pathophysiology of COPD can overlap with asthma, as most COPD patients have some reversibility of airflow limitation with bronchodilators. By contrast, some non-smokers with chronic asthma develop irreversible airway narrowing. However patients with complete reversibility of airflow limitation should be treated as asthma.

Differentiation of COPD from asthma is often difficult, and is best undertaken by a detailed history. COPD often presents later in life, with insidious and gradual onset of breathlessness, with less diurnal variation, associated with a history of exposure to noxious gas (usually cigarette smoking).

32.4.3 Causes of acute deterioration of COPD

- Acute bronchitis (viral or bacterial).
- Pneumonia.
- Pneumothorax.

- Pulmonary embolism.
- Left ventricular failure.
- Sepsis.
- Drugs (beta-blockers, NSAIDs, sedatives).
- Acute abdomen.
- Chest pain (trauma, rib fracture, osteoporosis).
- Post-operative sedation / retention of secretions.

32.4.4 Investigations

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

32.4.5 Severity Assessment in COPD

Make an immediate assessment of severity (see table below) 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 67 Severity Assessment in COPD

Other Categories	Mild	Moderate	Severe
	Emergency: respiratory arrest, unconscious patient, upper airway compromise		
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.

32.4.6 Management of COPD

General Principles

- Inhaled bronchodilators are effective treatments for acute exacerbations.
- Nebulized treatment is simpler to administer, but does carry a risk of spread of droplet infections such as influenza. An aerosol inhaler should be used with a spacer device in the acute setting if possible. If nebulizer therapy is used, this should be changed to inhalers at the earliest convenient time.
- 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 248).
- Exacerbations with clinical signs of infection (increased volume and change in colour of sputum and/or fever, leukocytosis) benefit from antibiotic therapy. Oral therapy is usually sufficient unless there is evidence of pneumonia.
- Controlled oxygen delivery (24-28% by Venturi mask) or 0.5 - 2 L/min by nasal prongs, aiming for sat.O_2 of 88-92% is indicated for significant 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 186).

Emergency Treatment of COPD

- **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 of COPD

- **Immediately obtain an arterial blood gas (ABG).**
- Commence controlled oxygen therapy (24-28% by Venturi mask) or 0.5 - 2 L/min by nasal prongs to maintain a PaO_2 >60 mm Hg or sat.O_2 88-92%. For more information, see *Oxygen Therapy* on page 255. Monitor for rising $PaCO_2$ (hypercapnia).
- Nebulized salbutamol 5 mg and ipratropium 0.5 mg stat and 2-6 hourly according to clinical response. Nebulize using compressed air if $PaCO_2$ elevated or there is concern the patient may be retaining CO_2.
- 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*, below.
- Consider oral antibiotics (IV if unable to take orally) if the patient has two out of three of the following: purulent sputum, increased sputum production, increasing dyspnoea.
  - Use single agent oral antibiotic e.g., beta-lactam, amoxicillin, macrolide, or doxycycline.
  - If IV therapy is necessary use single agent beta-lactam IV antibiotic - amoxicillin/clavulanate 1.2 g q8h or amoxicillin 1 g q8h.
- If consolidation on CXR, treat as *community-acquired pneumonia* (see page 259).
- 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 may be indicated, discuss with a Respiratory Physician. Guidelines for aminophylline dosage are available - search for "theophylline" on the CDHB intranet. 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 care 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 ABG 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 of COPD

- Oxygen (see *Oxygen Therapy* on page 255).
- Inhaled salbutamol or salbutamol/ipratropium, via a spacer device. 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 (IV if unable to take orally) are appropriate if there is evidence of two out of three of the following: purulent sputum, increased sputum production, increasing dyspnoea.
  - Use single agent oral antibiotic e.g., beta-lactam, amoxicillin, macrolide, or doxycycline.
  - If IV therapy is necessary use single agent beta-lactam IV antibiotic - amoxicillin/clavulanate 1.2 g q8h or amoxicillin 1 g q8h.
- If consolidation on CXR, treat as *community-acquired pneumonia* (see page 259).
- Consider chest physiotherapy.
- Anxiety reduction strategies and breathing control exercises are important, since anxiety often complicates admission with COPD.
- 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.
  - Peak Expiratory Flow Rate (PEFR).
- Adjustment of treatment: individual patient needs may change during the course of treatment including the frequency and dose of 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 may be useful to identify external factors such as health-care utilization behaviours that are amenable to change, to reduce likelihood of readmission (absence of self-management skills, involvement of Emergency Department or ambulance services without considering primary care / community options, high patient or carer anxiety).
- It is helpful to obtain spirometry and ABG 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. Rehabilitation programmes are now available around Christchurch. Details, including the referral process, can be found on HealthPathways.
- 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 186.
- 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 no longer required (unless home oxygen is indicated - see *Domiciliary Oxygen* on page 257).
  - 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., CREST (Community Rehabilitation Enablement Support Team), 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, have been developed as part of the Canterbury Initiative. These pathways can be accessed via HealthPathways.*

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. CG101 June 2010.

