HLABISA
HOSPITAL
HANDBOOK

PROTOCOLS

The Rural Health Initiative is a project of the SA Academy of Family Practice / Primary Care, designed to support rural practitioners in the delivery of appropriate healthcare to their communities.

LIVERPOOL SCHOOL OF TROPICAL MEDICINE

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SOUTH AFRICAN SUGAR ASSOCIATION

Africa Centre for Population Studies & Reproductive Health
HLABISA HOSPITAL HANDBOOK

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Dedicated to the memory of
Alma Buthelezi
and
Spence Alexander
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Disclaimer

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When in doubt, seek the assistance of a more senior colleague, or, when further information is required concerning drug indications or dosages, consult the SAMF, the current pharmaceutical package insert, or the relevant pharmaceutical company.

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HEALTH FACILITIES IN THE HLABISA DISTRICT
INTRODUCTION

Hlabisa is one of the larger health wards in northern Zululand with an estimated population of more than 200,000. It lies between latitudes 28° and 29° south, and is traversed from north to south by the National Road N2. The boundaries enclose four small towns, the informal settlements of Dukuwululwe and a large rural hinterland rising to 300-m above sea-level in the northwest. The Hluhluwe/Umfolozi Game Reserve, the oldest and third largest in South Africa, cuts the district in two. The major industries in the area are sugar cane, pineapple cultivation and forestry. The district is also the home of the widely exported Hlabisa basket. However, most workers are local/distant migrants and 50% of the population are unemployed. The average wage is estimated at R500 per month but some care-cutters receive as little as R25.00 per day.

The district endures many of the typical health problems of a developing country - epidemics of cholera, bacillary dysentery, malaria, tuberculosis and the most devastating, HIV/AIDS, have all occurred in recent years. The perinatal mortality rate is estimated at 31/1000 and the infant mortality rate at 16-1000 (facility-based estimates). 15% of children in the community are stunted and 7% are under-weight-for-age. Immunisation coverage up to 6 months is -70%. Demand for healthcare in the district often threatens to overwhelm the available resources.

The first clinic at Hlabisa was established by the Lutheran Mission Society in 1932. The hospital itself came into being in 1948, with the arrival of the first resident medical officer, Dr Erling Hestenes. Over the next 20 years he developed the clinic - more or less single-handedly - into a 300 bedded hospital, capable of meeting the needs of the local population.

The hospital was taken over by the KwaZulu government in 1978 and was run by staff from the Lutheran Mission Society and army doctors on alternative military service. By the 1990s, doctors at the hospital were caring for 420 inpatients and supporting thirteen community clinics. After the elections of 1994, local services, previously provided by the Natal Provincial Administration, were incorporated into the new province of KwaZulu-Natal.

The hospital has been staffed since 1992 by at least six Medical Officers, mainly expatriates, but more recently South African doctors on Community Service. They see 30,000 patients each year in OPD, about 10,000 of whom are admitted, 3,000 deliveries per year take place in the labour ward and approximately ten procedures are performed each day in the operating theatre. Almost 200 patients are enrolled in the community TB programme every month. Primary Health Care trained nurses run the community clinics, and are supported by fortnightly/monthly visits from the Medical Officers.

Antenatal, under 5s and TB services are free, though other patients are charged R7 for an OPD consultation. However, the hospital and its clinics are often the last port of call for many patients - traditional healers (izinyanga and izisangoma) are invariably the first to be consulted. There are also 13 GPs practising in the district, who are widely respected and charge –R60 for a first consultation. Some patients with medical insurance have access to sophisticated facilities and specialist consultation in Richards Bay/ Empangeni.

For most healthcare workers and their patients in the district, however, access to immediate specialist consultation is often impossible, and the range of clinical problems to which they are expected to respond is formidable. Few appropriate clinical resources exist to support them. Many rural hospitals have compiled books of protocols/standing orders designed to guide their staff, but these have rarely been subjected to wider review. The National and Provincial Departments of Health have also produced many helpful guidelines, but these lack comprehensiveness and sometimes need to be adapted to local conditions.

This project began in 1991 when doctors at Hlabisa Hospital attempted to establish their own protocols to improve the care of their patients. These protocols were updated in 1994 and have been an essential source for all new staff since. This new edition aims to build on the experience accumulated in the previous versions, by updating the management protocols, and supplementing them with a rationale, and with references to the best of local literature. We hope that this approach will empower rural doctors to make appropriate decisions in a critical way, in partnership with their referral centres, and so allow the continual evolution of the protocols, guided as far as is possible by the evidence available.

Gerry Davies - on behalf of the staff of Hlabisa Hospital - past, present and future

Further reading
1) Cooper 1 ’999 Why doctors choose to work in rural hospitals’ S.A.M J 89:736
2) Pelot, SJ ’889 The procedural skills of rural doctors” S.A.M J 86:759
ANAESTHESIA
The vast majority of our cases are emergencies and will usually be done the same or the next day. No elective cases will be done routinely on Fridays. Remember that the Monday list may be packed with cases from the previous weekend. In order to ensure the smooth running of lists and to minimise cancellations, wards must book cases prior to 07h30 on the day of surgery - booking slips arriving late may be cancelled for that day at the discretion of the surgeon (please make these decisions in the morning for the comfort of such patients). Priority in the morning session should be given to paediatric, O.P.D. and previously cancelled cases. Cases arising in O.P.D. should be discussed with the surgeon on duty in theatre that day.

It is the responsibility of the doctor who books the patient for theatre, to ensure that the patient is fit for anaesthesia, consent has been given (age of consent is 18 years; however, if patient has ID book and is in paid employment, age of consent is 16) and, if necessary, prescribed appropriate pre-medication. All but the most minor cases should arrive in theatre with a patient i.v. line. Blood for electrolytes and trauma cases should be ordered according to the patient's haemodynamic state. We do not routinely X-Match for Caesarean section unless the patient has a history of anaemia and/or looks anaemic clinically and check Hb < 8. Non-urgent surgery should not be contemplated in poorly controlled diabetics or patients with a diastolic B.P. of 110 mm Hg or greater. We still recommend a N.P.D. time of 6 hours prior to surgery wherever possible but this may not be necessary in healthy patients who have taken only fluids (2 hours safe).

All obstetric patients should be given Cimeti dine 800 mg stat p.o. + Sodium Citrate 0.314 ml 30 minutes prior to surgery to minimise the risk of aspiration. Should general anaesthesia be required. Patients for spinal anaesthesia should be pre-loaded with 1 litre of Normal Saline immediately pre-operatively.

Children should routinely be given Trimeprazine 2-3 mg/Kg 2 hours pre-op and Atropine 0.02 mg/Kg i.v. at immediately prior to induction - it can be mixed with Ketamine in the same syringe. However, do not give Atropine in the summer months if the temperature is > 30 °C as it may abolish sweating, and result in hyperpyrexia.

Adults for ketamine anaesthesia should be given Diazepam 0.2 mg/Kg p.o. 1 hour pre-theatre or alternatively the same dose immediately prior to induction i.v. (it cannot be mixed with ketamine or pethidine in the same syringe). This helps to prevent emergence delirium.

Further Reading
1) Carson et al. 1986 'Severity of anaemia and operative mortality and morbidity' Lancet i 727
2) Johnston JR 1982 'A haemorrhagic of coniinium as the sole oral analgesic in obstetric anaesthesia' Anaesthesia 37:33
3) Phillips et al. 1983 'Pre-operative drinking does not affect gastric contents' J. A. Anesth. 16:2

Both our theatres are equipped with Boyle's type anaesthetic machines that use piped oxygen as the carrier gas for the two other inhaled agents - Nitrous oxide from a separate pipeline and Halothane from a vaporiser. Each is fitted with a Humphrey A.O.E. breathing system and a ventilator. It is essential that you understand something about the equipment and how to check it in order to operate it safely.

THE MACHINE
Gas from the pipeline supply at high pressure enters the machine through pressure reducing valves. Back-up cylinders of Oxygen and Nitrous Oxide are attached at this point for use in case the bulk supply fails. The gases pass through a flowmeter that controls the flow rate and composition delivered to the patient. This mixture then flows downstream through the vaporiser before entering the breathing system. The vaporiser is flow-compensated and calibrated for Halothane only. A "flush" valve located by the machine fresh gas flow outlet delivers fresh oxygen at 35 l/min directly from upstream bypassing the flowmeter and vaporiser.
THE BREATHING SYSTEM

The Humphrey A.D.E. is a combined Mapleson A and D/E breathing system which can safely be used for both spontaneous and controlled ventilation in both adults and children. The configuration is selected by a single lever - A in the UP position and D/E in the DOWN position. The Heidebrink or "split" valve located on the top of the system must be fully open (unscrewed) in the A configuration, in order to allow the patient to exhale during spontaneous ventilation. A little positive expiratory pressure (i.e. valve partially screwed down) should be applied when hand ventilating an apnoeic patient.

A CONFIGURATION

The A.D.E. is a "low-flow" system - re-breathing is usually prevented and normocapnia maintained with a fresh gas flow (F.G.F.) from the machine of 50 ml/kg/min for spontaneous ventilation and 70 ml/kg/min for controlled ventilation. In practice, this means a minimum F.G.F. for an adult of 3-4 l/min in A configuration and 6 l/min in D configuration.

THE VENTILATOR

For general anaesthesia with controlled ventilation we use an Ohmeda 7000 series. This is driven by the oxygen supply and is designed to deliver a pre-set tidal volume to the patient. It is therefore, a "volume cycled" ventilator such that, when the required volume of gas has been pumped through to the patient, the inspiratory phase will be switched on and the expiratory phase will start. This also means that the airway pressure reflects the impedance of the patient's lungs and the breathing system.

The respiratory rate should be set on the dial at 12/min for an adult and slightly higher for a child. The required tidal volume is 10 ml/kg - multiply this by the R.R. to obtain the minute volume (8-10 l/min for most adults) and set this on the minute volume dial. Set the I:E ratio at 1:2 and confirm that the "sigh" switch is on - this delivers 150% of the set tidal volume every 64 breaths to minimise atelectasis.

SAFETY CHECKS

These should be performed at least daily for a routine list and before any major operation.

✓ Check the gas supply
1) Check the pressure gauges for the built supply outside the theatre - these should read ~ 400 kPa for gases and ~ 70 kPa for vacuum.
2) Disconnect the O₂ (white) and N₂O (blue) pipelines from the PMGV outlet on the ceiling behind the machine. Turn on the flowmeter - no flow should occur and the O₂ alarm will sound.
3) Turn ON the O₂ cylinder - there should be no leakage of gas at the back of the machine. The O₂ bobbin only should rise and the O₂ cylinder gauge should read full.
4) Turn ON the N₂O cylinder - there should be no leakage of gas at the back of the machine. The N₂O bobbin should rise and the N₂O cylinder gauge should read full.
5) Turn OFF the O₂ cylinder - BOTH bobbins should fall. The O₂ failure alarm should again sound.
6) Reconnect the N₂O pipe to the PMGV outlet - BOTH bobbins should rise, the O₂ failure alarm should be turned off.
7) Turn OFF the N₂O cylinder - the N₂O bobbin only should fall.
8) Reconnect the N₂O pipe to the PMGV outlet - the N₂O bobbin should rise again.

✓ Check for leaks
- Occlude the fresh gas flow outlet with your finger. The machine should leak only from the pressure-relieving valve at the back, with a characteristic noise.
- Close the spill valve fully in A configuration and occlude the Y-connector of the breathing system with your finger. The reservoir bag should expand steadily, the manometer rise and there should be no leaks from any part.

✓ Check the ventilator
- Switch the breathing system to D configuration. Fill the bellows with the O₂ flush and occlude the Y-connector - if the bellows falls > 100 ml/min a leak should be suspected.
- Check that the controller is plugged in at the back of the machine. Turn the ventilator on.
- Press “lamp test” to confirm all the alarms are functioning (NB the power failure alarm is audible only).
- Further test the high and low airway pressure alarms as follows:
  - Fill reservoir bag onto Y-connector (“Dummy Lung”)
  - Fill bellows
  - Allow ventilator to cycle a few times
  - Remove dummy lung
  - “Low airway pressure” alarm should sound on 2nd cycle
  - Now set tidal volume to 11
  - Put finger tightly over the end
  - The “set volume not delivered” alarm will sound and the bellows will re-expand before reaching the 11 mark.

✓ Check that the vapouriser is full

✓ Check the suction equipment

✓ Check the intubation equipment
- There should always be 2 laryngoscopes, a range of E.T. tubes and Guedel airways, introducer, tube changer and Ferguson’s gag available.

✓ Check that a self-inflating ambu-bag and free-standing oxygen cylinder are in theatre (should everything go wrong).

Further reading:
1) Humphry D 1996 'Towards simpler, safer and more efficient anaesthetic breathing systems' in M. Melos (ed.), Excerpta Medica. Recent Advances in Anaesthesia, Critical and Intensive Care, Hong Kong, 93-107
2) Omnide 7000 Manual (Omnide)

**Airway Management**

Control of the airway is the FIRST PRIORITY in anaesthesia - a patient will die more quickly from respiratory obstruction than from anything else. Thus it is vital to recognise when you are dealing with a patient with a potentially unsafe airway, and to be clear about the management of airway problems.

**RECOGNISING THE PROBLEM AIRWAY**

Many starved patients breathing spontaneously under general anaesthesia do not need intubation. Ketamine in particular, by virtue of its dissociative effects, preserves airway reflexes better than any other intravenous agent. Simple airway manoeuvres (chin lift, jaw thrust or insertion of the correct size Guedel airway) are usually all that is required in such situations. However, intubation will normally be necessary in the following cases (either to secure the upper airway or protect the lower from aspiration of gastric contents):
- Emergency abdominal surgery with a full stomach (<4 hrs post-fluids, <6 hrs post-solids)
- Obstetric general anaesthesia (> 18/40 and early post-partum period)
- Obese patients (especially those with symptomatic reflux)
- Muscle relaxation / controlled ventilation required
- Difficult mask airway (esp. edentulous, mandibular problems)
- Position other than supine / lithotomy
- Procedure longer than one hour (really for convenience of the anaesthetist).

Some intubations may be "difficult". However, several
useful predictors may help to identify such patients in advance (and enable you to ensure that help is at hand / refer the patient), such as:
  - Short neck/receding chin (<6 cm between the thyroid notch and lower border of mandible) alerts you to the possibility of an "anterior larynx" that may require a straight blade and cricoid pressure to intubate successfully
  - Immobile mandible (patient should be able to open mouth 3 fingers' breadth and protrude lower teeth beyond the upper)
  - Overbite (upper teeth get in the way)
  - Limited neck flexion / extension
  - Mallampati IIIVs (if you can only visualise the base of the uvula or just the hard palate when the patient opens the mouth wide and protrudes the tongue, intubation may well be a problem)
  - Deviated / immobile trachea.

RAPID SEQUENCE INDUCTION

Most of the intubations we do are for emergency patients at risk of aspiration as part of a Rapid Sequence or "crash" induction - this is the standard method of securing an unsafe airway for G.A. The important points are as follows:
  - Pass an n.g. tube prior to the operation and aspirate it / instil antacids before you begin.
  - Always have the following immediately at hand before you start: adequate suction, TWO working laryngoscopes, introducer, bougie and a tube of the correct size that you have checked and cut to length to minimise the risk of bronchial intubation - it is also a good idea to have another tube 0.5 cm smaller as a back-up.
  - (The correct diameter tube is, for a child, age/4 + 4.5 cm (this approximates to the size of the little finger), for an adult female 7.0-7.5 cm, and for an adult male 8.0-8.5 cm. The correct depth for the tube is age / 2 + 12 cm up to a maximum of 23 cm. Laryngeal oedema can be a problem in eclamptic patients and a smaller tube may be needed)
  - Always pre-oxygenate the patient with 100% Oxygen for 3-5 minutes. This creates a column of 100% O2 in the patient's airway and lungs, giving you almost two full minutes to intubate once the mask is removed, although less in obstetric patients owing to changes in lung volumes.
  - As you give the i.v. induction agent, get an assistant to apply cricoid pressure - it must not be removed until the trachea is intubated or the patient is again in a state to maintain the airway him / herself. The only exceptions are if the patient actually vomits (in which case maintaining cricoid pressure may lead to oesophageal rupture), and if you cannot pass the tube through the cords (when releasing cricoid pressure slightly may help).
  - Intubate, intubate the cuff, hand ventilate and listen in both axillae. The most reliable proof that the tube is in the trachea is that you saw it go in there! Other signs (mist in the tube, breath sounds etc.) can be unreliable and desaturation may be delayed for as long as 10 minutes. If you are genuinely unsure, try withdrawing air from the tube with an oesophageal detector device or make a second look with the laryngoscope.

FAILED INTUBATION DRILL

Do not persist for longer than two minutes if you can't intubate. Repeated attempts and prolonged hypoxia will kill the patient. Therefore recognise early that this intubation may be beyond your capabilities.

Call for help !

Maintain cricoid pressure

Lay the patient head down on the left side

Suck out any secretions / vomitus

Maintain a mask airway and hand ventilate with 100% Oxygen (ventilate relatively slowly and rapidly to avoid forcing the oesophagus open with consequent risk of aspiration).

If the airway is easy to maintain with a mask and more experienced help is available, induce anaesthesia with Fentanyl and attempt intubation - with a second dose of Succinylcholine if required. Be very cautious about a second attempt in non-emergency patients and cancel non-emergent cases.

If the airway is in doubt and/or the patient remains hypoxic DO NOT attempt re-intubation and NEVER give a third dose of Succinylcholine. Try a nasopharyngeal airway / tube and release cricoid pressure. It will also fail to an emergency needle cricothyrotomy and set up for formal surgical cricothyrotomy.

LARYNGEAL SPASM

Occurs most commonly during induction but may also occur after extubation, especially if mal-timed (i.e. when the patient is semi-anæsthetised). It may also be a complication of Ketamine anaesthesia. Children are especially prone due to the smaller size of their airway.

The stimulus is usually premature insertion of an
airway or laryngoscopy (especially after induction with thiopentone or ketamine) or incision while the patient is still "light". Pharyngeal secretions and vomitus in the airway may also initiate it. Laryngeal spasm manifests as inspiratory stridor but may be silent if complete, with falling saturations and paradoxical chest/abdominal movements.

Don’t panic!
- Remove the stimulus (i.e., discontinue surgery, do not poke things in the airway and don’t try to force the tube through the cords)
- Administer 100% oxygen by mask and apply CPAP by screwing down the spit valve a little
- If saturations can be maintained (which they will 9/10 allow the spasm to subside - if they cannot (rare) try a small dose of Suxamethonium (10-20 mg), which may relieve the spasm without abolishing spontaneous ventilation. If the airway is still compromised then consider a full dose of Suxamethonium and intubation.

EXTUBATION

"Awake" extubation is the rule in our practice (i.e., make sure the patient coughs or makes a grab for the tube before you remove it). However, "deep" extubation is smoother for patients with a safe airway and may be safer for asthmatics and smaller children (for fear of inducing broncho- or laryngospasm respectively). Some patients may bite the tube on emergence so always put in a Guadel airway as well to pre-empt this problem.

Further Reading
2) Turnbull ME 1976 “Failed intubation drill” Anesthesia 31:850
3) Wee MYK 1988 The oesophageal detector device: Assessment of a new method to distinguish oesophageal from tracheal intubation Anesthesia 43:27

**STAGES OF ANAESTHESIA**

<table>
<thead>
<tr>
<th>STAGE</th>
<th>RESPIRATION</th>
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<th>EYE REFLEXES</th>
<th>LARYNX &amp; RESPIRATORY REFLEXES</th>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
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<td>4. Overdose</td>
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**MONITORING**

"Critical incidents" occur in ~10% of anaesthetics, so is important that you feel prepared to deal with them. Most are non-life threatening and simply dealt with, as long as they are recognised early and dealt with effectively. The common ones in our practice are the short apnoeas that occur post-induction with thiopentone or ketamine and hypotensive episodes in patients undergoing spinal anaesthesia for Caesarian Section. Serious arrhythmias and loss of airway control seem to occur quite infrequently.

Monitoring of patients during general or spinal anaesthesia is essential for spotting and dealing with problems as they occur - it is facilitated by use of the anaesthetic record. All such patients should have B.P. and pulse recorded every 5 minutes and continuous pulse oximetry. It should be remembered that the "knee" of the O₂ dissociation curve lies at ~90% and that blood O₂ content falls off rapidly below this value - which should be regarded as the minimum acceptable during maintenance. Another important point is that normal saturations do not guarantee normocapnia, which may have clinical importance. The signal may be disturbed if the patient is very cold, moving, or if there is a lot of ambient light.

Depth of anaesthesia can be difficult to assess clinically. The classical stages described by Guedel are most obvious during a mask anaesthesia when no pre-medication has been given. They are not as useful under ketamine or when the patient is paralysed. The most reliable guides are the pattern of respiration (with spontaneous ventilation) and the autonomic response to surgical stimulation. Typical signs of "light" anaesthesia are sweating, hypertension and tachycardia with atypical voluntary movements in the unparalysed patient. The surgically anaesthetised patient breathes slowly, regularly and deeply with little/no response to incision. Patients under spinal anaesthesia should be attended at all times and observed carefully for any signs of a "high spinal".

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**COMPlications during anaesthesia**

**Hypotension**

Significant hypotension intra-operatively is defined as a systolic pressure of \(< 70\) mmHg in fit adults or \(90\) mmHg in the frail/elderly, or a fall of \(25\)% from pre-op levels. These figures relate to the limits of cerebral autoregulation. Hypovolaemia is the most common underlying/potentiating factor.

For procedures lasting less than one hour, and where blood loss is insignificant, no intra-operative fluid replacement is necessary. For longer procedures, usually complicated Caesars or Laparotomies, give \(-10\) ml/kg/hour maintenance crystalloid to compensate for evaporative loss/loss fluid shifts etc. Healthy adults with a normal pre-op haemoglobin can tolerate \(750 - 1000\) ml blood loss (20\)% blood volume) without transfusion - replace with crystalloids and colloids in a ratio of 2:1. Further losses should be replaced with a combination of colloid and packed cells to maintain an optimal haematocrit.

All anaesthetic agents (with the exception of ketamine, diazepam and opioids) are both negative inotropes and vasodilators and depress central cardiovascular reflexes. The increased intrathoracic pressure generated during positive pressure ventilation can also reduce venous return and result in hypotension.

<table>
<thead>
<tr>
<th>Hypovolaemia?</th>
<th>Yes</th>
<th>Replace significant haemorrhage vigorously, fluid challenge (10) ml/kg over 5 minutes.</th>
</tr>
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<tbody>
<tr>
<td>No</td>
<td></td>
<td>Gave 3 mg/kg of Epinephrine every 2-3 minutes until satisfactory response. Exclude an iatrogenic compression!!</td>
</tr>
<tr>
<td>Post-scapa?</td>
<td>Yes</td>
<td>Exclude anaemia! Reduce % Halothane. Expectant management if immediately post-induction</td>
</tr>
<tr>
<td>No</td>
<td></td>
<td>Exclude pneumothoraces. Reduce minute volume and vigorous IPPV</td>
</tr>
<tr>
<td>Drug effect?</td>
<td>Yes</td>
<td>Resuscitate vigorously with fluid challenge and transfusion as required. Administer an infusion if resistant</td>
</tr>
<tr>
<td>No</td>
<td></td>
<td>Check Fi(_O_2) using (O_2) monitor</td>
</tr>
</tbody>
</table>

**Hypoxaemia**

A systematic approach is helpful when the saturations start dropping!

Confirm that the pulse oximeter is reading accurately - but remember that it is considerably more sensitive at detecting desaturation/cyanosis than you are. Follow the algorithm):

- Is the airway secure? Yes \(\rightarrow\) Check position of and suction of ETT (ETT is correctly positioned or blocked). Check out integrity 
- Is ventilation adequate? Yes \(\rightarrow\) Lighter anaesthesia, hand ventilate. \(\rightarrow\) Check breathing system for disconnection. Check ventilator settings.
- Evidence of bronchospasm? (high inflation pressures, wheeze etc.)
  - Yes \(\rightarrow\) Give 10 puff of \(\beta\)-agonist down tube
  - No \(\rightarrow\) Give Amphobrine 5 mg i.v. i.e. effect of muscle relaxants or intubation? Withdraw larynx 1-2 cm and check random saturation. Deepen anaesthesia with 3-5% Halothane if severe. Give Adrenaline 0.3-0.5 ml in 1:1000 i.v. if signs of anaesthesia.

- Clinical signs of shock?
  - Yes \(\rightarrow\) Discontinue \(N_2O\) immediately. Needle thoracostomy followed by chest drain
  - No \(\rightarrow\) Resuscitate vigorously with fluid challenge and transfusion as required. Administer an infusion if resistant.

- Check \(F_iO_2\) using \(O_2\) monitor - the \(F_iO_2\) does not correlate with flowmeter setting, switch to cylinder if on pipeline supply and to emergency free-standing cylinder if cylinder supply exhausted

- Increase \(F_iO_2\) to compensate.

Intra-pulmonary shunting due to the effects of anaesthesia and ventilation but consider: tamponade, fluid or perivascular embolism.
ARRHYTHMIAS

We do not routinely monitor the E.C.G. since coronary artery disease is uncommon in our patients. However, high risk patients or a heart rate of < 60 or > 120 should be assessed using the monitor. The common causes of intra-operative arrhythmias are hypoxaemia, hypercapnia, hypokalaemia and autonomic stimulation (sympathetic stimulation on intubation and parasympathetic stimulation from visceral traction etc.). Pre-existing cardiac disease and medication esp. aminophylline and digoxin should also be taken into account.

BRADYCARDIA

- Healthy sinus bradycardia
- Effect of Halothane (dose-dependent suppression of SA node may lead to a junctional escape rhythm, or “Benign junctional block”) or Opioids Para-sympathetic stimulation (laryngoscopy, visceral traction)
- Late sign of hypoxia
- True heart block or sinus node disease

Treatment is not usually required if Rate is > 40/min and B.P./Perfusion maintained. Withdrawal of noxious stimulation and Atropine 0.25 mg i.v. will usually restore sinus rhythm and B.P.

TACHYCARDIA

- Light Anaesthesia
- Hypovolaemia
- Anaemia / Hypoxaemia
- Fever
- Drug effect - ketamine, suxamethonium, thiopentone, atropine, ephedrine
- Narrow complex tachycardias esp. P.A.T. and A.F.
- Broad complex tachycardias esp. V.T.

In all cases correct the underlying cause. Vagal maneuvers should be tried with P.A.T., but if there is no response and in cases of paroxysmal A.F., give Digi-oxin 0.5 mg as an i.v. infusion over 30 minutes. In cases of severe compromise, synchronised D.C. Cardioversion 10-50 J should be performed. Calcium antagonists and b-blockers are relatively contra-indicated under Halothane anaesthesia. Franks V.T. (uncommon in our parents) requires lignocaine 1.5 mg/kg slat i.v. followed by an infusion of 1-4 mg/min.

ANAPHYLAXIS

This occurs in ~ 1 in 10,000 anaesthetics, more commonly in female patients. Almost all anaphylactic reactions occur around the time of induction and the most likely culprits are thiopentone, suxamethonium or opioids. Reactions to ketamine and diazepam are very rare. The usual signs are an erythematosus rash, bronchospasm and/or cardiovascular collapse. Draw up 1 mg (1ml of 1:1000) adrenaline in 9 mls of water and give 0.3-0.5 mg stat i.v. and further 0.1 mg aliquots (amounts) until a satisfactory response is obtained. Give a 10 ml/kg bolus of fluid. If hypotension is resistant to bolus adrenaline (uncommon), give an infusion of 5 mg in 500 ml at 10 ml/hr initially. Persistent bronchospasm should be treated with hydrocortisone 500 mg i.v. and aminophylline 6 mg/kg i.v. over 20 minutes.

Further Reading
1) Hanna and Rees Williams 1983; Anaesthesia: A practical review. BMJ 311:367
2) Zaik PC and Davis HS 1968; "Cardiac arrhythmias during halothane anaesthesia" Anaesthesia 47:528
3) Morris WR 1993; "Desaturation diagnosis and treatment" Bailliere's Clinical Anaesthesiology 7(2):215
4) Fister MM and Bolda BA 1984; "Anaphylactoid reactions during anaesthesia" Clin Anaesthesiology 2:237
5) Scott DA and Davies MJ 1993; "Hypotension" Bailliere's Clinical Anaesthesiology 7(2):237

LOCAL ANAESTHESIA AND USEFUL PERIPHERAL BLOCKS

Local anaesthesia is the safest and most satisfactory method for a surprisingly large number of procedures, though it is usually inappropriate for children. In our hospital, the following are usually done under local - most debridements / revisions / suturing of small and superficial wounds, small abscesses, small skin grafts and most biopsies including lymph node and breast. Use of ring and brachial plexus blocks allows most of our hand / forearm surgery to be done without a G.A. (manipulations excepted). Ring block is also the method of choice for circumcision in adults. Lignocaine 1% (10 mg / ml), Lignocaine 2% (20 mg / ml) and plain 0.5% (5 mg / ml) Bupivacaine are available for use.
The maximum dose for a single administration of Lignocaine is 3 mg/kg.

The maximum dose for a single administration of Bupivacaine is 2 mg/kg.

However, "near-toxic" doses are used often in regional anaesthesia.

Both are removed from site into the general circulation with maximum plasma levels at ~30 minutes. In order to minimise the risk of significant systemic absorption, either aspirate before you inject or keep the needle moving constantly. Local anaesthesia is relatively ineffective in highly inflamed or infected tissue and may also be very rapidly absorbed from such a site. The rate of removal is related to the vascularity of the area and can be reduced by the use of vasoconstrictors.

Adrenaline should be added at a concentration of 1:200,000 (= 100 µg/ml) to 1:1,100 solution for each 20 ml of lignocaine solution. The maximum dose for an adult is 500 µg. It makes no difference to the absorption of Bupivacaine and need not be used. NEVER use adrenaline for anaesthesia of the nose, ear, penis or digits.

N.B. Absorption of adrenaline during halothane anaesthesia can lead to serious arrhythmias so be very cautious about converting to a G.A. after failed infiltration or regional anaesthesia involving adrenaline at any concentration higher than 1:400,000 - rather use a ketamine infusion.

When adrenaline has been added the dose of Lignocaine can be doubled. Large volumes of 0.4% Lignocaine can be made by adding 20 ml 2% to 80 ml of 0.9% saline and 0.5 ml 1:1000 adrenaline - this is very useful for anaesthetising large areas and augmenting a low or partial spinal block at Caesar. Lignocaine lasts for about 1 hour. The optimal concentration of Bupivacaine for infiltration anaesthesia is 0.25% so where more prolonged anaesthesia is required equal volumes of 1% lignocaine and 0.5% bupivacaine may be mixed.

TOXICITY

Hypersensitivity reactions are uncommon with amide-type agents like lignocaine and bupivacaine, but patients should always be monitored for the first 30 minutes for signs of systemic toxicity. This almost always results from intravascular injection and is characterised by anxiety, nausea/vomiting - convulsions may be followed by drowsiness and cardiovascular depression. Supportive care is all that is normally required although diazepam may be necessary to control fits and rarely bolus droshing of an adrenaline infusion may be required to support the circulation temporarily.

AXILLARY BLOCK

This is an extremely useful brachial plexus block and safer for the non-expert than the supraclavicular route (which carries a risk of pneumothorax of 1±4% even in the best hands).

The principal point is poor blockade of the musculo-cutaneous and intercosto-brachial nerves which leave the sheath proximally at the level of the coracoïd process. This may make use of an arterial tourniquet proximally uncomfortable.

Position the patient’s arm with the elbow at 90° - don’t overabduct the shoulder. Use 40 ml of 1% lignocaine or 0.5% Bupivacaine with adrenaline. Add 0.4 ml of 4% Sodium Bicarbonate (this speeds up the onset of the block). Apply a tourniquet - digital pressure distally to prevent leakage from the distal end of the sheath after injection. Slide a scalp vein or regional needle with the bevel directed downwards under the fibrous axillary sheath next to the axillary artery until it can be seen to pulsate strongly when released. Inject the solution and wait 15-20 minutes for the block to take. If you puncture the artery you need not withdraw, rather re-position the needle in the sheath, aspirate again carefully to confirm you are no longer in the artery and then inject. As you come out of the sheath, inject 2-3 ml of solution into the medial aspect of the arm to block the intercosto-brachial nerve.

The block can have a latent period of 30 minutes and spreads proximally to distally - so be patient! Test elbow flexion (musculo-cutaneous n.) and extension (radial n.) and grip strength (median/ulnar n.). Assess the sensation up and down the pre- and post-axial border of the arm. If

- Intercosto-brachial nerve
- Coraco-brachialis muscle
- Musculo-cutaneous n.
- Coracoïd process
- Axillary sheath
- Chloride

sensation is preserved on the lateral aspect of the forearm perform a rescue block of the musculo-cutaneous n., either by injecting 3-4 ml of solution along the lateral aspect of the biceps tendon 2cm proximally and distally to the flexion crease, or injecting 5-10 ml into the coraco-brachialis muscle under the coracoïd process. The block should last 2-4 hours.
Further Reading

**SPINAL ANAESTHESIA**

This is the method of choice in our hospital for virtually any major operation below the umbilicus - we use it for all routine caesarian sections and leg amputations. Hyperbaric bupivacaine solution is injected into the subarachnoid space to produce blockade of the spinal roots and ganglia. The level of the block is dependent on the dose injected, volume and baricity of the solution, level of puncture and position of the patient during the first 5-10 minutes. All types of nerve fibres are affected with large, myelinated nerve fibres the first to be blocked and the first to recover. Sensory fibres should be blocked for 2 hours. Sympathetic blockade extends - two segments higher than the sensory block and the resulting vasomotor paralysis is responsible for hypotension, the major immediate complication.

The contra-indications to spinal anaesthesia are:
- bleeding diathesis / platelets < 100
- hypovolaemia
- fixed cardiac output (e.g. severe mitral stenosis)
- local sepsis on the back
- raised intra-cranial pressure
- G.I. perforation (increased peristalsis due to unopposed parasympathetic action)
- proven diabetic autonomic neuropathy

**TECHNIQUE**

Patient sitting upright on table with nurse attending. All patients should be pre-loaded with at least 500 ml of NS/saline. Check B.P. before you start.

Perform lumbar puncture at L3-4 level (1 space above level of the iliac crests) wherever possible use a 22G (0.7 mm, black) standard spinal needle or smaller, if necessary with a large hypodermic as an introducer/sheath. Always insert the needle with the bevel directed laterally to split rather than cut the dura.

Ensure that CSF is freely flowing. You may also aspirate a little to ensure that the needle is not in a spinal/ dural vessel. Inject slowly (1ml every 5 seconds) and do not barkette. ONLY 0.5% Bupivacaine with 5% dextrose (s.g. 1.024) should be used - 1.5-2.0 ml (7.5-10 mg) for caesarian section, 1.0-1.5 ml (5-7.5 mg) for saddle block or leg operations. Morphine 0.5 mg or

**NEUROLOGICAL LANDMARKS FOR SPINAL**

Fentanyl 50 mg may be added to provide extra analgesia (though we do not do this in our hospital), but NEVER use adrenaline in an attempt to prolong the block. Note that pregnancy, obesity and old age are associated with a lower subarachnoid volume and cautious dosing may be necessary. Do not inject during a contraction as the increased intrathecal pressure could raise the level of the block.

Keep the patient in the initial position for 5 minutes to allow the solution to sink and "fix" to the tissues. Position the patient for surgery (15° left lateral tilt for Caesars (Crawford position), head supported by a pillow in all cases). Check B.P. every 3-5 minutes.
Determine and monitor the level of the block for at least 20 minutes - the patient should be attended at all times. The sensory level is easily assessed with a pair of forceps - the umbilicus is T10, the xiphoid T7 and the nipples T4. Motor blockade can be crudely assessed as 100% - can't move legs at all, 66% - flexes feet but not knees, 33% - flexes knees. If you find the sensory block has ascended above the level of the axillae, test biceps function (C5-6 myotome). If this is absent, prepare immediately to deal with a high spinal.

In order satisfactorily to anaesthetise the visceral peritoneum for abdominal surgery the Great Splanchnic nerve should be blockaded, which probably requires a block to T6 in most patients. In a supine/as head-down position 2 mls Bupivacaine will block to T4 or even higher with involvement of the respiratory muscles, but the level of the block is probably lower in the sitting position with a hyperbaric solution. 1.5 mls will usually block to T4 but may be as low as T6-10 and give a shorter duration of anaesthesia, which may be a problem for an inexperienced surgeon doing a C/S through a midline incision.

COMPLICATIONS

HYPOTENSION

Treat initially with a last bolus of i.v. fluids, give 100% Oxygen. If no response, raise the patient's legs but do NOT put her head-down. Dilute 1 vial of ephedrine (30 mg) in 20 ml of water. Give 3 mg aliquots every 2-3 minutes until the B.P. responds. If hypotension is associated with bradycardia due to cardioplegia from a T1 or higher block, give atropine 0.3 mg.

FAILED/INADEQUATE BLOCK

Always test the adequacy of your block before you cut! A totally failed block mandates a GA and if you don't feel capable of doing this safely, refer the patient. If the block is slightly too low or partial, you have two options:

- Paramedication - Give ketamine 0.25 mg/kg aliquots every 10-15 minutes (preferred before the cord is clamped) or Pethidine 50 mg i.m./50 mg i.v.
- Local infiltration - Make 100 ml 0.4% Lidocaine as follows - add 20 ml 2% Lidocaine to 80 ml sterile 0.9% Saline with 0.5 ml 1:100 Adrenaline (this makes a dilution of 1 : 200,000). Infiltrate two bands either side of the midline about two finger's breadth apart from the symphysis to 5 cm above the umbilicus. Infiltrate the sheath underneath through the already anaesthetised skin. After incising the skin and fascia, infiltrate the peritoneum underneath the sheath, before entering the abdomen. Don't try to pack, and be as gentle as possible.

"HIGH SPINAL"

Respiratory difficulties are usually the first sign (some doctors like to maintain constant verbal contact with the patient while they are operating to detect this), and may progress to a respiratory arrest if the C2-4 segments are involved, often accompanied by severe hypotension. The patient should be intubated if necessary and ventilation may be required with 0.5% halothane as sedation - even if this is the case the effects of the spinal will wear off after 2-3 hours, and the patient can be weaned off the ventilator. An adrenaline infusion may be required temporarily if hypotension responds poorly to ephedrine/atropine.

POST-SPINAL HEADACHE

We see this uncommonly. It is related to a persistent CSF leak after dural puncture. It may be delayed for up to 6 days and is characteristically occipital/frontal and worse on sitting/standing. It may last 6 weeks. Analgesia may suffice in mild cases but tight binding of the upper 8" of the abdomen or blood patching may be required.

Less common complications are meningitis, transient VI/VIII palsies and rarely, spinal cord compression from a paraspinous haematoma or abscess.

Further Reading
1) Wilkinson O 1955 "Low spinal anaesthesia for Caesarian Section" Update in Anaesthesia 6:26
2) Van Boeckel JF 1996 "Spinal Anaesthesia for Caesarian Section" Update in Anaesthesia 7:30
4) Chambers WA 1952 "Spinal anaesthesia with hypobaric bupivacaine: effects of concentration and volume administered" B.J. Anaesthesia 54(1):75
In principle, we aim to reserve general anaesthesia for cases where regional/local anaesthesia is difficult or unsatisfactory, or where good control of the depth of anaesthesia is required.

We use three distinct methods of general anaesthesia in our hospital:

1) **KETAMINE** is the method of choice for most patients requiring a G.A. in our hospital, because it is the safest. It produces a unique “dissociative” state in which the patient is insensitive to pain but retains muscle tone and protective reflexes (pharyngeal, laryngeal and corneal). Alone amongst i.v. induction agents it is a cardiovascular stimulant, and causes a rise in pulse and blood pressure. The depth of anaesthesia can sometimes be difficult to assess - movement/verbalisation does not always indicate pain but do not assume that surgical anaesthesia has been achieved until nystagmus and voluntary movement have been abolished. Usually children should be premedicated with atropine 0.02 mg/kg to prevent salivation and airway complications during the procedure, and adults with diazepam 0.2 mg/kg to attenuate emergence delirium.

Ketamine may be used as an i.v. bolus of -2 mg/kg, given slowly over about 30 seconds to avoid apnoea (which is usually transient in most cases). This should give 5-10 minutes of surgical anaesthesia after 60 s. Boluses may be given repeatedly if you have underestimated the extent of the procedure, but a better method would be to use an infusion of 0.5 mg/ml (250 mg ketamine in 500 ml of fluid) at a rate of 1.5-3.0 mg/kg/hour (use drip rate chart). Remember that at the end of the operation you still have ~ 10-15 ml's of solution in the drip set, so ensure that you always continue running N/Saline through for at least five minutes before the patient leaves the theatre esp. in children. Ketamine may also be given intramuscularly to children without a line up at 4-6 mg/kg which gives up to 25 minutes of anaesthesia after ~5 minutes.

The only absolute contra-indications to ketamine anaesthesia are hypertension, cardiac/cerebrovascular disease, and raised intra-cranial or intra-ocular pressure. It is not ideal but can be used in patients with a history of mental illness or alcoholism. In our practice, it is the ideal paediatric anaesthetic due to its safety and should be used wherever possible.

2) **THE MASK TECHNIQUE** is useful for patients where airway problems are not anticipated and good control of anaesthesia, or a relative degree of muscle relaxation are needed (e.g. some manipulations, hand surgery/failed axillary block). Neurovascular blockade and intubation are not required and spontaneous ventilation is preserved throughout. The technique is simple but requires more anaesthetic experience as the airway/ventilation must be closely monitored and controlled if necessary - this must be the job of a doctor or trained anaesthetic assistant.

The patient is induced with an i.v. agent e.g. Thiopentone 4-6 mg/kg - a “sleep dose” titrated to effect (the eyelash reflex is a reliable indicator with thiopentone) is used rather than the pre-determined bolus used in a crash induction. Anaesthesia is then maintained with Halothane, Nitrous Oxide and Oxygen given by mask and varied according to depth. Increase the concentration of Halothane from 1% in 0.5% increments every 5 breaths or so. For the first few minutes after the induction high concentrations of Halothane (~3%) should be administered to saturate the tissues (“Over-pressure”), but after 5-10 minutes 1-2% should provide adequate anaesthesia. Opiates should be used to provide extra analgesia e.g. pethidine 1mg/kg i.v.

Some children can have a gas induction using the Ayre's 'T-piece' if they will tolerate the mask as this is safer - starting with an O2 / N2O mix only. Halothane concentration should be increased from 0.5% by 0.5% every 3-4 breaths until surgical anaesthesia is reached (up to 5% may be required). In practice this even works well with a crying/shouting child, especially if the mother is present. However, the technique requires experience and should be used only when other methods fail.

3) **INTUBATED GENERAL ANAESTHESIA** is the method of choice for most emergency abdominal procedures (e.g. ecliposes, trauma and non-trauma laparotomies) which are technically difficult without muscle paralysis, often involve a full stomach/unsafe airway, and will in most circumstances last >1 hour. A spinal is safer for Caesarian Section due to the special risks involved with an obstetric G.A. (25% of maternal deaths in Zululand are related to General Anaesthesia).
1) Resuscitate the patient as fully as possible pre-op. pass an n.g. tube and aspirate it. Check the anaesthetic machine, draw up your drugs and label them. Check the ventilator settings.

2) Rapid sequence induction
   - Pre-oxygenate for 5 minutes
   - Thiopentone 5 mg/kg i.v. bolus (beware of giving too much (>2 mg/kg) in the elderly or shocked patient when ketamine 2 mg/kg is a better choice).
   - Apply cricoid pressure as the induction agent is given.
   - Succinylcholine 1 mg/kg i.v. bolus. Keep the mask seated on the patient’s face. Wait until the fasciculations stop and the patient’s jaw drops.
   - Intubate and confirm tube placement. Release cricoid pressure.

3) Atracurium 0.1-0.25 mg/kg i.v. as soon as you are happy that the airway is secure.

4) Turn the ventilator on.

5) Maintenance - N₂O / O₂ 70/30% + Halothane 1%. intravenous opiates (e.g. Pethidine 100mg i.v.) provide extra analgesia and reduce Halothane requirements. For an obstetric G.A. wait until the baby is delivered before giving opiates with a long half-life.

6) Reversal - Give Atropine 0.02 mg/kg + Neostigmine 0.04 mg/kg (in the same syringe) slowly i.v. Do not attempt to reverse the effect of atracurium earlier than 20 minutes after the last dose.
   - Turn halothane and nitrous oxide off. Give 100% Oxygen. (This should be continued for ~ 5 minutes after N₂O is stopped to prevent diffusion hypoxia).
   - Suction the airway, ensure a Guedel airway is in place (in case the patient bites the tube while emerging).
   - Exsufflate.

Further Reading
1) White et al. 1982 ‘Ketamine - its pharmacology and therapeutic uses’ Anaesthesia 37:119
2) White P.E. 1988 “What’s new in intravenous anaesthetics?” Anesthesiology clinics of N. America 6 (2) 297
3) Kelcham O.W. 1993 Where there is no anaesthesiologist: the many uses of Ketamine Trop. Doc. 29:183
Obstetric practice in our district is fairly typical of Southern Africa but may be quite different from the experience of many newly arrived doctors. Antenatal care is free and 95% of mothers in our district attend at least once, but 20-30% deliver at home without the aid of a traditional birth attendant. Some use traditional medicines to facilitate labour (isihlambezo, inembe, imbeleksane) and afterwards (ugqotho). Some of the plants included in these preparations e.g. khavia nunnata have powerful oxytocic effects. At home the cord may be cut with a razor blade but is traditionally cut with a reed (umlalanga) and may not be tied. Ochre or dung is sometimes still applied. However, though cases of neonatal tetanus are still seen, we see very few neglected third degree tears in our hospital. Of mothers delivering with a trained midwife, half deliver in clinics and half in hospital. Partographs are used for all.

The perinatal mortality rate in our district is 40 per 1000 and the maternal mortality rate probably - 150 per 1000. Common causes of perinatal mortality/morbidity in our district are APH, prematurity, obstructed/prolonged second stage of labour, eclampsia/pregnancy-induced hypertension, diabetes and syphilis/chorioamnionitis. Asymptomatic carriage of STDs can be demonstrated in - 50% of our antenatal attenders and - 40% are HIV +ve. Platypelloid and small Gynaecoid pelvis are prevalent amongst Zulu women. The pelvic brim often has a high angle of inclination and a restricted inlet which results in a late descent of the head during the third trimester first stage of labour. Despite cephalopelvic disproportion, though the head (caput) may be visible at the introitus, it is not uncommon to find that the head is still 3/5 palpable abdominally. Our rate of caesarean section for hospital clinic deliveries is currently ~ 15%.

Only about 2.5% of the Zulu population are Rhesus negative and the commonest Rhesus phenotypes are CD e so almost all of the few cases of haemolytic disease of the newborn are due to anti-D antibodies. Sensitisation to Kell, Duffy or Lewis antigens is extremely rare, though more of the Zulu population are negative for these antigens than Europeans.

- 15% of births are low birthweight and the incidence of twins is ~ 1 in 75 deliveries. The mortality rate for infants up to including 30/40 gestation managed in our nursery is 73%, though more intensive neonatal care is available at our referral centres. Breastfeeding is widely, though not universally, practised by mothers in our district.

The maternity services in our district rest primarily on clinic and advanced diploma midwives - they run the community and high-risk referral ANC clinics. At the hospital, the maternity M.O. reviews new and sick high risk cases daily - all low-risk waiting mothers are reviewed once a week. Other M.O.s provide on-call labour ward cover. The following protocols are aimed at everyone involved in perinatal care in our district. They are designed to complement the excellent already existing manuals by Drs Larsen and Breen and the Perinatal Education Programme which should be referred to if further details are needed.

Further Reading
3. Larsen J.V. 1996. 'The Differences between caucasian and black obstetric patients in South Africa.' OSF Forum p.36

HYPERTENSION

BP >140/90 mm Hg on two measurements 4 hours apart. Occurs in ~ 1 in 8 of the antenatal population in the second half of pregnancy. Antenatal is divided into two groups:

- **Aproteininuric** - may be chronic hypertension (present in the first half of pregnancy), or gestational. These patients are at lower risk.
- **Proteinuric** - + proteinuria is already a bad sign and patients with 2+ proteinuria should be urgently assessed by a doctor. Never attribute 2+ proteinuria or more to a UTI in a hypertensive patient. These patients are high risk and in danger of developing eclampsia, intracerebral haemorrhage, abruptio, IUGR and IUD.
Management

1) Initially admit anyone with a DBP ≥ 100 mm Hg.
   - 8 hourly BP chart, daily urine dipstick and kick chart.
   - If high B.P. is confirmed, treat with Methyl Dopa 500 mg t.d.s.
   - Nifedipine 10 mg p.o. for every B.P. spike >110 mm Hg.
   - US/S for dates and liquor volume.
   - Check urea, creatinine, urate and platelets.

2) If B.P. settles on treatment, patient is aprotininuric, U/Cr/Urare/Platelets are normal and foetal movements are good - may continue management as an outpatient.
   - Check B.P. and urine dipstick weekly at ANC.
   - Induce if necessary to avoid post-datism.
   - Re-admit if condition deteriorates.
   - N.B. Patients occasionally develop eclampsia without proteinuria but this would be very rare if F.M. good and urate normal.

3) If BP is difficult to control, increase anti-hypertensive medication as necessary
   - Methyl Dopa increasing to 750 mg 6 hrly
   - Hydralazine 25-50 mg 6 hrly
   - Nifedipine 10-30 mg 8 hrly
   - Prazosin 1-5 mg 8 hrly

4) In-patient management is required for all patients with any of the following features:
   - proteinuria
   - Urea >4.5, Cr >75, Urate >0.35 (the last reflects placental DNA turnover and cannot be interpreted in twins)
   - platelets <120
   - poor foetal movements
   - IUGR
   - severe oedema

ECLAMPSIA

The principles are simple: the mother’s condition takes priority over that of the foetus. A vaginal delivery is often possible, but if you do have to do a C/S get experienced help or transfer the mother once her condition has been stabilised.

1) Resuscitate the patient; if still fitting, use suction and left lateral position to prevent aspiration, give O₂ by mask, establish i.v. access. Give diazepam 10 mg boluses only if the fit has not yet stopped. Once the fit stops, nurse 30° head up (to minimise cerebral oedema).

2) Give all patients a loading dose of Magnesium Sulphate 4g i.v. and 10g i.m. (half into each buttock). Maintenance doses of 5g i.m. are then given 4 hrly until 24 hours post-delivery provided that the urine output is >30 ml/hr, respiratory rate is 16/min and foetal reflexes are present. Catheterise all patients.

3) Reduce B.P. to 140/90. Use nifedipine 10 mg p.o. 1/2 hrly p.r.n. for conscious patients. Give hydralazine 6.25 mg i.m. in unconscious patients (but beware signs of cardiac failure). Repeat in 30 minutes if no response but be careful not to precipitate a hypertensive episode.

4) Most eclamptics are hypovolaemic but very prone to pulmonary oedema. If the patient is oliguric, give a cautious fluid challenge of 300 ml N/Saline or Ringers repeated according to the JVP, state of the chest. If there are other complicating factors, especially haemorrhage, a CVP line is very useful - aim for a CVP of slightly below normal (~3-5 cm H₂O). In general, 100 ml/hour maintenance is about right - do not give 5% dextrose as this may precipitate cerebral oedema.

5) Examine the patient carefully with attention to the leasibility of vaginal delivery and condition/viability of the foetus. Order Hb, U&E, Cr, INR and platelets. Check the whole blood clotting time yourself.
6) Decide on a mode of delivery - this is the only way to stop the disease process, and must be achieved as soon as possible, and preferably within 12 hours. Unless the mother already has definite coagulopathy or a definite obstetrical indication for C/S (e.g. CPD), aim initially for vaginal delivery if not already in labour. Induce or administer using Pgs/AROM/Syntocinon as appropriate. This gives you time to stabilise the patient, who often makes a significant initial improvement (e.g. regaining consciousness). Reassess in 4 hours. If labour is progressing aim for vaginal delivery with assisted second stage. If there is no change, make sure that the mother’s condition is stabilised and prepare/refer for C/S (depending on available anaesthetic skills). As long as a mother has had magnesium sulphate and her B.P. is well controlled, she is unlikely to deteriorate during transfer. If you decide to do a G.A. remember the possibility of fetal oedema (have 7.0 ET tube at hand) and the effects of magnesium sulphate on other drugs - reduce doses of muscle relaxants and opioids accordingly.

7) Give Syntocinon NOT ergometrine post-delivery. Monitor the urine output and fluid status closely.

8) Low dose aspirin prophylaxis (75 mg daily) should be considered for all eclamptics and severe pre-eclamptics at the time of their next pregnancy, but especially those with onset <32w.

Further Reading
2) Collaborative Eclampsia Trials 1995 “Which anticonvulsant for women with eclampsia? Evidence from the Collaborative Eclampsia Trial”. Lancet 345: 1455

ANTE-PARTUM HAEMORRHAGE
P.V. bleeding in the second half of pregnancy.

Abortion typically painful, praevia painless.

The haemorrhage may however be completely concealed, so be suspicious in any woman with severe lower abdominal pain and a distressed dead foetus. If the baby is dead, the cause is invariably a severe abortion, not a vasa praevia.

Management
The principles of management are to resuscitate the mother aggressively and only to do a C/S for a live/ viable foetus or a placenta praevia.

1) Resuscitate the mother: Large bore i.v. access both arms. Give 1-2 litres of crystalloid +/- colloid fast and start cross-matched blood as soon as it is available. Check Hb, U&E, Platelets and INR. Check the whole blood clotting time at the bedside (normal 3-8 minutes). If coagulopathy is already present give freeze-dried plasma 4-5 units stat (this lasts about 1 hour in the circulation)

2) Decide on the cause: Do NOT do a p.v. until you have established the placental site by US/S and speculum examination! Remember that even a “heavy show” should be mixed with mucus. Beware of a diagnosis of abortion in a scarred uterus - always consider the possibility of a ruptured uterus especially if there is a sudden cessation of contractions/pain during labour. The presenting part typically disengages and may be easily palpable in the abdomen.

3) Decide on the mode of delivery: Consider the clinical and antenatal findings esp. cause/severity of APH and the gestational age.

- Placenta Praevia:
  - <37/40 manage conservatively - deliver by emergency C/S only if bleeding is continuous/frequent and heavy. Give dexamethasone 6 mg i.m. 12 hrly x 4 if <34/40.
  - ≥37/40 deliver by elective/emergency C/S even if the bleeding has stopped.

- Abruption:
  - Deliver vaginally if the foetus is dead and the mother is well-resuscitated. Do AROM and start a Syntocinon infusion. Prompt delivery can be expected in most cases due to the irritant effect of the intrauterine bleeding. If progress is evidently poor, consider referral at an early stage for C/S.
  - C/S is reserved for a viable foetus or a deteriorating mother. However, consider referral if the platelet count is <50. The WBCT is >8 minutes or there are signs of coagulopathy.
  - C/S for APH can be dangerous - you must at least know how to devascularise the uterus. Bleeding may be severe in the presence of coagulopathy or an abnormal placental site. Consider referral for C/S with an anterior placenta praevia and uterine scar - a caesarian-hysterectomy may be necessary owing to adherence of the placenta to the scar.

4) Post-partum: Prevent PPH with a 40u/l Syntocinon infusion but be prepared to explore in theatre if bleeding does not promptly settle - this may be due to uterine atony or rupture or resistant coagulopathy.
Further Reading
3) Hey JI et al. 1984 'The treatment of rupture of the pregnant uterus: Analysis of 93 cases treated in a rural hospital in Zambia' Int J Gyn and Obst 22:415

PRE-LABOUR RUPTURE OF MEMBRANES
NEVER DO A P.V.

Confirm the diagnosis - take a careful history and do a sterile speculum to see if liquor is draining from the cervix. Look at the quality of the liquor (is it meconium-stained or offensive?), exclude cord prolapse and note the state of the cervix.

Determine the gestation - if an early US/S has not been done and you are unsure clinically you can do a foam test on liquor obtained from the speculum examination or amniocentesis. Fortunately, the earlier in gestation that the membranes rupture, the longer it takes for labour to ensue - at 28/40 ~25% of mothers will still not be in labour after one week, whereas at term 80% will be in labour by 24 hours.

- <34/40 or Negative amnio
  - 4 hrly obs and pad checks
  - Dexamethasone 6 mg 12 hrly i.m. x 4
  - Ampicillin 500 mg 6 hrly i.v./p.o. for 1 week
  - Metronidazole 1g p.r. 8 hrly/400 mg 8 hrly p.o. for 1 week
  - If infection occurs, add Gentamicin and deliver
  - If the patient does go into labour spontaneously, do not suppress it.

- >34/40 or Positive amnio (1-4+)
  - Await spontaneous labour for 24 hours if no P.V. has been done and maternal and foetal obs are satisfactory
  - If not in established labour by 24 hours do a P.V. and induce with Pgs/Synfo depending on the state of the cervix. The exceptions are grandmultiparae or previous C/Ss in whom conservative management should be pursued for another 24 hours - if there is still no sign of labour proceed to C/S.

Further Reading
1) Von Hoender J, Steyn DW 1996 'Management of premature rupture of membranes after 34 weeks gestation - early vs delayed induction of labour' S A M J 86:282

PRETERM LABOUR (WITH INTACT MEMBRANES)
- Confirm the diagnosis - 'pseudo-contractions' may result from U.T.I. True preterm labour may be precipitated by twins, abortion or chorioamnionitis.
  - Do a P.V., dipest the urine and do a C.T.G. Reassess after 4 hours. If there is no change in the cervix 'false labour' is confirmed.
- Establish the gestation - 'Pre-term' ≤ 34/40. Do an amnio if you are in any doubt. There is no point in suppressing labour when the foetus is mature!
  - Suppress labour if definitely < 34/40 or amnio 0-1+ and the cervix is < 5 cm dilated (exception twins or known cervical incompetence when loxolysis may be effective even up to 8 cm) Ensure that the mother is not hypertensive or haemorrhaging and has no signs of cardiac disease.
- Hexoprenaline - 10µg i.v. slowly then 150µg in 1 litre at 30-45 drops/min inflated to the contractions and the maternal pulse (which must never exceed 120/min). Continue the infusion for 6 hours then reduce it half the therapeutic rate for 12-18 hours. Then continue oral Hexoprenaline 1 mg 6 hrly for the duration of pregnancy.
  - Indomethacin - 100 mg p.r. 12 hrly x 4 (if <32/40)
  - Dexamethasone - 5 mg 12 hrly x 4 i.m.
  - Ampicillin 500 mg 6 hrly x 4 doses i.v. then oral for 1 week
  - Metronidazole 1g p.r. 8 hrly/400 mg 8 hrly p.o. for 1 week
  - Vitamin K 10 mg i.m.

U.T.I
If MSU shows 10+ leucocytes and bacteriuria give Nalidixic acid 1g 6 hrly for 7 days.

VAGINAL DISCHARGE
In pregnancy, vaginal discharge may be more copious but should not be offensive or discoloured. True pelvic inflammatory disease is thought to be very rare in pregnancy but studies in our district have demonstrated high carriage of STD organisms in antenatal patients.
- If white and itchy - Econazole/Clotrimazole pessary
- If frothy, green/yellow - Metronidazole 2g stat
- If unresponsive to metronidazole or cervicitis seen - Erythromycin 500/500mg 6 hrly for 10 days + Metronidazole 400 mg 8 hrly for 7 days.
WR POSITIVE

The prevalence of syphilis in our antenatal clinics is 6.5% - it probably approaches 30% in unbooked patients. WR +ve mothers have an odds ratio of ~12 for adverse perinatal outcome in our district - in the untreated case the risk of congenital syphilis is ~10%.

- Benzathine Penicillin 8 ml i.m. weekly for 3 weeks as an outpatient
- If an inpatient, Procaine Penicillin 600 mg i.m. daily for 10 days
- If truly allergic to Penicillin (rare) - Erythromycin 500 mg 6 hly for 14 days (much less effective)

Further Reading

ANAEMIA

- Admit if Hb <7 or <9 close to term
- Check stools for parasites and urine for bilharzia
- Give ferrous sulphate 200 mg 6 hly and Folate 5 mg daily for the rest of pregnancy
- Recheck Hb 1 month after starting treatment
- Only transfuse if absolutely necessary (<6g in labour or <8g prior to C/S)

POLYHYDRAMNIO

The common associations are maternal diabetes, foetal abnormalities and twins but a cause cannot be found in half.

- Check the blood glucose and Rh factor status. US/S will detect twins and ~50% of foetal abnormalities - duodenal atresia is easily visualised as a 'double bubble' in the foetal abdomen whereas in oesophageal atresia the stomach bubble is conspicuously absent. Gastrochisis and anencephaly are also quite easy to visualise on US/S - these last three can be confirmed by a maternal serum alpha-FP and the last by x-ray.

Because of the risk of pre-term labour and cord prolapse the mother should have her cervical score checked fortnightly at the clinic, and be admitted if it is less than 1, or at 36/40.

Severely symptomatic polyhydramnios can be managed with Indomethacin 25mg 6 hly or repeated amniocentesis. The foetal lie should be checked and corrected daily on the ward and a controlled rupture of membranes carried out at 5-6cm when labour ensues.

Examine the child carefully and always pass an n.g. tube to check that the oesophagus is patent.

Further Reading

INTRAUTERINE DEATH

- Check Hb; glucose, WR, HIV
- Check INR and WBC weekly, although coagulopathy is very rare.
- Give erythromycin 500 mg 6 hly for 10 days.

Induce if the patient requests this, except in the case of grandmultiparas and previous C/Ss, where waiting for spontaneous labour is much safer. If coagulopathy develops, it can be reversed with Heparin, and the patient induced.

DIABETES

Defined as a fasting blood glucose >8 mmol/l on two occasions. In cases of 'impaired glucose tolerance' (>6 but <8 mmol/l) do a sugar screen (mini-GIT). Give the unfasted patient 50g glucose orally and take a blood glucose 1 hour afterwards. A glucose >8 mmol/l confirms the diagnosis.

All diabetic mothers should be admitted either at their first ante-natal visit or other first available opportunity for assessment of control. The target is a normal blood glucose throughout the pregnancy to avert foetal hyperinsulinaemia and its consequences. This will require insulin in almost all cases, requirements for which will approximately double as pregnancy progresses. However, some patients can be satisfactorily managed on oral hypoglycaemics in the first trimester.

- Do blood glucose profiles 2-3 week viz. 06h00 (fasting), one hour after lunch and supper and 22h00
- Use Actrapid and Monotard initially at 0.6-0.8u/kg/day (2/3 a.m. 1/3 p.m.) and adjust the dose to achieve a blood glucose <7 mmol/l at all times. Some mothers with mild diabetes can be managed on glithecpenamide initially but will require insulin after 36/40.

See at high-risk clinic fortnightly and induce at 38/40 as there is an increasing risk at birth and dystocia at term. Omit the morning insulin on the day of induction and start at 5% dextrose infusion at 40 drops/min - add about 1/3 of the daily insulin dosage as Actrapid alone to the bag and check blood glucose 2 hly - adjust the dose of insulin accordingly to the
OBSTETRICS AND GYNAECOLOGY

results. Post-delivery stop all insulin as requirements fall rapidly post-partum. Insulin is usually not required again for some days.

Be alert to the possibility of hypoglycaemia in the foetus - and paradoxically to respiratory distress which may occur despite the evident macrosomia (hyperinsulinaemia may delay lung maturation).

Further Reading
3) Casson IF 1997 'Outcomes of pregnancy in insulin dependent diabetic women; results of a five year population cohort study' B M J 315:275

BAD OBSTETRIC HISTORY

For previous stillbirth, early neonatal death or mid-trimester abortion where there is no evident cause, check Hb, WR, Htv, Glucose. Assess the cervix. All patients

- Folate 5 mg daily, vitamin C 100 mg, Slow Mg 2+ daily
- Erythromycin 500 mg 6 hrly for 14 days and Metronidazole 2g stat
- Condoms

Shirodkar suture may be inserted at Ngwelezane for cases of cervical incompetence.

LABOUR

ASSESSMENT OF THE WOMAN IN LABOUR

Review the antenatal and inpatient records before looking at the partogram. Take account of the antenatal risk factors and confirm that the partogram is correctly filled in.

Consider the mother's general state (hydration, hunger, mental state) and examine the abdomen before proceeding to P.V. In general if you are uncertain about your findings accept the more conservative estimate of progress and ask a more experienced colleague for help. Establish the lie, presentation and station of the head (or other presenting part) in fifth palpable per abdomen.

On p.v. note the degree of cervical effacement (very important in early labour), application and dilatation in cm. Feel the head for position, moulding at the sagittal and lambdoid sutures (add together for a score out of 5) and caput. If you can't feel the moulding at the lambdoid suture you must at least double that at the sagittal. If you can't feel any sutures at all due to caput assume a score of 6.

Make some assessment of the pelvis - the diagonal conjugate approximates the A-P diameter of the inlet + 1.5 cm. This should be ~ 11.5 cm in most normal Zulu women. If you can easily touch the sacral promontory suspect a small pelvis (most doctor's fingers are less than 1 cm long). The intertuberosus diameter should accept four knuckles and the subpubic angle three fingers (~90°) - otherwise suspect a small outlet. Usually however a large inlet means an adequate outlet.

ASSESSING THE DESCENT OF THE HEAD IN FIFTHS

Confirm foetal well being. Always listen to the foetal heart with the Pinard stethoscope - we also practice selective monitoring of high-risk foetuses antenatally and in labour with CTG but remember that this has low specificity. The normal F.H.R. is 120-160/min (can be as low as 110/min at term) with baseline variability of 10-25 b.p.m. Worrying traces are a falling baseline and prolonged Type 2 or Variable decelerations with poor baseline variability, tachy/bradycardia.

The presence of moderate/heavy meconium-staining of the liquor is consistently associated with an adverse perinatal outcome. In Zulu primigravidae, even light staining in early labour is associated with invariable CPD in ~ 50% of cases. It is our policy to rupture the membranes at an early stage in high-risk pregnancies to view the liquor. If no liquor is seen, push the presenting part up a little, you may obtain a rush of thick meconium which has been dammed up behind a tight head.

Further Reading
1) Lentock CE and Xweta BE 1995 'The partograph in community obstetrics' Tropical Doctor 25:56
3) Krogman MH and Malakong FE 1979 'The significance of meconium staining of liquor amnii during labour' S.A.M.J. 56:1069
FAILURE TO PROGRESS IN LABOUR

Failure to progress in labour is defined in the diagram. The causes differ according to the stage at which delay occurs, the position of the foetus and parity of the mother.

- **Delay in the latent phase**
  Ensure that the mother is in established labour (i.e. that there has been a documented change in the state of the cervix). If so, rupture the membranes. If there is meconium staining the cause is either CPD or a sick foetus and CS will usually be required. If the liquor is clear in a primigravida then the likely cause is primary hypotonic inertia and she should be given a trial of Syntocinon. Cervical dystocia is another rare cause.

- **Delay in the active phase**
  Be very suspicious in a multipara as the cause will usually be CPD. About 2/3 of foetuses initially in the occipito-posterior position will subsequently rotate 135° to the anterior and deliver and in some cases analgesia +/- Syntocinon (to overcome discoordinate contractions) may encourage this as long as there are no signs of CPD. Constriction ring is diagnosed when a space can be felt between the brim and the head during a contraction and a 'second cervix' can be felt p.v. ~ 5 cm above the real one. Primigravidae almost always warrant a trial of Syntocinon for hypotonic inertia as long as there are no signs of C.P.D.
  
  Be sure to examine the mother 2 hourly to 8 cm and hourly thereafter. No mother should be allowed more than thirty minutes in 2nd stage before steps are taken to expedite the delivery.

GUIDE TO MODE OF DELIVERY WITH CEPHALIC PRESENTATION AND LIVE FOETUS

<table>
<thead>
<tr>
<th>Vertex</th>
<th>Station of head</th>
<th>Moulding Score</th>
<th>Foetal Distress</th>
<th>Mode of delivery</th>
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<td></td>
<td></td>
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<td>CS</td>
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(Descent CS)
INDUCTION AND AUGMENTATION OF LABOUR

Never attempt to induce labour with Pgs or Synasilmin in grante multiparae in the presence of a uterine scar or where there is a high risk of C.P.D. In all cases confirm that the foetus is cephalic and has a satisfactory CTG prior to induction. All mothers undergoing induction/augmentation should be observed half hourly for at least two hours.

INDUCTION

If the cervix is <3cm dilated and >1cm long it must be ripened with vaginal Prostaglandin tablets. These should be placed in the posterior fornix next to the cervix. Patients must then lie on their side for 1 hour.
- P2/P3 - 50µg (1/4 tablets) once daily
- P2/P3 - 1mg (2 tablets) 8 hourly
- P4 - 0.5mg 12 hourly
Repeat CTG 1-2 hours after insertion.

Once the cervix is 3cm dilated and well-effaced, Amniotomy + 200ml Oxytocin infusion 10-60 drops/min should be started.

Induction of labour in P5 and upwards may present problems - use ntil methods are nspitation and "sweep and stretch". Balloon catheter induction may be used if these methods fail.

AUGMENTATION

Indicated for primary hypotonic inertia of the uterus, almost always in primigravidae. This may be manifest as a prolonged latent or active phase. Be suspicious of the multipara whose has delay in the active phase - she usually has secondary inertia which always has an associated cause (C.P.D. / Malspresentation etc.). The presentation must be cephalic and there must be no signs of C.P.D. or foetal distress (esp. meconium stained liquor). The aim is to achieve 3 contractions >40 s every 10 minutes.
- P0 Synasilmin 2u/m starting at 10 drops/min, increasing every 30 minutes to max 60 drops/min.
- Change to 10 u/m if satisfactory contractions not achieved, increasing in the same way.
- Abandon augmentation after six hours if no progress or sooner if signs of C.P.D. develop.
- P1-4 Synasilmin 2u/m ONLY increased from 10-60 drops/min as required.

If signs of hyperstimulation (4+ contractions in 10 minutes) and/or foetal distress develop - remove Pgs from vagina/switch off synto, put the mother in left lateral position, administer oxygen and give heparin/mamine 5-10 mg slowly i.v. If foetal distress persists CI/S will be necessary if vaginal delivery is not imminent.

Further Reading
Meredith DC et al 1995 "Induction of labour with intravaginal misoprostol in the second and third trimesters of pregnancy" S.M.J 85(10) 1068

OTHER INTRAPARTUM PROBLEMS

BREECH

We attempt ECV at term for all breeches. Vaginal delivery for all singleton breeches results in complications in at least 1 in 200 deliveries in the best hands in Europe - we do not use outlet forceps and have little experience of breech extraction, so our policy is to do C/S for all singleton breeches, preferably electively.

However, you may be called upon to deliver a failcompli breech in labour or one of twins, or the uncommon case of a multip with a well-fried pelvis having an elective vaginal delivery. Progress must be perfect and augmentation is never appropriate. Familiarise yourself with the mechanism of breech delivery, especially Loth's and Mauriceau-Planteau-Leveaux.

TWINS

The big cause of perinatal mortality with twins is premature labour. Even term twins are smaller than singletons, and trial of a labour is appropriate even with a scarred uterus, if the first twin is cephalic (65% of cases). Breeches are also usually safely delivered vaginally. Elective C/S should only be planned for twins where the first is transverse or other risk factors dictate otherwise.

Synasilmin may be used for the same indications as a singleton delivery, although there can be problems monitoring the second twin. After delivery of the first twin, clamp and cut the cord as usual and try to establish the head presentation of the second twin. If it is oblique or transverse correct to vertex or breech by external version, but keep the membranes intact. At this stage it is helpful to have 20u Synasilmin running, as the contractions may take a short time to return. Once the presenting part has descended into the pelvis (and this can take up to 60 minutes, so don't hurry) rupture the membranes. In the case of a buttock breech or a transverse lie that cannot be corrected by external version, grasp one foot firmly.
through the membranes and pull down, rupturing the membranes as you do so (internal version). The foetus can then be delivered by breech extraction.

If you fail with internal version, or if a mother is admitted from clinic with a retained second transverse twin and ruptured membranes, you will have to do a C/S, since the risk of rupturing the uterus with further attempts is high. However, try to avoid doing a C/S for a retained second twin in other circumstances, especially if the foetus is dead. C/S with a second twin is rare and it may be possible to do a V.E. or craniotomy.

CORD PROLAPSE

If the cord is still pulsating when this is detected and management is correct, a live baby should result. Deliver vaginally if the cervix is 9+ cm and the head well-down with vacuum if necessary. Otherwise prepare for emergency C/S -
- Push the head up and hold it away from the cord
- Give Hexoprenaline 10 mg i.v. slowly
- Insert a catheter, fill the bladder with 500 ml saline and clamp it.
- Take the mother to theatre with a hand in the vagina at all times displacing the head.
- At C/S drain the bladder at the last minute and remove the hand from the vagina as the uterus is opened.
- Resuscitate the baby as necessary.

SHOULDER DYSTOCIA

Usually heralded by a head that is slow to deliver and buries itself in the perineum, often after a rather tight V.E. Use McRobert’s Maneuver - get your assistants to apply extreme flexion to the mother’s hips so that her knees almost touch her shoulders and apply gentle traction on the head +/- suprapubic pressure from above. This is almost always successful. If you still cannot deliver the shoulders, place a finger in the posterior axilla and pull downwards. Push the elbow forwards and deliver the posterior arm. Traction on the head will then deliver the anterior shoulder. Always check the baby afterwards for brachial plexus injury.

RETAINED PLACENTA

A retained placenta is one that has not delivered one hour after delivery of the baby. However, if Syntocinon has been given it should be expected within 15 minutes. If it is lying in the cervix or the vagina, simply grasp and remove it. If it cannot be felt at all, removal in theatre under G.A. will be required – run a Syntocinon infusion of 20 u/h, resuscitate vigorously and inject 10 u of Syntocinon proximal to the clamp to encourage separation. Always check that you have removed all the cotyledons. Do not persist if the placenta is very adherent and difficult to remove - transfer early as hysterectomy is sometimes required.

POST-PARTUM HAEMORRHAGE

Prevent and be prepared for PPHs - especially grandmultiparae, twins, APHs etc. Act quickly as PPH can be brisk and life-threatening.
- Start an infusion of Syntocinon 20 u/h
- Resuscitate aggressively, with blood as soon as it is available.

If the bleeding does not stop promptly, explore the genital tract immediately under saddle block in theatre. Have good light, at least one assistant and two Sims’ speculums handy. Exclude:
- retained products (remove manually or with blunt curettes/forceps)
- atonic uterus or rupture (pack the uterus thoroughly with bandages and apply bimanual compression - refer for? hysterectomy in the latter case)
- lower tract lacerations (suture)
- coagulopathy (give FDPs)

THE HIV + VE MOTHER

Approximately 40% of the fertile female population in Hlabisa are HIV +ve. Strenuous efforts should be made to avoid unwanted pregnancies amongst these women. Perinatal complications which may be associated with HIV include low birthweight, IUGR, prematurity, puerperal sepsis and possibly increased fetal loss, though we haven’t yet seen a significant rise in the perinatal mortality rate in our district. The risk of transmission to the child is ~30% in our setting, about half which is related to breastfeeding.

PREVENTION OF VERTICAL TRANSMISSION

- Short course antiretroviral regimes are demonstrated to reduce the rate of transmission by ~40%, but neither zidovudine nor nevirapine are currently licensed in South Africa for this purpose.
- Multivitamin supplementation during pregnancy reduces the risk of adverse perinatal outcome. Vitamin A alone is not associated with any benefit and neither is capable of actually reducing transmission.
- For mothers in whom membranes are ruptured > 4 hours prior to delivery, cleansing of the birth canal
with 0.25% chlorhexidine 4 hourly until delivery may be effective at reducing transmission.

- Caesarian delivery has a relative risk of 0.46 for transmission in local studies, but we do not offer it for this indication at present because of the high rate of post-operative morbidity in the mother.
- Avoid amniocentesis, routine AROM in early labour, and routine episiotomy.
- Prophylactic antibiotics should be considered for all caesarean sections, in pre-labour rupture of membranes, and all clinical AIDS patients in labour.

**BREAST-FEEDING**

- Mothers should in general be encouraged to continue breast-feeding until at least six months since infants can rarely be weaned sooner and the risks of malnutrition and respiratory/gastrointestinal infections are greatly reduced in this period. If breast-feeding is prolonged, the risk of transmission probably outweigh the benefits conferred. Some mothers who have access to clean water and a sound understanding of the safe preparation of formula are capable of using this right from the start, but each case needs to be considered on its own merits.

Further Reading

2. De Cock K et al. 2000 'Prevention of Mother-to-Child HIV Transmission in developing countries: translating research into policy and practice' *JAMA* 283:1175
3. Fawzi WW et al. 1998 'Randomised trial of four vitamin supplements on pregnancy outcomes and T cell counts in HIV-1 infected women in Tanzania' *Lancet* 351:1477

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**ABORTION**

This is the commonest reason for admission to our gynaecological ward (~ 40% of admissions in 1996). It is difficult to ascertain what proportion of 'spontaneous' abortions in our district are in fact illegally induced but a multi-centre survey of hospitals in RSA concluded that at least 7.5% were. It was estimated that ~ 30% of incomplete abortions in black women showed evidence of infection/complications.

Until November 1995 when the *Abortion and Sterilisation Act* of 1975 was repealed, therapeutic abortion could be performed on very limited indications in South Africa. The new *Choice on Termination of Pregnancy Act* of 1996 provides for Termination on request up to 12/40 and from 13/40 to 20/40 if a medical practitioner is of the opinion that the continued pregnancy would 'significantly alter the social or economic circumstances of the woman' or 'pose a risk of injury to the woman's physical and mental health'. Implementation of the Act in our province has been extremely patchy and we do not at the present time perform abortion on demand in our hospital but patients can be referred to Empangeni Hospital for the procedure.

Ectopic pregnancy is the commonest indication for emergency laparotomy in our hospital - this is almost certainly due to the high prevalence of pelvic infection in our district, which is estimated to increase the risk of ectopic pregnancy 7-10x.

All incomplete abortions should be Amp Gent & Flagent IV and have Hb, WR and rhesus requested on admission. We still perform all our E.R.P.C.s in theatre under ketamine as this is the safest environment in our hospital.

**LOW ABDOMINAL PAIN AND/OR BLEEDING**

WITH POSITIVE PREGNANCY TEST

- Os open
- Os closed - Do US/S

**INCOMPLETE ABORTION**
- 4th E.P.C
- Anx test

**INEVITABLE ABORTION**
- Threatened abortion
- Medical abortion
- Medical abortion
- Medical abortion

**MISSED ABORTION**
- Ectopic pregnancy
- Medical abortion
- Medical abortion

**EMERGENCY LAPAROTOMY**
- Medical abortion

In all cases of abortion check the Rhesus status of the mother - if negative give 50 μg of Anti-D.
ECTOPIC PREGNANCY

A positive pregnancy test with suspicious findings on bimanual palpation + tenderness and cervical excitation should always prompt a pelvic US/S. A normal/slightly enlarged uterus with no evident gestation sac implies ectopic pregnancy even if no mass is visible in the adnexae (may be absent in ~ 60% initially even with skilled operators). Conversely a definite intrauterine gestation sac co-exists with an ectopic pregnancy in only ~ 1 in 30,000 cases. A confusing 'decidual cast' with small loculated areas in the uterus may sometimes be present in ectopic pregnancy but is usually easy to distinguish from a true sac. In the presence of these findings it is usually not necessary to confirm a haemoperitoneum by culdocentesis or peritoneal aspiration though this should recover unclotted blood in 85+%. It is possible to refer difficult cases for laparoscopy at Nqwelestone but we have only done one unnecessary laparotomy in the last four years using B-HCG and US/S findings alone.

Diagnosis

Do a general examination with attention to oestrogenisation (secondary sexual characteristics), virilisation, nutrition and thyroid status. Look for signs of Turner’s syndrome. Check visual fields and express for galactorrhea. Do a pelvic examination to rule out any major structural abnormalities.

Check Pregnancy test

Pelvic US/S should be done to assess the size of the uterus (small if poorly oestrogenized) and if possible the ovaries - in 70% of patients with P.C.O.S. the ovaries are increased in size (upper limit of normal ~ 2x2x4cm) and may contain multiple 6-8 mm cysts arranged peripherally. Ovarian tumours should also be evident - cysts that are complex and/or >6 cm in diameter should be referred early for exploration.

Order a screening hormonal profile (FSH, LH and oestradiol) in all patients who have normal p.v.s and no galactorrhea. If the patient is concerned about her fertility, also arrange a provera test at the first visit. Give 5 mg i.d.s. for 5 days. A withdraw bleed signifies a well-oestrogenised endometrium and probable supra-ovarian cause.

In patients with frank galactorrhea, order prolactin only. In very wasted patients be sure to exclude other systemic illness, especially H.I.V and hyperthyroidism.

Management

Post-depot - expectant management or try two or three cycles of COCP

Abnormal p.v. - imperforate hymen and haematocolpos can be managed by simple incision but other abnormalities need specialist surgical repair.

LH and FSH high, clinical Turner’s => Premature ovarian failure. Start Prempro-N

LH and FSH low => Hypogonadotrophic hypogonadism. Refer Endocrine clinic for combined pituitary function tests.

LH high, FSH and Oestradiol low + Large, cystic ovaries => P.C.O.S., Refer Endocrine clinic.

Normal gonadotrophins, High Prolactin => Probable Prolactinoma, Refer Endocrine clinic for C.T.

Further Reading
2) Crouseman EC. 1996 A practical guide to the diagnosis and management of amenorrhoea Drugs 52: 671

AMENORRHOEA

The vast majority of our cases are secondary and the clinical approach to amenorrhoea is essentially the same for both 1st and 2nd forms. Premature ovarian failure, polycystic ovary disease and hypogonadotrophic hypogonadism account for 70+% of cases in both groups.

1st amenorrhoea is defined as no periods at age 17 or no breast development /other secondary sexual characteristics at age 14. Uterine agenesis seems to have been the commonest structural abnormality in the primary group.

Lactational amenorrhoea, pregnancy and depot contraceptive use are probably the commonest causes of secondary amenorrhoea in our practice. Prolonged breast-feeding can prevent menstruation for up to 15 months post-partum. The mean duration to return of fertility post-depot is ~ 6 months, and 80% will have conceived by one year.
INFERTILITY

Most Zulu women have a child before marriage, often by a man they do not marry subsequently. That child will usually be left with the maternal grandmother when the woman does marry. Most Zulu men will have fathered children by other women before they marry. A married couple will only rarely have children from a previous union in their own kraal. It is not uncommon to see women with children from different pre-marital partners who present for investigation of secondary infertility (~65% of cases). Presentation may also be indirect with various non-specific lower abdominal complaints: direct questioning elicits the reason for the consultation.

It is vital to realise that the pressure on women to produce a child in their marriages is huge, and the possibility that failure to do so may be the fault of the husband is rarely considered. 11% of couples presenting to Ngwelezane Infertility Clinic report that the husband had had no previous children and in 90% of these cases he was found to be oligo/azoospermic.

30% of women presenting for investigation at Ngwelezane show evidence of tubal damage on HSG and ~40% are found to be anovulatory (some women have both problems). Only ~2% of women undergoing treatment at the Infertility Clinic ultimately become pregnant.

FIRST VISIT

1. Take a careful history enquiring about previous children (on both sides), menstrual history and contraceptive use, marital status and duration of infertility. Many newly weds are found to spend much time apart due to the husband’s migratory labour. Sensitive advice is then appropriate about the fertile period and no investigation is necessary for a further year.
2. Gynaecological examination to exclude an immediately obvious cause of infertility. Start with a PAP smear before doing a PV.