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

32.5 Asthma

Asthma is a clinical syndrome characterized 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. ABG in moderate or severe asthma.

At Christchurch Hospital the Asthma Admission Form should be used for ALL admissions.

32.5.1 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 68 Severity Assessment in Acute Asthma

	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 L
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, paradoxical breathing).* *See* **Life Threatening Asthma** *on page 252.*

32.5.2 Management of Asthma

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

Table 69 Immediate Management of Asthma

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.

Continued over…

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 - sodium chloride 0.9% 1 L 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. Pulse oximetry/ABG.
- Continuous ECG. Pulse, respiratory rate, BP 2 hourly. Special nurse.
  Serum potassium 12 hourly.

LIFE THREATENING Asthma: Management

Clinical

- FEV_1 or PEFR <33% predicted (or of usual best).
- Silent chest, cyanosis, or feeble respiratory effort [4].
- Bradycardia or hypotension.
- Exhaustion, confusion or coma.

Management

- High flow oxygen (40-60%).
- Salbutamol + ipratropium via oxygen driven nebulizer (initially continuous).
- Loading dose IV salbutamol 250 microgram with subsequent infusion (5 mg/5 mL salbutamol made up to 100 mL with 5% glucose , infuse at 10-30 mL/hr).
- CXR to exclude pneumothorax.
- ICU or Respiratory team review.

1. It is essential that all nebulized bronchodilators are given with oxygen 6-8 L/min.
2. Patients with life threatening asthma, or severe asthma not responding to initial treatment, and patients in whom there is any suspicion of a complication require a CXR. Complications 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 ABG measurement should be made in all patients admitted to hospital unless severity assessed as mild.
4. Patients with life threatening asthma sometimes may not appear distressed.

Note: *There is some evidence that IV magnesium may be helpful in severe asthma refractory to standard treatment. Seek Specialist advice. Nebulized magnesium is not recommended.*

Note: *A normal CO_2 in an asthma attack is a marker of severe disease.*

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.*

32.5.3 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.
  - Moderate to 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 nebulizer.
    - Consider using an intravenous bronchodilator.
  - Consider the possibility of a complication or an alternative diagnosis:
    - Pneumonia.
    - Pneumothorax.
    - Cardiac arrhythmia.
    - Left ventricular failure.
    - Laryngeal or tracheal obstruction.
    - Acute Respiratory Distress Syndrome (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:

- Hypoxaemia: 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.

32.5.4 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.

Interval Asthma Control

- 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.*

Note: *Patients with unstable features or poor compliance should be referred to a Respiratory Physician, preferably while in hospital.*

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.

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. Consider ***single inhaler therapy*** (see page 255).
  - Introduction to the Asthma Self-Management Plan and basic self-management skills.
  - An arrangement for ongoing follow-up and education as an outpatient.

Smoking Status

- Give nicotine replacement and smoking cessation advice and support if required. Current smoking is associated with reduced effectiveness of inhaled corticosteroids. See ***Nicotine Dependent Patients*** on page 186.

32.5.5 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.

32.5.6 Treatment on Discharge

- This will obviously vary from case to case but usually the patient will receive:
  - Inhaled corticosteroid - beclomethasone or budesonide 800-2000 microgram daily or fluticasone 500-1000 microgram 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**.
  - Short acting $beta_2$ agonist inhaler to use as required (NOT regularly).
  - Advice regarding common side effects of these medications:
    - Short acting $beta_2$ 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.*

32.5.7 Single Inhaler Therapy

'Single inhaler therapy' (otherwise known as single maintenance and reliever therapy or SMART™) is the use of a combination inhaled steroid / $beta_2$ agonist preparation as a preventer and a reliever. The only products able to be used in this way in New Zealand currently are those containing formoterol and budesonside (Symbicort™ and Vannair™). Seretide™ (salmeterol / fluticasone) should not be used in single inhaler therapy, because of the slower onset of action of salmeterol. For more information about the use of single inhaler therapy, see http://journal.nzma.org.nz/journal/121-1285/3346/content.pdf.

There is no evidence available to guide the decision when to restart single inhaler therapy following an exacerbation. Pragmatically it seems sensible to restart single inhaler therapy close to discharge, but to remind the patient to seek early medical advice if they are using more than 8 puffs of their combination product daily, or if symptoms are not improving.

32.5.8 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. Exhaled nitric oxide assessment can be ordered via the Respiratory Physiology Laboratory. For help interpreting the result, please contact the Acute Respiratory Physician on call.

32.6 Oxygen Therapy

Aim - to prevent 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 257).*

32.6.1 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.

32.6.2 Indications for Oxygen Therapy

- 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.

32.6.3 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.

32.6.4 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."
- 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 nebulizers. 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 longer 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.