If you suspect a pelvic infection treat it, and if you suspect a cervical malignancy, biopsy it.

If she has a fibroid uterus clinically/on US/S, refer to Ngwelezane with a view to myomectomy.

If history and examination are unremarkable further investigation may be indicated but four criteria must be met first.

1. The woman must be under 38 years.
2. There must be no living children from the marriage.
3. Both partners should be HIV negative.
4. The man must have normal semen analysis. This is done here in our lab on a fresh (2 hour) specimen produced 2 days after last intercourse.

(Normal volume 1-4ml. Sperm concentration >20 x 10^6 per ml. Total sperm ejaculate >50 x 10^6. Motility >50%. Liquefaction complete in twenty minutes. Leucocytes <5 x 10^6 per ml.)

Abnormality is only accepted after three specimens. Treating any infections can improve sperm function. Male infertility is very hard to treat but consider referral to Urology to confirm the diagnosis.

No further measures are taken until these criteria are met; if the man will not come forward for investigation we should not investigate the woman further.

Calculate the date of the twenty first day of the woman’s next menstrual cycle. Ask couple to return on this day.

SECOND VISIT

1. Review HIV and PAP smear results with the patients and counsel accordingly. If either partner is HIV positive further investigations are inappropriate.
2. Take bloods for: WR/FSH/LH/Prolactin/Day 21 progesterone + TFIs (the latter if clinically indicated).

THIRD VISIT

1. Review blood results. If results show normal gonadotrophins with an anovulatory progesterone, treat with an ovulatory stimulant (Tamoxifen 10mg od on day 2-6 of the menstrual cycle).
2. Refer to Endocrinology if hyperprolactinaemia or LH:FSH = >3:1 (probable polycystic ovary disease).
3. Refer to Ngwelezane for: Hysterosalpingogram if blood tests are normal.

Further Reading:
1) Van Zyl A 1980 ‘The infertile couple’ Parts I & II. S.A.M. J 57:446 and 485
2) Blumenthal NJ 1984 ‘Hysterosalpingography in the assessment of infertility in black patients’ S.A.M. J 85:654
4) Chimpembe Pi J et al. 1998 ‘Infertility profile at King Edward VIII Hospital, Durban, South Africa’ Trop. Doc. 28:160
PELVIC INFLAMMATORY DISEASE

This is a common cause of infertility and chronic pelvic pain in our setting. ~30% of cases are caused by C. trachomatis infection, which correlates strongly with subsequent infertility. The more severe the presentation the more often N. gonorrhoeae is isolated (60% of severe cases). This organism is now always penicillin-resistant and ~10% of isolates are also tetracycline resistant. Many other vaginal organisms may also contribute to ascending infection. The presentation may be toxic in HIV +ve women and quickly result in peritonitis.

The I.D.S.O.G. criteria are widely used to guide diagnosis. Acute P.I.D. is diagnosed in the presence of:
- Lower abdominal + cervical excitation + adnexal tenderness plus one or more of the following
- Fever >38°C
- WBC >10,000
- Pus on culdocentesis
- Inflammatory mass on bimanual palpation or US/S
- +ve gram stain for gonococci or antigen test for chlamydia or >10 leucocytes/field on cervical swab

In practice, the prevalence of chlamydial infection and other STDs in young women in Hlabisa is so high that even when these criteria are not met, you will rarely be wrong to treat for P.I.D. on suspicion. Remember that acute P.I.D. is uncommon if there has been no intercourse for 6+ months or if the patient is pregnant. The important differentials are ectopic pregnancy and ovarian cyst complications. Endometriosis and appendicitis are both uncommon in our patients. Therefore, always do a pregnancy test! Vomiting is rare even in severe P.I.D. and its presence should suggest another diagnosis. Intravenous tetracycline, previously the mainstay of therapy, has now been withdrawn and various antibiotic combinations are preferred. All are equally effective provided they have adequate anaerobic cover.

Management

MILD CASES
- Ciprofloxacin 500 mg stat p.o.
- Doxycycline 100 mg daily b.d. for 1 week
- Metronidazole 400 mg i.d.s. for 1 week

SEVERE CASES (defined as Fever >38°C, Pulse >100, peritonitis or tender adnexal mass)
- Ampicillin 1g 6-hourly i.v.
- Metronidazole 1g b.d. p.o.
- Gentamicin 240 mg daily i.v.
- Doxycycline 200 mg daily p.o. and Ciprofloxacin 250 mg stat p.o. when can take oral.
- Continue i.v. antibiotics until afebrile for 48 hours and abdomen soft. Continue doxycycline for 10 days
- For tubo-ovarian abscesses antibiotics should be continued for 14 days.
- Surgery is sometimes required for patients failing conservative management:
  - Generalised peritonitis failing to improve within 24 hours on i.v. abs
  - Tubo-ovarian abscess >6 cm which enlarges or fails to resolve after two weeks
  - Pouch of Douglas abscess
Posterior colpotomy or US/S guided percutaneous drainage may be possible for the two latter but laparotomy is the only option if a tubo-ovarian abscess has ruptured and is probably the easiest way to definitively drain a Pouch of Douglas abscess.

In all cases, ensure contact tracing and promote condom use where possible.

Further Reading
1) Hager WD et al. 1983 'Criteria for the diagnosis and grading of salpingitis' Obstet Gynecol 63(1):113
HPV INFECTION AND CARCINOMA OF THE CERVIX

HPV causes genital warts (condylomata acuminata) and is a cause of cervical and vaginal carcinoma. The lifetime risk of Ca cervix for a black South African female is currently estimated at ~1 in 20 and serological surveys in women from our district have demonstrated that serotypes 15 and 18 which are responsible for malignant transformation are widespread. More than 10% of women of reproductive age have dyskaryotic smears on screening. Cervical screening is currently a controversial topic in South Africa - we do not yet have the structures to implement any such comprehensive screening programme in our district, since coverage of 70% of over 25s would be required, and we are already struggling to implement effective primary healthcare in other areas. Even in an intensive pilot study in our district we were unable to get results to ~50% of those screened.

CHILDREN

It should be taken as a sign of sexual abuse. Histology and EUA is essential. Treatment is by electro-cautery and podophyllin or beta-adenept. These children need regular cervical smears from puberty.

WOMEN OF REPRODUCTIVE AGE

Appearance varies from the obvious warty growths to flat lesions. It may be difficult to differentiate them from condylomata lata (which disappear after a dose of benzathine penicillin), granuloma inguinale, and carcinoma so typical lesions should always be biopsied.

The preferred treatment for women is electro-cautery. This cannot be used on the penis however. Podophyllin 0.25% solution is a less effective alternative and should only be applied under supervision in 0.P.D./on ward. No more than 0.5ml should be used at a single application and it should not be left on for longer than 8 hours. Applications can be continued twice a week to a maximum of eight. Difficult, chronic and confluent lesions may need referral for vulvectomy or circumcision.

PREGNANT WOMEN

Often large secondary infected growths are seen. Treatment is with the secondary infection (Flagyl 2g stat + Erythromycin 500mg ads for five days) and review after one month. Never use Podophyllin in pregnancy! Most regress postnatally and unless they are a real mechanical problem are not a contra-indication to NVD (risk of juvenile laryngeal papillomatosis in the neonate is <1 in 1500 deliveries).

POST MENOPAUSAL WOMEN

Must always be biopsied to exclude malignancy.

'Burning Vulva Syndrome' is secondary to HPV infection and is treated by topical steroids or premarin cream - in difficult cases laser ablation of the area can be carried out.

C.I.N. AND CARCINOMA OF THE CERVIX

Any clinically abnormal cervix must be biopsied unless obvious cervicitis on speculum. (Take 2-3 biopsies from square-columnar junctions at different sites.) The only important differential of a hard, deformed cervix with contact bleeding is a biliardinoma which may respond to protriandazole and is uncommon.

- CIN 1 - treat for PIP and repeat in 3-6 months
- CIN 2/3 - refer colposcopy clinic Empangeni hospital
- Confirmed Ca - in all cases do Hb, U&Es, LFTs, CXR and renal US/S for staging and refer with histology report to Room 6 KEH VII (Gynaec Oncology Clinic) for assessment. Do not send cases with renal tract involvement or evident distant metastases.

Further Reading
1) Heyns FC et al, 1995 'Screening for cervical neoplasia in KwaZulu - lessons from an unscreened population' S.A.M.J. 85: 1169
2) McCoy & Baron D 1995 'Cytopathological screening for cervical cancer - what are the opportunity costs?' (Carnival) S.A.M.J. 86: 535

VAGINAL BLEEDING

Pelvic inflammatory disease and fibroids are the common causes of abnormal vaginal bleeding outside pregnancy in women of reproductive age in our practice. Endometrial hyperplasia and polyposis are also quite common but true endometritis is rare. Incidence rates for carcinoma of the cervix in black women in South Africa are among the highest in the world (43/year per 10,000, 40% of cancer deaths in black women). *Endometrial...
carcinoma is much less common, probably due to the high parity of local women and comprises <5% of cancer deaths among black women in RSA. Malignancy is responsible in ~50% of women presenting with post-menopausal bleeding. In all cases it is essential to exclude malignancy clinically/histologically before starting treatment. Once this has been done the basic principle of management is to correct a deficiency of progesterone in the second half of the cycle. Diagnostic D&C can be avoided in many cases with a little consideration of the likely underlying pathology - a survey of endometrial curettings at our hospital during 1995-1996 showed 60% to be normal/unrepresentative, 20% hyperplastic + polyps and the other 20% equally divided between malignancy, endometritis and evidence of recent pregnancy.

ABNORMAL VAGINAL BLEEDING

**CYCLICAL**

Menorrhagia

- Menorrhagia/inter-menstrual bleeding

- Always do a pregnancy test and rule out recent/diaper contraceptive use. Mid-cycle bleeding/ menstruation in young women is established.

- Mild menorrhagia in women in their 50s may bea result of temporary/intermediate bleeding.

- Menorrhagia is best managed with a combination of: Progesterone, Missed pill, IUCD and surgery.

- In all cases, check Hb and prescribe haematinics where appropriate. Transfusion may rarely be required in the acute stages.

- 3/4 of women on Depo-Provera will experience some irregular bleeding. Very heavy bleeding is due to a very atrophic endometrium which has ulcerated and is best managed with intramuscular Conjugated Oestrogens 25 mg repeated once after 6 hours if necessary, and followed by Ethinyl Estradiol 50 mg b.d. for 10 days. In most other cases the endometrium is proliferative - for acute bleeding that is so severe that it is resulting in significant anaemia or distress, Mirena 1-10 mg b.d.s. for 14 days may be prescribed.

- If bleeding continues after a recent abortion suspect hydatidiform mole. This probably occurs in ~ 1 in 2000 pregnancies in Zululand. The uterus may remain enlarged and grape-like vesicles may be passed. The diagnosis is confirmed by a 'snowstorm' appearance on US/S. All patients should be referred for evacuation (younger women) or hysterectomy (over 40 years). ~20% of women undergoing evacuation will develop invasive disease but as long as it is detected early a cure rate from chemotherapy of close to 100% can be expected. So good follow-up is essential. Urine β-HCG should be checked fortnightly and should be negative at 3/12. Serial dilutions of the urine can be carried out to get a crude titre during this time. Continue until the β-HCG has been negative for 3 consecutive fortnights. All that check the urine every 3 months for 2 years.

**NON-CYCLICAL**

- Always try to get a fairly detailed menstrual history - it is possible with a little patience and is crucial to deciding on management. Specifically ask about the possibility of pregnancy and contraceptive use. Carry out a full gynaecological examination.

- Management is determined by the age of the patient and nature of the bleeding.

Further Reading:
1) Bayer S and O'Connor A.P. 1993 'Clinical manifestations and therapy of dysfunctional uterine bleeding' JAMA 269:1823
2) Hall et al. 1982 'Control of menstruation by the cycle-oxygenase inhibitors naproxen sodium and mefenamic acid' Br J Obstet Gyna 94:555
CONTRACEPTION

Contraception is a key primary care service. RSA currently has a population growth rate of close to 4% / year. The average age of sexual debut is ~14 years - in surveys of rural schoolgirls less than 30% were using contraception. ~10% of deliveries in our district are of mothers under 17 years and we currently receive about two requests for termination of pregnancy per week. Uptake of family planning services is low in Hlabisa. Many factors contribute - misunderstanding concerning contraception, lack of an independent choice for many women and failure to integrate family planning into many hospital / clinic consultations.

Abortion is now legal in South Africa (since 1997) and the laws governing its use are some of the most liberal in the world. Work is ongoing to try to ensure that this option is available to rural women as well as urban women. However, acceptable and available family planning methods should help to give women more power to choose when to conceived in the first place.

FAMILY PLANNING FOR WOMEN

INJECTABLE CONTRACEPTIVES

We routinely use Medroxyprogesterone acetate ("Depo-Provera") or Noristerone Enanthate ("Noristeral") which are both extremely effective contraceptives. They have higher efficacy than the combined pill if given at the correct intervals, and the need to remember a daily tablet is removed. Depo-Provera is given every 12 weeks and Noristeral every 8 weeks. Both are suitable for most women but are contra-indicated with a history of past severe arterial disease or steroid-associated jaundice, current abnormal genital tract bleeding or current pregnancy. It should be given on day 1-5 of the period or within 6 weeks post-partum. It is safe immediately post-partum but a 4 to 6 week delay is recommended to avoid torrential post-partum bleeding. Protection is immediate. With depo, give the patient a date to return in 11-13 weeks and explain that if she doesn't return, she may become pregnant.

Benefits:
1. Highly effective contraceptives.
2. Do not affect breast milk volume (unlike the combined pill).
3. Reduce menorrhagia (and thereby anaemia) and dysmenorrhoea in most women.
4. Reduce incidence of PID.
5. Invisible once given and under the woman's control.

Side-Effects / Drawbacks:
1. Irregular bleeding is common on injectables and although this is usually light spotting it can be clinically significant. Patients need to be warned in advance of the possibility of irregular bleeding.
2. Heavy pv bleeding can be treated with Ethinylestradiol 50mcg daily for 21 days. (ie Ovran). 50% of women stop menstruating completely after 1 year on depo and this also needs to be carefully explained.
3. Slow return to fertility (average is 8 months after the last injection).
4. Possible loss of bone density with long-term use (therefore less suitable when nearing the menopause).

ORAL CONTRACEPTIVES

1) Combined Oestrogen / Progestogen Pills

These are excellent contraceptives (99% effective if taken as per protocol). They should be started on day 1-5 of menstruation (officially need barrier contraception for 7 days if started after day 2). All our pills contain seven dummy tablets so in all cases one pill should be swallowed at the same time each day (not one each time the woman has intercourse!). A withdrawal bleed will normally be seen during the week of dummy tablets. Protection is immediate if COC is started with menstruation.

Missed pills:
- If pill is taken within 12 hours of the normal time, no action is required.
- If a pill is missed completely it should be taken as soon as possible and the rest of the packet continued at the normal times. A simple rule after one or more missed pills is to recommend barrier contraception for 7 days after the omission.

Additional protection (eg barriers) are required if the patient has vomiting or diarrhoea, or if she is on antibiotics. Always give three packets of COC at a time and see the woman in 12 weeks to monitor satisfaction and understanding of the method. 6 monthly prescriptions thereafter.

While on rifampicin/TB treatment or anti-epileptics (enzyme inducers) double doses of COC or POP are required and depo should be given every 8 weeks.

Triphasit® or Nordette® are combined oral contraceptive pills suitable for non-lactating non-smoking women up to the menopause with no history of hyperension, thromboembolism, liver or breast disease and with a BMI of less than 40.
Benefits:
1. Highly effective and easily reversible.
2. Reduces rates of carcinoma of the endometrium and ovary. PID, fibroids, endometriosis, ectopic pregnancy, heavy periods (and thereby anaemia).

Side-effects/drawbacks:
1. Increased incidence of thromboembolism and hypertension, myocardial infarction and CVA
2. Small increased risk of breast carcinoma and liver adenoma.
3. Reduces breast milk volume and therefore not recommended while lactating.

2) Progesterone-only Pill

Microval® is a progesterone-only pill suitable for lactating mothers. One tablet is taken daily. POPs work by thickening cervical mucus and making endometrium un receptive to implantation. Unlike the COC they do not suppress ovulation.

Benefits:
1. Do not reduce breast milk.
2. No oestrogen-related side-effects e.g. thromboembolism, hypertension (therefore can be used by diabetics).

Side-effects/drawbacks:
1. MUST be taken within the same three hours every day or it is ineffective (less forgiving than COC).
2. Only 95% effective even if taken religiously.
3. May cause irregular bleeding.

TUBAL LIGATION

This is available to all women who request permanent birth control. It must be made clear to the patient that there is a failure rate of at least 1 in 500. A small operation is required under spinal anaesthesia which requires admission for 24 hours. In practice, most of our TLLs are carried out at C/S or immediately post-partum and this may be associated with a slightly higher failure rate. We use the Pomeroy method routinely.

EMERGENCY CONTRACEPTION

Must be taken as early as possible after intercourse and within 72 hours of intercourse to be effective. Give 2 Ovral tablets stat and repeat 12 hours later. The high doses of oestrogen often cause nausea and vomiting so give Meclopropramide 10 mg with each dose. If the tablets are vomited within 3 hours of consumption a replacement dose is required.

Consider levonorgestrel, 0.75mg repeated 12 hours later (this is 25 microval tablets repeated after 12 hours). This is as effective as the combined regime and does not cause nausea.

INTRA-UTERINE CONTRACEPTIVE DEVICES

Fitted as an outpatient and lasts for 4-5 years until removed. They are appropriate for women > 35 years in a stable relationship - in most other cases the risk of subsequent pelvic infection is probably too high. Women with IUCDs may experience heavier periods.

FEMALE CONDOMS

Preliminary surveys of this method in our district showed that acceptability was reasonable but uptake has been low and availability poor.

FAMILY PLANNING FOR MEN

VASECTOMY

Not very popular in our district! Can be done under local anaesthetic, but requires referral as we have little experience of the procedure.

CONDOMS

Increasingly acceptable to men in Hlabisa and widely available free in OPD and clinics. Since the relative risk of HIV acquisition in women using oral contraception in Africa is ~ 1.45, condoms should be recommended for almost all sexual contacts, even where the partner is already using an effective method.

Further Reading
1) 1983 Multinational comparative clinical trial of long-acting injectable contraceptive : medroxyprogesterone enanthate given in the dosage regiments and depot Medroxyprogesterone acetate. Final report Contraception 28:1
WOUND MANAGEMENT

A general understanding of the principles of wound management is helpful when dealing with surgical/traumatic wounds, ulcers and burns, both on the wards and in O.P.D. Humans have the capacity to regenerate their epithelia but the connective tissues (with the exception of liver) can only undergo repair. In nature therefore almost all wounds heal by second intention—an inflammatory phase is followed by proliferation of myofibroblasts and angiogenesis that form granulation tissue (the future dermis). This is then re-epithelised completely from the wound edges in 14-21 days. The scar becomes red and hard as it organises and contracts over the first 12 weeks but then gradually whitens and relaxes according to the progressive withdrawal of blood vessels, usually complete by 6 months. Mature scar tissue has ~70% of the strength of the tissues it has replaced.

Wound healing can be modified surgically by dressing, suture or skin grafting. Approximation of wounds by suture can lead to more rapid healing by ‘first intention’ and correct dressing, especially the provision of a ‘moist wound environment’ over large defects, facilitates epithelial cell growth and migration and prevents contamination/infection. Early or delayed skin grafting will often be necessary where the above cannot achieve satisfactory closure. However the timing of suture/grafting and need for prior preparation/inletion of the wound requires some careful consideration.

In all wounds ensure that there is no damage to deeper structures that might need attention and that there are no retained foreign bodies (e.g., glass).

SUTURING

Our general purpose suture materials are monofilament nylon for skin and chromic catgut for deeper tissues. The former is non-absorbable and supports less bacterial growth than braided sutures like silk or polyester, but has less forgiving knotting properties and should always be tied with surgeon’s knots. The latter can be used to close deeper tissue spaces and is fully absorbed by ~21 days but may encourage infection—in many cases good apposition can be obtained by monofilament mattress sutures alone. The timing of suture essentially depends on the degree of tissue trauma and contamination which determine the size of the bacterial inoculum in the wound at presentation. By day 3, the wound fluid is actively bactericidal and if frank infection is not established closure should be safe.

Immediate primary suture is appropriate for all clean/minimally contaminated wounds ≤8 hours old on the body and ≤24 hours old on the hands, face and scalp. However, it is almost never appropriate for:
- wounds on the foot
- crush injuries/severe lacerations with extensive tissue trauma
- bites
- heavily contaminated wounds
- tongue/lip wounds (which invariably heal well spontaneously unless severe)

If there is some doubt as to the status of a wound rather do a wound toilet and arrange for delayed primary closure at 72 hours, though in some cases lacerations presenting early it may be possible to excise the edges/toilet and close immediately. Secondary suture is usually indicated following a wound infection or very late presentation and may require undermining of tissues to achieve good approximation. Note that a wound is as likely to become infected if it is closed too late as too early (due to colonisation).

Sutures are usually removed at the following intervals:
- Face - 4 days
- Scrotum/Labia - 5 days
- Scalp, hand - 7 days
- Trunk - 10 days
- Leg - 14 days
- Abdomen - transverse 5 days, midline 7-10 days

Most sutured wounds will quickly develop a protective fibrous crust and a dry gauze dressing for the first few days is usually all that is required. A clean wound should be treated as little as possible and only changed if signs of wound infection or ‘strike-through’ by exudate occurs, providing a path to the wound for bacteria. Otherwise the dressing should be removed when the sutures are removed.

DRESSINGS

There is almost no dressing that needs to be changed more frequently than every 72 hours! Sterile saline is preferred for general purpose cleansing of wounds as it has no tissue-toxicity and does not cause hypersensitivity. Gauze is our general purpose dry dressing and is used alone for surgical wounds and with povidone-iodine ointment or cream for many other infected wounds e.g. after debridement, drainage. If left in place for too long the fibres easily become entangled in the healing tissue—such dressings should be soaked off with normal saline.
Paraffin gauze (telfa gras) is our principal non-adherent dressing. It should not be used on infected wounds. Its main use is for dressing skin grafts and clean wounds where gauze alone would easily stick esp. abrasions.

Semi-permeable membrane dressings (polyurethane film) are available principally for dressing burns - they allow such wounds to exude whilst protecting them from infection and allowing re-epithelialisation.

Occlusive hydrocolloid dressings (Sodium carboxymethylcellulose e.g. granuflex-E) are useful for ulcers esp. bed sores - they expand into the wound cavity and apply pressure to the ulcer floor whilst absorbing exudate. They may encourage auto-debridement of some sloughy wounds but should never be used in the presence of infection and are no substitute for a surgical debridement where indicated. They may be left in place for up to a week but should always be changed when they leak.

Povidone-iodine is an iodophor - it releases inorganic iodine in a more controlled way than the older, more toxic tinctures. It is used as a 7.5% surgical scrub 5% cream and a 10% ointment. It has a broad spectrum and is sporicidal. The cream has desloughing properties.

Chlorhexidine is available as a 20% surgical scrub and a 25% aqueous solution - the latter is diluted with 70% alcohol to 0.5% for hand-washing and with sterile water to 0.05% for cleansing of infected wounds. It is also useful for overgranulated wounds as it inhibits formation of granulation tissue.

Asepsin is a mixture of malic and other organic acids + propylene glycol which causes differential swelling of organic and inorganic proteins and consequently separation of adherent sloughs. Glucose powder/caster sugar may be equally effective. Again, for heavy sloughs the treatment is surgical.

Silver Nitrate sticks are used for severely overgranulating wounds.

SKIN-GRAFTING

Most defects smaller than 2x2 cm on non-critical areas will close by secondary intention. However, SSG may be required for larger defects or if located in an area where a contracture could be a problem - e.g. face, fingers etc. Grafting in this situation should be carried out early (at about 3 days).

TETANUS PROPHYLAXIS

Though immunisation coverage in our health ward is currently high, only ~70% of children receive all three doses of DTP and many older patients will never have had a primary immunisation series for tetanus at all. Therefore for any wound that is >6 hour old and/or evidently contaminated in a patient of any age who cannot produce evidence of a complete primary series and/or a booster within the last ten years, give 0.5 ml of Tetanus toxoid i.m. at presentation.

For heavily contaminated/devitalised wounds in non-immunes also give Human Tetanus Ig i.m. (250 i.u. for adults,125 i.u. 5-10 years and 75 i.u. under-5s) i/v.

Benzathine Penicillin 2.4 MU i.m. In all cases where the patient seems never to have had toxoid before arrange for a further booster at 6 weeks and again at 6 months.

Further Reading
3) Ryan TJ 1997 'Global curriculum for wound management' Tropical Doctor 27 Suppl 1: 31
5) Una H & Alberg C 1975 'Tenons strengths of twelve types of knot employed in surgery, using different suture materials' Act Chir Scand 142:1

AMPUTATIONS

With the exception of digit amputations, we only attempt classical Above and Below-knee amputations in our hospital. Most are two-stage procedures - a guillotine followed by fashioning and closure of the stump after 48-72 hours. In the face of pre-existing ascending infection, guillotine amputation as distal as possible to the site of infection followed by a below-knee revision results in a subsequent above-knee amputation 4x less often than an attempted primary B.K.A. Broad spectrum perioperative antibiotics can reduce amputation sepsis rates to ~15%. Always use a tourniquet unless you think you are operating for definite ischaemia.

After delayed primary closure of the stump a plaster shell should be applied to prevent contractures. This should be removed when the sutures come out (~10-14 days). Physiotherapy and continued bandaging of the stump to encourage a conical shape should continue until the stump is ready for limb fitting at about 6 weeks.

An amputation is usually considered for one of the four following reasons -
TRAUMA

Limb salvage may be possible even when your first impressions are highly unvarnished! About 78% of Gustilo IIIc injuries will require a secondary amputation from resulting complications but only -20% of IIIb’s.

Assess the limb according to the ‘Mangled Extremity Severity Score’ (M.E.S.S.) -

A. Skeletal/Soft tissue injury
   - Low energy (Stab, Low velocity gunshot, simple #) 1
   - Medium energy (Open/multiple #s) 2
   - High energy (High velocity gunshot, crush) 3
   - Very High energy
     (Above + gross contamination/avulsion) 4

B. Limb Ischaemia
   - Reduced pulse but variable 1
   - Pulseless, Paraesthetic 2
   - Cool, paralysed, insensate 3
   - N.B. All scores DOUBLE for ischaemia > 6 hours

C. Shock
   - Systolic B.P. always > 90 mm Hg 0
   - Transient hypotension 1
   - Persistent hypotension 2

D. Age
   - < 30 years 0
   - 30-50 years 1
   - 50+ years 2

A M.E.S.S. score of 7+ is an indication for PRIMARY AMPUTATION.

Less severe injuries should be thoroughly debrided and stabilized as for any open fracture - consider early transfer to Ngwelezane for fixation of the more severe but potentially salvageable injuries esp. tibias.

DIABETES

About half our amputations are related to complications of this - although the weight distribution of the African foot seems to have a protective effect, prevention of foot infections can be difficult in an area where many people do not wear shoes! A limb-threatening infection is usually indicated by the presence of a full-thickness ulcer ≥ 2 cm of surrounding cellulitis and resistant hyperglycaemia. A paradoxical policy of ‘aggressive conservatism’ should be adopted viz. all such foot infections in diabetics not responding to antibiotics (Amp, Gent, Meti) within 12-24 hours must be urgently debrided because of the often unsuspected extent of the underlying infection - if a foot is left to suppulate an amputation will usually inevitably follow. Primary amputation may sometimes be indicated where infection has already destroyed the function of the limb.

Aggressive glycaemic control, continued antibiotics and especially repeated debridement where indicated can salvage a diabetic limb. The presence of osteomyelitis is an important factor - the ability to reach bone with a probe at debridement and X-ray changes are helpful but insensitive guides. Most deep diabetic foot infections are polymicrobial, the commonest isolates are Staphylococci, Enterobacteriaceae (esp Proteus, Klebsiella, coliforms), Streptococci and anaerobes (mostly bacteroides and clostridia). Intravenous antibiotics should be continued for at least 48-72 hours after debridement and continued orally for a total of 4 weeks.

Where control of the infection is not achieved by these methods, amputation should be performed at the lowest functional level that does not transgress the ascending cellulitis. Macrovascular disease is still relatively uncommon in our population but if there are good reasons to suspect it refer the patient for investigation of the other limb and revascularisation.

ISCHAEMIA

An increasing problem - though our patients may have a more favourable lipid profile and greater fibrinolytic activity than Europeans, smoking is beginning to take its toll. Peripheral atheromatous aneurysms may be more common in black patients but the distribution of disease is otherwise similar. Non-atheromatous peripheral vascular disease may also occur, usually Takayasu’s disease or idiopathic intimo-medial degeneration of the abdominal aorta (the majority of abdominal aortic aneurysms in black patients are due to non-atheromatous causes). Most of our patients present late with advanced complications - a complaint of claudication is exceptional. Revascularisation is often hampered by significant disease beyond the bifurcation.

Though many more below knee amputations become ambulant with the proper support, there is nothing more demoralising for the patient than repeated revisions or a second amputation. Unfortunately, the extent of the ischaemic lesion, the presence/absence of the popliteal pulse, amount of intraoperative bleeding and even Ankle-Brachial Indices/Angiograms may not accurately predict stumps healing (though an absolute ankle pressure of 70 mm Hg or an ankle-brachial index of at least 0.3 may be helpful). Without the benefit of transcutaneous Oxygen pressures a failure/revision rate ≥ 50% can be expected.
Sepsis in the stump is an important contributory factor - the organisms commonly recovered include S. aureus, and anaerobes including B. fragilis and clostridia. In chronic cases, consider referral for a "revascularisation/amputation" at KEM which may achieve a better functional level.

'VROT-FOOT'

This is an idiopathic neuropathic foot invariably found in males <60 years with a history of alcoholism sometimes associated with frank pellagra. The proximal vessels are always patent - there is an endarteritis of the foot arteries/smaller vessels with marked vasodilatation sometimes resembling a tumour blush on angiogram. It is usually associated with a peripheral sensory neuropathy and destruction of the bones of the foot, in which infection may play a role. The clinical appearance is of a clawed foot often with missing or stubby toes, subluxed metatarsal heads and ulceration of the sole. The lesion is chronic, unlikely to respond to revascularisation and conservative management should be pursued as far as possible. Diabetes, leprosy and malignancy should be excluded.

Further Reading
2) Huizinga et al 1993 "Prevention of wound sepsis in amputations" S.A.M.I. 63 3.7
3) Pollack and Ernst 1993 Use of Doppler pressure measurements in predicting success in amputation of the leg Am J. Surg 163 303
4) Caggo-Jack S 1991 "Pattern of diabetic foot ulcer in Port Harcourt, Nigeria" Practical Diabetes Digest 2 75
5) Trope GE and Crookes RC 1975 "Idiopathic neuropathic foot in blacks" S.A.M.I. 49 2157

Management
Cover the wound, give tetanus toxoid and immunoglobulin in severe cases with uncertain immunisation status. Give antibiotics immediately - Penicillin and Cloxacillin i.v. for Grade 1 injuries and Pen/Gent/Metro i.v. for more severe injuries. Antibiotics need not be continued beyond 72 hours unless frank infection is established.

Grade 1 injuries may be treated in O.P.D. under L.A. without laying open which would probably only contaminate the wound further - apply a sterile dressing and apply a backslab to allow dressing/review of the wound.

Grade 2 injuries should be formally debrided in theatre immediately. Clean the surrounding skin then remove the dressing. Irrigate copiously and continuously ('dilution is the best disinfectant') - start with the bucket and then use sterile saline. Use a tourniquet only if bleeding is severe - an oozc will slowly abate and tells you which tissues are viable! You may need to extend the wound longitudinally for access to contaminated recesses. Remove foreign material and debride in layers to ensure adequacy - be conservative with skin but trim ragged edges, excise non-viable fascia and muscle more aggressively. Leave large bone fragments if they have any soft tissue attachments. Severe injuries should be referred to Nkwelezane within the first 48 hours so that external fixation/flap closure can be effected at the 48 hour wound inspection. In less severe cases, leave the wound open and review at 48 hours in theatre - clean wounds may

GRADE 2
- Wound 1-10 cm with moderate soft tissue damage
- Can usually be closed by simple means (the temptation is often to do a primary closure).

GRADE 3
Wound >10 cm with associated extensive devitalisation of skin/muscle. Communion / wide displacement of the #. High energy injury from the history.
Subclassified as -
- 3A - adequate cover of bone /#
  (= skin loss requiring SSG)
- 3B - periosteal stripping/exposed bone
  (= skin loss requiring IGA)
- 3C - associated arterial injury
  (requiring repair for limb preservation)

OPEN FRACTURES
A surgical emergency! Debridement must wherever possible be carried out within 6-8 hours - an open # (fracture) is essentially a 'wound complicated by a broken bone' and the quality of your initial wound management may well determine the ultimate outcome. May be classified by the Gustilo method as follows -

GRADE 1
- Skin puncture by bone (usually from within out)
- Wound <1 cm
- Usually low energy injury with minimal contamination but beware the occasional high energy wound e.g. pedestrian hit by a vehicle where a small wound may be associated with crushing/degloving.
All Grade III injuries should be treated as an emergency to Ngelezeane for the primary debridement.

Stability of the underlying # is crucial in reducing the incidence of delayed sepsis. Suitable methods for our hospital are:

- Calcaneal pin traction and below-knee backslab for tibial #s
- Perkin's traction for femoral #s
- Narrow, mid-arm sling or light U-slab for humerus #s
- Closed methods for forearm (usually incomplete bushknife injury)

Primary/early internal fixation is indicated for ankle, olecranon and unstable forearm injuries. External fixation may be required at a later stage especially in severe tibial #s with bone loss/severe comminution.

Further Reading
1) Marks RK 1964 'Management of the open fracture - lessons learned' *Trauma and Emergency Medicine* 11.1 pp.963
3) Antrim RN and Solomon J 1987 'A review of prophylactic intravenous antibiotics for open fractures' *Orth. Rev.* 16:246
4) De Wandel CM 1995 'Management of open tibial fractures' *Trauma* 25:48

ANKLE FRACTURES

Generally result from a combination of external rotation and either abduction/adduction. The simplest classification is that of Weber (see diagram). Talar shift always implies disruption of the syndesmosis and consequently a B or C # (fracture). Due to the shape of the articular surfaces, even a mm shift implies a loss of joint congruity. Be sure tenderness is absent from the posterior 6 cm of tibia and fibula. A fracture is unlikely unless X-ray is necessary. If a # is present clinically, obtain an A-P in 10° internal rotation and lateral views.

'Sprained' lateral ligaments should be managed with a support bandage and early mobilisation unless severe pain and swelling are present when a below-knee P.O.P. should be applied for 3 weeks.

Lateral Ankle Fractures

Unilateral As and Bs with no talar shift or evidence of medial ligament damage may also be managed in a below-knee walking P.O.P. as an outpatient for 6 weeks. If reduction is maintained, the patient the P.O.P. should be maintained for a further 4 weeks (total 6 weeks in plaster).

Internal fixation is indicated for:

- Failed conservative management
- Displaced #s involving a significant part of the weight-bearing surface (e.g. large medial or posterior fragments)
- Direct/compound injuries (e.g. MVAs).

In children, epiphyses disrupt before ligaments so the congruity of the mortice is usually preserved. Salter type I-II injuries are satisfactorily managed by P.O.P. +/- reduction, but the rare 'triplane' # usually requires fixation of the medial malleolar fragment to prevent a severe varus deformity developing.

Further Reading
1) Lindojp BJ 1985 'Classification of ankle fractures: the Laug-Keane or AO systems?' *Clin Orth* 199:12
2) Shand et al 1985 'Multicentre trial to introduce the Ottawa ankle rules for use of radiography in acute ankle injuries' *BMJ* 311:598
3) Bewes PC 1985 'The management of ankle fractures' *Trauma* 25: Suppl 1:58
TIBIAL AND FIBULAR FRACTURES

The tibia is the most commonly fractured long bone in the body and the most likely to be open due to its long subcutaneous surface. #s (fractures) are often spiral/oblique and rotated when the # results from torsional forces. A useful guide to assessing this is the relation of the anterior superior iliac spine, middle of the patella and big toe (which should all be roughly in a straight line).

The principles of treatment are to allow early weightbearing in order to hasten union whilst controlling rotation and angulation of the distal fragment. Management of fibular #s is usually determined by the co-existing tibial # since they invariably have the same mechanism of injury except where caused by a direct blow. Isolated fibular #s (midshaft or below) may be managed in below knee walking P.O.P. for 6 weeks. (However, examine the ankle carefully to exclude an associated ankle #).

Reduction is required for any other tib/fib #s which are significantly shortened, rotated or off-ended (‘bayonet position’). Persistent angulation in the first 6 weeks can be corrected by wedging the plaster. Calcaneal traction with a backslab is useful in the early stages with very displaced open #s. It should never be maintained for more than 3 weeks however and most patients with this kind of # will be transferred to Nurgerie after their primary debridement. Patients with Grade III open #s will usually have an external fixator applied to stabilise the #. Mean time to union is 16 +/- 4 weeks and depends on the # pattern (spiral (fastest) < transverse < oblique (slowest)). Initial management is weightbearing on crutches in an above - knee P.O.P. (with walking heel and the knee in 15° flexion) for 6-8 weeks.

Test for signs of union at 6-8 weeks and X-ray to radiograph. If union is advancing and there is good callus apply a below-knee/Sarmiento plaster. If union is poor/absent and there is little callus retain the above knee plaster. Review the patient again in 6-8 weeks.

Knee stiffness and small effusions following tibial #s are common and ankle swelling and stiffness the rule. The patient should be encouraged to exercise the leg right from the start of immobilisation and to continue after the plaster is removed.

Further reading:
1) Dohle and Nix 1981 “The treatment of fractures by direct weight-bearing” J Trauma 1:514
2) Laushelve PH and Shangula K 1995 Assessment and management of common tibial injuries in a district hospital” Top Doc 25:12

KNEE INJURIES

These are a fairly common presentation at weekends especially during soccer tournaments! The history is all important and is worth taking some time over – ask particularly about the mechanism of injury, ability to weightbear after the injury, time of onset and progression of swelling, any ‘popping/snapping or locking’. The x-ray will invariably be normal but severe soft tissue injuries may still be present especially if there is a haemarthrosis. If you immobilise any knee always encourage early quadriceps exercises.

HAEMARTHROSI

Usually a rapidly swollen knee which is often quite tense, unlike a traumatic effusion. The blood can remain liquid for up to 2 weeks but early aspiration should always be performed both to relieve pain and for diagnosis - fat globules floating on the aspirate signify an intra-articular fracture. The exact diagnosis is often not evident but – 50% will be due to anterior cruciate tears. The Lachman tests are probably better than the anterior draw at detecting an ACL injury in the acute anaesthetised knee. Other causes include capsular/synovial tears, osteochondral #s (often accompanied by a patellar dislocation and a loud ‘snap’ at the time of injury with usually only a small if any bony fragment visible on the x-ray) and meniscal injuries. A modified Robert-Jones’ bandage should be applied and the patient reviewed in 2-3 days to check for instability or need for re-aspiration. Serious associated injuries should be managed as below.

UNSTABLE KNEE

Collateral ligament injuries may be graded simply in the following way:
- Grade I - pain/tenderness but no instability
- Grade II - pain and mild/moderate instability
- Grade III - severe instability but surprisingly little pain

This is a paradoxical situation where the more severe injury may present later with fewer symptoms so be wary. Lateral ligament injuries may involve the common peroneal nerve. In the case of Grade III injuries always test for varus/varus instability with the knee fully extended - if it is present the posterior capsule of the joint has also been damaged. If there is some doubt clinically as to the presence/absence of instability it can be assessed on stress x-rays under anaesthesia/sedation. Grade III injuries should be managed in a ‘slow – pipe’ P.O.P. at 30° flexion for 6-8 weeks.
grade III injury with a posterior capsule tear should be referred for repair. In all cases instability after conservative management should be referred for assessment at orthopaedic clinic. Cruciate ligament injuries may be obvious from the posterior sag/ve posterior draw of the flexed knee in posterior cruciate tears and from a +ve anterior draw in anterior cruciate injuries. The latter is usually associated with internal rotation in an isolated injury and no rotation where a medial collateral tear co-exists. A +ve anterior draw and external rotation may signify a medial ligament injury alone. In our setting ACL tears are managed in a stowe-pipe for 4 weeks unless the patient is a very keen sportsman and/or there is a large avulsed fragment of the anterior tibial spine in the joint which could be secured by internal fixation. PCL tears are managed in a stowe-pipe at 60° flexion for 4 weeks.

'LOCKED KNEE'

Often follows a blow to the flexed knee after which the patient is unable to weightbear. The usual cause is a meniscal tear though a loose fragment from an osteochondral fracture or fibres from a torn cruciate may be responsible. On examination there is a 'springy block' to full extension/loss of hyperextension, joint line tenderness and clicking on McMurray's manoeuvre etc (though these tests are often difficult to interpret in the acutely injured knee unless done under anaesthesia when an attempt to unlock the knee may be made).

Initial management is conservative - an MRI bandage should be applied and the patient should be partial weight-bearing on crutches for 2 weeks. If there is no improvement during this time he should be referred for arthroscopy and menisciectomy (the results of delayed selective arthroscopy are no worse than immediate indiscriminate arthroscopy).

PATELLAR DISLOCATIONS & FRACTURES

Have a high index of suspicion for these where there has been a direct blow to the knee. Dislocations should be reduced and managed in a plaster cylinder for 3 weeks. Fractures where the extensor mechanism is intact (i.e. can they raise/hold their leg straight off the bed?) can be managed in the same way but where it is damaged refer for repair +/- tension band wiring of the patella.