32.6.5 Adjusting the Dose

- Does the ABG show evidence of chronic CO_2 retention, i.e., a compensated respiratory acidosis (elevated HCO_3 level), together with chronic hypoxaemia? If so, take care to avoid making CO_2 retention worse.
- Using a pulse oximeter as a monitor, adjust the flow rates:
  - For nasal cannulae in 0.5 - 1 L/min steps.
  - For variable concentration masks in percentage increments.
  - For standard masks in 2 L/min steps.
- Get the haemoglobin oxygen saturation to about 90%, wait about five minutes at each step for those with COPD.
- Once stable, if there is any risk of CO_2 retention, check the blood gases about 30 minutes later.

***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.*

32.6.6 Monitoring

- **Pulse oximetry provides an estimate of capillary haemoglobin oxygen saturation. It does not assess the adequacy of ventilation nor the gas exchange status.**
- ABG 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 ABG monitoring. If the patient is at risk, monitor ABG 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.*

32.7 Long Term Oxygen Therapy, Domiciliary Oxygen

32.7.1 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 in the stable state.

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 (fax 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.

32.7.2 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 to Respiratory Services for advice.

32.7.3 Portable Oxygen

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

32.8 Spirometry

- Spirometry testing should be considered for all patients with symptoms or signs of lung disease such as cough, dyspnoea, 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.

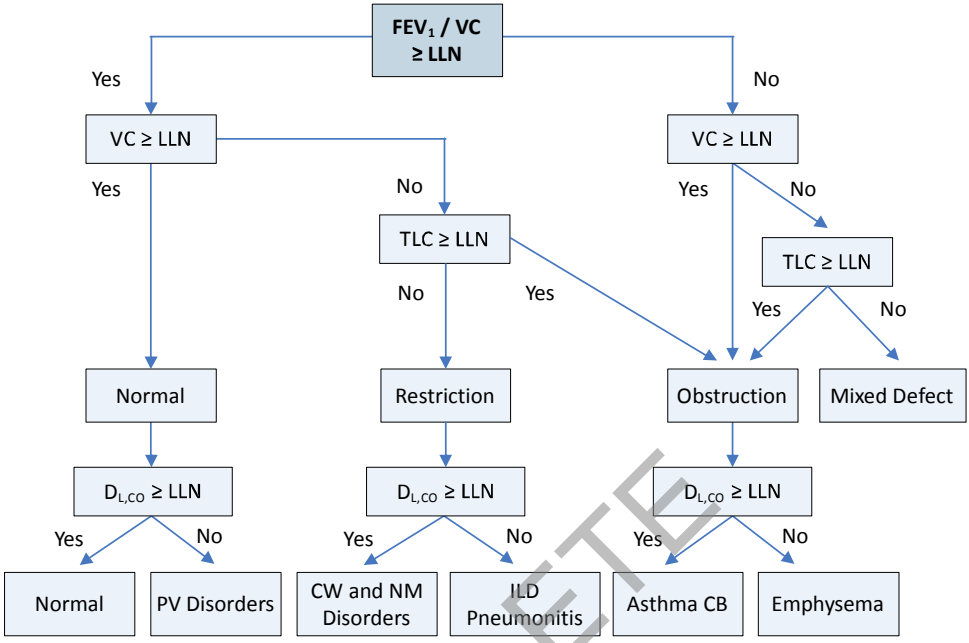
Once you diagnose obstructive lung disease on the FEV_1/FVC pattern, the FEV_1% predicted can be used to assess the severity of obstructive lung disease. The American Thoracic Society/European Respiratory Society (ATS/ERS) recommends the following severity classification in their 2005 standardization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

Table 70 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 71 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.*

32.9 Community Acquired Pneumonia (CAP)

32.9.1 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.
- *S. 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)**.

32.9.2 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 intra-abdominal pathologies such as pyelonephritis and acute cholecystitis can mimic CAP.*

32.9.3 Investigations

- Refer to the *description of the CURB-age score* (see page 261).
- 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 PCR, urinary antigen (*L. pneumophila* serogroup 1 only) and serology (acute and convalescent).
  - ZN stain and culture for TB.
  - Stains for *Pneumocystis jirovecii* (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 PCR especially if influenza is suspected.

32.9.4 Additional Investigations

- *Diagnostic pleurocentesis* (see page 55):
  - Should be performed when a significant (>1 cm on lateral decubitus CXR) parapneumonic effusion is present on CXR. Ultrasound guided aspiration is now recommended for pleurocentesis.
  - 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, preferably before pleurocentesis is performed. See *Criteria for parapneumonic effusion and empyema* (see page 265).
- Bronchoscopy. Indications include:
  - Immunosuppressed patient.
  - Life threatening pneumonia.
  - Multiple CXR changes.
  - Deterioration despite appropriate initial treatment.
  - Contact the Respiratory Physician on call.

32.9.5 Management of CAP

Resuscitate

- **A**irway
- **B**reathing
- **C**irculation

See *The ABCs* on page 63.

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 72 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):

- Mortality <2%.
- Consider outpatient management if no significant comorbidity and adequate social supports.
- Use single agent oral antibiotic e.g., beta-lactam, amoxicillin, macrolide, or doxycycline.