DISLOCATED KNEE

Usually associated with severe soft tissue injuries and possible vascular compromise - they must be reduced promptly! Immobilisation should be maintained in a stowe-pipe P.O.P. at 90° flexion for 4 weeks.

Further Reading

NECK FRACTURES

Most helpfuly thought of as intracapsular ('subcapital', 'transcervical') or trochanteric / extracapsular ('inter- trochanteric', 'pertrochanteric' and 'subtrochanteric'). The principal risk in the first group is avascular necrosis, the likelihood of which increases the nearer the # (fracture) is to the head; since the blood supply arises from the anastomosis of the femoral circumflex arteries distal to the margin of the articular cartilage. The typical patient is unable to weightbear but walking does not exclude the diagnosis by any means - some patients may present ambulant after 3 weeks following a minor fall! The usual signs are shortening, external rotation and a reduction of varus on x-ray from 145° to 90° (in trochanteric #s), Bruising is a late sign normally only seen in extracapsular #s. Initial management is analgesia and gentle skin traction (~3 kg). Probably the best method for splinting during transfer etc. is to strap the patient's legs together. In general mortality in the elderly is high (7-33% at 3 months) often related to complications of traction and one of the best predictors is the patient's mental state. Intracapsular #s are usually divided into displaced and undisplaced #s. Undisplaced #s presenting early in a younger patient should be treated with a dynamic hip screw (D.H.S.). Older patients, especially late presenters, can often be managed satisfactorily by immediate/early mobilisation once pain has subsided (some will already have done this themselves!) but refer potential surgical candidates for assessment in any case. Displaced #s are conventionally managed by hemiarthroplasty in older patients (the patient herself will usually wear out before the acetabulum....) but for younger patients open reduction and insertion of D.H.S. is probably a better solution. Trochanteric #s may be in 2, 3 or 4 parts. The definitive management is internal fixation with a sliding pin and plate but stability is related to the degree of comminution and some 4 part #s may need up to 4 weeks of non-weight-bearing post-op. Patients not fit for surgery or who were not in any case ambulant prior to the injury should be managed by 4-6 weeks of skin traction.
and mobilised as far as is possible.

Subtrochanteric # s in elderly patients should always raise the suspicion of metastases but may also be the result of a high-energy MVA injury in younger patients. They are usually suitable for Perkin's traction if undisplaced but should rather be put in 90° - 90° - 90° traction if the proximal fragment is very flexed by the ilio-osseous muscle.

SHAFT FRACTURES

These are usually high-energy injuries so be on the look-out for associated injuries, especially knee instability, hip dislocation, pelvic #s etc. In children up to the age of 2 years and/or 15 kg gallows traction should be applied and in older children extension skin traction. Union usually takes 1 week per year of age (subtract one week if the # is a spiral pattern or in the lower third of the shaft) to a maximum of 8 weeks. In patients over 12 years, Perkin's traction is our preferred management for most uncomplicated femoral shaft #s - some shortening (up to 4 cm) will often have to be accepted with this method but can be well compensated with a shoe-raise or hip-tilt. It is essential that quadriceps exercises are started immediately to prevent wasting and knee stiffness. Traction should normally be maintained for 8 weeks followed by 4 weeks partial weight-bearing. Some patients however should be referred to Ngwelezane within 2 weeks for insertion of a Russell-Taylor interlocking nail - principally compound injuries, where other fractures co-exist or in elderly patients who may not tolerate traction for long periods.

CONDYLAR FRACTURES

Perkin's traction for 4-6 weeks followed by a stove-pipe P.O.P. may be suitable for some of these injuries e.g. very comminuted #s in the elderly, unfractured supracondylar in young patients, unrotated uncondylar fractures. However, in adults supracondylar #s are often flexed by the gastrocnemius and Perkin's traction with the knee in flexion/supported may not control the flexion adequately. In this case, as in most cases of intercondylar #s involving the articular surfaces and in rotated uncondylar #s, the patient should be referred to Ngwelezane for internal fixation with an intramedullary nail inserted via the knee joint.

Further Reading
1) Wing R and Hingle P 1996 The functional management of femoral shaft fractures" S A Family Practice 17 9 406
2) Beves P 1974 Fractures of the femur in a tropical context: a revaluation of Perkin's traction" Trans. Doc. 4 84

TORSO TRAUMA

This may be the result of an MVA (high-speed shearing crushing of thorax etc.) or penetrating trauma from knife/gunshot wounds (the latter carrying about 3x the mortality of the former). In South Africa currently, death from the latter is twice as likely as from the former. Many severely traumatised patients in our district never reach hospital so our trauma population is somewhat selected - however, torso injuries account for a high proportion of hospital trauma fatalities - correct diagnosis and management is essential in reducing this. Civilian injuries are in general less destructive than military ones, and a policy of selective conservatism for many cases is now generally accepted.

Further Reading
Muckart DJ et al. 1995 The changing pattern of torso trauma in KwaZulu/Natal - a clinical and pathological review" S A M J 85 1172

CHEST INJURIES

Ensure the patient's ABC.

Attend to life-threatening injuries first:
- immediate needle thoracostomy for tension pneumothorax
- an occlusive dressing taped on three sides for sucking/blowing wounds
- splinting of flail segments/placing the patient with the flail side up
- control any severe external source of haemorrhage e.g. intercostal artery

Re-assess the patient more closely and identify the injuries. Consider the position of penetrating wound/s in relation to underlying structures esp. the mediastinum and upper abdominal contents. Subcutaneous emphysema usually but does not always indicate pleural penetration. Rib #s (identified clinically by # [fracture] site tenderness and A-P chest compression) are useful predictors of underlying injury - splenic injuries may accompany # L. 10th, rib # 1st rib is often associated with major neurovascular/tracheobronchial injuries. # of 7 or more ribs carries a risk of infradiaphragmatic injury of 50%. Sternum #s are similarly cause for caution as they may be associated with a myocardial contusion.

PNEUMOTHORAX

May be managed conservatively only if <20% and the patient is asymptomatic. Repeat the film if you are suspicious on other grounds (e.g. rib #s or s.c.
PULMONARY CONTUSION

Often the result of a deceleration injury or high velocity gunshot wound (spalling; splintering; perforation; implosion). Invariably localised to a lobe, segment and usually occur immediately after the injury – they may however be inapparent for 24 hours. Uncomplicated contusions resolve in <7 days but 50-80% will become infected and may form empyemas/lung abscesses – antibiotic prophylaxis is indicated. The contusion may resolve into a more sharply defined nodule suggesting haematoma formation – these can be managed expectantly and will resolve in 4-6 weeks.

STABBED HEART

Carries a dismal 85% mortality even in the best trauma centres. In those who reach hospital, Beck’s triad (hypotension/shock, distended neck veins and “distant heart sounds”) will be present in 90% reflecting the probable tamponading effect of cardiac tamponade. Pericardiocentesis is a temporary measure and emergency cardiology must be carried out as soon as possible – our only option would be to resuscitate, leave a catheter in the pericardium and airlift the patient to Durrban. Emergency room thoracotomy even in moribund patients or those who arrest on arrival is NOT indicated in our circumstances as the mortality is 90% even with a cardiothoracic surgeon attending.

Myocardial contusion resulting from blunt trauma may be diagnosed from the mechanism of injury and S-T/T wave changes on E.C.G. – this is thought to be the most common undiagnosed visceral injury leading to delayed mortality in trauma patients. Bed rest is advised until the changes resolve. Inotropes may sometimes be required.

Further Reading
2) Demetriades D 1984 “Cardiac penetrating injuries. personal experience of 45 cases” B J. Surg. 71:95

RUPTURED DIAPHRAGM

Usually the result of an M.V.A., more rarely penetrating trauma. Invariably left sided due to hepatic protection of the right. Bowel sounds in the chest and a markedly scapheal abdomen (Gibson’s sign) suggest the diagnosis which can be confirmed on X-R – An N.G. tube will be seen in the chest. Severe respiratory compromise can result and the patient should be transferred for immediate operative repair.
MEDIASTINAL INJURIES

Require a high index of suspicion - be wary if a gunshot track crosses the midline. The patient may have pain on swallowing, hoarseness, haemoptysis or interscapular pain but the presentation may be non-specific. Look for unexplained shock/toxicity, neck-swelling/surgical emphysema, upper limb hypertension/missing pulses, mediastinal ‘crunch’/murmurs on auscultation etc: CXR may show mediastinal widening and emphysema +/- associated pleural effusion. Most of these injuries (oesophageal perforation, rupture of the descending aorta, tracheo-bronchial injury) are serious and require referral for assessment (contrast swallow, angiography, bronchoscopy) and repair.

Further Reading
1) James KW 1986 'Thoracic trauma' Surgical Clinics of N. America 66:4 pp 957
2) Graham JM 1979 'Penetrating trauma to the Lung' J of Trauma 19:665
3) Mudrack DJ et al 1984 'Penetrating injuries of the pleural cavity' Thorax 39:789
4) Thomson SR et al 1990 'Prospective study of the yield of physical examination compared with chest radiography in penetrating thoracic trauma' Thorax 45:616

ABDOMINAL INJURIES

Blunt trauma can produce rupture of a viscus without marking the abdomen. On the other hand, even proven peritoneal penetration need not result in surgery. Remember that a negative laparotomy in an unstarved, intoxicated patient in difficult circumstances may carry significant morbidity/mortality (1-3%) and that repeated series have firmly established selective conservatism as best practice. However, repeated critical clinical assessment is essential as it alone determines the management - in the hands of trauma surgeons, sensitivity of the initial clinical assessment can be as high as 90%.

Management
1. Examine the patient carefully for signs of peritonism and other evidence of visceral injury especially shock, pyrexia etc. Be especially wary in the presence of associated injuries, especially head and spinal cord injuries, which may mask signs.
2. Pass an n.g. tube, pull up an i.v. line and keep the patient n.p.o. Resuscitate where appropriate prior to surgery.
3. Explore the wound under L.A. to establish whether peritoneal penetration has occurred, toilet and close the wound. However, the peritoneal breach may be distant from the skin wound so this test is quite insensitive. Eviscerated omentum should be ligated, amputated and pushed back into the abdomen.
4. Peritoneal lavage is only really useful in blunt injuries. Abdominal/Chest films are unhelpful as the presence of intra-abdominal air is not in itself an indication for laparotomy.

5. Immediate indications for laparotomy -
   - Peritonism (not localised wound tenderness alone)
   - Shock (with no other obvious cause)
   - Evisceration (unless omentum only)
   - All gunshot wounds (much higher incidence of major visceral injury, especially liver and colon)
   - Suspected diaphragmatic injury (better transferred)

All other patients should be observed and re-examined every 4-6 hours. Do NOT prescribe antibiotics!

6. Delayed laparotomy should be carried out if signs of peritonism or haemorrhage subsequently develop (note that the absence of bowel sounds is a late finding with a haemoperitoneum vs. perforation). Most patients not requiring surgery can be discharged after 48 hours.

Further Reading

PELVIC INJURIES

These are generally high energy injuries resulting from a tractor or car accident, though isolated pubic rami #s may occur in the elderly after a fall. Mortality is high and may approach 50% in compound #s, often from other associated injuries. The associated haemorrhage will often be in excess of 2 litres especially in compound injuries where it cannot be lamponaded by the soft issues. Look for buttock/periurethral/inguinal bruising and assess the femoral pulses. Resuscitate vigorously! A huge retroperitoneal haematoma may be palpable abdominally and make diagnosis of associated abdominal injuries difficult without peritoneal lavage.

Palpate the whole pelvis and apply antero-posterior and lateral compression/expansion to assess the stability of the pelvic ring in the lateral and vertical planes. If there is significant movement/displacement the ring must be disrupted in at least two places. Vertical displacement of one side may be obvious from asymmetry of the hips/legs. If there is obvious instability and signs of shock immediately stabilise the pelvis by tight elastoplast strapping in OPD - protect the pressure areas with 2-3
layers of orthopaedic soft/abdominal swabs.

#s (fractures) of the anterior pelvis (especially butterfly #s) will be associated with bladder or urethral injury in 1
in 20 male cases. However, if the patient can pass
macroscopically clear urine, a clinically significant injury is
extremely unlikely. On the other hand, blood visible at the
urethral meatus is considered diagnostic of urethral
laceration/rupture. Rectal examination may reveal a swollen
perineum and/or displaced prostate. If there is an
obvious urethral injury, insert a suprapubic catheter - if there
is some doubt, wait for the patient to void for 2-3 hours. If no
urine is passed do an emergency urethrograms - inject
(Detripaque 5-10mls, mixed with KY jelly if necessary to
increase viscosity) either via syringe and tines or 12F
Foley blown up to 2-3mls in the urethral meatus.
Extravasation suggests a urethral injury and a suprapubic
catheter should be inserted. If the urethra is intact then a
urethral catheter can be passed - if urine is still not produced
suspect a ruptured bladder.

Pelvic #s may be graded according to the Tile
classification:
A. Stable (No disruption of pelvic ring or <1cm
displacement)
B. Rotationally unstable but vertically stable (Posterior
Sacro-ilac ligaments intact)
  - anteroposterior "open book #"
  - lateral "closed book #"
C. Rotationally and vertically unstable (Posterior
Sacro-ilac ligaments disrupted)
  - unilateral
  - bilateral
  - with associated acetabular #

Management
- Type A #s require bed rest until pain subsides or 1-2
  weeks and then mobilisation.
- Type B #s can be managed in the same way if there is
  <2.5 cm opening of the anterior pelvis. If >2.5 cm then open
  reduction and internal fixation are probably required.
- Type C #s should be referred immediately to
  Ngwelezane and are generally managed conservatively
  as operative morbidity is high. A pelvic sling or
  external fixator is used to reduce the pelvic bones +
  - lower limb traction to correct vertical displacement
  of a hemipelvis for 4-6 weeks.

Further Reading
1) TIE M 1983 Pelvic ring fractures - should they be fixed? J Bone & Joint
Surgery 70:1
2) Trillo PG 1990 Pelvic ring injuries. Surgical Clinics of N. America 70:655

Surgery

BECXOZELE

Penetrating Neck Injuries

The neck can be divided anatomically into
three zones:

Zone I
  Angle of
  Mandible
  Thyroid Cartilage

Zone II
  Cricothyroid
  Membrane

Zone III
  Cricoid Cartilage
  Clavicle

Wounds in zones I and III usually result in severe
vascular injuries and the tight fascial compartmentalisation
of the neck (carotid sheath formed from deep cervical
fascia which merges with the thyroid/cricoid cartilages
anteriorly and pericardium inferiorly) means that rapid
airway compression may result. However, careful
assessment of zone II injuries allows 30-40% to be
managed conservatively. The jugular veins are injured in
≤15% of penetrating neck injury and the carotids (usually
the common) in ≤10%.

Management
Whatever the appearance of the wound, first secure
the airway and resuscitate the patient vigorously - he
may have bled profusely prior to admission. Always insert
i.v lines, check B.P. in the opposite arm (to avoid proximal
venous extravasation), sit the patient up (to prevent air
embolism and lower the pulmonary pressure), and NEVER
probe the wound with your finger to establish whether it
has penetrated the platysma as you may release
tamponade from a major vessel. Control any external
haemorrhage with a pressure dressing or a proximal
pressure point. Severe bleeding can be temporarily
tamponaded with a Foley catheter inflated in the wound.

Examine the neck (surgical emphysema, haematoma,
tenderness along the trachea etc) all the upper body
pulses, the neurological system and the chest. CXR may
reveal a widened mediastinum or associated
haemopneumothorax (try to include the neck right up to
the mandible to see the upper airway silhouette).

Absolute indications for surgical exploration are -
• Gunshot wound
• Evidence of vascular injury (active bleeding/shock, large/expanding/pulsatile haematoma, absent pulse)
• Evidence of airway/pleural penetration (blowing/bubbling wound, progressive surgical emphysema or respiratory distress)
• Haemoptysis/hematemesis.

All other patients, including those whose haemorrhage has stopped, those with non-progressive surgical emphysema and those with isolated nerve/orchial plexus injuries should be admitted. Angiography may be indicated for zone I and III injuries esp. if the mediastinum is widened or there is a bruit in a haemodynamically stable patient. A short period of observation will help to eliminate the possibility of an occult oesophageal injury (suggested by dysphagia, pain on swallowing, wound track towards the midline with surgical emphysema/signs of mediastinitis) - this is a difficult injury to rule out without oesophagoscopy as the sensitivity of barium swallow is only 70-80%. Brachial plexus or other nerve injuries should undergo repair at an interval.

Further Reading
1) Assensio et al. 1991 'Management of penetrating neck injuries' Surgical Clinics of N. America 71: 257

FOREARM FRACTURES

These are demanding #s (fractures) - multiple muscle attachments in the forearm tend to rotate the fragments and holding a reduction by closed methods is difficult. Late slipping due to muscle wasting/stripping of the cast is a constant problem. However, in children, if a reasonably accurate reduction can be achieved, the # will always unite and remodeling may be able to improve any residual deformity. However, this process is most efficient in the metaphysis/epiphysis area, and only occurs in the direction of movement of the adjacent joint so mid-third and very rotated #s are unlikely to remodel well. 60% of children lose 20-60° of rotation due to significant angulation but angulation in favourable areas can improve at ~10°/year until the epiphyses fuse. In adults with both radius/ulna #s however there is a high risk of non-union and closed methods invariably fail so almost all our cases are referred for primary internal fixation with Rush rods or compression plates.

Accurate assessment of the injury is the key to correct management.

Rotational deformity - check the relative widths of the radius at the # site (if different, one of the fragments must be rotated) and the relationship of the bicipital tuberosity to the radial styloid process. Angulation - if there is significant shortening of one bone the other MUST be dislocated so ensure that X-rays always include the elbow and the interior radio-ulnar joint to exclude Monteggia or Galeazzi #/dislocations. In practice however about 90% of injuries are 'apex anterior' and best maintained in mid- or full pronation. Above elbow POP is required in all #s above lower 25% of the arm to control rotation. Always apply a broad arm sling (not a collar and cuff) or a traction loop on the proximal part of the forearm (not at the wrist!). It is often possible to re-manipulate for angulation under anaesthetic up to 3 weeks if unsatisfactory reduction/failure to hold - however significant displacement cannot be corrected by closed methods after the first week.

COLLES' AND SMITH'S FRACTURES

Indications for reduction of Colles' #s are severe clinical deformity, >15° posterior tilting of the distal radius on lateral X-ray (normal = anterior tilt of ~11°) and displaced ulnar styloid (indicates severe disruption of inferior radio-ulnar joint). Salter II epiphysial injuries are reduced in the same way. Apply a backslab and complete at 48 hrs post-reduction with the hand in full pronation, full ulnar deviation and slight palmar flexion.
Smith's #s can be reduced and held in a split full arm POP with the hand in dorsiflexion and supination. Take a check X-ray at 1 week to exclude slipping. If reduction cannot be achieved or held refer for re-manipulation/internal fixation. In both cases assess for union at six weeks.

**ISOLATED ULNA/MONTEGGIA FRACTURES**

The mechanism of injury is usually the same - the patient's arm is struck while bending off a blow. Examine the X-rays carefully to exclude a radial head dislocation whenever you see a significantly displaced ulnar #. If the ulnar # is truly isolated apply POP (half arm lower 1/3, full arm upper 1/3) for 3-6 weeks and then mobilise from a sling. If the radial head is also dislocated refer for internal fixation of the ulna # and open reduction of the radial head.

**ISOLATED RADIUS/GALLEAZI FRACTURES**

Usually a # of the distal 1/3 with complete soft-tissue and the proximal fragment pulled into mid-pronation by pronator teres. Check for an associated dislocation of the inferior radio-ulnar joint (ulnar head distal to radial). #s higher up the radial shaft are associated with varying degrees of supination of the proximal fragment - a rough guide as to how much can be gauged from the position of the radial tubercle. Closed reduction may succeed in children and can be held in a full arm POP in mid-pronation/supination (according to the position of the proximal fragment) for 6 weeks. X-ray weekly for three weeks until you are sure the reduction will be maintained. Compression plating of the radius is preferred for adults and failed closed methods in children (esp. if epiphyses are about to fuse).

**RADIUS AND ULNA FRACTURES**

Attempt reduction of #s in the lower 2/3 of the arm in children. This is best done with the arm suspended from a drip stand on clove hitchies. Apply a split above-elbow POP for six weeks (X-ray weekly for 3 weeks). Adults and proximal 1/3 #s in children should be referred to Nagwezane - they may continue conservative management in a straighter AE POP with 30° of elbow flexion but internal fixation is often carried out.

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**ELBOW FRACTURES**

The common injuries are supracondylar fractures, dislocations of the elbow, lateral condylar injuries, medial epicondylar injuries and fractures of the proximal radius and ulna.

**OSSIFICATION OF EPIPHYES AROUND ELBOW JOINT**

**SUPRACONDYLAR FRACTURES**

Peak 6 years. Majority follow a fall onto the extended arm but 5-10% are of the flexion or reversed type. Usually gross swelling but with a tender, mobile lower humerus. Always assess the radial pulse but more importantly the perfusion and response to passive stretching of the fingers (to detect forearm ischaemia). Neuropraxia may also be a complication (radial>median>ulnar). The undisplaced # fracture may be treated in a collar & cuff for 3 weeks +/- an above-elbow backslab. Indications for reduction are ≥15-30° of backward tilting of the supracondylar complex, severe valgus/varus deformity and <50% contact between the fragments.

No more than 3 M.U.A.s should be allowed and if the first is unsuccessful (usually due to severe swelling) extension traction can be applied or a repeat attempt delayed for 2-3 days. If success seems unlikely without screening refer to Nagwezane for the third attempt within the first week. Lateral and axial views should be taken to check the reduction and a collar and cuff and backslab in as much flexion as the pulse will allow (preferably >110°) should be worn for three weeks. Some persistent backward tilting or cubitus varus ('straight arm') should be accepted - a supracondylar osteotomy is possible later but some severely displaced and irreducible #s should be referred for percutaneous pinning.
DISLOCATED ELBOW

May occur at any age. The elbow is grossly swollen but fixed and the normal triangular relationship between the epicondyles and olecranon is disturbed. The dislocation is invariably posterior, frequently accompanied by other fractures esp. medial epicondyle and there may be vascular problems in 10%. Ensure no bony fragments are in the joint. Reduction should be prompt and maintained in a collar and cuff + backslab for 1-2 weeks.

LATERAL CONDYLE FRACTURES

A fracture with a bad reputation, peak 3-4 years. Essentially a Salter II or IV injury depending on how proximal the fracture line is. <2 mm displacement can be managed in a collar and cuff + backslab at 90\(^\circ\) flexion but it is essential to review at one week since further displacement can occur. >2 mm displacement requires internal fixation with a K-wire (late complications of which may be avascular necrosis and tardy ulnar nerve palsy).

MEDIAL EPICONDYLE FRACTURES

The medial epicondyle only ossifies at 6 years so it is fortunate that this # usually occurs in older children (9-14 years) - but it can be missed in younger ones esp. in association with a dislocated elbow. It is caused by avulsion of the common flexor origin during forced abduction of the arm. The epicondyle may become trapped in the joint in which case, if it is ossified, it will be absent on the AP and visible on the lateral (the reverse of the normal situation). Always assess the ulnar nerve and check for valgus instability.

Minimally displaced #s may be treated in collar and cuff + backslab for one week. Open reduction and internal fixation is indicated for >5 mm displacement with valgus instability, epicondyle incarcerated in joint, ulnar nerve palsy, associated dislocation. An epicondyle in the joint for >4 weeks should be left alone.

PROXIMAL RADIUS FRACTURES

"Pulled elbow" seems to be quite uncommon in Zululand - it is due to subluxation of the radial head under the annular ligament. Supination is lost and there is tenderness over the radial head. X-rays are normal. Reduction is normally easily effected by firm pronation and supination. In children the usual # is through the neck <30\(^\circ\) of angulation or 4 mm of displacement can be managed in collar & cuff and backslab for 2 weeks. Greater displacement requires reduction of the proximal fragment with the joint strained in valgus. If this is unsuccessful refer for open reduction +/- insertion of K-wires. In adults the common injury is a # of the radial head. Check that the distal end of the ulna is not also subluxed - it is the interosseous membrane is completely torn and the # is very unstable (Essex-Lopresti #). Undisplaced #s are best treated with a broad arm sling for 3 weeks with early movements. Displaced or comminuted #s should have a trial of active movements for 4-5 days but many will require excision of the radial head so refer early for review at Ngw.

OLECRANON FRACTURES

Undisplaced /minimally displaced #s are managed in a full arm POP (3 w child, 6w adult). More marked displacement requires tension-band wiring.

Further Reading:
2) Wilson NJ et al. 1968 "Treatment of fractures of the medial epicondyle of the humerus" Injury 13 342
SHAFT OF HUMERUS FRACTURES

Always assess the radial nerve (injured in ~10%). Use a 10cm narrow sling under the distal forearm only supporting the elbow at 90° so that the weight of the forearm acts to reduce overlap and angulation. A bandage or very light U-slab fixed to the shoulder may be added for protection ("sugar-tong splint"). The only indications for manipulation are gross angulation or a cold pulseless arm - in the latter case reduce the fracture and apply traction. Radial nerve injuries are managed expectantly as they are invariably in continuity and will start to improve after ~6 weeks. Show the patient elbow exercises which must begin within 2-3 days. Check for union at 4-6 weeks. If the # is clinically united, discard the arm splint but continue to mobilise the arm from the sling for another 4 weeks. If there are still no signs of union at 12 weeks refer for internal fixation. Delayed repair of the radial nerve can be carried out at this time if there has been no recovery of function.

HEAD OF HUMERUS FRACTURES

The key principle with these #s is early movement to prevent permanent loss of function due to adhesive capsulitis. As the fragments unite in almost any position especially if impacted. Therefore the usual management is a broad arm-sling (impaired) or collar and cuff (angulated #s) with elbow exercises for 6 weeks (3-4 weeks in children). The exceptions are:
- Severely displaced two-part #s(Neer III) and fracture-dislocations (Neer IVs) require closed reduction and sometimes internal fixation.
- Avulsions of the greater tuberosity (Neer IVs) attempt closed reduction but may require internal fixation with a screw.

DISLOCATED SHOULDER

Try gravitational traction but most require reduction under GA (Ketamine seems to be satisfactory in most cases). We usually use Kocher's method. Keep the arm in a broad arm-sling for 3 weeks. Beware the elderly patient with a dislocation - consider that it may have been present for some weeks with capsulitis. It may be irreducible by closed methods by this stage so don't persist if reduction is not easy, as there is quite a risk of iatrogenic damage.

CLAVICLE FRACTURES

Broad arm-sling for 3 weeks or until the clavicle is no longer tender.

Further Reading

HEAD INJURIES

50% of all trauma deaths are associated with head injury - about 60% of head injury deaths occur at scene/in transit but the mortality for severe head injuries (GCS <8, 'coma') reaching hospital is still 40%. Many patients are multiply injured and attention to ABCs is crucial - many severe head injuries are hypoxic and/or hypotensive. Admission and analysis of patients who ‘talk and die’ implicates such preventable factors in ~50%. In Natal, 88% of head injury deaths demonstrate hypoxic neuronal damage at post-mortem. It is therefore crucial to adopt a systematic approach.

GLASGOW COMA SCALE (GCS)
(Incorporating Adelaide Coma Scale for children)

Eyes open
- spontaneously 4
- to speech 3
- to pain 2
- none 1

Best motor response
- obeys commands 6
- localises pain 5
- withdraws to pain 4
- flexes (decorticate) 3
- extends (decerbrate) 2
- None 1

Best verbal response
- oriented 5
- disoriented but appropriate words 4
- inappropriate words/vocal sounds 3
- incomprehensible sounds/cries 2
- None 1

Normal scores:
>5 years 15
2-5 years 14
1-2 years 13
6-12 months 12
6 months 10
Management

Ensure the patient's airway and ventilation. Intubate any patient with GCS <8 or a potentially difficult airway esp. facial injuries - be aware that the incidence of arterial hypoxaemia in head-injured patients breathing spontaneously without signs of respiratory distress is ≤65% and that time to intubation in comatose patients relates to outcome. Use the pulse oximeter. Maintain cervical spine immobilisation. Hypotension in head-injured patients is due to hypovolaemia until proved otherwise and the source of haemorrhage may be concealed - resuscitate aggressively with Ringer's/haemaccel and blood as soon as available. As well as correlating with poor outcome, hypotension may predict later rises in I.C.P. Do a neurological assessment with special attention to the GCS, pupils and any localising signs/signs of raised intracranial pressure. Examine the skull for evidence of a vault or basal skull # (CSF leak, haematoxylinum, mastoid bruising/Panda sign).

Control seizures (occur in 4%) with Diazepam 0.2 mg/kg and Phenytoin 15 mg/mg/kg i.v. at a rate of not >50 mg/min. X-ray cervical spine and skull.

Nurse the patient 30°-head-up, toilet and close the scalp wound even if there is an underlying skull #. Though this will help to prevent any further contamination of the #, give Penicillin/Sulphadiazine both 500 mg 6 hourly i.v./p.o. for 10 days as prophylaxis against meningitis. Do not remove any bone fragments or weapons as these may be tamponading intracerebral vessels and you may not be able to control the resulting haemorrhage.

Skull X-ray is indicated for:
- any loss of consciousness / amnesia
- neurological signs
- CSF / blood from nose or ear
- suspected penetrating injury
- scalp bruising or swelling
- difficult to assess (children, seizures, alcohol)

NB. Patients with a GCS 5-10 do not need SXR pre-CT.

Raised intracranial pressure manifested clinically by deteriorating GCS + papilloedema and Cushing reflex (slowing pulse and rising B.P.) should be treated with Mannitol 1g/kg and Frusemide 80 mg i.v. - this may buy time for transfer. Any patient with GCS <15 (risk of Intracranial Haemorrhage – 1:121), localising signs or skull # (risk of I.C.H. 1:32 if fully conscious and 1:4 if not) should be admitted for observation. A fully conscious patient with any history of loss of consciousness before arriving in hospital should not be discharged unless she has a normal SXR (risk of I.C.H. >1:5000). Remember, however that a small number of patients with initially normal findings will subsequently deteriorate so always try to ensure that they are discharged to the care of a responsible adult with clear instructions.

Urgent consultation with Wentworth Hospital (WWH) neurosurgery.

- GCS 5-10
- any fall in GCS (be careful to eliminate any non-neurosurgical factors)
- GCS 11-14 + skull fracture or neurological signs
- patients with a fixed dilated pupil

Resuscitate and re-evaluate before consultation.

- Shocked and/or hypoxic patients
- Patients with an initial GCS of 3-4 from blunt trauma (probably have irreparable diffuse axonal injury (mortality/severe morbidity 95+%), and should not be transferred immediately, unless <10 years (children may demonstrate surprising improvements)). Bullet wounds to the head will not be amenable to neurosurgery.

Non-urgent consultation (During working hours, next neurosurgical OPD – Mon, Weds)

- GCS 15, but stabbed head / deeply indrawn bone fragments
- GCS 15 and localising signs (usually a cerebral contusion, often following a knuckles injury which does not require surgery)
- GCS 11-14 at 48 hours and no # or focal signs (<20% will have an abnormal C.T. scan)

Emergency burrholes are almost NEVER indicated in our situation - the only conceivable situation where it might be justified would be a rapidly deteriorating patient where air transfer would not be immediately arranged (e.g. during the night).

Further Reading
SPINAL INJURIES

The common causes of spinal cord injuries in our district have been gunshot wounds and MVAs. These patients present a serious challenge and many of our efforts at community rehabilitation have been unsuccessful. In recent European series, survival for paraplegics now approaches 20+ years but our experience has been much less optimistic with frequent deaths from urinary sepsis/renal failure and pressure sores.

In general, a spinal injury accompanied by any neurological involvement is by definition 'unstable' as are those where the posterior elements of the spinal column (pedicles, laminae, facet joints and posterior ligaments) are disrupted on X-ray. The only spinal injuries that can be considered definitely stable and that do not require reduction are:

- **Anterior wedge** #s to <2/3 the height of the posterior vertebral border (>2/3 or loss of height of the posterior border implies associated disruption of the posterior elements and for a burst #).
- **Isolated transverse or spiral process #s** (but be sure to exclude associated unstable injuries)
- **Type I odontoid peg #s** (involve only the tip, commonly missed on poor quality X-rays)
- **Extension injuries** of the cervical spine (though paradoxically a significant number of these patients will be quadriplegic at presentation).

Any suspicion of spinal trauma mandates total immobilisation until X-rays exclude an unstable injury. Immediately apply and maintain cervical spine immobilisation in any trauma patient with neck pain, torticollis or who has a reduced GCS and do not examine neck movements. Injuries to the thoraco-lumbar region should be sought by log-rolling the patient with in-line neck immobilisation and examining the back for areas of deformity or swelling (evidence of damage to the posterior ligament complex).

When the patient's other injuries dictate, take her to X-ray. ~90% of significant cervical spine injuries will be visible on a lateral/pull-down view which must include C7/T1. Request an AP and open mouth view as well. If a pull-down film still fails to show T1 request a swimmer's view. Other oblique films are only necessary if the lateral shows over-riding of two vertebral bodies suggesting a unilateral/bilateral facet joint dislocation. Thoracic and lumbar injuries usually result from hyperflexion and ~ 2/3 occur at the thoraco-lumbar junction between T12 and L2. Injuries of the upper eight thoracic vertebrae almost always involve the contralateral sternocostal joint so make sure you examine the sternum.

If neurological signs are present, the critical decision is whether the patient has a complete or incomplete injury. Complete transections are characterised by a sensory level, flaccid paraplegia, neurogenic shock and anatomic bladder. Bulbocavernosus and anal wink reflexes and priapism may occur. If there is ANY neurological function below the level of the injury the lesion is INCOMPLETE - pay particular attention to the S1-S5 sensory dermatomes and knee flexion (L5/S1). Incomplete transection syndromes are especially common in cervical spine injuries - the *Central cord syndrome* which often follows hyperextension injuries of the neck is characterised by weakness greater in the upper limb, patchy sensory loss and urinary retention. Anterior cord syndrome = quadriplegia + mild spinothalamic impairment and preserved proprioception. Posterior cord syndrome = dysaesthesia in neck/lumbar/trunk + mild motor impairment of the upper limbs. All patients with neurological signs (complete or incomplete) presenting within 8 hours should receive Methylprednisolone 30mg/kg/hour for a further 23 hours. Open spinal injuries (gunshot, stabs) should receive Penicillin and Sulphadiazine i.v. for at least 72 hours.

Stable cervical spine injuries (wedge #s, hyperextension injuries without neurological signs, Type 1 odontoid peg #s) may be managed as an outpatient in a hard collar for ~6 weeks. Minimally displaced Jefferson (bow-out) #s of C1 and hyperextension injuries with signs should be admitted and observed in a hard collar for 3–6 weeks. Most other cervical spine injuries require reduction under screening or skull traction and should be referred to Ngwelezane. Wedge #s of the thoraco-lumbar spine (commonest injury) require ~1–2 weeks bed rest/anaesthesia + extension exercises. Unstable thoraco-lumbar injuries will generally be reduced by extending the spine using a pillow under the patient's back. These patients should be referred to Ngwelezane for assessment as some will require surgery if reduction cannot be achieved by conservative methods.
Patients with complete transection clinically must immediately be started on the following regime:

- **2 hourly turns.** The paraplegic patient should be encouraged to take responsibility for her own pressure areas as soon as possible and this should become a lifelong habit. Quadruplegics require diligent turning.
- **Passive movements of the affected joints at least twice per day.**
- **Chest physiotherapy.**
- **High fluid intake.**
- **Silastic urethral catheter.** This is on free drainage for 1st 48 hours then clamped and released 4-6 hly by the patient. After 4 weeks, paraplegic patients with lesions higher than T10 should attempt to ‘tap and express’ (tap the bladder to initiate the detrusor reflex and apply suprapubic pressure to empty it) or provoke emptying by stroking their thigh/scrotum/falina. If the patient can do this well, this should be adequate. If not and for LMN (bladder lesions) check a residual immediately after he has expressed - if there is a significant residual intermittent ‘semi-sterile’ (!) catheterisation is taught. The patient should self-catheterise 6-8 hry with a Jacques catheter lubricated with soap and water, and either bol or apply antiseptic to the catheter afterwards. In practice many patients fail to cope with ISC and request an indwelling catheter - this is a temptation that should be avoided at all costs! (Urinary sepsis is the major cause of death in paraplegics). Quadruplegics will require a long-term urethral catheter.
- **Sloot softener (liquid paraffin +/- glycerine suppositories) with intermittent manual evacuation as required.**
- **Subcutaneous heparin 5,000 u. hly.**

Patients with incomplete transections must be transferred to Nkuziazane / King George Hospital in appropriate immobilisation, either on a long spinal board and/or in a hard collar.

**Further Reading**

3. Hall VB & Davies WJ 1988 A swing to intermittent clean self-catheterisation as a preferred mode of management of the neurogenic bladder for the tetraplegic spinal cord patient. Paraplegia 26:405

We see ~150 burns patients in O.P.D. each year - more than three quarters are aged less than 6 years and burns comprise 25% of all paediatric surgical admissions. Though much cooking in our district is done over open fires or paraffin stoves, 60% of burns in our hospital are related to scalding from hot water/tea/cook. Though burns probably contribute only 3% of accidental childhood deaths in RSA the resulting morbidity is considerable - furthermore, in an audit of burn patients in Hklusa, many of those with a prolonged hospital stay, repeated surgery and those ultimately requiring transfer were considered to have been inadequately managed in at least one respect.

**Assessment**

1. **Attend to A.B.C’s first.** All burns >10% or signs of shock require an i.v. line immediately. Wrap children in a warm blanket.
2. **Assess the extent of the burn according to Lund and Browder charts.**
3. **Make some estimate of the depth of the burn viz.**
   - **Superficial:** blistered, hypenaemic, painful and moist with normal hair.
   - **Indeterminate:** waxy, white with few hairs.
   - **Deep:** leathery eschar, dry, painless with thrombosed vessels and no hairs.

**LUND AND BROWDER CHARTS**

**IGNORE SIMPLE ERYTHEMA**

<table>
<thead>
<tr>
<th>Age</th>
<th>1-5</th>
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<th>10</th>
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<tr>
<td>Head</td>
<td>10%</td>
<td>9%</td>
<td>7%</td>
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<td>Thigh</td>
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<td>Leg</td>
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All adults with >15% or children with >10% burns or burns to the hands/feet, perineum or face should be admitted. Fire and electrical burns are usually indeterminate or deep and may conceal unsuspected visceral injury and should usually be admitted.

Management

I.V. fluids are indicated for all adult burns >15% and all children's burns >10% or in any patient with signs of shock. Fluid requirements are calculated as follows - % B.S.A. of burn x Body wt. (in kg) ml.

FOR CHILDREN (<6 YEARS) MULTIPLY THIS VOLUME X 2

This volume of fluid should be administered:
- in the first 6 hours
- again in the succeeding 16 hours
- again in the following 24 hours

It is important to remember that these timings are calculated from the time of the burn.

Though about 2/3 of our patients present within 24 hours, be aware that their fluid deficit may already be 1-2 litres by the time they arrive. Always catheterise >30% burns and any patients with 10-30% burns whose urine output <0.5 ml/hr.

Give Morphine 0.2 mg/kg i.v. or 4-6 hourly titrated to analgesic effect. Tildine drops (1 drop per year of age - max. 10 drops) given sublingually 3-4x/day are effective and convenient for children.

Keep the patient warm.

Do not prescribe antibiotics routinely. It is usual for a 'burns fever' to persist for up to 72 hours. Penicillin and Gentamycin should however be given if the patient becomes systemically unwell during this period (possible early beta-haemolytic streptococcal infection).

Beware of the potential for contractures especially in flexural burns - employ splints (or less reliably) passive stretching exercises early in an effort to avoid these.

Circumferential deep burns of limbs or trunk should be released by immediate escheratomy - this is a simple procedure which does not require anaesthesia/analgesia in most cases and will prevent ischaemic respiratory complications developing.

A high energy/protein diet should begin as soon as possible but many burns patients have delayed gastric emptying which may make feeding difficult. Our policy has been to start oral intake as soon as the patient can tolerate it but in severely burned patients early n.g./n.j. feeding is preferable. Use Ensure supplements or a high calorie diet supplemented with eggs/milk/oil.

Check Hb, U&Es and albumin in patients with extensive burns. Blood loss and haemolysis may occur in the early stages so repeat after 2-3 days. An albumin <15 and Hb <8 will almost certainly mean an unsuccessful graft.

Wound Management

There are three options for dressing burns in our hospital -

1. Exposure to air with application of Povidone iodine ointment.

2. Application of thick occlusive dressings for the duration of healing (not less than 10 days). These 'burn sandwiches' must be at least 2 cm thick to prevent 'strike-through' and bacterial superinfection of the wound.

3. Application of semipermeable occlusive dressings (e.g. Omiderm) for the duration of healing.

The first cleaning/debridement of a burn and application of any occlusive dressing is best done in theatre under ketamine and sterile conditions. Subsequent infection should be treated with topical 0.05% Chlorhexidine solution. Silver Sulfathiazine cream is preferred to Povidone-iodine on the face.

Hands with superficial burns are best managed with a glove/plastic bag filled with Povidone-iodine and passive stretching. Deep burns on the hand should be referred early to Clainwood for excision and grafting.

Perineal burns are a special infection risk. Change the dressing with each nappy! Very problematic perineal burns have occasionally required referral for a defunctioning colostomy.

All superficial burns should heal satisfactorily in 21 days (or at least show significant re-epithelisation).

Indeterminate burns should be observed and debrided as required. Povidone-iodine 5% cream or Aserine may be useful for light sloughs, but surgical sloughectomy will often be required when the slough is mature at 7-10 days. Watch for any signs of early streptococcal infection or later gram negative colonisation. Proper wound care will help to prevent these from converting to deep burns. If healing is not apparent by 21 days, SSG is required. This type of burn is our most common indication for grafting.