If **CURB-age** score 2 (Moderate CAP):

- 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 PO azithromycin).
  - Older patients (>50yrs) COPD, smokers: **IV amoxicillin/clavulanate 1.2 g q8h** (alternative IV cefuroxime).
- Consider early switch to oral antibiotic - see below.

If **CURB-age** score 3-4 (Severe CAP):

- Mortality 10-50%.
- Inpatient management.
- **Dual antibiotic therapy:**
  - **IV amoxicillin/clavulanate 1.2 g q8h + IV clarithromycin 500 mg q12h**.

If **CURB-age** score 4-5 (very severe CAP), consider ICU referral and wider spectrum antibiotic cover:

- **IV clarithromycin 500 mg q12h + IV ceftriaxone 2 g q12h + IV gentamicin 5 mg/kg (initial dose).** 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 sodium chloride 0.9% 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 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.
- Physiotherapy may be needed if sputum retention likely.

Failure to respond to initial antibiotic and supportive therapy

- Consider the presence of penicillin resistant *S. 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* (see page 186).
- 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. It is currently recommended locally that it should be undertaken** if the patient remains unwell or has risk factors for underlying malignancy (significant smoking history, asbestos exposure). These will be funded through Community Referred Radiology.
  - British Thoracic Society guidelines also include those over 50 years of age, however age alone is a controversial indication and not funded through Community Referred Radiology.

Note: *CXR may take up to 3 months to clear especially in older patients and those with COPD.*

32.9.6 Common Complications

- Parapneumonic effusion - seen in up to 40% of cases. Should always be aspirated to exclude empyema and complicated *parapneumonic effusions* (see page 265).
- Large simple parapneumonic effusions (>1/3 of hemi-thorax), all complicated parapneumonic effusions and all empyemas should be immediately referred to the Respiratory Service.

32.9.7 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.

32.10 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.

32.10.1 Investigations

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

32.10.2 Management

- Physiotherapy - especially if patient has underlying lung disease.
- Oxygen if indicated.
- Bronchodilators if history of airflow obstruction.
- Antibiotics:
  - **Mild/moderate:**
    - Amoxicillin/clavulanate 1.2 g IV q8h.
    - Add clarithromycin 500 mg q12h IV if patient immunocompromised (e.g., 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):
    - piperacillin/tazobactam 4 g/500 mg IV q8h **and**
    - gentamicin 5 mg/kg IV initial dose **and consider**
    - clarithromycin 500 mg q12h IV.

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

References:

British Thoracic Society Guidelines. Thorax 2009, Guidelines for the management of community acquired pneumonia in adults: update 2009 http://www.brit-thoracic.org.uk/Portals/0/Clinical%20Information/Pneumonia/Guidelines/CAPGuideline-full.pdf

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/

32.11 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.

32.11.1 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.

32.11.2 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: amoxicillin/clavulanate, penicillin and metronidazole or clindamycin. Attention must be directed towards underlying gastro-oesophageal reflux, and gingival disease.

32.12 Pleural Effusion

See BTS Guidelines published in Thorax 2010;65 suppl II http://www.brit-thoracic.org.uk/guidelines/pleural-disease-guidelines-2010.aspx.

32.12.1 Classification

The differentiation between **exudates and transudates** is the essential first step in the diagnostic evaluation. See *Exudate/Transudate* (see page 265).

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 pleurocentesis. If you are uncertain whether to carry out a chest aspiration, contact the Respiratory Team on call.

32.12.2 Investigations

- Diagnostic pleurocentesis. May be undertaken by medical staff with appropriate experience. Ultrasound guided aspiration should be performed, 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 55). Ultrasound should be performed at the bedside, rather than as an "x marks the spot" in the Radiology Department.
- Measure plasma total protein, glucose and LDH levels for comparison with pleural fluid.

32.12.3 Contraindications for pleural aspiration

- Unwilling or uncooperative patient.
- Abnormal bleeding tendency. Check history, medications, e.g., anticoagulants, examination, INR, APTT, fibrinogen, and platelets. If in doubt, discuss with Consultant before proceeding.
- Insufficient pleural fluid.
- Chest pyoderma or herpes zoster.

32.12.4 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).
- Consider additional tests as below depending on clinical context.

32.12.5 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, seek cause, e.g., heart failure, cirrhosis, nephrotic syndrome, acute glomerulonephritis, peritoneal dialysis, myxoedema

  Note: *5% of malignant effusions are transudates.*
- 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). See *Tuberculosis* on page 154.
- 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 268).
  - 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**.

32.12.6 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.

32.13 Spontaneous Pneumothorax

See BTS Guidelines published in Thorax 2010;65 suppl II http://www.brit-thoracic.org.uk/guidelines/pleural-disease-guidelines-2010.aspx.

32.13.1 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.

32.13.2 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.

32.13.3 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.

32.13.4 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.
- 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 a total of >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.

3 way taps must never be used in drains inserted for the management of pneumothorax.

- Inpatient cases with pneumothorax should be managed by either the Respiratory Service on the Respiratory Ward or by the Cardiothoracic Surgical team if traumatic.