Deep burns >2x2 cm should ideally undergo early excision and SSG. Excision is commonly accompanied by haemorrhage but this usually responds to the application of gauze soaked in Adrenaline 1 in 15000 solution. Sequential excisions may be necessary esp. in small children who tolerate blood loss poorly. In practice, deep burns can be grafted piecemeal as different parts of the eschar mature/separate as long as the underlying granulations are healthy.
SSG at Hlabisa is feasible/appropriate for up to 15% burns. Maximise the chances of success by ensuring that the wound is clean, well-prepared (properly debrided and not over-granulated) and the patient is not anaemic.

Burns >60% have a very poor prognosis and palliative care is most appropriate. Patients with 15-60% burns should be discussed with Ngwelezane/King Edward Hospital. Ensure that the patient is adequately resuscitated prior to transfer.

FURTHER READING
2) Somamito GO 1993 'Burns care in Africa: reducing the misery index' J Burns Care and Rehab 14:586

LIGHTNING INJURIES
These are super high voltage (10 x 10^6 V) D.C. discharges of very brief duration. Fortunately the extremely high current (5 x 10^3 A) generated usually 'flushes over' rather than through the body and the effects are therefore different from other high voltage electrical burns. About 65% of victims survive a direct lightning strike but deaths occur with some regularity in Zululand during the summer months and a considerable body of folklore is connected with protection from lightning. Cardiopulmonary arrest usually from asystole but sometimes from ventricular fibrillation is a frequent occurrence. Sinus rhythm will often quickly resume spontaneously but apnoea due to respiratory centre paralysis may persist for longer than 15 minutes - though very few of our cases reach us within this period, prolonged resuscitation under such circumstances may be appropriate. Persistent myocardial damage is uncommon, but ST changes will be evident on ECG in 10-30% and arrhythmias may occur - admission for 24 hours for observation is recommended.

Burns are typically superficial with a splashed-on appearance (Liebenberg's figures) and entry/exit wounds are rarely seen. Muscle damage with associated compartment syndromes or rhabdomyolysis is uncommon. However, a Potassium and Creatinine should be checked during admission and the urine dipsticled for myoglobin.

Neurological complications are frequent and include peripheral (usually motor) neuropathy, transverse myelitis and encephalopathy. Recovery may follow within hours or days but permanent disability may result. Spinal compression and long bone #s esp. hip may follow tetanic spasms and trauma during the injury. Cataracts may also develop acutely and at an interval.

FURTHER READING

THE FORESKIN
Removal of the foreskin is usually requested for one of four reasons -
- 1. Tight phimosis in a child - usually accompanied by spraying, ballooning of the foreskin or pain on micturition. The parents should be reassured that it is normal for the prepuce and glans to be adherent in infants, and only 50% of foreskins are retractile by the age of two years. In infants, operation should wherever possible be deferred until six months or older. In the case of a very tight foreskin that doesn't pout at all on attempted retraction surgery should be considered at an earlier stage. Gentle dilatation of the prepuce with a soft plastic suction catheter may be a helpful temporizing measure.
- 2. Acute paraphimosis should be reduced - unless a dorsal slit is required to relieve it, the foreskin is already infected or sloughing or the condition recurs, a circumcision need not follow.
- 3. Neglected or complicated STDs - this is sometimes appropriate for severe genital warts but poor hygiene is also a major factor 'you wouldn't cut off your ears just because you couldn't be bothered to wash behind them'. In many cases, washing with soap and water/gentle daily retraction may be all that is required and determined treatment for STDs/balanitis should be pursued before or after operation is considered.
- 4. Less clearly specified sexual/cultural reasons - though ritual circumcision was abolished in the Shaka era some rural people do still circumcise and there seems to be a quite widespread belief amongst Zulu men that circumcision will protect them against STDs. Though there is some evidence that this indeed the case the patient should be advised that barrier methods of contraception are far more effective.

HYDROCOELE
Primary vaginal hydrocoele in older men are a common presentation in OPD - they often obtain massive proportions! They are generally idiopathic but may be related to chronic epididymo-orchitis esp. T.B. or malignancy (in which case they are usually smaller and the testicle may be palpable). Conservative management may be attempted in older patients with repeated drainage. Always palpate the testicle after drainage - if it is abnormal or tender treat as per urethritis, get a testicular
US/S and α-FP, β-HCG. Order EMUs for AFBs in the older patient. Where conservative management fails, the patient can be referred for hydrocelectomy (younger), or adhesion can be carried out (instil 250 mg Terracycline in 15 mls of Na/Saline + 5 ml lignocaine 2% into the hydrocele).

In infants, congenital hydrocele due to a patent processus vaginalis usually subsides spontaneously and only requires hernioplasty if persistent at one year. The condition is commonly bilateral, the swellings transilluminate and the testis is easily palpable. It must be distinguished from an incarcerated indirect inguinal hernia. These should be referred promptly for repair within two-three weeks since the risk of early complications is high.

THE TESTICLE

Epididymo-orchitis is common in our patients so be cautious about a diagnosis of torsion in sexually active patients - however a clear history of trauma, an elevated firm, tender testicle with little epididymal tenderness and an absence of penile discharge or pyuria on MSU warrants an exploration. In children (<12 years) always assume the worst. In either case the sooner the operation is carried out the better - however some patients come inexplicably late and there is certainly no rush to explore the testicle after 24 hours. We have found that the best policy is to remove the tooted testicle and do a contralateral orchidopexy at the same time, as few patients can be persuaded to return at an interval when the risk of infection is less.

Undescended testis is found in - 5% of full term infants. This is of no concern unless the testis remains impalpable at one year or there is an associated hernia which requires immediate attention, when they should be referred for assessment/orchidopexy.

RETENTION OF URINE

Both prostatic disease and urethral stricture (due to STDs/Trauma) are common in our patients. Prostatic Carcinoma is the 2nd commonest tumour of black males in RSA with an incidence rate of ~ 15 per 10,000/yr (about 2/3 rate in white males). The age distribution is similar to other populations. However, ~ 50% are histological stage D at presentation and a higher Gleason grade than in European patients can be expected. This may well be due to late presentation since most patients in our district with prostatic disease first present to the hospital in retention. Acute retention of urine is characterised by pain and bladder tenderness - beware of catheterising the patient with chronic retention and overflow, as a spectacular diuresis and pre-renal failure may follow. Given the high incidence of urethral stricture, do not persist or use an introducer if passage is difficult - suprapubic puncture is safer. For all cases, check Hb, U/E and Lumbar Spine/CXR. Treat for U.T.I. in all cases as almost all have a precipitating infection. Do a P.R. when the bladder is drained. Where a urethral stricture is suspected refer for optical urethrotomy or urethralplasty at King Edward Hospital.

If the prostate is clinically normal send a P.S.A. and arrange for a trial without catheter in 1 week. If the P.S.A. >4 then do a prostatic biopsy (P.S.A. <4 has a 90% negative predictive value but poor positive predictive value). If the T.W.O.C. fails many patients will elect to keep their catheter in which case a Foley should be changed every 4 weeks and a Silastic every 3 months. T.U.R.P. can be arranged electively at Ngwelezane if desired.

If the prostate is suspicious do a prostatic biopsy and arrange a trial without catheter in 3-4 weeks when the histology is back.

If the patient elects to keep his catheter or if there is evidence of metastases, he should be prescribed Ciprofloxacin 100mg i.d.s or a subcapsular orchidectomy can be performed

If there is no evidence of metastases and the patient wants the catheter out, then refer for a Channel T.U.R. and Orchidectomy at Ngwelezane.

STONE DISEASE

This is not unknown in Zulus though we see few convincing presentations of renal colic - usually mixed stones resulting from Primary Hyperoxaluria or struvite stones from repeated UTIs. The vast majority of patients will pass their stone with conservative management in 24-48 hours. If the pain persists and no stone is passed do an US/S or I.V.U. (depending on the Ct). Patients with stones causing obstruction should be referred to King Edward Hospital for ureteroscopy or nephrostomy.

Further Reading
1) Steenkamp and De Cock 1994 'Epidemiology of urethral stricture at Tygerberg hospital' SAMJ 84:267
2) Bereczky 1967 'A pathological analysis of clinical adenocarcinoma of the prostate in black patients' (letter) S.A.M.J. 97:344
4) Uzassa M et al 1997 'Male circumcision and susceptibility to HIV infection among men in Tanzania' AIDS 11:73
HAND INJURIES

FRACTURES
Finger #s usually take 3 weeks to unite. The key principles of management are active movements as soon as possible for all stable #s and avoidance of rotational deformity - all the fingers should point towards the scaphoid when they are flexed.

All phalangeal shaft #s not transtressing a joint are suitable for early mobilisation with neighbour strapping. Those which are very displaced or angulated should be reduced first.

Intra-articular #s can be referred for insertion of K-wires unless very comminuted but adequate results can also be achieved with splinting.

Flexor avulsions of the terminal phalanx should be referred for internal fixation but extensor avulsions (mallet finger) are satisfactorily managed in a plaster mallet splint (PIP free to move, DIP fully extended).

Metacarpal #s are intrinsically stable because they are splinted together by strong ligaments - if there is a lot of rotation or overlap reduction should be attempted. A crepe bandage for protection is usually all that is required. A cock-up splint is better if the # is very angulated and requires reduction. Multiple MC #s are less stable and internal fixation is preferred.

Distocated #s usually result from extreme hyperextension and must be urgently reduced. Splint MCP joints flexed and IP joints extended if the reduction is unstable. Neighbour strapping and early active movements can be started if stable. MCP dislocations frequently ‘button-hole’ through the volar plate and become irreducible - they usually need to be referred for open reduction.

TENDON INJURIES
The usual cause of these in our practice is a #knife wound though they may be part of a much more extensive injury. We attempt simple extensor and zone I flexor repairs as a primary procedure in our hospital.

A modified Kessler suture (4/0 Ethibond) + a running 6/0 Prolene epitendinous suture is used (this probably disturbs the tendon’s blood supply least).

Extensors - E. digitorum communis is supplemented by E. indicis proprius and E. digitii minimi but repair of the last two is not essential so almost all repairs on the dorsum of the hand involve one layer of tendons. Since they have a common muscle bulk the tendons tend not to retract too far. Be sure to exclude an acute boutonierre injury by Elson’s middle slip test - flex the PIP joint over the table-edge and ask the patient to extend it against resistance. If the middle slip of the extensor expansion is intact, the finger lifts and the DIP joint is lax. If it is not, there is no PIP extension and the DIP joint stiffens and extends. These injuries should be referred.

Flexors - Zone I repairs (distal to the proximal IP crease) are distal to the insertion of E. digitorum superficialis and only involve the profundus tendon. Always check that superficialis is intact by doing Apley’s test-anchor the other fingers flat on the table and ask the patient to Flex the PIP joint. If this is impossible (FDP cut/difficult (FDP intact) and the DIP joint is tense the superficialis tendon is interrupted.

Simple Zone V injuries with no vascular or neurological involvement can also be attempted and

STEADY THE PHALANX

ELSONS MIDDLE SLIP TEST
If the middle slip is ruptured, there is no extension of the P/P joint and the D/P joint stiffens and extends a little.

3 SLIPS OF THE EXTENSOR EXPANSION.
The diffusely swollen hand - is probably the commonest presentation we see. It often follows a human bite or a thorn prick and presentation has invariably been delayed for some days. Deep space suppuration is almost always established by this time so exploration is usually warranted. X-rays may already show changes of osteomyelitis. Look specifically for Karavel's sign of flexor tendon sheath infection (flexed swollen finger, pain on passive extension and pin-point tenderness over the sheath). Certainly if there is no response to Clavulacillin 1g 6hrly i.v. Gentamicin 240 mg o.d. i.v. + Metronidazole 1g b.d. p.r. m 12-24 hours surgery is mandatory. Explore the thenar and mid-palmar space and if necessary expose and irrigate the tendon sheaths (butterfly needle tubing is about the right size). Leave corrugated drains in the infected spaces. Splint the hand in the James position immediately post-op (30° dorsiflexion at the wrist, 90° at the MCP joints and fully extended IP joints) with a volar POP slab. Elevate the hand post-op and give daily hand baths. Start physiotherapy with appropriate analgesia on the second day. If progress is clearly slow re-exploration/debridement of necrotic tissue should be done - in practice this is often the stage at which an IP amputation or MCP disarticulation is carried out for a clearly non-viable/useless digit.

Further Reading
1) Menken U & Hawelis CJ 1991 ‘Human bite-bite injuries of the hand. A study of 100 cases within 18 months’ J Hand Surgery 16: 431
2) Nevisser RJ 1978 ‘Closed tendon sheath irrigation for pyogenic flexor tenosynovitis’ J Hand Surgery 3: 452
3) Lindsay JR 1995 ‘Tendon injuries: how to recognise and treat them’ C M E 14: 963
4) Evans JD et al 1995 Results of extensor tendon repair performed by junior accident and emergency staff Injury 26: 107

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**SNAKEBITE**

Incidence - 80 per 105/year in our health ward with peaks in spring and summer. Though the majority of snakebites (65% of cases admitted) require no specific treatment, some local snakes are highly venomous:

- **Puff adder** (Bitubousa, Bitubula) - severe cytotoxicity sometimes progressing to haemalological effects especially thrombocytopenia
- **Mozambique spitting cobra** (Bitubula) - similar to the above and may also cause snake venom ophthalmia.
- **Black and green mambas** (Field): rapidly progressive and potentially lethal neurotoxicity.
Most of our patients are seen more than 8 hours after the bite and will often have had a tourniquet applied and traditional remedies (isibiBala) administered. Almost all are children and they, in particular, should be managed aggressively - in 1994 the case fatality rate from snakebite in our hospital was 5% and all these deaths occurred in children.

**GENERAL POINTS**

- Treatment is based on the clinical effects of envenomation and not on a presumptive or even positive identification of the snake involved.
- All children with ANY signs of envenomation must get an i.v. line.
- Admit to I.C.U. - all severe cases and all under-fives with anything but minimal swelling.
- All other cases with any degree of local swelling should be observed in O.P.D. overnight, or at the ward during the day, until stabilised (usually 24 hours). Local swelling may subsequently progress in ≤25% so baseline and repeated observations (leg circumference/level, B.P. Pulse) are crucial.
- Only cases without ANY clinical manifestations of envenomation and with normal blood tests can be discharged from observation in O.P.D. after 8 hours.

**Assessment**

The vast majority of bites are cytotoxic with local pain and local swelling most prominent. A severe bite is defined as:

- swelling of the whole hand or foot 1 hr
- swelling to the elbow or knee 6 hrs
- swelling to the shoulder or hip 12 hrs from time of bite

Any neurological symptoms such as confusion, blurred vision or neurological signs such as weakness, paresis, dyspnoea or hypersalivation should be taken seriously and antivenom given.

All bites regardless of severity should have FBC, U & E, Prothrombin time and Partial Thromboplastin time taken. A twenty minute whole blood clotting time should also be done and reviewed before the patient goes to the ward.

**Treatment**

Analgesia - paracetamol +/- codeine for routine use. Stronger opiates with caution in the more severe cases. Elevate the bitten part. i.v. fluids for ALL children and in adults with anything but minimal swelling.

Antibiotics are not necessary in the majority of cases. Only cases with severe swelling as defined above should get antibiotics initially but they should also be started in cases with later tissue necrosis. The current recommendation is Chloramphenicol, Gentamicin and Metronidazole for 5 days.

Give tetanus toxoid where appropriate. Small children, under the age of 5 years, with moderate or severe bites whether requiring antivenom or not, should be observed on I.C.U. until clearly improving.

Polyvalent antivenom is effective against all the most dangerous snakes (except the boomslang or vine snake, bites from both of which are characterised by delayed bleeding problems and for which a specific antivenom can be obtained on request from S.A.I.M.R. - Tel. (011) 882 9948). The earlier it is given the better and only in exceptional cases should it be given more than 12 hours post - bite. There is a high incidence of anaphylaxis however and it should be reserved for the indications listed above. Shocked patients need may need vigorous resuscitation prior to antivenom though rarely cardiovascular collapse may be a direct effect of the venom itself and may not resolve until antivenom is given.

Absolute indications for anti-venom are:

- Neurotoxicity
- Severe Cytotoxicity as defined above
- Clinically important systemic bleeding

Relative indications are:

- Bites on fingers (risk of local necrosis)
- Bites on face
- Sub-severe swelling with other features of non-specific envenomation esp. haematological disturbance.
- Small children with signs of envenomation

Antivenom is only ever administered in I.C.U. with resuscitation equipment immediately to hand. The standard dose is 4 VIALS for all patients including children. In cases of neurotoxic envenomation 10 VIALS should be given. Dilute the antivenom in 200 mls N. / Saline and start the infusion at 1 ml/minute. Some doctors give 0.25 ml 1:1000 subcutaneously into the forearm immediately prior. Stay with the patient for 15-30 minutes.
The most common adverse reaction in our experience has been urticaria but this may presage more serious anaphylaxis. In such cases, stop the infusion and give adrenaline 1 in 1000 i.m. or s.c. (NOT i.v.) 0.1 ml/kg up to age 5, 0.5 ml 5-12 years and up to 1 ml in over 12s. + Promethazine 25 mg stat i.m. If the indications for antivenom are strong, the infusion may be continued under cover of repeated doses of adrenaline as required, but should hypotension, bronchospasm etc. develop, it must be discontinued.

Patients with neurotoxic bites should also be given atropine 0.6 mg (adult) or 0.05 mg/kg (child) followed by adrenalinium 10 mg (adult) or 0.25 mg/kg (child) in order to establish whether neuromuscular blockade is complete. If improvement can be demonstrated this may be possible to maintain the patient on Neostigmine 0.05-0.1 mg/kg 4 hours whilst the antivenom is administered.

However, in some cases, temporary intubation and ventilation will be required. 10-20% of snakebites require surgery in our setting. This is usually manifested by early blistering but debonding/exploration is best delayed until 7 days, when necrotic tissue has had time to demarcate and the systemic effects have subsided. The typical pattern of necrosis of a bitten limb is of dead skin extending up the dorsal aspect overlying an often extensive area of fat necrosis which can lead to continued infection if not debrided. Tendons may be deranged and subsequently necrose. Fasciectomy for compartment syndrome in the early phases of snakebite is virtually never required and may be hazardous. Closure by secondary suture or SSG is the rule and careful splitting of the limb is necessary to prevent contractures/ deformity/stiffness.

Patients with neurotoxic bites should be admitted for five days as delayed relapse of toxicity is described. Serum sickness may occur 7-10 days after administration of antivenom with a rash, arthralgia and fever. It should respond to a short course of prednisolone.

Further Reading
4) Pramanikadchene AF et al 1999 “Low dose subcutaneous adrenaline to prevent acute adverse reactions to antivenom serum in people bitten by snakes: randomised placebo-controlled trial” BMJ 318:1041

**DOG BITE**

This is a common problem in O.P.D. especially in children and usually presents no serious problems. However, KwaZulu-Natal experiences regular epizootics of synaletic rabies and since 1976 dog rabies has spread from Swaziland and Southern Mozambique, establishing itself in many peri-urban settlements. Approx. 20 cases per year are reported in the region (~90% of cases reported in RSA) and at least four have occurred in the Hlabisa district since 1994. The risk of contracting rabies from the bite of a known rabid animal is ~50% overall. Most bites occur in children and often when passing/entering a neighbour’s kraal. Treat a complaint that the dog is immunized with some suspicion unless the owner has been able to produce a certificate - more than 1000 dogs are immunised each year in our district but the majority are not.

There are only 20 reported cases of vaccine failure worldwide, including one in South Africa, mostly amongst patients in whom vaccination was delayed or incomplete. All four forms of rabies virus (Classical, Mokola, Lagos bat and Duvenhage) occur in RSA but vaccination is thought to confer protection against them all. Be wary of head/neck and upper limb bites – ensure they are reviewed early. The overall infection rate from bacteria in dog bites is probably around 5%.

**DO NOT TAKE CHANCES** in management of dog bites!
Rather subject a child to 3-4 injections than admit her six weeks later with rabies.

The history is all important and determines the management. Always ask the following questions -

**IS THE DOG KNOWN TO YOU?**

1. **NO**
   - Behavior abnormal?
     - Unprovoked bite?
     - RABIES PROTOCOL
   - Dog well?
     - Explanatory bite?
     - Known, aggressive or immunised dog?
     - EXPECTANT MANAGEMENT

2. **YES**
   - Behavior abnormal?
     - Unprovoked bite?
     - RABIES PROTOCOL
Wound Management (for all patients)

Clean thoroughly under L.A., encourage bleeding. Some larger/dirty wounds will require a formal debridement in theatre.

Delayed primary suture should be the rule. Certainly do not suture if >8 hours old, frankly infected or there has been suspected rabies exposure. Give *Latanus toxoid* where appropriate. *Antibiotics* - Give prophylactic Ampicillin and Clavulanic for 3-5 days if the wound is clinically contaminated or if patient presents <6 hrs after the bite (not usually required). This is NOT a substitute for an adequate debridement!

### RABIES POST-EXPOSURE PROPHYLAXIS

Indicated as above but categorised according to the severity of the bite:

<table>
<thead>
<tr>
<th>Category 1</th>
<th>Category 2</th>
<th>Category 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Touch/lick of intact skin</td>
<td>Touch/lick of broken skin</td>
<td>Full thickness bite (Transdermal, after with bleeding)</td>
</tr>
<tr>
<td><strong>Expection</strong></td>
<td>Rabies Vaccine</td>
<td>Rabies Vaccine (Anti-Rabies Immunogloobulin)</td>
</tr>
</tbody>
</table>

**Rabies Vaccine**

We are now using human diploid cell vaccine according to the standard (Essen) schedule viz. 1 ml i.m. into the deltoid (adults) or thigh (children) on days 0, 3, 7, 14 and 28. An extra dose should be given on day 90 for Category 3 exposures since immunoglobulin is thought to interfere with immunisation. If vaccine is given >48 hours after the bite, double or triple the first dose. If there is doubt about follow-up, it may be appropriate to admit the patient for the first 2 or 3 doses. The schedule can be stopped if the suspect dog remains well after the third dose, or if it is subsequently killed, and found to be free of rabies.

**Anti-Rabies Immunoglobulin**

Give 20 i.u. /kg bodyweight (1/2 deep into the wound, 1/2 i.m.). N.B. Dose in ml = Body weight (kg) x 0.13

This may still be effective if given <1 week after exposure. Do not repeat as anaphylaxis can occur and other live immunisations may be hampered.

Always phone the Dept. of Agriculture in cases of suspected rabies exposure. 0358 381044

They will destroy/quarantine the animal and examine it for rabies.

**Rabies Cases**

Hopefully you will never be called upon to manage one of these. The incubation period is <90 days in 80-90% of patients. In South Africa the mode is 34 days. A brief prodrome may occur often with paraesthesiae at the site of the bite. This is followed by agitation/hyperactivity/hallucinations reflecting rabies encephalitis, although rarely a 'paralytic' form occurs in man. Death is inevitable, often following within hours of admission, and heavy sedation (diazepam, phenothiazines) should be administered until this occurs. Administration of immunoglobulin or vaccine at this stage is contra-indicated. Rabies is a notifiable disease and it is vital that post-mortem specimens are obtained for submission to The National Institute of Virology - if the family decide consent authority may be sought from the magistrate or medical superintendent under the Human Tissue Act of 1983.

Further Reading

1) Department of Health 1997 Guidelines for the medical management of Rabies in South Africa
WOUNDING ENERGY

Kinetic Energy = ½ MV^2

The formula suggests the greater seriousness of high velocity wounds but ‘high’ and ‘low’ are relative terms and wounding energy is also highly dependent on transfer to the tissues e.g. contact with a high density tissue like bone dissipates a lot of energy (see diagram).

A-B: Small Entry and Exit, minimal tissue damage
A-C: Small Entry and Large Exit (Classic High Velocity Wound)
A-D: Small Entry and 2-3cm Exit (common, needs debridement)
C-D: Large Entry and 2-3cm Exit (Unstable bullet, shrapnel)

- HIGH VELOCITY wounds (e.g. AK-47) cause greater tissue damage through tumbling/yaw and the ‘cavitation effect’ affecting 10x the diameter of the bullet and sucking in debris via the exit/entry wounds. However, many of the technically ‘high-velocity’ wounds that we see are ‘through and through’ limb injuries that may only dissipate 10% of their wounding energy and cause little damage.

- LOW-VELOCITY wounds (e.g. pistol, home-made weapon) are much more common. They usually create an uncomplicated track with little or no concealed damage. The bullet often lacks sufficient energy to exit the skin and may be felt subcutaneously.

- SHOTGUN wounds vary in their seriousness according to range. At <5 m the wound is compact and may penetrate deeper tissue with wadding and cartridge plastic embedded. At longer ranges the wound is more scattered, and the pellets cause less damage, usually just embedding themselves in the skin.

Tissue effects vary - the more dense and less elastic, the more damage it sustains. Lung, fat and skin are therefore relatively preserved whereas muscle, bone and liver may suffer more severe damage.

Management

SIMPLE WOUNDS
(no bony or neurovascular involvement)

The track does not require debridement as long as the wounds are <1cm in size. Admission is only indicated for functional reasons e.g. can’t walk. Give penicillin and tetanus toxoid.

COMPLICATED WOUNDS
(usually bony involvement)

No matter how comminuted the # if the wounds are <1 cm in size, treat as a closed injury without debridement.

If the wounds are >1 cm in the presence of a fracture or neurovascular injury, the injury should be debrided - aim to leave a clean, viable wound suitable for reconstruction. Loose pieces of bone stripped of soft tissue attachments should be removed and tendons without synovial cover should be excised. Early inspection of the wound at 48 hours with a view to repeat debridement is important. Closure should normally be delayed primary or by SSG unless the wound is highly favourable after the first debridement. Give Penicillin/Cloxacillin iv initially then oral for 3 days.

Vascular injuries of minor vessels require haemostasis only but interruption of a critical trunk or clear evidence of compromised perfusion requires the urgent attention of a vascular surgeon.

Nerve injuries are often in continuity (~80%) and may be managed expectantly - a delayed repair at 3 weeks may be attempted if no improvement occurs, but failure to improve usually signifies a nerve that has been irretrievably damaged. However, a large interrupted trunk discovered at initial debridement which is not completely blown apart should probably undergo primary suture.

Internal fixation will often be the method of choice for shattered femurs but most other #s should be managed with traction/closed methods.

The only bullets that should be removed are those under the skin or inside joints.

Further Reading
1) Kingsley-Brown A. ‘Gunshot wounds - then and now’ J.R.C.S. Edin. 12/89 302-305
2) Swan and Swan 1931 ‘Principles of ballistics applicable to the treatment of gunshot wounds’ Surgical Clinics of N America 11:2 221
3) Coupland R ‘War wounds of limbs’ J.R.C.S.
Bone and Joint Sepsis

Septic arthritis usually presents as an acute monoarthropathy of a weight-bearing joint (30% lower limb - knee commonest site in adults, hip > knee > ankle in children). *S. aureus* is the dominant pathogen (40%+ in children), though streptococci are responsible for 10-20% of cases in all age groups and may be associated with a slow response to treatment and joint destruction. *N. gonorrhoeae* must be considered in sexually active adults. *H. influenzae* is an important pathogen in children under 2 years. HIV +ve patients often have atypical pathogens especially *S. pneumoniae* and Gram-negative organisms. Polyarticular involvement may accompany a slash/strep bacteraemia and be present in 40-50% of infants. About a third of cases have an evident primary focus for the bacteraemia and in some other serious sepsis e.g. pneumonia may be present.

Haematogenous osteomyelitis typically affects the metaphyses of long bones in children but vertebral osteomyelitis/discitis can occur in the over 50s - 90% of cases are caused by *S. aureus*. Infection spreads through the cortex causing thrombosis of the intramedullary vessels and ultimately emerging on the bone surface as a subperiosteal abscess. This interrupts the blood supply from the periosteum and infects the involved cortex. In infants, vessels cross the growth plate and infection rapidly spreads into the epiphysis/ neighbouring joint. In older patients the growth plate has the effect of containing the infection except in the hip where the very distal envelopment of the femoral head by the capsule makes spread into the joint very likely.

Pyomyositis is sepsis in the deep tissues of a limb often following minor trauma - it is common in our practice and is increasingly seen in young adults who are often HIV +ve. *S. aureus* is invariably responsible.

Diagnosis

Is difficult as the patient/parent will usually give a history of prior trauma and gait marks may already have been made by a traditional healer - do not assume that it is those that have caused the inflammation! Important differentials of acute monoarthritis in adults are crystal synovitis and reactive arthropathy. In children be on the look-out for Rheumatic fever (classically migratory) and Perthes disease. Beware of a diagnosis of irritable hip/ transient synovitis as this seems to be uncommon in our patients. Also treat a diagnosis of cellulitis in an adult with suspicion especially if slow to respond to treatment and virtually never entertain it in a child! Fluoroscint in pyomyositis is a late sign so don't let its absence delay surgery.

The diagnosis of bone/joint sepsis is essentially clinical as laboratory investigations (WBC, ESR) have low sensitivity - Diagnostic aspiration of the joint *should* always be undertaken when there is the slightest suspicion of septic arthritis, usually under GA in smaller children.

Gram stain and culture should be performed on all aspirates/surgical specimens (both to confirm the diagnosis and identify non-staph organisms).

Do NOT expect x-ray changes - these do not usually occur for at least 5 days after the onset of symptoms in neonates/infants and 10 days in adults. The only appearances to be expected in acute septic arthritis are early soft tissue swelling/joint space widening. The sequence of changes in osteomyelitis is metaphyseal rarefaction > periosteal elevation > avascular necrosis (usually 'normal'-looking area of bone as the rest becomes rarified) > periosteal new bone formation > sequestrae/cloacae. X-rays are however helpful to exclude other causes of bone pain (e.g. slipped epiphysis, fracture).

<table>
<thead>
<tr>
<th>Volume</th>
<th>NORMAL</th>
<th>INFLAMMATORY</th>
<th>SEPTIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>ml</td>
<td>3.5</td>
<td>&gt;3.5</td>
<td>&gt;3.5</td>
</tr>
<tr>
<td>Viscosity</td>
<td>High</td>
<td>Low</td>
<td>Variable</td>
</tr>
<tr>
<td>Colour</td>
<td>Clear</td>
<td>Yellow, translucent</td>
<td>Yellow-green, turbid</td>
</tr>
<tr>
<td>WBC/mm³</td>
<td>&lt;2000</td>
<td>2-10 x 10³</td>
<td>&gt;10 x 10³</td>
</tr>
<tr>
<td>PNMLs</td>
<td>&lt;25%</td>
<td>&gt;50%</td>
<td>&gt;75%</td>
</tr>
<tr>
<td>Glucose = serum</td>
<td>&gt;25 mg/dl</td>
<td>&lt;25 mg/dl</td>
<td></td>
</tr>
<tr>
<td>Gram stain</td>
<td>Negative</td>
<td>Negative</td>
<td>Positive</td>
</tr>
</tbody>
</table>

Management

1. Analgesia, Splintage (skin traction for hips)
2. Give **Clavulanic Acid 150 mg/kg/day i.v. + Erythromycin 30 mg/kg/day p.o.** (two anti-staph agents combined to prevent resistance emerging.) for suspected osteomyelitis. **Substituto Ampicillin** for erythromycin in septic arthritis. **Ceftazidime** is preferred for children under 2 years and for treatment failure in adult gonococcal arthritis.
3. **Septic Arthritis - Initial management** for almost all joints should be immediate arthroscopy - needle
aspiration is probably only effective if the patient presents earlier than 72 hours, and needs to be repeated every 2-3 days according to the response and re-accumulation of fluid. We have preferred S
cor 
drainage and insertion of a corrugated drain for 48-
72 hours as most of our patients present late. All 
septic joints must be splinted post-surgery to prevent 
contractures. Hip joint sepsis requires referral to 
Ngwelezane as surgery is technically difficult and the 
risk of complications high (e.g. necrosis of the femoral 
head with subluxation/pathological #).

4) Pyomyositis - exploration and drainage is always 
required. Long-term complications are rare.

5) Acute osteomyelitis - cases presenting within 48 hours 
can be managed with i.v. antibiotics alone but be wary 
of attempting this in our hospital, as patients rarely 
present so early and a sub-periosteal abscess is almost 
always already present - thus almost all our patients 
require operation. If pus is present in the soft tissues 
and the bone underlying the periosteum is smooth, 
the abscess has already formed a sinus and 
discharged. A drain inserted in the abscess cavity 
may still be helpful however. The bone should be 
drilled only if no such pus is apparent in the soft 
tissues. If there are clinical signs of relapse within the 
first week, seek the patient for a "second look" in 
th there. After the acute stages have settled the patient 
should be discharged on Co-Tetracaine for 6 weeks 
and reviewed at the end of the course.

6) Chronic osteomyelitis - if X-rays demonstrate signs 
of chronic osteomyelitis relapse at some stage is 
inevitable and the patient must be reviewed. Any 
intercurrent relapses are treated with 2 weeks Co-
Tetracaine and incision and drainage of soft tissue 
collections if necessary.

The prognosis of chronic osteomyelitis in the under 
4s is usually excellent - absorption of the sequestrum 
and formation of a strong involucrum progresses rapidly, 
often with full cure in 6 months. In older patients, surgery 
will usually be required at some stage as the sequestrum 
will usually continue for much longer with persistent 
infection. Patients should be referred for assessment at 
Ngwelezane at 12 months for lower limb cases and 6 
months in the upper limb. When the involucrum is ripe, 
sequestrectomy/sauceration is performed + antibiotic 
irrigation/implantation of gentamicin beads. This is 
followed at an interval by packing of the cavity with 
cancellous bone grafts or placement of a muscle flap to 
fill the bony cavity.

Further Reading
1) Morgan OS et al. 1996 "An 18 year clinical review of septic arthritis 
from tropical Australia" Epidemiology and Infection 117(3) 423
2) Learmont ID et al. 1984 "Acute osteomyelitis and septic arthritis in 
children. A simple approach to treatment" S.A.M.J. 65(4) 117 (Jan 28)
3) Serieux A et al. 1997 "HIV infection as a risk factor for septic arthritis" 
Br. J. Rheum. 36(3) 333
4) Chiebere LC. 1979 "Pyomyositis: a review of 206 cases in 112 patients" 
Arch. J. Surg. 137 235

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**OPHTHALMOLOGY**

A community survey of the Inkuvuma district in 
1993 found that 1% of the population were blind, with 
a further 1.4% suffering from impaired vision. The 
commonest causes of blindness were age-related cataract 
(50%) and chronic glaucoma (22.9%). Chronic open 
angle glaucoma is commoner in Africa than anywhere 
else in the world and may present late in relatively young 
patients, but we are currently seeing only 1-2 cases/ 
month. Serious eye infections and penetrating trauma 
are also common causes of morbidity. Of 100 penetrating 
eye injuries referred to the ophthalmology service at 
Edendale, 44 were rendered completely blind in the 
affected eye and only 6 regained an acuity of 6/12 or 
better. There is a serious lack of ophthalmologists in 
the public sector in South Africa. Most rural patients 
depend on the provision of mobile Sight-Saver clinics, 
which last visited Phalabora in 1999. Our eye services are currently 
co-ordinated by a trained ophthalmic nurse practitioner.

**EYE INFECTIONS**

- Chloramphenicol 1% eye ointment applied 6 hourly will 
  usually suffice for uncomplicated bacterial 
  conjunctivitis. Don't pad the infected eye!
- If there is an associated urethral discharge, also give 
  Ceftriaxone 125 mg st 1/2
- For keratitis or frank corneal ulceration revealed by 
  fluorescein staining, add fortified gentamicin 0.3 % 
  drops and consider subconjunctival injection of 20 
  mg of gentamicin, repeated daily as necessary for up 
  to 5 days.
- Where staining shows the characteristic dendritic 
  pattern of herpes simplex, start aciclovir 3% drops 4 
  hourly.
LID PROBLEMS

- Generalised lid cellulitis or infection localised to the lacrimal gland (dacyroadenitis) can usually be treated with oral Penicillin V 500 mg 6 hourly for 5 days.
  Infection of the lacrimal sac (dacycystitis) requires iv Penicillin 500000 iu 6 hourly for 2-3 days, followed by oral Penicillin for a further 5 days. Incision and drainage of abscesses may be required if progress is slow or the lid starts to point.

- Lesion at the margin of the lid may point externally (styes) or internally into the conjunctival sac (meibomian gland abscess - painful - or chalazion - painless). Styes can be managed by epilating the affected lash. Meibomian gland abscesses can be incised and drained and chalazions incised and curetted via the conjunctival surface of the eyelid. All these procedures should be followed by Chloramphenicol eye ointment 6 hourly for 5 days.

- Blepharitis (scaling, ulceration or maceration of the lid margin accompanied by intense itching) can also be treated with Chloramphenicol ointment applied to the lid margins 6 hourly for 2 weeks, and saline bathing to remove scale. Resistant cases sometimes benefit from 1% hydrocortisone cream.

EYE TRAUMA

- Simple corneal abrasions resolve in 48-72 hours: pad the eye for pain relief and give prophylactic Chloramphenicol ointment.

- Foreign bodies can usually be removed simply under topical anaesthesia with a needle or moist cotton-wool bud. If you can't find one, remember to exert the lid! Afterwards treat the eye as above.

- Penetrating trauma to the globe requiring surgical repair requires broad spectrum iv antibiotics, an eye pad and referral within 24 hours for ophthalmological assessment.

- If you suspect an intraocular foreign body, order views of the orbit with the patient looking up and down. If nothing is visible and the history is suspicious refer early for slit lamp examination.

CATARACTS

All mature cataracts with absent red reflex are routinely referred for surgery to Ngwelezane. Most patients undergo Extracapsular extraction (ECCE) and insertion of a posterior chamber intraocular lens if they can afford it (fortunately we have a supply of low cost Indian lenses at Hlabisa), though some have either an intracapsular extraction (ICCE) or failed insertion of IOL after ECCE and need aphakic glasses afterwards. Insertion of anterior chamber lenses has been associated with <30% persistent uveitis and formation of anterior synechiae in ~16% in a KwaZulu-Natal series and is not generally recommended.

GLAUCOMA

- The classical symptoms/signs of chronic (open angle) glaucoma are visual field defects, cupping of the disc on fundoscopy and raised intracocular pressure. Acute (closed angle) glaucoma is usually much more dramatic with severe headache, ocular pain, visual loss and conjunctival injection with a shallow, hazy anterior chamber.

- In acute cases give Acetazolamide 250 mg 4 times daily + Timolol 0.5% drops 12 hourly. When the symptoms have subsided and in cases of chronic glaucoma, give Timoptic 1-4% drops B.I.D.

- Refer all acute cases for trabeculectomy. Many chronic cases will already have such a degree of visual loss that surgery is unlikely to be helpful and medical therapy is often preferred.

GLASSES

Supplies of glasses for refractive errors from -3 to +4 and Aphakic glasses are available from the hospital for R50 (c/o Hilliar's Optometrists in Pietermaritzburg).

Further Reading
2) Cook CD et al. 1993 Prevalence and causes of Low vision and blindness in Northern KwaZulu. S.A.M.J. 83:596
3) Cook CD et al. 1998 Is anterior chamber lens implantation after Intracapsular lens extraction safe in rural blacks patients in Africa? A pilot study in KwaZulu-Natal, South Africa. Eye 12:321
4) Cook CD et al. 1991 Perforating eye injuries in KwaZulu S.A.M.J.
<table>
<thead>
<tr>
<th>CONDITION</th>
<th>CLINICAL APPEARANCE</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FUNGAL INFECTION</strong></td>
<td></td>
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</tbody>
</table>
| Candidosis                     | **Pseudomembranous** Creamy white/yellow plaque that wipes off to leave a red/bleeding surface, most commonly occurs on the buccal and labial mucosa, tongue and hard and soft palate.  
May involve any part of the oral mucosa. | Nystatin pastille (100,000 units, one to two pastilles dissolved in mouth 4-5 times a day), vaginal troches or fluconazole 50-100mg qds for 1-2 weeks/Fluconazole 150mg stat. |
| **Atrophic**                   | Red lesion that appears on any surface of the oral mucosa, chronic, midline demarcation of tongue and hard palate (C11-MIP)                                                                                           | As above.                                                                                     |
| **Hyperplastic**               | While plaques that cannot be wiped off, most commonly occurs on the buccal mucosa                                                                                                                                    | As above.                                                                                     |
| **Angular cheilitis**          | Red tissues radiating from the corners of the mouth                                                                                                                                                                  | Nystatin/Amphoteric B ointment applied 4 x daily.                                             |
|                                |                                                                                                                                                                                                                       | Meloxicam gel.                                                                               |
| **BACTERIAL INFECTIONS**       |                                                                                                                                                                                                                       |                                                                                               |
| **LGE**                        | Fiery red band along gingival margin well with the amount of plaque present.                                                                                                                                         | Remove all local irritating factors:  
- Rinse with 0.2% chlorhexidine for 2-4 weeks  
- Also use metronidazole 300mg tds for 1 week  
- If significant bone loss is seen patients require regular assessment and removal of sequestered bone.  
- Alternatively, augmentin, 1 tablet 8 hourly can be taken. |
| **NUG**                        | Destruction and necrosis of interdental papillae, spontaneous bleeding and halitosis present.                                                                                                                      |                                                                                               |
| **NLP**                        | Rapid and progressive soft tissue loss, destruction of the lower bone.                                                                                                                                                |                                                                                               |
| **NS**                         | Ulcerative-necrotic lesions with exposure, destruction and sequestration of bone is seen                                                                                                                                |                                                                                               |
| **VIRAL INFECTION**            |                                                                                                                                                                                                                       |                                                                                               |
| **Herpes simplex** type 1 & 2  | Oral ulceration and vesicles that are painful and found on the hard palate, attached gingiva, buccal ridges - ulcers are shallow round and typical and coalescence may be chronic. | Acyclovir 400 - 800mg 5 x daily for 10 days                                                   |
| **Varicella zoster**           | More than one dermatome may be affected - multiple shallow, small ulcers, unevenly distributed along the 5th cranial nerve usually the palate - pain, neuropraxia and tenderness are usually present. | Acyclovir 800mg 5 x daily for 10 days                                                        |
| **Cytomegalovirus**            | Single ulcers with initial formation of vesicles - both shallow/deep ulcers may be seen                                                                                                                               | Acyclovir 800mg 5 x daily for treatment of systemic disease                                   |
| **OHL**                        | Unilateral/bilateral white lesions that do not wipe off. Found commonly on the lateral surface of the tongue, may extend dorsally/ventrally, asymptomatic.                                                                  | No treatment indicated.  
Or Acyclovir 800mg tds for 2 weeks.                                                          |
<p>| <strong>HPV</strong>                        | Oral warts may be exophytic, granular/cornflower like, asymptomatic and commonly isolated.                                                                                                                            | Surgical excision, laser therapy/laser surgery.                                              |</p>
<table>
<thead>
<tr>
<th>CONDITION</th>
<th>CLINICAL APPEARANCE</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>NEOPLASMS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• mucous carcinoma</td>
<td>Blue, black flat or keloidal lesion seen on the hard palate and gingiva, can occur at other sites</td>
<td>Vinblastine intralesional 0.2mg/ml or 0.1mmol of tumour</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Surgical excision</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Radiotherapy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Improve oral hygiene</td>
</tr>
<tr>
<td>• lymphoma</td>
<td>Maculopapule ulcers anywhere in mouth</td>
<td>Chemo and radiotherapy</td>
</tr>
<tr>
<td>ORAL ULCERS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• aphthous ulcers</td>
<td>May be minor/major/keratotic, may be persistent</td>
<td>Treatment depends on severity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Topical steroids 0.1% triamcinolone acetonide loz applied to lesion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Beclometasone spray – 1 pull 3 to 4 times day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Beclometasone 0.5mg tablet dissolved in a tablespoon of water – use mouthwash 3-4 times day for 2-3 min</td>
</tr>
<tr>
<td>SALIVARY GLAND</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• xerostomia</td>
<td>Dry mouth</td>
<td>Sugarless gum/candy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bethanechol 25mg tid po</td>
</tr>
<tr>
<td>• parotid swelling</td>
<td>Enlarged parotid glands uni/bilateral</td>
<td>No specific treatment</td>
</tr>
</tbody>
</table>
Hlabisa lies at the unstable southern edge of the African Malaria belt. Transmission is typically hypoenemic, usually January to April depending on rainfall, but may be becoming more enemic in adjacent health districts bordering Mozambique, due to widespread cross-border migration of index cases and the upsurge of Anopheles gambiae by re-emergent pyrethroid-resistant Anopheles funestus. Plasmodium falciparum is the dominant species with <1% Plasmodium ovale - though smears in the community do sometimes show asymptomatic parasitemias. All our patients should be considered non-immune. Limited local data suggest 20% chloroquine resistance and rising in vitro MICs to Fansidar and other first line drugs. From an average of 100 cases in 1997, annual caseload in Hlabisa increased to 2000 in 1999 with transmission continuing into the winter and an in-patient case fatality rate of ≤7%. Most cases are from areas of the district lying below 500m, traditionally Hluhluwe and St. Lucia/Dukuduku, but increasingly from Mbulula and environs. The National Malaria Surveillance Programme oversees residual spraying and other control measures in the district - all cases should be reported to their office in Jozini.

CLINICAL FEATURES
COMMON SYMPTOMS
- Fever (39.4°C as a rule but may be aiyerial on admission, don't expect a tertian pattern in the acute phase)
- Headache
- Characteristic "paroxysms"
- Mild diarrhea and vomiting
- Prostration

However, a flu-like illness may be the only presenting feature - be especially wary in season of women with spontaneous abortions and children with sudden onset seizures and rapidly decreasing consciousness. A long history (>7 days) correlates with severity.

SIGNS OF SEVERE MALARIA
- Altered consciousness (confusion, seizures, irritability, coma)
- Non-ambulant
- Intolerant of oral medication
- Poor urine output (/&/ U>20 and Cr >260)
- Jaundice (/&/ Bil>30)
- Clinical anaemia (Hct<20)
- Tachypnoea
- Hypotension

Consider pregnant women and children under 1 year as potentially severe even if none of the above signs is present.

DIAGNOSIS
Confirm suspected malaria with a rapid antigen test (Kalquick Pf. currently). If negative and clinical suspicion is strong, repeat the test in 4 hours and ask for at least a thick film to detect non-falciparum species. Parasitaemias are only semi-quantitatively graded in our lab and treatment decisions are invariably based on the clinical features. All malaria patients that are admitted must have FBC, U+E and Bilirubin reviewed by MO on the day of admission.

MANAGEMENT
NON-SEVERE CASES
It is no longer possible to admit all cases for observation. Patients with no signs of severe malaria should be treated as outpatients with a three day course of Co-artem® (Co-artem® contains 20 mg arteether and 120 mg lutemfantrine per tablet) according to the table below. (Use weight if available rather than age.)

<table>
<thead>
<tr>
<th>WEIGHT</th>
<th>AGE</th>
<th>NO. OF TABS</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 - 14 kg</td>
<td>1 - 5</td>
<td>1</td>
</tr>
<tr>
<td>15 - 24 kg</td>
<td>6 - 8</td>
<td>2</td>
</tr>
<tr>
<td>25 - 34 kg</td>
<td>9 - 12</td>
<td>3</td>
</tr>
<tr>
<td>≥35 kg</td>
<td>≥13</td>
<td>4</td>
</tr>
</tbody>
</table>

It is essential that patients complete the 3-day (6 doses) treatment course: the first dose given on diagnosis, the second after 8 hours, and then twice daily for two days. Tablets should be taken with food or water. If the patient vomits within one hour of administration, a repeat dose must be taken. Patients who are unable to eat within 48 hours should be carefully monitored as they are at an increased risk of treatment failure. If unwell or febrile after three days, they should be admitted for treatment with quinine.

Co-artem® is contra-indicated in pregnant women and children <1. These patients must be referred to hospital and treated with quinine. In these and all severe cases, a loading dose of quinine should be given before transfer (see below).

SEVERE CASES
- A loading dose of quinine must be given as soon as possible. At the clinic this must be given intramuscularly prior to transfer (20mg/kg in 5ml N saline - half in each thigh).
On arrival at hospital all severe cases should be admitted to a medical ward (or ICU when the ward is full). They should be catheterised and routine observations including RR and HCT recorded hourly.

If no loading dose has been given at clinic, give IV 20mg/kg in 500ml over 4 hours followed by 500ml fluid over 4 hours. Then start maintenance doses 8 hourly - 10mg/kg in 1 litre over 8 hours. The dose may be halved as soon as 48 hours if the patient shows a clinical response.

The fluid of choice is N-Saline with 50mls 50% dextrose added per litre, as quinine causes insulin release. Each dose should go in at least 250mls. Supplementary fluids may be required. Review fluid status after initial resuscitation. In children 4ml/kg/hr should suffice. Some polyuric adults may require >2l extra per day.

As soon as patients can tolerate it start oral quinine. If the patient is well enough to go home, before the completion of a seven day course, stop quinine and give a full course of Co-artem®. A minimum of 8 hours must pass between the last quinine dose and the first dose of Co-artem®. Do not give quinine and Co-artem® at the same time.

COMPLICATIONS

- Oliguria/patients with malaria are almost always hypovolaemic, so all should receive an adequate fluid challenge (200-500 ml over 20 minutes). If this does not provoke a diuresis re-assess the fluid status, if possible with a CVP line. Repeat the fluid challenge if the patient still seems dry. If hydration seems satisfactory, then give escalating doses of frusemide (80mg and 120 mg boluses followed by a 500ml infusion), followed by dopamine 2.5mg/kg/min if necessary. Refer EARLY if diuresis is lacking likely - in very ill patients two to three passes of peritoneal dialysis prior to transfer may be life-saving.

- Blood transfusion is indicated for HCT <15% or Hb<5.

- Thrombocytopenia is common in malaria and is not in itself an indication of severity. Platelet transfusion is only likely to be required if Pts<20 or there is overt bleeding.

- Treat convulsions with diazepam and give 10-15mg/kg phenobarbitone as secondary (never primary) prophylaxis.

- Paracetamol, fanning and tepid sponging are often required as quinine has little antiflagellar effect in itself.

- If fever has not subsided after 72 hours, re-examine the patient carefully. Repeat thick smears and blood culture (the Kalquick will still be positive at this stage even in successfully treated cases). Empirical treatment for associated gram negative sepsis is indicated, if there is evidence of clinical deterioration.

Further Reading
3) Sharp and Le Serer 1995 'Malaria in S. Africa - the present and selected implications for the future' S.A.M. J. 86:93

**TUBERCULOSIS**

South Africa currently has one of the worst T.B. problems in the world - 140,000 new cases are notified each year (probably a third of the true number) and the annual risk of infection as estimates on tuberculin surveys is ~1.4% for the black population (suggested intense transmission). In KwaZulu-Natal, we have experienced a rise in case-load of 400% over the last 8 years with an estimated incidence in 1995 of ~400 per 100,000. The T.B. programme is now handling 175 cases each month and 25-30% of beds in our hospital are currently devoted to diagnosis and management of this condition.

Our approach to management since 1991 has been to institute Directly Observed Treatment (DOT) in the community as at early a stage as possible, using twice weekly high dose therapy administered via all the available horizontal PHC structures including clinics, CHWs and voluntary lay supervisors. This policy has achieved completion rates of between 78-89% (80% in 1995). 97% of these patients are cured and rates of drug resistance in the district are low - the rate of initial drug resistance was 7.3% in 1994, but only 0.3% of isolates were resistant to more than one drug (1.4% for R.S.A. as a whole). Acquired resistance was even lower, but a small number of multi-drug resistant cases have occurred in the last 3 years. We do not at the present time use a separate regimen for re-treatment cases.

The diagnosis of T.B. rests on **sputum microscopy**, CXR and clinical findings (in order of importance). Patients with a cough for <3 weeks and no other suggestive features (e.g. weight loss, night sweats/fever, suspicious CXR changes etc.) may be prescreened. **Amoxicillin 250 mg t.d.s.** for five days from O.P.D. but must be requested to return for admission if their condition does not improve. In practice, many will already have consulted a G.P. or clinic and had similar treatment. Always ask!
All patients with suspected T.B. are processed via the medical wards with the exception of those withpleural T.B. or those who have a prior diagnosis from another district (which should normally be accepted), who may be admitted directly to T ward.

Order 3 sputum smears for AFB, CXR (need not be reviewed in OPD before going to ward) and Hb/Automated differential WCC.

The doctor who makes the diagnosis should notify the following patients (using form GW 17/5) -
- All Sputum +ve PTB's and any sputum -ve with typical CXR appearances
- Other smear or culture positives (Histology, CSF etc.)
- Children under 5 years with a confluent or ulcerating test

Only the following cases are re-notified -
- Previously completed a full course of treatment and been shown to be smear culture/negative at completion and
- smear/culture positive again

All deaths from T.B. should also be notified on the same form. Note that the vast majority of clinical diagnosis of T.B. and Extra-pulmonary T.B. including pleural cases will not be notified (in 1995 the specificity of a diagnosis of PTB at Mbabane as compared with T.B. culture results was 77%). Details are not re-notified.

The medical care of patients with T.B. is not over when the sputum is positive - 60% are H.I.V. +ve and many will have other management problems. Be on the look-out for intracranial bacterial infections and adverse drug reactions.

T.B. patients in our district are managed solely through the O.O.T. programme - pharmacy do not dispense T.B. drugs to patients from O.P.D. or community clinics. After 10-14 days in hospital, patients are either transferred to T ward prior to discharge or discharged directly from the medical wards. After entry in the district T.B. register each patient is transported with a full course of treatment to his/her nominated supervisor. The supervisor is then visited each month to monitor the patient's compliance.

PLEURAL T.B.

In our setting approximately two-thirds of unselected pleural effusions are due to T.B. - in patients under 35, with an effusion filling 50% of the hemithorax and a serous aspirate which is exudative and lymphocytic on microscopy, the diagnosis is almost always T.B. and we do not usually confirm the diagnosis by pleural biopsy. Important differentials of an exudative effusion are parapneumonic (smaller, shorter history, often polymorphic aspirate), empyema and malignancy. Transudative effusions may be due to heart failure, nephrotic syndrome etc. in patients who are very symptomatic, therapeutic pleural aspiration should be carried out, which is a quicker method of relieving acute symptoms than a short course of oral prednisolone. Neither intervention reduces the incidence of post-effusion adhesions significantly and most of our pleural patients will probably not benefit from corrective surgery due to their sero-status. The T.B. empyema/pyopneumothorax can be the very problematic especially in H.I.V. +ve patients - if re-expansion is poor following closed intercostal tube drainage, our approach has been to insert a malecot tube thoracostomy and review for decortication/pneumonectomy at the end of treatment - early removal of a dry thoracostomy is often the best policy as ~25% of patients at King George Hospital will be felt with a drain after decortication/pneumonectomy and require thoracoplasty.

Further Reading
2) Lee et al. 1983 "Carbamic acid in the treatment of tuberculous pleurisy: a double-blind, placebo-controlled, randomized study" Chest 83:1256
LYMPH NODE T.B.

This is the commonest form of non-respiratory T.B. and may be present in up to 20% of H.I.V. +ve T.B. patients. Generalised lymphadenopathy in our patients will usually represent follicular hyperplasia as a manifestation of H.I.V. - in cases where it is asymmetrical or matted-feeling consider T.B. (though note that generalised lymphadenopathy from T.B. can be a manifestation of haematogenous dissemination and bacillary load in such nodes may be high). Wide needle (19G) aspiration of large nodes may yield a +ve A.F.B. smear in ~30% of cases of proven T.B. lymphadenitis and should be done on the ward or in O.P.D. before proceeding to formal L.N. Biopsy. Macerotic caseation of the node to the naked eye has a 98% +ve predictive value but sensitivity of <80% for culture/histologically proven T.B.. In such cases treatment should be started whilst histology/culture is being processed. Another useful strategy is to smear the cut surface of the node on a slide for AFB staining (yield ~50%). Nodes may enlarge, suppurate and form sinuses even on treatment and may still be enlarged in 15% at completion.

Further Reading
2) Perenboom et al. 1994 "Diagnosis of tuberculous lymphadenitis in an area of high infection and limited diagnostic facilities" Trop Geo Med 49:5 pp:288

PERICARDIAL T.B.

This is an increasing problem and often the most prominent feature of disseminated disease. It usually presents as a large pericardial effusion sometimes with tamponade but an effusive-constrictive or straightforward constrictive picture may also occur. The clinical signs of tamponade (Beck's triad - hypotension, raised jugular venous pressure and distant heart sounds) should always be sought. Signs that should suggest an element of constriction are prominent dependent or pulmonary
oedema, rapid x and y descents in the JVP (which is always raised) and a palpable 'pericardial knock/early S3' just after the second heart sound. CXR will demonstrate cardiomegaly after ~250 ml have accumulated and calcification on laterals in some cases of constriction. US/USG diagnosis rests on demonstration of both anterior and posterior echo-free (dark) areas beyond the right and left ventricular free walls and the echo-bright pericardium which persists throughout systole and diastole - this is often easiest to appreciate on M-mode. Some effusions demonstrate prominent fibrinous adhesions/masses on the epicardial surface. Precise quantitation of effusions is not possible but an echo-free space of >2 cm on either side certainly represents a large effusion and a space of <1 cm a 'small' one. Collapse of the right-sided chambers during diastole is a highly specific and at least 80% sensitive echo sign of tamponade but not necessarily aspiration in itself; clinical signs are definitely absent. Signs of constriction on echo are difficult to interpret for the non-expert but a pericardium which is 4 mm thick and a pericardial sac full of echobright material are highly suggestive.

Our policy has been to treat all patients conservatively initially with standard T.B. treatment and prednisolone 60 mg daily and to aspirate only for signs of tamponade at presentation or developing during treatment. Definitive diagnosis really requires pericardial biopsy as the culture of aspirates has a yield of only 60%. Prednisolone at reduced dose should be continued for no longer than 3 weeks. Patients with refractory effusions or constriction probably require referral for placement of a pericardial window or pericardietomy at an interval.

Further Reading
1) Gitimsy NH et al. 1983 'Abdominal tuberculosis - a ten year review' S.A.M.J. 64:22 op.449
3) Jain R et al. 1995 'Diagnosis of abdominal T.B. - sonographic findings in patients with early disease' Am. J. Roentgenol 165:139

**T.B. MENINGITIS**

This is now the commonest form of bacterial meningitis in Cape Town children and is a manifestation of early disseminated disease. We are also seeing an increasing number of cases in young adults with H.I.V. It is characterised pathologically by thick exudate in the basilar cisterns and vasculitis of the cerebral vessels which may result in hydrocephalus and cerebral infarcts respectively. Symptoms are often very non-specific in the early stages (malaise, headache, fluctuating consciousness level/personality) and will invariably have been present for more than two weeks. As the disease progresses, the conscious level steadily deteriorates and neurological signs appear - commonly Bell's, VIth and VIIth palsies and peripheral paresis often in conjunction with extrapyramidal signs. Signs of raised intracranial pressure should be carefully sought and monitored in all patients. Spinal arachnoiditis may also be a complication and is characterised by lower motor neurone weakness in a radicular distribution, usually in the lower limbs.

The CXR is abnormal in at least 50% of patients in most series. The typical CSF findings are: -raised protein (90% of cases, typically 2-4 g/dl), moderately high cell count with lymphocytic predominance (<500 cells/mm3 in 50%, note that PMNs may predominate in <40% on initial L.P.) and low glucose. AFBs will be detected on smear in <20% unless large volumes of CSF are taken (<10 ml - usually safe in TBM but not in other more acute meningitides) and concentrated. The yield on culture is
>65% and is highest on the first specimen.

TBM may be staged according to the British MRC system viz. stage I - undisturbed consciousness, no neurological signs, stage II - disturbed consciousness but not coma and local neurological signs, stage III - coma +/- neurological signs (the chances of a full recovery in the last group are only ~20%). Standard chemotherapy is adequate to ensure CSF penetration and should be continued for at least 12 months (substitute ethionamide for ethambutol in children). Cerebral tuberculosis, however, may develop/progress during treatment and may require 18-24 months. Prednisolone 1 mg/kg for adults and 2 mg/kg for children given for at least four weeks is indicated in patients with stage II or III disease and in patients with a CSF protein >5 g/l (From's syndrome) and/or signs of arachnoiditis clinically. Patients who deteriorate with suspected hydrocephalus should be transferred for CT scan and assessment for V-P shunting.

Further Reading:
1) Donald PR 1996 'Pneumonia meningitis in the Western Cape Province of South Africa' J Trop Paeds 42: 258
2) Alarcon F et al. 1990 'Tuberculosis meningitis - short course chemotherapy' Arch Neurol 47: 1313

SPINAL T.B.

This shows a bimodal age distribution with predominantly paediatric or elderly cases. It presents with chronic back pain, a characteristic 'gibbus' deformity (~80% +ve predictive value, most marked in thoracic lesions) and varying stages of spinal cord involvement resulting from vertebral collapse or compression by a paraspinous abscess. The radiological appearances include disc space narrowing ('discitis'), anterior wedging of two adjacent vertebrae (from spread under the anterior longitudinal ligament) and skip lesions with intervening normal vertebrae - the average patient has 2-3 involved. However, it is destruction of the posterior elements that render the spine unstable. Soft tissue swelling may be seen where there is a large accompanying cold abscess. Definitive diagnosis can only be made on bone biopsy at Ngwelianze or King George Hospital.

Patients with Frankel A or B neurological lesions should undergo spinal cord decompression as soon as possible and patients with severe wedging of two adjacent vertebrae will almost always require surgery to remedy the imminent spinal collapse. All other patients (including Frankel C3 and D1) are treated with a month's trial of standard chemotherapy and reviewed at King George Hospital. Many of these patients will be offered surgery according to their fitness for operation and nutritional status. Delayed surgery may be indicated for persistent instability or collapse causing a severe deformity or neurological deterioration - the 'Hong Kong' procedure (radical anterior debridement and bone grafting) is the most frequently performed and successful operation. Most patients are then maintained in a plaster jacket for 12-24 months until the bone graft is judged to have fused at King George Hospital.

Further Reading:
1) Becchie-Adjiri O and Squillante RG 1996 'Tuberculosis of the spine' in Orthopaedic clinic of N. America 22:1 pp 95
2) Ogle JV 1994 'Ankylosing spondylitis as an indication of the prevalence of Pott's Disease in Transkei' SAMJ 84:614

DRUG TREATMENT

In Hospital

<table>
<thead>
<tr>
<th>Adults</th>
<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISONIAZID (H)</td>
<td>250 mg</td>
<td>300 mg</td>
</tr>
<tr>
<td>RIFAMPICIN (H)</td>
<td>450 mg</td>
<td>600 mg</td>
</tr>
<tr>
<td>PYRAZINAMIDE (Z)</td>
<td>1500 mg</td>
<td>2000 mg</td>
</tr>
<tr>
<td>ETHAMBUTOL (E)</td>
<td>800 mg</td>
<td>1200 mg</td>
</tr>
</tbody>
</table>

Given daily whilst in hospital; all drugs are given in the morning before food (if possible). Children under 12 are not prescribed ethambutol due to the risk of optic neuritis.

In Community (Twice weekly directly observed therapy)

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<thead>
<tr>
<th>Adults</th>
<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td>ETHAMBUTOL (E)</td>
<td>2500 mg</td>
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Children:

<table>
<thead>
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<th>Adults</th>
<th>Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>ISONIAZID (H)</td>
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<td>1200 mg</td>
</tr>
<tr>
<td>ETHAMBUTOL (E)</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

Further Reading:
1) Davies GR et al. 1993 'Twice weekly Directly Observed Treatment for HIV infected and uninfected tuberculosis patients: Cochrane study in rural South Africa' AIDS 13: 611
2) Connolly C et al. 1998 'Repose and remission amongst HIV infected and uninfected patients with TB successfully treated with twice weekly Directly Observed Therapy in rural South Africa' AIDS 13: 1543
CHALLENGE DOSES FOR SEVERE HYPERSENSITIVITY

Many cutaneous reactions especially mild itching (which is usually related to rifampicin) only require promethazine 25-50 mg acute and/or camphor cream/calamine lotion. The same however is not true for hepatitis, which should always be taken seriously - neglected hypersensitivity hepatitis in the community can be fatal so always try to identify the offending agent! Oddly however, jaundice often does not recur when the patient is re-challenged.

<table>
<thead>
<tr>
<th>Day 1</th>
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<th>Day 3</th>
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<tbody>
<tr>
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</tr>
<tr>
<td>RIFAMPICIN @</td>
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<td>300 mg full dose</td>
</tr>
<tr>
<td>PYRAZINAMIDE (Z)</td>
<td>250 mg</td>
<td>1000 mg full dose</td>
</tr>
<tr>
<td>ETHAMBUTOL (E)</td>
<td>100 mg</td>
<td>500 mg full dose</td>
</tr>
</tbody>
</table>

Escalate to full dose of one drug before adding the next.

Further Reading
1) Girling D.1992 “Adverse effects of anti-tuberculous drugs”. Drugs 23:56

SEXUALLY TRANSMITTED DISEASES

These are very common in Khulisa as in much of the developing world. As well as causing an acute illness, which is in itself unpleasant, STDs may become chronic and complicated. The burden of these complications falls largely on women and includes ectopic pregnancy, infertility, perinatal wastage and chronic pelvic pain. Furthermore, STDs of all kinds facilitate the transmission of HIV and it has been demonstrated that improved management of STDs at the primary care level can reduce the incidence of HIV infection.

The incidence of STD in the population aged 15-49 years is estimated at 9%/year and we estimate that ~25% of adult females in the district are harbouring an STD at any one time. 50% are asymptomatic and only 250 women seek treatment at clinics or GP surgeries each year - of those only 65% were judged to have been treated adequately according to current provincial guidelines. STDs are widely held to be ukulwa kwabantu phe excellence and the first consultation will often be with a traditional healer - remember that the patient's perception of the illness may be very different from yours in this case and clear counselling is essential.

The prevalence of syphilis in antenatal women is ~5% and this seems to play a large part in our perinatal mortality. Other bacterial STDs such as chancroid (50% of genital ulcers) are also very common. KwaZulu-Natal is an endemic focus for Granuloma Inguinale which may cause chronic and destructive genital/groin ulceration - Gentamicin may be an effective alternative for this organism in cases of treatment failure with erythromycin. All isolates of N. gonorrhoeae in our area demonstrate high grade penicillin resistance but ~90% remain ceftriaxone sensitive.

It is important to realize that clinical examination alone cannot effectively discriminate between most STDs and that at least 25% of patients have more than one infection. Syndromic management is by far the most cost-effective and efficient method, besides being much more convenient for the patient.

1) GENITAL ULCERATION (BOTH SEXES)
Benzathine penicillin 2.4 Mu i.m stat + erythromycin 500 mg q.d.s. p.o. for 5 days
In those allergic to penicillin, erythromycin 500 mg q.d.s. for 14 days
Aspirate any fluctuant glands

2) URETHRAL DISCHARGE / SWOLLEN TESTES
Ciprofloxacin 500 mg p.o. stat + doxycycline 100 mg b.d. p.o. for 7 days

3) VAGINAL DISCHARGE
SEXUALLY ACTIVE, NON-PREGNANT
Ciprofloxacin 500 mg p.o. stat +
Doxycycline 100 mg b.d. for 7 days + Metronidazole 2g p.o. stat

PREGNANT, PERI / POST-MENOPAUSAL
Methronidazole 2g p.o. stat (not in first trimester)
Ask to return - If not better:
Ceftriaxone 125 mg stat +
Erythromycin 500 mg p.o. q.d.s. for 7 days

If clinical evidence of candidiasis: Ciproflox 500 mg pessary

4) LOWER ABDOMINAL PAIN
Ciprofloxacin 500 mg stat + Doxycycline 100 mg b.d. for 7 days + Metronidazole 400 mg b.d. for 7 days (all p.o.)
Treat partners of 3 and 4 as 2, and partners of 2 as 3.
As important as the drugs are the following points -
- Do a WB on all patients and give a date for follow-up if the results will not be available the same day. If we give Benzathine Penicillin 8 ml i.m. weekly for three weeks.
- Above all, ENSURE PRIVACY AND ADOPT A CARING, POSITIVE ATTITUDE.
- Try to be aware of your own preconceptions and take the time to listen to any queries the patient has.

COUNSELLING
- Emphasise that the disease is transmitted specifically by sexual intercourse.
- That someone suffering from such a disease may be asymptomatic.
- That STDs can increase the risk of contracting HIV. Offer a test.
- That you should take ALL the treatment to ensure that you are cured, even if your symptoms go away.
- That it is possible to infect your partner again if you do not.
- That for this reason you should abstain from sex or use condoms during the period of treatment.

CONDOM PROMOTION
- Get a condom out of the packet and demonstrate how to use it.
- Give an adequate supply and tell the patient where they are available.
- Explain that condoms can prevent both STDs and HIV.

CONTACT TRACING AND TREATMENT
- Ask about the number and present location of all partners within the last three months.
- Explain that they have no symptoms they are probably all infected.
- Give contact cards and/or treatment for the patient to give to the partners.

Further Reading
1) Grosskurth H et al. 1995 'Impact of improved treatment of sexually transmitted diseases on HIV infection in rural Tanzania, randomized controlled trial' Lancer 345; 530
3) Harrison A et al. 1992 'Improving quality of STD management in rural South Africa' AIDS 12: 2329

LIVER DISEASE
Liver disease is a common cause of admission to the male medical ward. Liver biopsies at Hlabisa 1995-1997 demonstrated cirrhosis in 33%, steatosis in 20%, siderosis in 15% and hepatitis/portal tract inflammation or fibrosis in 33%. Hepatocellular carcinomas were twice as common in this series as either cholangiocarcinomas or metastases, and are probably under-represented. Various aetiological factors contribute to the prevalence of liver disease in our hospital.
- Viral hepatitis and its complications - the HepB carrier rate is ~10% and most of the community acquire the infection horizontally before the age of 4. It is now the target of a national immunisation programme. The HepC carrier rate is ~1% but this virus has recently been demonstrated to contribute disproportionately to chronic liver disease/cirrhosis at King Edward Hospital.
- Alcohol - although isizulu (sorghum/maize beer) has relatively low alcohol content (2-3%) other more potent home-made spirits (30%+°) are widely available e.g. isiziquha.
- Siderosis (traditionally common feature of liver biopsies in African men, possibly related to iron overload from cooking pots and a polygenic predisposition. Porphyrin cutanea tarda may be an associated clinical feature.)
- Other less common aetiologies of chronic liver disease include granulomatous hepatitis resulting from schistosomiasis or tuberculosis and veno-occlusive disease (i.e. veins and Budd-Chiari type syndromes).
- Painful hepatomegaly should always raise the suspicion of amoebic liver abscess or hepatocellular carcinoma. Tuberculosis is also a relatively common cause of unexplained hepatomegaly in adults. In children, mild hepatomegaly is a common finding and will often be the result of kuppfer cell hyperplasia from repeated infections/infections.
- Biliary obstruction in younger patients may be related to wandering ascariads but in older patients more often to gallstones or pancreatic malignancy.
- Our patients often present late with gross ascites but excepting gynaecomastia, leukonychia and subtle skin changes, signs of chronic liver disease will usually be absent.
Investigations

- Routine work-up should always include LFTs, U & Es and INR. Important differentials of primary liver disease in the jaundiced patient are malaria (ParaSight F) and haemolytic anaemia (Hb, Retic count and dipstick urine for urobilinogen/bilirubin). Always exclude cardiac failure/constictive pericarditis in cases of ascites and dipstick the urine for protein to eliminate nephrotic syndrome.

- Liver US/S is useful in almost all cases excepting the jaundiced patient under 20 with typical transaminitis who almost always has an acute HepA or B hepatitis. Interpretation of parenchymal patterns needs some experience but a grossly fatty liver or miliary granulomas produce a bright echogenic enlarged liver and are hard to miss. Cirrhosis is characterised by a small bright liver with an irregular "mosaic-code" appearance to the edge, a relatively enlarged caudate lobe and atrophy of the gall bladder bed. Signs of portal hypertension are - portal vein >1.5 cm, splenic vein >1 cm and splenomegaly (>5 cm in contact with the abdominal wall).

- Biliary obstruction is characterised by the appearance of the "parallel channels"/"double-barrel shotgun" signs in the parenchyma representing normally invisible 3rd bile ducts running alongside peripheral portal veins. The upper limit of normal for the common bile duct diameter is 0.5 cm - gall bladder dilatation is not a reliable sign of biliary obstruction.

- Cysts in the liver are characterised by the following properties - they appear round irrespective of beam direction, the posterior wall is distinct with posterior enhancement of echoes due to passage of the beam through fluid. Many cysts have no internal echoes but the most common cause in our practice is amoebic liver abscess which frequently demonstrates shifting material inside the cyst.

- Metastases and hepatocellular carcinomas vary in appearance depending largely on their size. There are no absolutely pathognomonic appearances of hepatoma and it can be easily confused with metastases especially if advanced with haemorrhage and necrosis giving rise to a "mosaiciform" or "bulls-eye" appearance. On the other hand large central cavities are uncommon in HCC and typically result from metastatic pelvic malignancy.

- Ascitic tap should be performed under aseptic conditions in most cases of ascites as a diagnostic manoeuvre -

  - Protein estimation
  - Cell Count, Glucose

  ![Diagram of protein estimation]

  Serum-Accites albumin gradient <1.1

  ![Diagram of serum-ascites albumin gradient]

  Serum-Accites albumin gradient >1.1

  ![Diagram of serum-ascites albumin gradient]

  >250/mm³
  -PNMLs
  -Glu low
  +ve Gram
  or culture

  <250/mm³
  -Lymphocytes
  +ve cytology
  -Glu normal
  +ve A.F.P
  -ve peritoneal biopsy

  Cirrhosis

  Hypoalbuminaemia

  Bacterial

  T.B. Malignancy

  From other causes

  Peritonitis

- Certain 2nd line investigations can be carried out in Durban. α-lactalbumin is available in cases where H.C.C. is suspected and is +ve in 60%, especially younger patients. Where a viral aetiology is suspected order HepA IgM, HepB SAg etc. and HepC ELISA.

- Liver biopsy is the definitive investigation in most cases but may be withheld where there is a clear history of alcohol excess or obviously advanced malignancy (esp. if α-F.P.+ve). It should not be done in jaundiced patients with deranged clotting or in the presence of biliary obstruction.

- Peritoneal biopsy under L.A. is the quickest way of definitively diagnosing patients who present with undiagnosed exudative ascites and features suspicious of T.B. on US/S e.g. thickened peritoneum, abdominal lymphadenopathy. It is essential that the ascites is well-controlled pre-op.

Management

- Ascites should be managed by bed-rest and a 500 mg/day salt restriction initially. Patients who fail to respond may be managed with Diuretics (Spironolactone 100 mg/day titrated to response up to 400 mg/day is most effective. Avoid diuretic combinations and don't aim to diurese faster than 1 kg/day except in patients who have peripheral oedema.) Paracentesis (No more than 1.5 l at a time in the non-oedematous patient unless you also give albumin 40 g after each tap but up to 5 l may be safe
in the patient with peripheral oedema. In both cases, monitor the B.P. afterwards and have a line in.)
- Total abstinence from alcohol. Temporary admission with diazepam prophylaxis and thiamine supplements may be the only way to achieve this.
- Careful prescribing!
- Specific management is possible only for a minority of our patients (T.B. treatment, relief of biliary obstruction at E.R.C.P., chemotherapy/aspiration of A.L.A.). Patients with viral hepatitis are not routinely followed up as HbeAg +ve patients spontaneously revert at a rate of 5-10% per year and interferon - therapy can only be offered to young patients with severe chronic active hepatitis B who have a good chance of cure. The prognosis of patients with cirrhosis can be estimated from the Child's - Pugh classification -

<table>
<thead>
<tr>
<th>Points scored for increasing abnormalities</th>
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<tbody>
<tr>
<td>Encephalopathy (grade)</td>
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<tr>
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<tr>
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<td>3/4</td>
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<tr>
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<td>Mod/Severe</td>
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<td>Bilirubin (µmol/l)</td>
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<td>25 - 40</td>
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<td>1-4</td>
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<tr>
<td>4-6</td>
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<td>&gt;6</td>
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Grade A = 5-6, Grade B = 7-9, Grade C = 10 - 15

Patients admitted with decompenated/end stage liver disease should be admitted to I.C.U. Give:
- oral neomycin 1-2 gms 6 hourly
- lactulose 10-30 mls 8 hourly
- protein - free diet
- intravenous vitamin K 10 mg/day
- vitamin B complex, 2 mls in each bag of fluid
- continuous dextrose 10 % infusion
- stop all diuretics
- precipitants such as S.B.P. or G.I. bleeding should be looked for. Cebriloxone should be started if S.B.P. is suspected clinically/on tap.

Further Reading
2) Bezuidenhout et al. 1996 "Tuberculosis peritonitis in Lesotho" Tuberculosis 67:47
3) Abdul Karim et al. 1993 "Hepatitis C Infection in urban and rural Kwazulu-Natal" S.A.M.J. 83:161

CARDIOLOGY

CARDIAC FAILURE

This condition accounted for ~5.2% of non-TB medical admissions and was twice as common in women in a survey conducted at the end of 1996. 35% of cases were admitted in AF. Almost 40% were attributed to hypertensive heart disease (high BP at presentation, LVH) and ~25% to rheumatic heart disease. (Murmur,echo evidence of valve abnormalities). The remaining 35% were probably due to Idiopathic dilated cardiomyopathy (DCM). It was notable that in women peri-cardial TB accounted for almost as many admissions as all other causes of acquired heart disease put together (see TB section for management).

- Ischaemic heart disease remains rare in our district but is gaining in prevalence in urbanised black South Africans. We also see a significant number of patients with right-sided failure due to pulmonary hypertension often from advanced valvular or primary lung disease.
- Rheumatic Heart disease accounts for ~20% of cardiac disease in RSA with a prevalence of ~7/1000 in the age group 2-18 years. Mixed mitral valve disease/pure mitral regurgitation are the commonest lesions. Hypertensive heart disease is diagnosed in 15-20% of autopsies for cardiovascular disease in RSA. A proportion of such cases present clinically in a non-mitral phase and can be difficult to distinguish from idiopathic dilated cardiomyopathy - this is a common entity in black South Africans and is thought to result from a combination of malnutrition, alcohol excess and possibly viral myocarditis. In males it will often be associated with liver disease. It is not due solely to thiamine deficiency and the classic presentation of 'wet beri-beri' is rarely seen but many of our patients have predominantly right-sided symptoms. "Soshun beri-beri" with a low-output state has also been recognised in urban black South Africans and responds dramatically to thiamine. Peripartum cardiomyopathy (~3 months post-partum) occurs 1/1000 deliveries in Kwazulu-Natal. These cases may just reflect the underlying disposition of the community to develop idiopathic cardiomyopathy under the haemodynamic stress of the peri-partum period - although it will resolve in two-thirds, about 50% will relapse in subsequent pregnancies.
Management

ACUTE
- Sit the patient up, give 35% Oxygen by mask
- Give Furosemide 40-80 mg slowly i.v.
- Add Morphine 5-10 mg slowly i.v. if severe left-sided sympotms.
- Glyceryl Trinitrate by continuous i.v. infusion (at an initial rate of 100μg/min and decreased according to clinical response and the patient’s B.P.) may be used for resistant pulmonary oedema where the systolic B.P. >90 mm Hg. If this is not available substitute one 5mg tablet sublingually.
- If BP is high and not controlled by GTN infusion, use Hydralazine 25-50 mg b.d. orally to bring diastolic down <100 mm Hg.
- If Systolic BP <90 mm Hg start a ‘cardiac dose’ Dopamine infusion at 10μg/kg/min and increase by 5μg/kg/min every 15 minutes to a maximum of 20 μg/kg/min until systolic >100 mm Hg. If response is poor, start an Adrenaline infusion 0.05 - 5μg/kg/min. Either inotrope should ideally be given via a central line.
- If a precipitating arrhythmia is present it is usually Atrial Fibrillation. Confirm this on ECG. If the patient is not already digitalised give 1 mg Digoxin in 100 ml of fluid i.v. over 2 hours. If the situation is not so acute consider rapid oral digitalisation instead (0.5 mg 12hrly X2 then 0.25 mg 12 hrly X4). Maintain with a single daily dose of up to 0.25 mg according to rate.
- In suspected peri-peri give Thiamine 50 mg i.v. 8 hrly for 24 hours then 10 mg t.d.s. orally.
- In ‘last ditch’ cases consider venesection of 250 ml blood.

CHRONIC/MAINTENANCE
- Establish the diagnosis - the most reliable indicators of pre-existing hypertension are retinopathy, hypertension at presentation/arising after stabilisation and Left Ventricular Hypertrophy on Echo (LV septal or free wall thickness >11 mm). The S1 may be loud and an S4 may be present. The typical signs of dilated cardiomyopathy on echo are gross dilatation (End diastolic diameter >5.6 cm) and a low cardiac output (Ejection Fraction <40%, flat aortic root motion) with a normal wall thickness. S1 is typically soft and an S4 may be heard. Regurgitant valve lesions are commonly present in severe DCMO but the presence of obvious stenosis or valvular thickening definitely establishes the aetiology as Rheumatic.

- ACE Inhibitors should be considered in all patients with chronic cardiac failure - be cautious if the creatinine is >200μg/l or the B.P. <100 mm Hg and remember to withdraw any potassium sparing diuretics. ACE inhibitors are contra-indicated in patients with stenotic valvular lesions. Enalapril 2.5 mg initially increased to 5-20 mg once daily is the drug of choice.
- With the exception of patients with symptomatic Aortic Stenosis who should be urgently referred, the timing of surgery for valvular lesions depends on the patient’s NYHA grade*. All patients with Rheumatic Heart Disease should however be booked for assessment at Wentworth at presentation (appointments for new patients often take 3 months). Pure Mitral stenoses will usually undergo balloon valvuloplasty whereas in MMVD/MR where the valve annulus is damaged open repair is attempted. In many cases this is not possible and the success of repair depends on how well the repair is controlled pre-op. MV replacement is in practice often carried out at an early stage even in young patients, usually with a St. Jude prostheses (porcine grafts have a life span as short as 2 years in younger patients due to severe calcification).
- Refractory Cardiac failure - Where diuresis with loop diuretics and ACE inhibitors is not satisfactory consider adding low dose thiazides (e.g. Hydrochlorothiazide 25 mg or Metolazone 2.5 mg alternate days), or low dose spironolactone.
- In some intractable patients addition of low-dose β-blockade may be beneficial (through up-regulating β Receivers or as prophylaxis vs. VT) but should only be started in hospital e.g. metoprolol 12.5 mg once daily.
- Digitalisation for patients in sinus rhythm may have a +ve inotropic effect in <25%.
- Anticoagulation in patients with cardiomyopathy should probably be reserved for those with suspected embolic phenomena or frank mural thrombosis on echo. Patients with valve replacements need only be maintained at an INR of 2.0-2.5 (+/- Dipyridamole) - this level is probably associated with a rate of valve obstruction due to thrombosis of <1%/year without serious risk of bleeding. In very unreliable patients, low dose (2.5 mg) anticoagulation without INR control should be considered.

* New York Heart Association Functional Classification
- I Symptom free for normal activity, II Minor limitation of activity, III Marked limitation of activity, IV Symptoms at rest or on minimal exertion.
Further Reading
4) Jessete et al 1989 'To what extent do patients in sinus rhythm benfit from digoxin therapy: A systematic overview and meta-analysis' J Am Coll Cardiol 14: 279
5) Wiesenbauch et al 1994 'Predictors of outcome after valve replacement for rheumatic mitral regurgitation in the era of chordal preservation' Circulation 90: 2

RHEUMATIC FEVER AND HEART DISEASE

Rheumatic heart disease is the most common form of acquired heart disease in children/young adults in our practice and the major cause of cardiovascular death in the first five decades of life in RSA. The average age at presentation is ~23 years. 30% of patients with acute Rheumatic fever are under 15 though the disease is rare below age 4 years. Carditis is more severe and carries a higher mortality in developing countries. African patients with acute Rheumatic fever rarely demonstrate nodules, areas of erythema marginatum, which should be remembered when applying the Jones Criteria (see below).