32.13.5 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 2-3 weeks of resolution (Aerospace Medical Association, Air Transport Medicine Committee. Medical guidelines for air travel. Aviat Space Environ Med (2nd edition 2003)) and scuba diving (contraindicated).

32.14 Intercostal Tubes

The insertion and management of intercostal tubes is a complex and specialized area which should only be undertaken by trained staff. 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.

32.14.1 Indications

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

32.14.2 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 is 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).

32.14.3 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 – see above. If present, this should be removed and the chest tube connected directly to the underwater seal. 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 sodium chloride 0.9% 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.

32.14.4 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.

32.15 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 haematocrit, haemothorax is diagnosed.
- Check coagulation profile.
- 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).

33. Rheumatology, Immunology and Allergy

33.1 Rheumatology and Immunology Department Information

Main Office

3nd Floor, Riverside, ☎ 80953, fax 80201

Inpatient Services Ward 23

- Dr Peter Chapman, Dr John O'Donnell, Professor 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.

33.2 Immunology and Allergy: recommended referrals

- Anaphylaxis:
  - See *Anaphylaxis* (see page 72).
- 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.

33.3 Acute Swelling of a Single Joint - Septic Arthritis, Gout etc.

The cause of the acute swelling must be established before any rational form of treatment can be given.

33.3.1 Possible Causes

- Trauma ± haemorrhage.
- Infection (septic arthritis signs may be modified if on steroids or in the presence of chronic arthritis e.g., rheumatoid arthritis).
- 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.

33.3.2 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 polarized 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. gonorrhoeae*). 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.

33.3.3 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. Refer to ***Penicillin Allergy*** (see page 148).
- 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 a sign of toxicity not a side effect.

  ***Note**: A lower dose regimen has also been shown to be effective (colchicine 1 mg stat, then 0.5 mg 1 hour later and no further doses for 3 days).*

  ***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 2-4 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 73 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:

- Immobilize 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.

33.4 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 emphasized 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.

33.4.1 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).

33.4.2 Clinical Assessment

Clinical assessment with frequent reviews should be the main guide to investigation.

33.4.3 Differential Diagnosis

- Infections (e.g., bronchiectasis, bacterial endocarditis, abdominal abscess).
- Malignancy (especially renal cell carcinoma and lymphoma).

- Connective tissue disease/primary necrotizing vasculitis (consider systemic onset rheumatoid arthritis, polymyalgia rheumatica, giant cell arteritis, granulomatosis with polyangiitis (GPA, formerly called 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 minimize their use as far as possible).

33.4.4 Investigations

- CBC + diff.
- CRP or ESR.
- Na, K, creatinine, urate, Ca, PO_4, alb, bili, ALP, AST, ALT.
- CK, thyroid function tests.
- Urinalysis including microscopy.
- Blood cultures (x3 sets with 10 mL of blood per bottle).
- Urine culture, including TB if unexplained pyuria.
- 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.
- *Tests for TB* (see page 156).
- 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.*

33.5 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.

33.5.1 Investigations

- CRP or ESR.
- CBC + diff.
- Urinalysis, microscopy, protein, culture.
- Na, K, creatinine, urate, Ca, PO_4, alb, bili, ALP, AST, ALT.
- CK, thyroid function tests.
- Vitamin D.
- Other investigations as determined by clinical assessment.

33.5.2 Treatment

- Generally PMR responds to low dose prednisone 15-20 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 may be considered 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 211).

33.6 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 211).

33.7 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), B lymphocyte depleting agents (rituximab), or anti-IL-6R monoclonal antibodies (tocilizumab) 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, ALP, AST, ALT, blood cultures, urinalysis, sputum culture, CXR.
  - ***Consider culture/stains for AFB*** (see page 156).
  - Other diagnostic imaging may be required to determine the source of infection.
- Empiric treatment:
  - Immunosuppressive therapy should be minimized 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.

33.8 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 74 Acute Pulmonary - Renal Syndrome

Suspected diagnosis	Screening tests
A: Idiopathic Vasculitis	
Granulomatosis with polyangiitis (Wegener's)	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	CBC and blood film
C: Infective (a rare cause)	
Mycoplasma	Nasopharyngeal swab for PCR, IgM antibody
Legionella	sputum for PCR, urinary antigen (*L. pneumophila* serogroup 1 only) and serology (acute and convalescent)
Mycobacterium	Sputum ZN stain
S. pneumoniae	Sputum Gram stain

1. Request "urgent vasculitis screen".

34. Spinal Injuries

Reception ☎ 99850 or 03 383 6850, fax 03 383 6851.

Burwood Spinal Unit Consultants

- Mr Balraj (Raj) Singhal (Clinical Director)
- Dr Rick Acland, Dr Angelo Anthony, Dr Xianghu Xiong
- Spinal surgeons: Mr Kris Dalzell, Mr Jeremy Evison, Mr Grahame Inglis, Mr Rowan Schouten

34.1 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 investigation (refer to *Spinal Cord Compression* on page 180). 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 mobilize 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**.