REVISED JONES CRITERIA FOR RHEUMATIC HEART DISEASE

Major Manifestations
Carditis
Polyarthritis
Gastroenteritis
Erythema marginatum
Subcutaneous nodules
Minor manifestations
Previous Rheumatic Fever/Heart Disease
Fever
Arthralgia
Prolonged P-R interval in absence of carditis
Leucocytosis, raised ESR
plus
+ve throat swab, +ve A.S.O., scarlet fever

About 2/3 give a history of sore throat in the preceding 3 weeks. Throat swabs are +ve for Gp A, B-haemolytic strep in <20% but may only represent carriage. ASO titres >250 have a sensitivity of ~85% but a specificity of at best 85% in children in developing countries. Some patients may present late with bona fide active carditis with few/no other manifestations. The dominant lesion is usually MR and in the acute phase is generally poorly tolerated with an unprepared left atrium - it may sometimes create a vicious circle of ongoing rheumatic activity until surgically corrected. Mitral valve prolapse in our patients is usually related to Rheumatic heart disease - it is a less benign condition with a different pathology from that found in Europe: it is commonly associated with de facto MR (Barlow's syndrome). Patients with acute Rheumatic Fever should be bed rested until the ESR is normal for 2 weeks (90% subside within 12 weeks). Aspirin may be used for analgesia but does not influence the course of the illness. The only indication for steroids is life-threatening carditis. All patients with valvular lesions clinically should be referred for assessment at a Cardiac clinic (WWW).

Penicillin 250 mg q.d.s. should be given to eliminate any concomitant streptococcal infection at the time of presentation. The most effective means of long term prophylaxis is Benzathine Penicillin 1.2 MU i.m. 3 weekly - we have generally used Penicillin V 250 mg b.d. p.o. (but even with active follow-up at least 15% will default treatment by one year). It is recommended that this should continue until age 21 following an attack of uncomplicated Rheumatic Fever and < 30 years in the presence of frank Rheumatic Heart disease.

Further reading

INFECTIVE ENDOCARDITIS

Always a threat in a population with such a high incidence of rheumatic valvulopathy and poor oral hygiene - however, it may also occur in patients with mitral valve prolapse or aortic sclerosis. In a U.K. series underlying heart disease had not been detected previously in ~40% of patients - don't let the source of the bacteraemia (e.g. pyomyositis, pelvic sepsis) put the possibility of endocarditis out of your mind. It remains a disease of young people in our setting - in African series, the presentation is often late with even half presenting in heart failure and a mortality of 40-60%.

The Duke Criteria may be quite useful in making a logical clinical diagnosis. Recovery rates from blood cultures at Halaisha have been poor - where such facilities are available ~95% are +ve on the first culture. We have had to rely more on clinical signs - fever, proximal splinter haemorrhages, microscopic haematuria and ESR (raised in 90%).
may be an accompanying hypergamma globulinaemia and +ve Rheumatoid factor in <50%. B-Mode echo has a sensitivity of <80% in detecting the presence of vegetations (esp. >10 mm mitral) relates to the risk of embolisation and mortality.

**DUKE UNIVERSITY CRITERIA FOR INFECTIVE ENDOCARDITIS (ABBREVIATED)**

**MAJOR CRITERIA**

1) +ve blood culture with typical micro-organisms i.e. strep. viridans, staph. aureus etc.- persistently positive on repeated cultures.
2) Evidence of endocardial involvement:
   - +ve echo with oscillating intracardiac mass (on valve/supporting structures or prosthesis in the absence of an alternative explanation) or abscess or valve dehiscence.
   - New regurgitant murmur (not change of a pre-existing murmur)

**MINOR CRITERIA**

- Predisposing heart condition
- Fever >38°
- Vascular phenomena: arterial embolism, septic pulmonary infarcts, mycotic aneurysm intracranial haemorrhage, Janeway lesions
- Immunological phenomena: Osler's nodes, Roth spots, Gonococcal meningitis
- Echo consistent with I.E. but not meeting major criteria
- +ve blood culture but not meeting major criteria

**Definite endocarditis = 2 major /3 minor +1 major / 5 minor / 1 E rejected = firm alternative diagnosis**

Clinical resolution in <4 days

Subacute cases with no evidence of embolisation (probably streptococcal) should receive Penicillin 500/6 MU q8h, Gentamicin 3mg/kg once daily for 4 weeks. In more severe cases (possibly staphylococcal) give Cloxacinil 2g q8h + Gentamicin as above. Patients with prosthetic valve endocarditis should be referred as should patients with worsening cardiac failure or those whose fever persists or recurs after 4 week of treatment.

Further Reading

**COMMUNITY-ACQUIRED PNEUMONIA**

The principal pathogens are *S. pneumoniae* and non-encapsulated *H. influenzae*. *Moraxella* spp. constitute <1% of isolates, and though *Klebsiella* spp are recognised as causing community-acquired severe lobar pneumonia in ill young adults in South Africa, these are also recovered in <1% of all cases. Serological studies suggest that <20% of CAPs may be related to atypical organisms *esp C.pneumoniae* but their significance is difficult to interpret. There is virtually no high-grade penicillin resistance amongst local pneumococci and since *Moraxella* and encapsulated *Haemophilus* species are so uncommon, penicillin remains the drug of first choice for CAP in our district.

Clinical indicators of severity are - R.R. >30/min, Systolic B.P. <90 mmHg, T°C >38.3°c, altered consciousness, cyanosis and involvement of >1 lobe. Admit all such patients to I.C.U.
- If non-severe give Amoxicillin 500 mg p.o. 8 hury.
- If severe give Benzylpenicillin 2-4 Mu (1.2-2.4 g) i.v. 6 hury.

In the over 60s or in patients with chronic chest disease add Gentamicin 4 mg/kg in a single daily dose or switch to Ceftriaxone (Gram negative colonisation/pneumonia is probably more common in this group).

**CHRONIC COUGH**

In patients presenting with cough lasting >3/52, TB is always the first differential. Although more than half of our chest patients are smokers, rates of bronchial carcinoma in black men are still relatively low (13 per 100/yr). However, we do often see patients with Chronic Obstructive Airways disease. There are also plenty of respiratory cripples in our district with complications of prior childhood chest sepsis or TB. This is usually manifest clinically as bronchiectasis with night-time/morning sputum production or the typical tuberculous ‘destroyed lung’ with dry cough and dyspnoea. Be sure to take an occupational history as silicosis etc. are compensable.

In our patients who are smear negative for TB, a failed trial of Amoxycillin (assessed according to the clinical response alone) in a patient with an Hb <10 g/dl has a +ve predictive value of 81% for culture +ve TB. However there are certain clinical provisos to be remembered when applying this approach.
In older AFB negative smokers with evidence of hyperinflation on the CXR and wheezing, the next diagnostic manoeuvre is an inpatient trial of steroids +/− bronchodilators.

- An AFB negative smoker with a collapsed lobe which fails to respond radiologically to antibiotics in three weeks should be referred for bronchoscopy.

- Patients with bronchiectasis will sometimes demonstrate typical tram-line/gloved finger appearances on CXR and characteristic postural expectoration especially at night/in the morning. They are also typically clubbed although this may also occur as a manifestation of TB alone. These patients usually respond poorly to Penicillin owing to gram-negative especially Pseudomonas colonisation – our only option in this situation is oral Ciprofloxacine 500 mg b.d. (although a combination of Ceftriaxone and Gentamicin may be effective against other gram negative organisms).

- Chronic destructive pneumonia due to anaerobes is a recognised entity in South Africa. It may mimic tuberculosis, but responds to addition of chloramphenicol or metronidazole.

In practice, it is quite often decided to initiate a ‘trial of TB treatment’ – be clear that this is not a final diagnosis and that comparison of our clinical performance against TB culture results in the past shows that we are wrong almost 50% of the time! Such patients should be critically reviewed three weeks after starting their treatment and further investigations initiated as appropriate.

Inferior Segment
Upper Lobe

Superior Segment
Lower Lobe

ASPIRATION - VULNERABLE BRONCHOPULMONARY SEGMENTS

EMPHYMA

All pleural effusions should be tapped to exclude this. Most will be related to complications of bacterial pneumonia but we recover AFBs on smear from sputum or pus in a number. All empyemas should be drained to dryness by chest tube and given Penicillin. The tube should be at least 26F and inserted no lower than the 4th/5th I.C.S. (− nipple level) in the mid-axillary line - this should drain the pus as effectively as a tube sited any lower provided the tip is placed in the most dependent area. In some cases re-expansion of the lung is not possible and these patients should be referred to Thoracic Surgeons at King George Hospital for assessment re. decortication and/or thoracostomy.

LUNG ABSCESS

This condition is thought to relate to aspiration of infected secretions during periods of unconsciousness and the typical risk groups are alcoholics and epileptics - we do see cases in previously well people and this may be due to the generally poor state of dental hygiene in our patients. The typical sites are the superior segment of the lower lobe and the posterior segment of the upper lobe, usually on the right. It may be often complicated by empyema or massive haemoptysis. Give Penicillin G 2-4 Mio I.V. i.v until definite clinical improvement. If progress is slow, add IV chloramphenicol 1 gm 6 hourly. Continue oral amoxicillin for up to 3 months.

FURTHER READING

ANAEMIA

- Anaemia of chronic disease Normocytic (MCV 80-95) Extremely common and associated particularly with TB and HIV. No evidence that vitamins/iron are helpful, although they are often given. A normocytic anaemia in the absence of disease may be due to mixed iron and B12/folate deficiency. In patients with chronic chest symptoms, a normocytic anaemia is a helpful diagnostic indicator of TB.

- Microcytic anaemia. (MCV <80, MCH <27). Iron deficiency. Most commonly due to dietary deficiency. Hookworms contribute, but are probably not sufficient to cause anaemia in the absence of malnutrition. Meats and green vegetables are the important sources of iron.
but in meat it is much more easily absorbed. Absorption of iron from vegetable sources is improved by vitamin C i.e. citrus fruits. Consider schistosomiasis, megaloblastosis, or GI loss, particularly in the elderly. Oral iron therapy should be given for long enough to correct the anaemia and replenish body stores, which means 4-6 months. FeSO4 given b/c is better tolerated than tabs. The Hb should rise by about 2g/dl every 3/52.

Indications for transfusion are:
- continuing blood loss
- requiring operation/procedure
- cardiac failure or severely debilitated with marked anaemia.

There is no a priori reason that an Hb as low as 4 needs to be transfused in the absence of the above.

**Macrocytic anaemia** (MVC >5)
Always ask about alcohol intake. Other causes should be considered than B12 and folate deficiency - reticulocytosis (haemorrhage/haemolysis)
- liver disease aside from alcohol
- phenylalanine (interferes with folate)
- aplastic anaemia and myelodysplastic syndromes and myeloma

Do not transfuse these patients as this can cause circulatory overload and they respond quickly to vitamin replacements.

Ask for a blood film (with reticulocyte count normal <2%) and B12 and red cell folate levels before starting vitamins. If the blood film shows hypersegmented neutrophils, or if the B12 level is low, then consider pernicious anaemia and send blood for antiparietal cell and intrinsic factor antibodies.

Treat with vitamin B12 strong tablets. Be aware that in pernicious anaemia, and small bowel disease (blind loop syndrome, crohns etc), there will be no response. High dose folinic acid 5mg od for four months and hydroxyzobetamin 1mg im daily for one week can be given and the patient reviewed with the vitamin levels. A Schilling test should be booked in Durban in B12 deficiency, and GI investigation may be warranted. Giving parenteral B12 will not interfere with the Schilling test.

**Haemolysis.** There is an exhaustive list, but acquired haemolytic anaemias esp. malaria and autoimmune due to infection e.g. pneumococcus or lymphoma/leukaemia/SLE should be considered. Methyl dopa can cause a drug induced AIHA. Check the reticulocyte count, urinary urobinogen and then Coombs test in suspected AIHA. Discuss with haematologist.

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**ASTHMA**

Asthma is a common and potentially fatal condition in our district. Africa asthmatics have traditionally been found to have low IgE levels and a low incidence of allergic rhinitis and eczema though ~75% demonstrate prick test positivity to at least one antigen. Rural populations may be relatively protected by high levels of blocking IgE antibodies to gut helminths but urbanised black asthmatics show clinical features not dissimilar to Europeans. Atopy in our patients is uncommon and most patients have late-onset disease. ~65% of asthmatics attending our chronic medical clinic have previously been admitted and most patients with severe asthma present late. Control in the community presents considerable problems. A survey in 1997 demonstrated that - 15% of outpatients could be judged as 'controlled' on the basis of symptoms especially nocturnal coughing and that half of these patients were on maintenance prednisolone. Only 27% of patients previously admitted were on inhaled steroids and in most the dose was only 200μg/day. Many patients struggle to master inhaler technique but most all are capable of using a spacer device.

**Diagnosis**

Chronic dry cough is the most common symptom. especially at night. Wheeze without cough is very uncommon. Not all that wheezes is asthma!

The important differentials are:

- **Childhood wheezing** - as many as 50% of the under 5s wheeze at some time, almost always related to respiratory virus infection. The vast majority will be wheeze free by age 5 and cannot be considered to have asthma. However the more severe and persistent the wheeze the more certain a diagnosis of subsequent asthma becomes.

- **Tuberculosis** - this is subsequently found to be the diagnosis in as many as 8% of asthmatics at referral centres and could be even higher in our district.

- **Lcrofll's syndrome** - may be a common cause in children and will be characterised by a high eosinophil count and pulmonary infiltrates. Ascaris ova may be present in the stools.

- **Occupational** - especially miners and sugar workers. They may be entitled to compensation. No patient should be started on oral/inhaled steroids without critical clinical evaluation and a CXR. Give a 1-2 week
trial of Prednisolone (0.5mg/kg in children or 30mg in adults - no need to tail off dosage) and review in OPD/CMC clinic. A good response to steroids indicates airways reversibility and consequently asthma.

**Management**

**CHRONIC ASTHMA**

- Base your treatment decisions on the patients' abilities, but in general, inhalers are the ideal. All inhalers should be given with a spacer (made from large plastic bottle) initially until good technique can be clearly shown. Spacers reduce pharyngeal deposition of particles and reduce dysphonia and oral candidiasis with inhaled steroids. Any patients who require more than a few puffs/day of β₂ agonist, or who have troublesome night-time symptoms require inhaled steroids. Giving a dose of salbutamol before the steroid bronchodilates and improves lung deposition of steroid.

  - Inhaled β₂ agonist (salbutamol 100µg MDI) 2 puffs p.r.n. 6hrly
  - Inhaled steroids (Budesonide 100µg MDI) 2 puffs 12hrly
  - Increase the dose of inhaled steroid up to 2000µg daily
  - Add slow release theophylline tablets b.d.
  - Prednisolone (lowest dose possible)

*Beclometasone 100µg MDI preferred in children under 6 years

- Always check inhaler technique and reinforce importance of inhalers esp steroids. Patients seen in the CMC have been taught to give 2 puffs with a spacer and take a deep, slow breath, hold, exhale, and take another deep breath. Children have been taught to breathe via the spacer after 2 puffs for a count of 20 seconds.
- Avoid prescribing beta blockers and NSAIDs.
- For patients who cannot use inhalers and young children, Theophylline tablets or syrup can be used in preference to oral β₂ agonists.

- 250 mg b.d. for adults (5 mg/kg/day in elderly, cardiac/liver patients, 15 mg/kg for healthy smokers)
- 25mg/5 ml syrup (over 8 weeks of age 1-3 mg/kg 6 hrly, 1-9 years 5 mg/kg 6hrly)
- However theophyllines have a narrow therapeutic index and the greatest potential for serious toxicity of all the anti-asthma drugs.
- Alcohol, smoking and phenyltol and rifampicin reduce levels.
- Liver impairment, heart failure, ciprofloxacin, erythromycin and cimetidine raise levels.
- Signs of theophylline toxicity include nausea, abdo discomfort, tachycardia and seizures - check theophylline levels and reduce dose in any patient with these warning signs.

- **Maintenance prednisolone** should be reduced to the lowest possible dose e.g. 2.5 mg all days. In brittle asthmatics consider rescue course of prednisolone (2 days worth) to take home and give advice about doubling dose of inhaled steroid and reporting promptly to hospital in the event of a deterioration. Make sure that patients know that they SHOULD NOT CONTINUE TO GET PREDNISOLONE AT CLINIC, but should come to hospital ASAP.

- Peak flow meters can be a useful guide to management if the best peak flow is recorded.

**ACUTE ASTHMA**

The key to the management of these patients is regular review - they should never be put on the ward until improved and stable.

Assess severity -

**Severe asthma**

- unable to speak a sentence in one breath
- RR>25/min
- pulse>110/minute

**Life threatening**

- silent chest
- cyanosis
- exhaustion, confusion, bradycardia, hypotension
- Sit up
- O₂ 40-100% by mask (be wary about giving any >28% in older smokers!) Use the pulse oximeter to gauge the minimum F₁ O₂ that maintains saturations >90%.
- Salbutamol 5 mg O₂-driven nebulisers every 20 minutes until there is clear improvement then 2-4hrly
- Prednisolone 30-60mg stat p.o. or hydrocortisone 200mg iv
- Add Ipratropium bromide 0.5 mg nebuliser 6 hrly if poor response
The prevalence of hypertension defined as 160/95 in community surveys of rural Zulus has been estimated at 6.3% and was found to be more common in females. Hypertension is more prevalent in urban Zulus and related to obesity and salt intake. There are few local data on outcome, but in European series the two year mortality for a DBP of 120-130 is -16%. Post-mortem studies in black patients in Durban demonstrated cerebrovascular complications in 41.9%, 32.9% congestive cardiac failure and 25% renal complications. The benefits of reduction of blood pressure are most evident in preventing stroke but hypertension accounts for -35% of cases of End Stage Renal Disease in SA blacks - the commonest preventable cause. In Hlabisa only -68% of hypertensives are controlled at primary care level.

Malignant hypertension accounts for -2-7% of hospital admissions for hypertension in South African series - -50% have advanced, usually acute renal failure and the case-fatality rate is 25%. The disease is thought to be more common in the black population and we quite commonly see patients referred from clinic with DBP >120 mm Hg but no retinopathy or organ failure.

Physiological studies in African patients show that many cases have lower renin levels and a relatively greater degree of LVH than Europeans. For this reason, β-blockers and ACE inhibitor monotherapy are widely thought to be relatively ineffective in our population.

**AIMS**

BP <160/100 in patients without complications.
BP <130/85 in patients with renal failure.
This can only be achieved by means of:
- *regular follow-up at C.M.C. and clinics*
- *repeated education (poor compliance is the commonest cause of refractory hypertension)*
- *simple regimes (compliance with t.d.s. regimes is 59% vs. 80% for q.d. regimes)*

**MEASUREMENT OF B.P.**

- *Cuff size. The cuff should encircle 80% of the arm (12.5 cm cuff for ordinary adults). Use a large cuff in obese arms as the B.P. can be overestimated by 30% with too small a cuff.*
- *Systolic is taken as Korotkoff 1 (first appearance of sounds) and diastolic as Korotkoff 5 (final disappearance of sounds).*
- *Position. The arm should be supported on a table or bed and be at the same height as the heart. Failure to do either can lead to errors of <10 mm Hg.*

The patient can be sitting or lying.
- *Record the result to the nearest 5 mm Hg.*

**Management of Uncomplicated Hypertension**

Very rarely does treatment need to be initiated on a single measurement. Check HbG and diaphoretic urine on all patients. If abnormal, take random blood glucose and/or U&Es/Creatinine.

- *In all patients, try non-pharmacological measures first*:
- *lose weight*
- *reduce alcohol intake*
- *regular exercise*
- *reduced salt diet* (No Knorr stock cubes or Aromat!)

- If diastolic <110 mm Hg or systolic <200 mm Hg, give advice and follow-up for 3/12.

- Figures above these should be given advice and seen in one month. Very high blood pressures (diastolic >120 and/or systolic >220) should have their B.P. checked in one hour, before commencing treatment. Many of these will be poorly controlled hypertensives already on some form of treatment.
When drugs are required follow the protocol -

**Hydrochlorothiazide 25 mg/triamterene 50 mg ("Dyazide") tab daily**

- Review in one month
- Ensure Compliance

**Add Reserpine 0.125 mg 1/2 tab daily**

- Review in one month
- Ensure Compliance

**Change to Hydralazine 20 mg b.d.**

- Review in one month
- Ensure Compliance

**Hydralazine 50 mg b.d.**

If still uncontrolled consider changing to / adding 2nd line agents e.g Prazosin 1 mg b.d. increasing to 5 mg b.d. or Enalapril 2.5 mg increasing to 20 mg daily.

Management of Complicated Hypertension

- **Hypertension in Diabetes**
  Dyazide can worsen hyperglycaemia and should be avoided. If adequate control can be achieved with ACE Inhibitor alone this is the drug of choice as it slows the development of renal complications.

- **Renal Impairment**
  Still much debate as to whether this can be caused primarily by mild/moderate hypertension per se. However, every effort should be made to slow progression to End-stage renal disease as we have no access to long-term dialysis. Tighter B.P. control is necessary to achieve this and the target should be 130/85 or a little less.

- **Heart Failure**
  ACE Inhibitors preferred, hydralazine has little negatively inotropic effect and can be used for acute control.

- **Malignant Hypertension**
  Defined as KWB Grade 3 or 4 retinal changes (flame haemorrhages and/or papilloedema) with a DBP >120 mm Hg. Admit for bed-rest and aim to reduce the DBP to 110 mm Hg over 24 hours. Use oral medication in the first instance as i.v. overdosing could seriously drop the BP and result in stroke. Use Hydralazine 25-50 mg t.d.s. or nifedipine 10-20 mg t.d.s. In all cases investigate for a 2 cm cause of hypertension which will often be present (1 renal disease, phaeochromocytoma etc). If the patient also demonstrates impaired consciousness (possible hypertensive encephalopathy) however aim to reduce the BP more quickly with hydralazine slowly i.v. 10-20 mg repeated after twenty minutes if no response.

- **Following a stroke**
  Rapid lowering may worsen the stroke. Treat if BP 200/120 after 48 hours.

- **Young hypertensives**
  Consider secondary causes:
  - renal disease
  - coarctation
  - endocrine
  - vasculitis/autoimmune.

Further Reading:
1) Orie LH & Sleyn K 1995 'Rationale for the hypertension guidelines for primary care in South Africa', S.A.M.J. (12 Pt 2) 1325
2) Seccaf YK & Mackland DB 1994 'The prevalence of hypertension in 4,993 rural Zulus' TRSTHM 78:785
3) Parag KB & Seccaf YK 1990 'Do Angiotensin Converting Enzyme Inhibitors work in Black hypertensives?' J. Hum. Hypertens. 4(4):450
5) Seccaf YK 1996 'Is the pathogenesis of hypertension different in black patients?' J. Hum. Hypertens. 10:53:37

**DIABETES MELLITUS**

Prevalence generally <1% in rural Africa but approaching 4% in urban Zulus. 'IDDMS' shows a bimodal distribution in age of onset - at 20-30 years (10 years later than in Europe) and at 45-50 years. The disease is virtually unknown <10 years of age and 80% present after age 30. In this older group, C-peptide levels are relatively high, suggesting that many patients on insulin may not actually require it. This situation and prima facie 'NIDDM' usually arise as a complication of obesity and insulin resistance, especially in females. Renal failure is probably the most common long-term complication in
black South Africans but hyperglycaemic emergencies and infections are responsible for most mortality (case fatality rate for DKA at Baragwanath 10-25% and increases to <50% in over 70s).

**Diagnosis**

The diagnosis should always be confirmed by a **formal** blood glucose as BM stix readings may be inaccurate under some circumstances. Diabetes Mellitus is defined as:
- **Random blood glucose >10.0 mmol/l**
- **Fasting blood glucose >6.7 mmol/l**

Be wary of making the diagnosis during a concurrent illness (re-lacte hyperglycaemia is especially common in children) and look for causes especially drugs e.g. Thiazides and Steroids. The aim of management should be control of symptoms and avoidance of hyper/ hypoglycaemic crises - we are in no position to attempt prevention of long term complications except in the exceptionally motivated patient.

**Management**

- **Dietary manipulation** should wherever possible be attempted as definitive management alone and should always accompany other forms of treatment. Local maize meals contain plenty of viscous fibre - the coarser forms of samp have the best glycaemic indices. Patients should in general eat chicken rather than beef and eat plenty of green vegetables. No more than one teaspoon of refined sugar in each cup of tea should be allowed (see the diet sheets available in OPD).

- **Metformin** is the drug of choice for obese patients (500 mg b.d. with food increasing to 1g t.d.s. where necessary). The principal side effect is diarrhoea but lactic acidosis is also a recognised complication.

- **Glibenclamide** is preferred in the non-obese patient (2.5 mg initially once daily increasing to 15 mg mane as required).

- **Insulin** should be reserved for patients who fail maximum oral therapy as an in-patient or young patients presenting with life-threatening DKA. The only subcutaneous insulin used in our hospital is Actraphane - a fixed dose human insulin combination containing 30/70 short/medium-acting forms. The initial daily insulin requirement should be in the range 0.5-0.7 u/kg/day with 2/3 of the dose given in the morning and 1/3 in the evening. Ensure that patients know how to rotate their injection sites. Few of our patients have a fridge but most can keep their monthly supply in the next best cool place (often a hole in the ground but a beer pot filled with water is preferred). Many NIDDM patients may require insulin during a concurrent illness but can usually be weaned after it subsides.

- Patients should have a simple evaluation every year for the presence of complications. This should include fundoscopy, assessment of their feet and a dipstick for proteinuria.

**DIABETIC EMERGENCIES**

**KETOACIDOSIS**

In ~25% of cases this occurs in patients who have not previously been diagnosed - infection and poor compliance are often important precipitants. It can occur in NIDDM patients. The diagnosis is usually simply confirmed by a *high* blood glucose and the presence of ketonuria - however, the condition is strictly defined as +ve serum ketones and arterial pH <7.3 - blood glucose may be <11.1 mmol/l in a small proportion of cases especially in pregnant women and starving/vomiting patients. There is usually an increased anion gap.

The diagnosis should always be excluded in any unconscious patient and prompt, correct management is essential -
- **If the patient is shocked** give 0.9% Saline as fast as is necessary to restore the circulating volume - if not give 1 litre over 1 hour followed by a further 1 litre over 2 hours. Continue at ~ 250 ml/hour for a further 16 hours (the usual volume deficit is 5-7 litres). Fluid replacement alone leads to a fall in blood glucose and osmolality and slower, isotonic fluids minimise the risk of cerebral oedema (often signalled by a fall in the plasma Na). Hypotonic solutions e.g. Darrow's should only be used when the Na >150 mmol/l and only then after 2l of 0.9% Saline have been given to restore the circulating volume. 5% dextrose should be substituted for Saline when the blood glucose is <15 mmol/l.

- The average K deficit in DKA patients is ~500 mmol though 25% present with a K >5.0 mmol/l. Administration of insulin leads to redistribution of K into the intracellular compartment and may cause a rapid fall in K levels so *almost all patients need supplementation* - this should be started immediately unless the initial K is <6.0 mmol/l or the patient is...
shocked. Check the K+ 1 hour after starting insulin. The rate of replacement should be varied to maintain the K+ level >4.0 as follows:

<table>
<thead>
<tr>
<th>serum K+</th>
<th>replacement rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;3.0</td>
<td>40 mmo/l</td>
</tr>
<tr>
<td>3.0-4.0</td>
<td>30 mmo/l</td>
</tr>
<tr>
<td>4.0-5.0</td>
<td>20 mmo/l</td>
</tr>
<tr>
<td>5.0-6.0</td>
<td>10 mmo/l</td>
</tr>
<tr>
<td>&gt;6.0</td>
<td>none</td>
</tr>
</tbody>
</table>

- Low dose i.v. insulin inhibits the uncontrolled lipolysis and gluconeogenesis of the ketotic state - give 5 u Actrapid i.v. and then add to the infusion bags to give a constant rate of 5u/hr. If there is no fall in the blood glucose in 2 hours, double the infusion rate. You are NOT aiming to normalise the glucose as fast as possible - maintain it at around 10-15 mmo/l with 5% dextrose for the first 24 hours. Start subcutaneous insulin as soon as the patient can eat unless control is very poor, when a sliding scale could be used temporarily.

- Bicarbonate is of no benefit in DKA.

- Catheterise and pass an n.g. tube in unconscious patients. Colloid may be required in persistently hypotensive patients. Antibiotics should be prescribed where there is evidence of infection as a precipitant.

HYPEROSMOLAR NON-KETOTIC COMA
(H.O.N.K.)

Principles of management are the same as in DKA with the following caveats:

- Patients are much more insulin sensitive and an infusion rate of ~ 2u/hour should suffice.
- Hypertenaemia is more common and hypotonic fluids needed more frequently. Dehydration is typically more severe and up to 1l/hour initially for 4 hours may be required.
- Smaller amounts of K+ are needed.
- Thrombotic complications are more common and s/c heparin 5000 u. b.d. should be given.

HYPOGLYCAEMIA

This is the most common diabetic coma - about a third occur on oral therapy. The commonest precipitants are missed meals and alcohol. Remember to advise all patients on diabetic medication never to be without an emergency source of ‘last glucose’. Give 50 ml 50% dextrose followed by something to eat. Prolonged hypoglycaemia may require admission and dextrose infusion for 24 hours. Fatalities are exceptional but do occur.

Further Reading
2) Huddle and Gill 1993 Hyperglycaemic admissions amongst diabetic patients in Soweto, South Africa Diabetes 10:181

EPILEPSY

The estimated prevalence in the Hlabisa district is 0.15%, which may be an underestimate as the condition is thought to be twice as common in developing countries as in Europe. Partial seizures secondarily generalised are the most common form and ~ 20% of cases are related to previous head trauma. Birth injury and CNS infection also play a big role in childhood. Neurocysticercosis is probably a rare cause in our district (~ one case/year) but has been demonstrated to be a common factor in other parts of RSA. Management presents problems - compliance with medication is often poor and monitoring of drug levels very difficult to apply (the usual level is 0.00 mmol/l) - one study of patients compliant on a standard dose of Phenobarbital 300 mg/day who had neither side effects nor seizures demonstrated 'potentially toxic' levels in 36%. Epileptics in our community suffer considerable morbidity, especially from burns, and most are dependent on some sort of disability grant.

Diagnosis
Il is vital to obtain a good, witnessed history of the fit to detail the type of seizure.

- Always ask about alcohol intake to exclude withdrawal seizures. These typically occur within 2 days of abstinence and may be associated with frank delirium tremens, but seizures may also be precipitated by acute intoxication and may continue even after the
patient dries out.
- 70% of those who will suffer post-traumatic epilepsy will have their first fit within two years of the injury - this is more likely if there was a penetrating injury or skull #neurosurgery. However, seizures can occur some years after the injury.
- Fovite convulsions typically occur between 9 months and 5 years, are single, generalised and last <15 minutes. They occur in ~3% of the under 5s with a long-term risk of developing epilepsy of 1%. However, any atypical features (repeated, neurological abnormalities on recovery, meningism) warrant LP and ideally the child should be admitted /observed overnight in OPD.
- Neurocysticercosis may present abnormal neurological signs or calcified cysts on x-ray of the soft tissue of the thigh.
- Do a full neurological examination including fundoscopy, check the BP and diabetst the urine for protein. Check HCT and WR.

Further Investigations
- Not appropriate after a single generalised afebrile seizure with no sequelae - the risk of recurrence in childhood is only 50%. Though anticonvulsants given immediately after a first seizure probably reduce the risk of recurrence by ~30%, the risks to the patient (e.g. builder, lorry-driver etc) should be considered carefully on an individual basis since he/she could be embarking on many years of treatment.
- Referral to the epilepsy clinic at KEH (children) or WWH (adults) should only be for those with local/ poorly controlled fits or neurological signs.

Treatment
- Start after two seizures (or one, if local, as the likelihood of a structural lesion is higher, and therefore a recurrence greater).
- Counsel that:
  - drugs must be taken regularly, probably for life
  - alcohol must be avoided
  - patients should not sit close to an open fire
  - swimming/bathing in deep water alone is dangerous
  - no driving/cycling/operating machinery is safe
  - interactions of phenytoin with OCP may decrease efficacy and need for toluate in pregnancy.
- Carbamazepine is the first choice for all fits in children and young adults and women of child-bearing age.

Start at 100mg bd (adults) and increase up to 800mg bd until control is achieved or the drug not tolerated. Side effects dizziness, ataxia, nausea, diplopia. Not uncommonly causes hypotension, and rarely causes agranulocytosis.
- Phenytoin is preferred for older adults and those in whom compliance may be a problem. Use 200-300mg nocte. Because of its non-linear kinetics, small dose increments may lead to large rises in serum concentration so the dose should be increased cautiously.
- Valproate is reserved for fits that have failed to respond to the above. It may also be used as a first line for some forms of childhood epilepsy e.g. juvenile myoclonic. Use 200mg bd up to 4g bd.
- Drug levels. Used primarily to check compliance before deciding to change treatment and to monitor Phenytoin levels in the upper dose range. Carbamazepine levels are less useful and side effects are a guide to dosage.
- Aim for monotherapy. If fits are not controlled, check compliance and drug levels. If seizures are still occurring at therapeutic levels then add a second drug, increasing the dose at successive visits until optimal. Then reduce the first drug. Phenytoin can be reduced over 2 weeks and CBZ over 1 month. If monotherapy is not working, then there is a 10-15% chance that a combination will control fits, but monitor levels closely as interactions are common and dangerous.
- We do not change patients already controlled on Phenobarbitalone to another drug unless they are experiencing troublesome side-effects
- Stopping treatment. In practice, achieving a seizure-free period of even two years has been exceptional in our clinic since compliance is so poor. In the largest European study, of adult patients stopping treatment after such a period 43% relapsed within 2 years compared with 10% who remained on drugs. This risk increased if the patients were over 15 years old or had seizures that were difficult to control (on more than one drug, repeated seizures on treatment). Since symptomatic epilepsy is probably much more common in our district, we generally wait for a five-year remission before withdrawing treatment. In children however, the risk of relapse after a two year remission is ~25% for generalised convulsions, so treatment can be withdrawn sooner.
STATUS
EPILEPTICS

Status is defined as a generalised tonic-clonic fit lasting more than 30 minutes, or repeated fits without recovery of alertness in between.

- Insert a guedel airway and give oxygen.
- IV access and diazepam 10mg (adults).
- Check HGT and give 50% dextrose if <5mmol.
- Repeat diazepam if no response.
- Give IV phenytoin at 18mg/kg over 20 mins. Then 100mg 8 hrly i.v. until taking oral. Do not give if the patient is on phenytoin and known to be compliant.
- Phenobarbitone 15mg/kg IV no faster than 100mg/min or until fits controlled.
- If all else fails then paralysed GA and ventilation.
- Check electrolytes +/- Ca²⁺ and consider a cause e.g. infection/ SCL if a de novo presentation. Note that cells will be raised in the CSF after fit.

Further Reading
1) Newton CR & Gero BT 1984'The epilepsies amongst rural blacks' S.A.M.J. 65:23
2) Dansay et al. 1982 'Seizures and neurocysticercosis in black men' S.A.M.J. 81:424
3) McArdiey MC et al. 1990 'The relevance of a first world therapeutic drug monitoring service to the treatment of epilepsy in third world conditions' S.A.M.J. 78:987
4) The First Seizure Trial Group 1993 'Randomized clinical trial on the efficacy of anti-epileptic drugs in reducing the risk of relapse after a first unprovoked tonic-clonic seizure' Neurology 43:428
5) MRC Antiepileptic drug withdrawal study group 1993 'Prognostic index for recurrence of seizures after remission of epilepsy' R.M.J. 306:1374

CEREBROVASCULAR DISEASE

Probably the commonest cause of death in our population attributable to chronic non-communicable diseases. This is widely thought to be much more common due to haemorrhage than infarction in our population. Atherosclerosis in black South Africans chiefly affects the cerebral circulation but is closely related to the severity of co-existing hypertension (though this may change as other risk factors become important e.g. smoking). Cerebral embolism is also much more common as a cause of stroke than in Europe, constituting <30% of cases. The source is usually cardiac. Strokes in young black patients are a well-documented phenomenon - some may be related to meningovascular infection (bacterial, Y.B., syphilis) or Takayasu's disease (possibly a hypersensitivity vasculitis related to T.B.) - however a significant number still go unexplained.

Diagnosis

A Stroke is defined as a sudden onset of localising neurological signs related to a vascular territory lasting >24 hours and a Transient Ischaemic Attack as the same pattern lasting <24 hours. Consider alternative diagnoses especially if meningism and fever are present. Headache, vomiting and deteriorating conscious level are significantly associated with a haemorrhagic aetiology. Listen carefully for a source of emboli.

Management

- Most stroke patients should be admitted. Meticulous nursing care and avoidance of bedsores are PARAMOUNT! Patients MUST be turned every two hours. The gag reflex is often absent initially, but only 2% remain dysphagic 1 month. Naso-gastric feeding may be poorly tolerated, and does not prevent aspiration pneumonia.
- Hypertension should be managed cautiously as a disturbance of cerebral auto-regulation at this stage could lead to an extension of the deficit. If B.P. >160/120 after 48 hours bad rest start gentle therapy.
- Passive movements should be started within 48 hours to prevent contractures. Independence should be encouraged as early as possible and the family involved right from the start as few facilities are currently available in our district for professional rehabilitation.
- It is difficult clinically to distinguish the different aetiologies of stroke but we refer selectively for C.T scan/further investigation -
  - patients with a sudden onset deficit complete within minutes and a possible source of emboli (i.e. murmur or bruit)
  - patients under 40 with no obvious aetiology (e.g. H.I.V./H.R. -ve, no cerebral infection)
  - suspected subarachnoid haemorrhage ( Nimodipine and/or surgery may be indicated if drowsy/confused or mild focal deficit)
- The subsequent relative risk of completed stroke in patients with T.I.A.s is ~7. Patients with a definite source of cardiac emboli should be warfarinised (either fully (INR ~3) or low dose depending on compliance) and those without should be prescribed Aspirin 150 mg daily.
MYELOPATHY

'Idiopathic myelopathy' is a common problem for African physicians - half of cases go unexplained. 'Konzolo' is a non-progressive spastic paraparesis resulting from myocytic toxicity due to improper processing of bitter cassava that occurs in Mozambique. However, this does not seem to occur in RSA. HTLV-1 infection is prevalent in KwaZulu-Natal (2.6% population prevalence in Empangeni) and 2-3 cases of tropical spastic paraparesis occur in our hospital each year, usually in association with HIV infection. Tabes dorsalis, multiple sclerosis and sub-acute combined degeneration of the cord are considered to be rare in black patients.

Management

- Check Hb, RPR, urine/stool for schistosomes ova, X-ray the relevant spinal level and examine the C.S.F.
- Send serum for HTLV-1 and HIV serology
- If the patient is anaemic +/- macrocytic, take blood for serum B12 and give 1000 µg B12 stat i.m. daily for a week + Folate 5 mg daily. Though established spinal cord damage from megakaryoblastosis is usually irreversible, this will prevent the lesion progressing.
- Refer for C.T./Myelogram if short history, signs progressing
- There is no response to a trial of Vit B12/Folate
- HIV, HTLV-1, RPR all negative, no evidence of TB/bacterial spondylitis or schistosomiasis.

Diagnosis

Document the neurological signs carefully. The pattern will often not be as clearcut as suggested below. Important differentials of spinal cord disease are:

- Ascending polyneuropathy (Guillain-Barre syndrome - predominantly ascending weakness)
- Spinal Cord Compression (Extradural bacterial/TB abscess, malignant deposits - typically radicular pain, sacral-sparing sensory loss progressing to hemisection/transaction)
- Intrinsic lesions/Transverse Myelitis (Tumours, HIV, Schistosomiasis, Vascular - classically bilateral dissociated sensory loss)
- Posterior column syndromes (Tabes Dorsalis and Sub-acute combined degeneration - characterised by +ve Romberg's sign and absent ankle jerks)
- HTLV-1 myeloneuropathy (usually spastic weakness and absent ankle jerks with little sensory loss)

PERIPHERAL NEUROPATHY

Many in-patients complain of burning dysesthesia/aching feet - in many cases this probably reflects a nutritional sensory neuropathy. This may be related to a deficiency of almost any of the B vitamins (even if the patient is not on T.V. treatment) so a trial of these is usually worthwhile (iii tablets 8 hourly) - other causes include diabetes, HIV, porphyria cutanea tarda and rarely leprosy (-1 case every 2 years). Nerve conduction studies can be carried out in Durban but are rarely indicated if the aetiology is clear. Neuropathic pain is relatively opiate-resistant and adjunctive Amitriptyline (25-50 mg nocte) may be helpful.