Low velocity injuries with cervical spine dislocations with neurologic symptoms and signs (rugby, trampoline, playfighting, gymnastics) should be reduced within the first 4 hours as neurological outcomes are much better if done so. Please refer them straight to the Spinal Consultant on call.

34.2 Traumatic Spinal Cord Injury

34.2.1 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).

34.2.2 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). Turns every 2-3 hours.
2) Ensure airway, breathing and circulation - treat neurogenic shock (with features of low blood pressure but bradycardia) with goal of maintaining systolic pressure >110 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 subcut once daily. For spinal injuries without cord injury, give enoxaparin 40 mg subcut 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. Check regularly for occiput, sacrum, and heel pressure sores.

34.3 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.
- 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 beats and atrioventricular conduction abnormalities.

Note: *If AD is not recognized and treated* ***promptly*** *the hypertension may escalate to dangerous levels resulting in intracranial haemorrhage, seizures, cardiac arrhythmia or death.* ***This is a medical emergency.***

34.3.1 Management

- Recognize the signs and symptoms of autonomic dysreflexia (AD).
- Check the patient's blood pressure (BP).

  Note: *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 BP not raised and the cause has not been identified, seek Consultant advice.
- 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, catheterize 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.
  - 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 can cause hypotension if the patient has been given drugs to lower BP.*
- If acute symptoms of AD persist, including elevated BP, suspect faecal impaction.
- If BP is ≥150 mm Hg systolic, consider administering glyceryl trinitrate (GTN) spray x 2 sublingually.

  Note: *Monitor patient for signs of hypotension.*

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.*

34.4 Common Presentations of Non-Acute/Long-term Spinal Cord Injury Patients

34.4.1 Respiratory Infections

Assessment

- Arterial blood gas essential.
- Sat.O_2 alone is inadequate.
- Chest X-ray.

Treatment

- Assistance with coughing in order to clear secretions and obtain sputum. Get Physiotherapist involvement as patient may not be able to cough.
- Antibiotics and oxygen if indicated.
- Care of skin is critical. Ensure patient is turned regularly.

34.4.2 Urinary Tract Infections

Assessment

- Urine for culture: bag urine not recommended, so bladder puncture for microscopy and culture will be needed.
- May need bladder catheterization and anticholinergic drugs.
- If urinary tract stones are likely, consider plain X-ray (KUB) for kidney, ureter and bladder. This is more likely if the patient has autonomic dysreflexia. Discuss with the Consultant if you think ultrasound is indicated.

Treatment

- Prescribe antibiotics.
- If tetraplegic, exclude an upper respiratory tract infection.

34.4.3 Lost Suprapubic Catheter Tract

If a spinal cord injury patient, who has had a suprapubic catheter placed for bladder management, has sluggish urinary drainage, or the suprapubic catheter comes out accidentally:

- Check for a urinary infection.
- Give antibiotics according to any recent cultures/sensitivities. If no cultures available on admission, give amoxicillin 1 g IV, and gentamicin 160 mg IV, both stat, before the insertion of a urethral catheter.
- Insert urethral catheter to allow bladder drainage.
- If not already taking an anticholinergic drug, give oxybutynin 5 mg BO PO.
- Call the Urology Registrar urgently (they may be able to reinsert the suprapubic catheter).

34.4.4 Constipation - with or without Abdominal Distension

Assessment

- Ask patient or caregiver to describe the bowel pattern for the last few days.
- Plain abdominal X-ray to exclude obstruction volvulus. May require CT abdomen.
- May be secondary to medications. Exclude a urinary tract infection.

Treatment

- Consider a gentle enema. Review drug treatment.

Contact the on-call Spinal Consultant via the hospital operator if there any questions on management.

35. Urology

35.1 Urology Department Information

Main Office

- 3rd Floor, Parkside West, Level 3, ☎ 81009, fax 80936

Inpatient Care

- Urology unit, enter from Parkside Level 2
- Ward 21 and 22 (Paediatrics)

Spinal Injury Care

- Burwood Hospital

Consultant Urologists and On-call Registrar

- Consultants: Kevin Bax (kevin@urology.co.nz), Nicholas Buchan (nick@urology.co.nz), 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.
- To contact the Urological Service on call, contact the Consultant (0800 - 1700) or Registrar (after-hours) via the operator. Contact the Consultant Urologist via cell phone through the Christchurch Hospital operator. Consultants do not use CDHB email. The Consultants use their addresses at Urology Associates as listed above.
- Secretaries: Rose Webber (rosew@cdhb.health.nz, ☎ 81009), Sue Lockie (☎ 81009).

35.2 Renal Colic

35.2.1 History

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

35.2.2 Examination

- If there is a high fever >38°C + significant renal tenderness, infection may be present.

35.2.3 Differential Diagnosis

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

- Aortic and iliac aneurysms.
- Testicular torsion.
- Pyelonephritis.
- Peritonitis, including appendicitis and diverticulitis.
- Biliary colic.
- Clot colic usually secondary to malignancy in kidney.

35.2.4 Investigations

- MSU: haematuria is present in only 85% of patients with renal colic.
- CBC + diff (the white cell count is often raised even when there is no infection), creatinine, electrolytes, calcium, phosphate, uric acid.
- Blood cultures if patient is febrile.
- *CT urogram and plain X-ray (KUB) for kidney, ureter and bladder* (see page 279).

35.2.5 Management

- IV access for analgesia + fluids.
- Adequate analgesia, opiates usually required.

Note: *Patients with infection and obstructed kidneys may develop urosepsis.* ***Use gentamicin, initial dose 3-5 mg/kg IV****. If further doses are required, see the gentamicin/tobramycin dosing guidelines in the Pink Book.*

35.2.6 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 as an outpatient. 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.
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) **Ultrasound**:
   - When CT is contraindicated (e.g., pregnancy).
   - Will detect larger (>5mm) stones, particularly in the proximal and distal ureter but only poorly visualizes 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).

35.2.7 Subsequent Management

- The majority of patients do not need admission and will pass a stone.
- The decision to admit the patient must be taken by the Urology on-call team 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 opioids and NSAIDs.
- Recurrent attacks of colic with repeated visits to the Emergency Department.
- Any ureteric stone in a solitary kidney.
- Creatinine >200 micromol/L.
- Admission may be required in other circumstances. Discuss with Urology on-call team.

When discharged:

- Send a referral to the Urology Outpatient Clinic.
- If the stone is 5 mm or less at the vesico-ureteric junction and becomes asymptomatic then no follow up is required.
- If the stone is >7 mm, the patient will be booked directly for semi-urgent surgery.
- Other stones will be followed in outpatients in 4 weeks.
- In all cases send the referral to Urology and a letter will be sent to GP and patient with advice and instructions.
- Advise patient to return if they develop a fever or become unwell.
- 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.

35.3 Macroscopic Haematuria

35.3.1 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?

35.3.2 Examination

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

35.3.3 Investigations

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

35.3.4 Management

- Discuss need for hospital admission with the Urology on-call team.
- If there is macroscopic haematuria with clot retention:
  - Catheterize 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.
  - If it does not resolve, discuss admission with the Urology on-call team.
  - If it does resolve, remove catheter and refer for outpatient assessment.

35.4 Microscopic Haematuria

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

- CBC + diff, creatinine, Na, K.
- MSU.
- Renal ultrasound.
- Flexible cystoscopy.
- Urinary cytology.

35.5 Testicular Torsion

35.5.1 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.

35.5.2 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.

35.5.3 Management

- Make an urgent referral for acute testicular exploration, detorsion and bilateral testicular fixation.

35.6 Acute Urinary Retention

Once you have determined the patient is in retention, catheterize without delay for pain relief.

35.6.1 History

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

35.6.2 Examination

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

35.6.3 Investigations

- CBC + diff, Na, K, creatinine, PSA.

35.6.4 Management

- *Catheterize patient* (see below) as soon as possible: urine specimen to laboratory.
- Record volume drained.
- Do not have more than 2 attempts to pass a urethral catheter.
- If unsuccessful a supra-pubic catheter may need to be inserted.
- If the catheter drains more than 1.5 L, monitor fluid and electrolyte balance.
- Send referral to Urology Department for consideration of TURP.

35.7 Urethral Catheterization

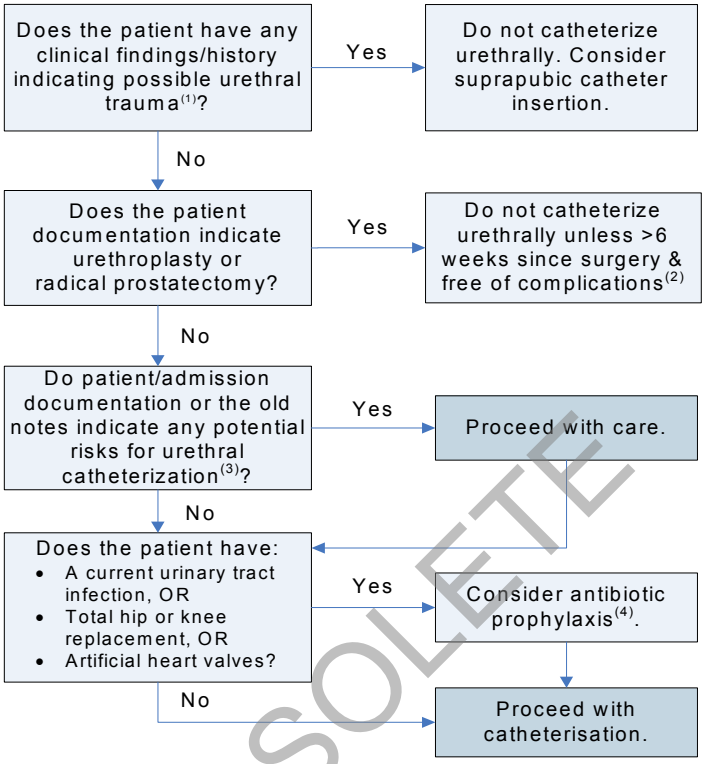
35.7.1 Indications for Urethral Catheterization

- Acute retention.
- Unconscious or sedated patients unable to void.
- In the operative and perioperative setting.
- Patients with prolonged epidural anaesthesia e.g., in labour.
- Chronic retention if associated with impaired renal function or infection.
- Incontinence.

Note: *Urethral catheterization for incontinence needs to be carefully assessed in light of social situation.*

35.7.2 Male Urethral Catheterization

Table 75 Algorithm for Male Urethral Catheterization



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. Catheterization 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 catheterization with care using 14 Fr catheter. If unsuccessful, insert a suprapubic catheter.
3. Potential risks for urethral catheterizations:
   - 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 catheterization previously
4. Bacterial endocarditis prophylaxis: for full details, refer to *Infective Endocarditis Prophylaxis* (see page 46). For other situations, seek Consultant advice.

35.7.3 Insertion of Urinary Catheter

- **Explain the procedure to the patient and gain informed consent.**
- **Select appropriate catheter (bigger is usually better, e.g., 18-20 Fr).**
- **Set up equipment following recommended best practice.**
- **Infection prevention:** Catheter associated infections are the most common hospital-acquired infection. Risks can be reduced by adhering to standard precautions, including the 5 moments of hand hygiene and using an Aseptic Non-Touch Technique (ANTT).
- **Choose catheter size and type according to reason for catheterization**. Choose the catheter that will suit the purpose of the catheterization:
  - **Use catheter size 16 - 18**:
    - 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.** Seek advice.
    - **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 circumcized**, return the foreskin to its natural position after catheterization.
- Clearly document in clinical notes:
  - Date/consent given.
  - Reason for catheterization.
  - Catheter type, length and size.
  - Amount of water in balloon.
  - Any problems with procedure.
  - Description of urinary drainage on insertion.
  - If specimen sent for further investigations.
  - Review date/catheter change or removal.
- **Indications for Suprapubic Catheterization:**
  - Failed urethral catheterization
  - Long term management of patients with neuropathic bladders

Note: 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.

35.7.4 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.

36. Notifiable Diseases (New Zealand)

36.1 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 a 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

Locate and print the notification form - ask the ward clerk or search for "notifiable diseases" on the CDHB intranet. Complete the form and fax it 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 that should be taken by staff dealing with the patient.

36.2 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 who require follow-up.

36.3 Diseases Notifiable in New Zealand to the Medical Officer of Health

Table 76 Diseases[1] Notifiable in New Zealand to the Medical Officer of Health

- Acquired Immunodeficiency Syndrome (AIDS)
- Acute gastroenteritis[2]
- Anthrax
- Arboviral diseases
- Brucellosis
- Campylobacteriosis
- Cholera
- Creutzfeldt-Jakob disease and other spongiform encephalopathies
- *Cronobacter* species
- Cryptosporidiosis
- Cysticercosis
- Decompression sickness
- Diphtheria
- Giardiasis
- *Haemophilus influenzae* B
- Hepatitis A
- Hepatitis B
- Hepatitis C
- Hepatitis (viral) - not otherwise specified
- Hydatid disease
- Influenza - Highly Pathogenic Avian Influenza (HPAI)
- Influenza - Non-seasonal influenza (capable of being transmitted between human beings)
- Lead absorption equal to or in excess of 10 microgram/dL (0.48 micromol/L)[3]
- Legionellosis
- Leprosy
- Leptospirosis
- Listeriosis
- Malaria
- Measles
- Meningoencephalitis - primary amoebic
- Mumps
- *Neisseria meningitidis* invasive disease
- Pertussis
- Plague
- Pneumococcal invasive disease
- Poisoning arising from chemical contamination of the environment
- Poliomyelitis
- Q fever
- Rabies and other lyssaviruses
- Rheumatic fever
- Rickettsial diseases
- Rubella
- Salmonellosis
- Severe Acute Respiratory Syndrome (SARS)
- Shigellosis
- Taeniasis
- Tetanus
- Trichinellosis
- Tuberculosis (all forms)
- Typhoid and paratyphoid fever
- Verotoxin-producing or Shiga toxin-producing *E. coli*
- Viral haemorrhagic fevers
- Yellow fever
- Yersiniosis

1. 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.
2. 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*, *Vibrio parahaemolyticus* or *Clostridium perfringens*.
3. Blood lead levels to be reported to the Medical Officer of Health (≥10 microgram/dL or 0.48 micromol/L) are for environmental exposure. Where occupational exposure is suspected, please notify OSH through the Notifiable Occupational Disease System (NODS).

37. 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/Rehabilitation 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.health.nz.

38. 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 cover and first page.

The Blue Book is 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/Resident Doctors Support 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 smartphone and tablet devices.

Updates and Amendments

The Blue Book is updated and reprinted in November every second year.

There will be no amendments to the printed version during the next 2 years.

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Canterbury
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Te Poari Hauora ō Waitaha

Management Guidelines for

Common Medical Conditions

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