Further Reading

1) Sishigee J 1983 'Prevalence and transmission of HTLV-1 infection in KwaZulu Natal' S.A.M. J 83:655
2) Van der Rysel E and Smith NJS 1994 'A high prevalence of HTLV-1 antibodies in HIV-1 seropositive patients with neurological disease' S.A.J Epidemiology and infection 91 p 20
WHO CLINICAL STAGING SYSTEM FOR HIV INFECTION/DISEASE

CLINICAL STAGE 1
- Asymptomatic
- Persistent generalised lymphadenopathy
- &/or Performance scale 1: asymptomatic, normal activity

CLINICAL STAGE 2
- Weight loss <10% of body weight
- Minor mucocutaneous manifestations (seborrheic dermatitis, prurigo, fungal nail infections, recurrent oral ulceration, angular cheilitis)
- Zoster within the last five years
- Recurrent URTIs (including bacterial sinusitis)
- &/or Performance scale 2: symptomatic, normal activity

CLINICAL STAGE 3
- Weight loss >10% of body weight
- Unexplained chronic diarrhea for more than 1 month
- Unexplained fever (intermittent or constant) for more than 1 month
- Oral Candidiasis
- Oral hairy leukoplakia
- Pulmonary tuberculosis within the last year
- Severe bacterial infections (pneumonia, pyomyositis)
- &/or Performance scale 3: in bed <50% of the day during the last month

CLINICAL STAGE 4
- HIV wasting syndrome
- Pneumocystis Carinii Pneumonia
- Toxoplasmosis of the brain
- Cryptosporidiosis with diarrhea for more than one month
- Cryptococcosis
- Cytomegalovirus disease of an organ other than the liver, spleen or lymph nodes
- Herpesvirus infection, mucocutaneous for more than one month or visceral (any duration)
- Progressive multifocal leucoencephalopathy
- Any disseminated endemic mycosis
- Candidiasis of the oesophagus, trachea, bronchi or lungs
- Atypical mycobacteriosis
- Non-typhoidal salmonella septicaemia
- Extra-pulmonary tuberculosis
- Lymphoma
- Kaposi's Sarcoma

Diagnosis

HIV testing is available for emergencies on site using a validated single-rapid technique for adults, but most routine tests are double ELISAs at the regional laboratory in Ngwelezane. The WHO (Bangui) clinical case definition should not therefore be applied without confirmatory serology but all patients undergoing a test should be pre- and post-test counselled by appropriately trained nursing staff. We have used the WHO Staging system which comprises four prognostic groups -
HIV Encephalopathy
s & or Performance scale 4 : in bed >50% of the day during the last month.
(N.B. Both definitive and presumptive diagnoses are acceptable.)

We also use two surrogate prognostic markers - the haematocrit and the total lymphocyte count - this is most accurately assessed by formal blood film but we have had to rely on the small cell count from our Coulter counter owing to constraints on lab time - this will generally correlate well except in the case of a significant monocytosis. A TLC <1000 has a +ve predictive value of 0.97 for CD4 <200 and in the presence of T.B. a TLC <1250 has a +ve predictive value of 0.96 for CD4 <200. Another suggestive biochemical marker is the presence of a high globulin fraction with a normal total protein - related to polyclonal B cell activation.

Screening syphilis serology may be valuable at presentation since the prevalence in our health ward is 8.4% and there is evidence of an early progression to neurosyphilis in HIV +ve patients.

Management

It is important to appreciate that most of our efforts are directed to management of early/intermediate HIV disease with common- or-garden 'high virulence' infections which can be treated successfully and economically, since <40% of our patients present to the hospital in Stage 4. The presenting illness is TB in ~37%, weight loss/wasting in ~24% and STDs in ~10%. We do not at present offer routine follow-up as we are not in a position to offer anti-retroviral therapy and the efficacy/feasibility of co-trimoxazole and isoniazid prophylaxis remain unproven in our patients. All patients should be counselled about the risks of unsafe sex and early treatment of STDs.

MUCO-CUTANEOUS MANIFESTATIONS

Multidermatomal Herpes Zoster has a +ve predictive value of 90% for HIV infection in our setting and is usually an early manifestation. Dissemination of the virus and post-herpetic neuralgia are fortunately uncommon. Give strong analgesia and a topical anti-septic (povidone iodine, gentian violet) and reassure the patient that the eruption will subside in 2-3 weeks.

The occurrence of oral candidiasis strongly predicts imminent progression to AIDS independently of CD4 count - it may appear as plaques, flat erythematous patches or angular cheilitis. First line treatment is Nystatin oral suspension 100-500,000 u. 6 hrly - if this fails try gentian violet 0.5% solution 8 hrly. For resistant or oesophageal candidiasis we have a limited stock of ketoconazole (200-400 mg once daily for 7-10 days).

Ichy rashes of various kinds are a common and distressing problem for many patients. Important differentials are:

- Scabies (very prevalent infestation locally, typical distribution - give Benzyl Benzilate 25%)
- Folliculitis (staphylococcal infection usually on the chest/arms in males - give oral cloxacillin)
- Seborrheic dermatitis (caused by biostaphilococcal form of Pityrosporum orbiculare, the same organism implicated in pityriasis versicolor. Often inflamed /crusted lesions in the seborrheic areas. Acute lesions need wet soaks to dry and remove the crusts followed by clo-trimazole 1% cream applied 8 hrly +/- hydrocortisone 1%. 2% Selenium sulphide shampoo may be useful for recurrences.)
- Secondary syphilis (can take various sometimes bizarre forms)

Drug reaction (esp. hypersensitivity to TB drugs)

'Prurigo' (idiopathic mononuclear/leukocytoclastic infiltration of subcutaneous tissues) - systemic amphotericin (promethazine 25-50 mg nocte) are most effective but camphor or menthol containing ointments /creams may be helpful. A trial of a topical antifungal may sometimes be successful and some rashes respond to hydrocortisone.

KAPOSI'S SARCOMA

Antibodies to human herpes virus - 8 are found in 30% of adults in Harare, though KS has only become a significant clinical problem in the last few years. 'Benign' lesions (small lesions confined to skin without oedema or ulceration) causing symptoms may be treated with palliative local field radiotherapy - <50% of lesions will disappear with a recurrence-free period of ~5 months. Local palliation is also appropriate for the swollen painful limb. 'Malignant' lesions (extensive skin, lymph node or visceral involvement) are a pre-terminal event - systemic combination chemotherapy achieves a short partial remission in only 40% and symptomatic relief in <10% - it cannot be generally recommended for our patients.

Further Reading
MYCOBACTERIAL INFECTIONS

Tuberculosis is often the point of first contact with an HIV +ve person – refer to the T.B. protocols for diagnosis and management. Mycobacterium avium intracellulare is rarely documented in African patients though it is common in the environment - possibly because infection is inhibited by the acquisition of M.tuberculosis. Similarly Mycobacterium other-than-tuberculosis (M.O.T.T.s) though prevalent in South African miners, are exceedingly uncommon in our patients.

BACTERIAL INFECTIONS

* Invasive pneumococcal disease (-lobar pneumonia, bacteraemia meningitis, sinusitis) is a common cause of mortality in our patients. *Non-typhoidal salmonella and E. coli may also cause fatal septicaemia. Promp recognition and treatment with Ampicillin 500 mg 6 hourly and Gentamycin 240 mg daily - unless confirmed pneumococcal disease) is essential - even with treatment the mortality of such episodes may reach 30%. Treatment should be continued for 2 weeks where salmonella infection is suspected or confirmed. Bacterial infections of all kinds in HIV patients commonly *relapse* and Amoxicillin or Co-trimoxazole prophylaxis should be considered in these patients.

Further Reading
2) Angeler X et al. 1999 'Early chemoprophylaxis with Co-trimoxazole for HIV-1 infected adults in Abidjan, Cote d'Ivoire: A randomised trial.' *Lancet* 353: 1463

CHRONIC DIARRHOEA/WASTING

Access to safe water and adequate nutrition are essential in avoiding this problem but neither are available to many of our patients. Mealie meal is an easily digested high-energy food but may need to be supplemented with vitamins. Patients should boil their water and know how to make O.R.S.

In most cases of chronic diarrhoea stool examination will be negative or show cryptosporidiosis. However, a significant minority will have either a bacterial infection (salmonella, shigellosa or campylobacter) or a treatable infestation (isosporiasis, amebiasis, giardiasis). Management of patients with chronic diarrhoea should proceed as follows -

STOOL MICROSCOPY

- **Fever**
  - No Fever
    - Faecal Leucocytes
      - Assume bacterial
        - Co-Trimoxazole 966 mg b.d. for 5 days
          - Co-Trimoxazole 650 mg q.d.s. for 10 days
        - Consider Clofazimine 500 mg b.d. for 5 days
          - Metronidazole 400 mg u.d.s. for 10 days
      - Assume parasitic
    -粪便细胞
      - Febrile
      - Faecal Leucocytes
      - Assume parasitic
      - Co-Trimoxazole 966 mg b.d. for 5 days
        - Co-Trimoxazole 650 mg q.d.s. for 10 days
      - Consider Clofazimine 500 mg b.d. for 5 days
        - Metronidazole 400 mg u.d.s. for 10 days

A positive diagnosis of isosporiasis or cryptosporidiosis may be made on modified Ziehl-Neelsen stain of the stool.

Give Albenza 2 tabs stat to all patients as 40-80% will be infected with trichuris - a course of 800 mg twice daily for 2 weeks may also help to eliminate microsporidiosis.

If there is *no clinical response, the most likely pathogen is cryptosporidium* - symptomatic treatment with codeine phosphate 15-60 mg 6 hourly should be started.

Further Reading
1) Batchelor B et al 1998 'Microbiology of HIV associated bacteraemia and diarrrhoea in adults from Nairobi.' *Epidemiol. Infect.* 117: 139

ACUTE NEUROLOGICAL PROBLEMS

Treatable differentials here are *bacterial meningitis, cerebral T.B. (Meningitis and intra-parenchymal tuberculomas) and neurosyphilis*. We have diagnosed few cases of cerebral toxoplasmosis to date but 50% of our population have evidence of past infection. Cryptococcal meningitis occurs sporadically (~5 cases/year) and has been difficult to treat in our hospital.

Patients without localising signs should undergo L.P. where at all possible and a Gram stain, India ink and W.F. should be ordered on the C.S.F. in addition to the routine examination. Toxoplasma serology and Cryptococcal antigen may be sent to Durban.

Patients with localising signs should be commenced on Sulphadimidine/Pirimethamine 500mg/12.5 mg i.i M. and then i.i b.d. The differentials are toxoplasmosoma, tuberculoma and vasculitic stroke. If there is no clinical response to toxoplasma treatment after 2-3 days the
patient should be referred for C.T. scan and/or T.B. treatment should be commenced.

T.B. meningitis is treated with standard T.B. therapy + Prednisolone 60 mg daily.

Neurosyphilis (C.S.F. R.W.) is treated with 12-24 

Mu Benzylpenicillin daily for 14 days.

Cerebral Toxoplasmosis should be treated as above for 6 weeks and Co-Trimoxazole prophylaxis commenced.

We have usually offered symptomatic treatment alone for cryptococcal meningitis in the past, but with expanded availability of fluconazole, specific therapy should be feasible. Traditionally patients with impaired consciousness are treated with Amphotericin B 0.7 mg/kg iv for 7-14 days though oral Fluconazole 400 mg daily is otherwise adequate. If there is a clinical response within the first two weeks treatment can be continued with Fluconazole 200 mg daily for a further two months. Secondary prophylaxis with 100 mg daily or perhaps 2-3x/week is indicated. In non-responders morphine is often indicated for headaches, and for raised intracranial pressure, therapeutic LP/acetazolamide should be considered.

**PNEUMOCYSTIS PNEUMONIA**

This is as prevalent an infection in African patients as in Europeans but disease occurs uncommonly - in patients without smear +ve T.B. the highest recovery rate at bronchoscop has been <20% from HIV +ve patients with pulmonary complaints and a small series at our hospital failed to diagnose any cases. A typical presentation would be a patient with severe dyspnoea/Resaturation on pulse oximetry out of all proportion to the CXR appearances (which may be clear or show a 'ground-glass' appearance). Cysts may be demonstrated on Giemsa stain of the sputum in ~50%. Treat with Co-Trimoxazole 120 mg/kg/day in 4 divided doses p.o. iv. for 14 days + Prednisolone 40 mg daily if severely hypoxic. Secondary prophylaxis with Co-Trimoxazole 960 mg daily or 3x/week is indicated.

**CARE AT THE END OF LIFE**

When employment has clearly become impossible, the provision of a disability grant is an important supportive measure and will entitle the patient's children to a maintenance grant after his/her death. Preparations for terminal care are heavily dependent on the patient's disclosure of his/her HIV status to potential carers at home - thus far many patients have opted for hospital admission. Difficult issues may arise at this time - especially if the patient herself or relatives and friends are unaware of the diagnosis. Sensitive counselling is essential. The health team have a duty to encourage disclosure especially where it might have implications for the patient's partner or family but protection of the patient's confidentiality remains paramount. An often expressed anxiety concerns insurance - reassurance can be given that most life insurance policies taken out prior to 1985 and most funeral plans do not contain an AIDS exemption clause (though it is important to check the small print). Where the patient has remained in a state of denial until the time of death it may be prudent to avoid writing 'AIDS' on the death certificate though the patient's serostatus cannot legally be withheld from an insurance company if she has signed a waiver and the death should be notified confidentially to the Department of Health.

**HIV TESTING POLICY**

- All patients undergoing an HIV test should receive pre- and post-test counselling, ideally by a trained counsellor. This should aim to ensure that the patient understands the purpose of the test, its advantages and disadvantages, why the doctor has requested it, what impact it could have on his/her treatment and the likely psychosocial consequences.

- Informed consent should be obtained. It is preferable that this should be written and recorded in the notes.

- Testing without consent may be permissible in the following situations -
  - *Patient unconscious / too unwell to be counselled*: if emergency treatment is required that will be guided by an HIV test and will be in the patient's best interests and the patient has not previously expressed a wish not to be tested.
  - *Child <14 years with no parent/guardian available*: if emergency treatment is required that will be guided by an HIV test and will be in the patient's best interests. In a child clinical situation the permission of the

**STDS**

Always ask and look for these! Treat according to protocol though they may be more severe in HIV +ve patients. They are a source of great discomfort and a key factor in transmission of the virus.
Minister of Health under the provisions of the Child Care Act must be sought.

- **Mental illness** if consent cannot be sought from spouse, parent and curator (person appointed by law to look after them).
- In each case, consent must be approved by the Medical Superintendent in writing.

- **Disclosure of results** -
  - *Other Health Workers* consent is required from the patient to disclose the results to other health workers even those working in the same institution.
  - This restriction should only be waived
    i) after considering the decision carefully
    ii) after explaining the decision to the patient and
    iii) after accepting full responsibility for the decision.
  - *Sexual partners*: If the patient refuses to disclose to partners at risk the health worker must inform the patient of their duty to protect third parties and warn that he/she may be forced to break the confidentiality of the patient-health worker relationship. If the patient continues to refuse, the partner must be informed directly by the health worker.
  - **Post-mortem**: There is currently some dispute regarding the patient's right to confidentiality after death. This is an issue that obviously requires some sensitivity and judgement - family members not at risk need not be informed but this will usually be necessary if AIDS is mentioned on the death certificate.

- **Insurance** -
  Many Life Insurance companies are now being forced to drop HIV exclusion clauses but may find other pretexts for non-payment. Some policies are available which require the patient to test HIV-ve at the time of taking out the policy and demand expensive premiums. If the patient subsequently sero-converts the policy reduces to 10% of its quoted value. In general, patients should be advised to check the small print very carefully before taking out any life insurance policy. Unit Trusts and other social welfare payments are alternatives. We have so far experienced few difficulties with family funeral plans which provide only for funeral expenses.

Doctors are under no legal obligation to release information about a patient who has died unless he/she has signed a waiver of confidentiality from the company. The family are not legally empowered to give consent if the patient has refused.

Further Reading
3) Seldel G 1995 'Confidentiality and HIV status in KwaZulu/Natal, South Africa: implications, resistances and challenges' Health Policy and Planning 11:418
4) Lawyers for Human Rights 'AIDS and the Law'

**FOR FURTHER ADVICE PHONE**
- AIDS Law Project: 011 4036918
- Lawyers for Human Rights: 033 3421130
- Dr. Eddie Barker (MASA Ethics Committee)
  031 322111 Ex 518/9

**SCHISTOSOMIASIS**

Hlabisa district lies within the endemic belt of this disease. *S. haematobium* infestation rates may be as high as 25-50% (in Umboombo area around the Jozini dam) and we are at the southernmost extent of the distribution of *S. mansoni* with rates of up to 2-3% recorded in northern KwaZulu. *S. matthei*, an unengulate schistosome is also prevalent and can hybridise with *S. haematobium*, infecting man. Schistosomiasis is generally held to be a milder disease in South Africa than more northerly areas and we see serious complications of disease uncommonly. The usual presentations are -

- painful haematuria in a child
- 'bilharziasis' of the genitals esp. cervix
- urological problems in adults (obstructive uropathy, bladder polyps/calcification/carcinoma)

We have also seen cases of Symmer's Fibrosis with portal hypertension and periportal/abdominal lymph node involvement.

**Diagnosis**

- **S. haematobium** - Collect 3 urines voided at lunchtime (it is not necessary to exercise a sick patient beforehand!) Also ask for a dip test of the same urine.
- **S. mansoni** - 3 wet stool preparations +/- rectal snips/ scroaches. Biopsy of abnormal cervix, cutaneous lesions or liver.
Ova will be identified on microscopy, viability can be assessed by a simple on-the-slide hatching test if necessary. We do not routinely do egg counts but 3+ haematuria or 1–2+ haematuria with 3+ proteinuria on dip-test strongly predicts a count of >200 ova/10 ml—a 'moderate' to 'heavy' infection. Symptoms will often also be in proportion to the intensity of infection.

I.V.U. should be carried out on patients with suspected urological problems only. Transient abnormalities are common (<40% of asymptomatic patients). 'Active' disease is characterised by polypoid lesions in the distal ureters and bladder—the classic 'moth-eaten bladder' on post-mic film whereas 'inactive disease' typically involves obstructive uropathy and bladder calcification. Bladder carcinoma should be considered as a complication in the 4th-5th decade.

**Management**

- **Praziquantel** 40 mg/kg as a single dose.
- Treatment does not prevent the acquisition of immunity though it may reduce its potency.
- 'Light' infections do not need treatment unless symptomatic.
- 'Active' lesions on I.V.U. should respond well to Praziquantel but 'inactive' lesions with calcification on X-ray and dead eggs in urine usually do not. Surgery is however rarely required even in 'inactive' cases.

Further Reading


Infestation with soil transmitted helminths is common in our patients—in surveys of under-5s in the Umzimba/Inyanya districts the prevalences of infection with Trichuris, Ascaris and hookworm were 54%, 50% and 37% respectively. Probably 90% of children in our area have at least one helminth infection. Flagellate infestation is also endemic—rates for Endolimax nana, Chilomastix mesnili and Giardia lamblia were approximately 7%, 4% and 3% respectively. Taenia ova are identified in only 1.5% of single stool samples. These findings are in contrast to other areas e.g. South Coast where hookworm infestation may be less but taeniasis considerably more common. Cryptosporidium has recently been identified as a common infestation of children in Durban, recovered from 2.4% of unselected and 9% of paediatric gastroenteritis admissions. Strongyloides and Enterobius are both unfortunately uncommon (<1%).

The nutritional significance of these infestations in children is the subject of some debate. but the is at present our policy to de-worm children only with frank malnutrition, anaemia or other possibly related symptoms. Pilot programmes into mass deworming are currently ongoing in the province. Other complications are probably rare but may include:

- Loeffler's syndrome (weezy children with transient pulmonary infiltrates and eosinophilia, caused by migrating ascariid larvae—do not de-worm at presentation as verminous pneumonia may result when the larvae die)
- Small bowel obstruction due to ascariid 'worm balls'—this seems to be becoming less common though at King Edward Hospital, during 1950–1970, 7 children per month were admitted with this condition. Management is to drip and suck until the obstruction resolves and only then de-worm.
- Neurocysticercosis may be an important treatable cause of epilepsy though probably not as significant in our patients as studies from other areas might suggest
- Trichuriasis may present with a 'whipworm dysentery' and/or rectal prolapse
- 'Roundworm' as a cutaneous larva migrans caused by a canine hookworm (Ancylostoma braziliense) which local children pick up after handling/stepping on dog faeces.
Myiasis caused by the larvae of Calliphoridae flies is occasionally encountered in small children or neglected wounds in adults. Cordylobia species may be easily expresssed from their holes after application of liquid paraffin but more aggressive species not responding to this treatment may require debriement +/- irrigation with chloroform or ether.

Treatment

Treat according to the instestation identified on stool microscopy - though in clinics/O.P.D. it is acceptable to treat blindly for 'roundworms' which are often well described by mothers/patients.

- PIPERAZINE 120 mg/kg (to a maximum of 4 g) as a single dose - for Ascaris (roundworms)
- NICLOSAVIDE 2g as a single dose (1g in under-6s) - for Taenia (tapeworms)
- ALBENDAZOLE 400 mg as a single dose (200 mg in under-2s) - for Trichuriasis/hookworm (consider a second dose the following day in heavy hookworm infections)
- TOPICAL THIABENDAZOLE solution 4x/day for 1 week - for 'sandworm'

Further Reading

Nephotic Syndrome

This is probably the commonest presentation of primary renal disease that we see - its prevalence may well be related to the risk of type 3 hypersensitivity reactions in a population who are constantly exposed to microbial antigenemia. In adults and children, the commonest cause is membranous or membranoproliferative glomerulonephritis and may be related to Hepatitis B/C carriage, syphilis, schistosomiasis mansoni or SLE. We have also seen it in patients with HIV/TB. In both age groups 'minimal change' disease is rare.

The syndrome is defined for our purposes as:

- Proteinuria > 3.5 g/24 hours/1.73 m² (>360 mg/mmol creatinine on 24 hr collection)
- Serum Albumin < 30 g/dl
- Peripheral oedema

The usual findings are: ascites, pleural effusions and gross peripheral oedema often with an enlarged liver - it is therefore an important differential of 'chronic liver disease'. Hypertension and renal impairment may also be present depending on the underlying disorder.

We usually only carry out a single 24 hour urine to confirm the diagnosis - check U&Es, Albumin, Hepatitis B and HIV serology, RPR and auto-immune screen in females. In older patients consider a myeloma screen. Do a US/CT to assess the renal size (a gross discrepancy may indicate renal vein thrombosis).

Management

- Restrict salt to approx 1500mg/day.
- Give Frusemide to mobilize the oedema (dose up to 500 mg 0d may be required).
- Consider an ACE inhibitor where proteinuria is heavy - this may achieve >50% reduction in proteinuria but at the expense of a 25% reduction in GFR!
- Special diets are probably unnecessary and are unlikely to be followed by the patient after discharge. In any case it seems likely that most of our patients are already subsisting on a relatively low-protein diet.
- Be on the lookout for complications -especially venous thrombosis and infection (pneumococcal pneumonia and streptococcal cellulitis are a particular problem).
- All our adult patients should be referred for biopsy - according to the results the renal physicians will decide whether any specific treatment is indicated (e.g. immunosuppressants).
Further reading
2) Schlegel P et al. 1993 ‘Progress in untreated patients with idiopathic Membranous Glomerulonephritis’ Nephron 63:67

ACUTE RENAL FAILURE

It is important to appreciate that this is not usually due to primary renal disease and should be reversible in the majority of cases if managed correctly. It is more prevalent in the developing world - 60% is ‘medical’ (hypovolaemia, esp. diarrhoeal disease, nephrotoxic drugs, sepsis), 25% surgical (obstructive uropathy, stone disease) and 15% obstetric (either early septic abortion or inability to deliver a viable baby). Mortality is still high (~50% at 1 week) but usually results from complications (sepsis, bleeding) rather than renal failure per se. Common causes at Hlabisa have been severe malaria, burns, septicaemia and haemolytic-uraemic syndrome.

The diagnosis requires a high index of suspicion - up to 50% of cases are non-oliguric (esp. post-calciation of the tubules) or chronic retention. Physical signs are non-specific, so you may miss the diagnosis if you don’t check the U&E! Consider the possibility of pre-existing chronic renal failure (pigmentation, anaemia, small kidneys on US/S).

Distinguishing reversible ‘pre-renal failure’ from irreversible ‘acute tubular necrosis’ is difficult biochemically - suggested markers of the ‘pre-renal’ state are a disproportionately high urea, urinary sodium <20 mmol/l and a urine/plasma creatinine >40. These markers do not relate to the prognosis and the clinical picture will often tell you more.

Management
- If the patient is clinically volume-depleted, give a fluid challenge of e.g. 200 ml over 5 minutes +/- CVP monitoring if you are genuinely unsure of the fluid status. Continue until signs of volume depletion disappear.
- If there is no response or the patient is clinically fluid overloaded give a bolus of 80 mg Furosemide (the prognosis is better if you can avoid prolonged oliguria) - follow by an infusion of 2-4 mg/min (max dose 1g/day) if no urine is produced. Add dopamine at 2.5 µg/kg/min if oliguria persists and adrenaline 4mg (4 ampoules of 1 in 1000) in 200 mls saline titrated with the BP. If persistent hypotension seems to be responsible.
- Take an E.C.G. if the potassium is >6.0 mmol/l - any flattening of the T wave or widening of the QRS complex must be promptly treated. Give Calcium Gluconate 10 ml 10% slowly i.v. over ~1 minute (repeat until after effect) followed by i.v. insulin / dextrose (10 u Acrapid + 50 ml 50% dextrose repeated every 15 minutes) - the potassium should fall by 1-2 mmol/l over the next 30-60 minutes. As a follow-up or as sole therapy in cases without E.C.G. changes prescribe sodium polystyrene sulfonate 15g oral or 30g rectal 6 hly.

- Treat pulmonary oedema immediately with morphine 5-10 mg slowly i.v. and oxygen. Venesection of 100-200 mls blood or giving mannitol 300-400 mg/kg of 25% solution once only may be temporising measures but if you are not going to abandon the patient at this stage you must start peritoneal dialysis or arrange for emergency transfer.

- Restrict daily fluid intake to output over the last 24 hours + 500 mls, examine the patient regularly and weigh her if possible daily. Prescribe with caution!

- High protein diet is tolerated. Give enteral feeds to prevent stress ulceration.

- Be alert to the possibility of secondary sepsis.

- Hypertension should respond to Nilidipine.

- Patients without an obvious precipitant and a persistently active urinary sediment, hypertension etc should be referred for renal biopsy (≤5% of cases may be related to acute glomerulonephritis) and specific therapy.

Though mortality in the acute stages is high, in patients under 70 full recovery of renal function is the rule beginning after a median of 10-14 days, though it may be delayed for some months. Patients with a surgical aetiology have the worst prognosis.

Further Reading
3) Graziano et al. 1984 ‘Dopamine and Furosemide in acute renal failure’ Nephron 37:38
4) Sedeel et al. 1984 ‘Acute renal failure in Blacks and Indians in South Africa - Comparison after 10 years’ Nephron 64:188
DERMATOLOGY

"If it's wet dry it, if it's dry wet it and if neither works try steroids"! In most cases this approach won't do any harm and will usually work but there are notable exceptions and these will be covered below. The commonest conditions encountered in OPD will be covered as well as those rarer conditions which are important to recognise and manage.

ECZEMA/DERMATITIS

The terms are interchangeable and refer to inflammation of the skin due to various endogenous and exogenous factors. In the acute phase it is characterised by redness, erythema, papules and vesicles with exudate and crusting. As the condition becomes more chronic, skin thickening, excoriation, hyper- or hypo-pigmentation, scaling and fissuring occur. Itching and burning are prominent through all stages of the condition. Lesions can be complicated by secondary infection.

- **Contact Dermatitis (or Exogenous Eczema):** Increasing westernisation exposes people to more and more irritants and chemicals, causing a steady rise in the incidence of contact dermatitis.

- **Irritant contact dermatitis** is caused by a direct toxic or damaging effect of chemicals, such as acids, alkalis, solvents, detergents and cleansers, on the skin. This can occur either after a single exposure if strong enough, or after repeated exposure with less noxious substances (e.g., washerwoman's hands, napkin rash, vaseline dermatitis of the face).

- **Allergic contact dermatitis** is due to delayed hypersensitivity to various allergens. Once sensitised, dermatitis can occur within 24 hours of exposure. The most common allergens are zinc (jewellery, coins, keys, clothing fasteners and studs), rubber, cosmetics (a multitude of offending compounds), drugs, and plants (often children or there is a clear history of gardening).

- The site of the lesions often gives a clue to the aetiology, however, allergic dermatitis can become disseminated and occasionally the lesions can occur distant from the site of exposure. An occupational history is an essential part of diagnosis. It is important to realise that contact dermatitis may be superimposed on any other skin condition; once the skin is damaged or inflamed, its ability to act as a barrier is reduced and irritation is much more likely.

- Treatment is primarily the identification of the causative agent and its strict avoidance. This is often possible using occupational history and the distribution of the rash alone. Diagnostic patch testing may be useful in allergic contact dermatitis and can be arranged by referral to KEH. It should obviously not be done in the acute phase of the eruption.

- In the acute phase saline soaks and or weak steroids (1% hydrocortisone; the absorption of steroids is increased when the skin is inflamed) may be helpful.

- The restoration and maintenance of a suitable barrier between the skin and irritants is an important factor in management. Regular use of a barrier cream may be sufficient to avoid further episodes, ameliorating ointment applied twice daily to hands and affected skin as well as after every hand wash is very effective but its greasiness is often unacceptable. Aqueous cream is a less effective but more tolerable alternative.

- **Atopic Dermatitis:** This is familial and associated with asthma, hayfever and urticaria. We see this very rarely. It often begins at four to six months of age with itchy papules on the scalp and face, upper trunk, napkin area and limbs. It is intensely itchy. As the child ages it localises to the flexures where constant scratching leads to ichthyisation and hyperpigmentation. The extensor surfaces may be involved. Nearly half of all cases will have resolved by 18 months of age and all but the worst will clear by age 25. Stress is a significant exacerbating factor.

Treatment is with weak steroids, 1% hydrocortisone, once or twice daily followed by emollients once the inflammation has settled, e.g., aqueous cream, emulsifying ointment. **Antihistamines** e.g., promethazine 25-50 mg nocte or chlorpheniramine 1 m.g. 2-4x/day in children are extremely useful especially at night, more for their sedative effect than antipruritic effect. Avoidance of stress is important.

- **Seborrhoeic Dermatitis:** Probably due to the fungus Pityrosporum ovale, this condition was generally a condition of poverty and poor hygiene until the advent of the HIV epidemic. We see it quite commonly. It appears as greasy or powdery scale on an erythematous or dusky background. It is localised to the sebum producing areas i.e., the scalp, eyebrows, peri-auricular area, naso-labial folds, neck, axillae, groins and presternal and intrascapular regions. It can occur in infancy as well as adolescence. Itch is mild to moderate.
■ Treatment of scalp lesions is with seborrhoea scalp ointment applied twice daily. It is probably an idea to advise the patient to cut their hair very short or to shave it all prior to treatment. A medicated or anti-dandruff shampoo may be adequate and more acceptable in mild cases. Other lesions should be treated with an anti-fungal cream such as Whitfield's ointment (Compound Benzoic Acid Ointment) or 1% clotrimazole cream. Nystatin is ineffective. 1% Hydrocortisone may be added in severe cases; oral ketoconazole may be necessary in AIDS cases. The condition often relapses but usually responds to further treatment and can be kept under control by improved hygiene and occasional treatment.

■ Xerotic Eczema: Basically just dry, cracked skin common in the elderly, malnourished and those who frequently use strong soaps or disinfectants. It is quite common in HIV. Treat with emollients and avoid frequent washing and strong soaps.

■ Exfoliative Dermatitis: This is a severe generalised erythema with oedema, vesication and scaling. It requires urgent and intensive therapy. It can arise from excessive or inappropriate treatment of eczema or other dermatoses; as a drug reaction to e.g. sulphonamides, streptomycin; as a cutaneous manifestation of systemic disease e.g. haematopoietic disease or internal malignancy; as a severe complication of any dermatitis or de novo. It is one of the few dermatological emergencies, though seldom gets them out of bed! Patients are often systematically unwell and may be in shock from a combination of severe inflammation, fluid depletion and sepsis.

■ Treatment is vigorous fluid replacement (fluid loss through inflamed skin can be considerable), intravenous corticosteroids, hydrocortisone 100mg q.d.s. and systemic antibiotics e.g. ampicillin and cloxacillin. Topical steroids should be avoided initially as systemic absorption can be huge. Emollients should be applied frequently. Oral or topical therapy can be instituted as soon as an improvement is evident.

■ Lichen simplex arises from an initially pruritic lesion of any cause which is repeatedly scratched. This causes lichenification and thickening of the skin, which in turn causes further itching, thus creating a vicious circle. Treatment is aimed at breaking this cycle with an occlusive dressing over either potent steroid ointment e.g. 0.1% betamethasone or coal tar and salicylic acid ointment.

■ Dermatitis artefacta: This does occur and should be considered in any bizarre skin lesions or ulcers. It can be diagnosed by demonstrating healing by occlusion only. This requires admission and observation, usually by other patients who make reliable informants. Psychiatric help is often necessary.

■ Dermatitis may complicate any underlying skin disease and is often seen in scabies infestations and is exacerbated by treatment. Emollients and/or weak steroids should be given in conjunction with treatment.

FUNGAL INFECTIONS

Dermatophytoses are extremely common in Africa. Hot and humid weather predisposes to infection as do host factors such as diabetes, HIV, malnutrition and prolonged steroid therapy.

Diagnosis is clinical, with confirmation by microscopy of scrapings from lesions prepared with KOH. Clean the site with an alcohol swab, then scrape, gently collecting the skin or nail flakes on paper (hair can be plucked in cases of T capitis). The samples are transferred to a slide and a few drops of 10-40% KOH are added then topped with a cover slip. After 5-10 mins the slide is examined under low power and low illumination. Fungal hyphae are seen as long branching threads. In T. capitis fungal spores may be seen inside or outside the hair.

■ Tinea Corporis (Ringworm of the body): Usually presents as annular, single or multiple, ring-like lesions which spread peripherally. The central area may be scaly or as the lesion grows may become smooth and depigmented. The border is usually purple or red, raised, well defined, vesiculo-pustular or scaly. Lesions may coalesce forming large irregular patches. The distribution is asymmetrical. Lesions grow slowly over weeks or months. Pruritis is common and severe. Treatment is with Whitfield's ointment or 1% clotrimazole cream. Extensive or refractory cases may require systemic therapy with ketoconazole or griseofulvin.

■ Tinea Cruris (Ringworm of the groin): This affects the moist warm folds of the groin, especially in males. Pruritis is marked. There is a peripheral active border, which is diagnostic. In acute stages treatment consists of potassium permanganate (1 in 10 000) soaks, in the sub-acute phase Merthiolate is helpful as is 1% clotrimazole cream. Griseofulvin and ketoconazole may be used.
**VIRAL SKIN CONDITIONS**

- **Shingles:** Reactivation of latent varicella zoster virus usually affecting one, unilateral dermatome causing a painful pustular rash. The rash may just cross the midline in immunocompetent individuals but should not involve more than one dermatome. Underlying immunocompromise, especially HIV, should be suspected in extensive shingles affecting more than one dermatome. Treatment is symptomatic as acyclovir is expensive and most patients present too late to receive any benefit from it. The use of acyclovir should be reserved for cases of severe ophthalmic shingles only. The pain of shingles is extremely unpleasant so adequate analgesia, e.g., with ibuprofen 200–400 mg t.d.s. is essential: amitriptyline 50 mg once works well for this type of neuralgic pain (increasing the dose does not increase the analgesic effect). Gentian violet 0.5% should be applied to the rash to prevent secondary infection, which is relatively common. Remember babies and young children can catch chicken pox from an adult with shingles.

- **Warts:** Various papovaviruses are responsible for the different types of warts, including common warts, plantar warts and genital warts. There is no specific treatment for warts, i.e., no virucidal therapies, all treatments are aimed at debulking the wart and stimulating an inflammatory reaction in the hope that the host immune system will clear the virus. Most warts will in fact disappear spontaneously after a few years, so aggressive surgical treatment with the risk of scarring is seldom indicated. Curettage under local anaesthetic followed by gentle cautery is effective. Weekly application of podophyllin 20% in benzoin tincture can be used - cover the normal skin with vaseline, apply an occlusive dressing and wash the application off after no longer than 6 hours. Podophyllin should never be given to patients for self-treatment as it invariably goes horribly wrong. Extensive vaginal warts should be referred to the gynaecological clinic for treatment.
IMPORTANT MISCELLANIAE:

- **Psoriasis**: This is rare in our population. Seen mainly in young adults, it is generally a much milder condition than in the West. It presents with oval or round, erythematous, scaly macules and papules, which converge to form silvery, grey plaques. The lesions are typically found on extensor surfaces - the elbows, scalp, and shins and behind the knees. The course is one of relapse and remission. Treatment is with 2% salicylic acid in emulsifying ointment applied 2-3 times daily to affected skin or 100ml of coal tar solution added to a bath daily. Exposure of the lesions to sun is also very helpful.

- **Albinism**: Congenital absence of pigment from the skin, hair, iris and retina. Most of our patients seem to be of the partial type with yellow hair and blue eyes (autosomal recessive melanosomal deficiency). These individuals are extremely sensitive to the damaging effects of solar radiation. There is a high incidence of solar keratoses and later squamous cell carcinomas of the sun-exposed skin. Photophobia and nystagmus are common. Every opportunity should be taken to advise about the avoidance of prolonged sun exposure (e.g. sunglasses, long sleeves, umbrellas etc.) and total sun block should be provided regularly for head, neck and hands.

- **Cheloid**: A hypertrophic scar can be distinguished from a true keloid by its remaining within the boundaries of the wound whereas keloids tend to extend beyond the original lesion. The former tends to regress spontaneously whereas the latter tends to enlarge. Keloids are relatively common in black Africans and can be disfiguring, especially in young women. Treatment is with oral intralesional triamcinolone, 5mg/ml, at monthly intervals, if there is no response after 6 months treatment should be stopped. It should be reserved for severe lesions on exposed areas only as it is expensive and the outcome is often disappointing.

- **Melanoma**: The acral type of melanoma is the most common variety seen in black Africans. It appears as a patchy pigmented macule commonly at the proximal nail fold, heel or sole: subungual lesions are also common. The depth of the lesion at presentation is the most important prognostic indicator (Breslow thickness). Any suspect lesions should be excised completely with a full thickness biopsy and a wide excisional margin. Care should be taken to ensure the edges of the biopsy are perpendicular to the skin i.e. the defect left after excision should be U-shaped and not V-shaped.

- **Pemphigus**: One of the few dermatological emergencies. We have seen two patients with this condition in our district. It starts with blisters in the mouth that burst leaving painful ulcers then crops of bullae appear on the trunk, head and neck. The blisters burst to leave a raw ulcer that is slow to heal. Diagnosis is based on biopsy of a fresh, intact blister, preferably sent fresh, not in formalin, to the lab for immunofluorescence. Untreated the condition is fatal within two years. Adequate treatment will lead to a near normal life span. Large doses of prednisolone (60-100mg/day) are needed to bring the condition under control and other immunosuppressants (e.g., dapsone, azathioprine or cyclosporin) may be used for their steroid-sparing effects. All cases should be referred to the dermatologists for follow-up.

- **Porphyria Cutanea Tarda**: Occurs relatively commonly in Zulus and is thought to be due to a combination of alcoholic liver disease, hepatic siderosis and maize diet. Skin fragility with blistering and scarring of sun-exposed skin, especially the coronal of the hands is characteristic. It is treated by repeated venesection (500ml every 14 days) and chloroquine 200mg twice weekly.
TYPHOID

Notifications of typhoid fever from the northern KwaZulu hospitals have been falling for some years but we continue to see sporadic cases in our district, typically in the first half of the year. On average at present, each health district in Northern KwaZulu-Natal can expect to notify ~10 cases of typhoid/year. About two thirds of households have access to a toilet of some sort (pit latrine or flush toilet) but >50% still get their water directly from local rivers. At present, there is virtually no chloramphenicol resistance amongst isolates from our area.

Most cases occur in young people and the disease is considered to be greatly under-diagnosed and severe in children. It is important to appreciate that typhoid is a systemic illness and that symptoms referable to the G.I. tract are commonly absent. The most common symptoms are severe headache, dry cough and profound malaise. However, diarrhoea may be a prominent feature, especially in late presenters and HIV +ve patients. *Salmonella* is also characteristic in the third week and typhoid should always be considered in a young confused patient.

The cardinal signs are high fever (39°C) which is typically sustained. Relative bradycardia (the normal response is an increase of ~15 b.p.m./°C increase in temperature) is a highly specific but insensitive sign. Rose spots are hardly ever seen in our patients and splenomegaly is said to occur in only 15% of cases. Don’t be confused by chest signs - these are commonly present.

The White Cell Count is low in ~25% and a White cell count ~6 correlates significantly with complications.

Studies of the Widal test in South Africa have demonstrated that a titre of 1:200 + (O and/or H) has a specificity of >90% for bacteriologically proven typhoid but a sensitivity of only 75%. It appears that titres are frequently positive in the first week in South Africa and a fourfold rise in titre is rarely demonstrated. Furthermore, both O and H titres are thought to remain high for about 2 years after infection.

Blood culture has a high yield throughout the illness and stool and urine cultures after the second week (the latter is also important to exclude schistosomiasis which may permit a prolonged carrier state).

Chloramphenicol 50-75 mg/kg (~1g 6 hrly for an adult) is the drug of choice. I.V. is preferred initially but start oral as soon as appellate for 48 hours. Give 14 days total.

Monitor the patient closely for complications which may present insidiously. Perforation should be managed surgically as 75% are single and can be oversewn simply. This may be necessary on site if the patient is not fit for transfer.

In practice it has been impossible for us to confirm bacteriological cure and since only 3% of patients are likely to become chronic carriers we have not made strenuous efforts to follow-up patients discharged after admission for typhoid.

Further Reading

AMOEBAIOSIS

KwaZulu-Natal has for a long time been considered the ‘Amoeba capital of the world’ - the disease assumed epidemic proportions in South Coast slum areas during the 1950s. We see the disease much less frequently and it has certainly declined in importance as a cause of dysentery since the arrival of the shigellosa pandemic. Stool surveys identify *Entamoeba histolytica* cysts in <10% of our population whereas the non-pathogenic *Entamoeba coli* can be recovered in ~60%. Recent work has shown that many of these cysts may in fact be of another non-pathogenic ‘dead-ringer’ for *E. histolytica* called *Entamoeba Dispar*. Amoebic liver abscess is the principal serious manifestation in our health ward, although a few cases of colitis are picked up. Stricture formation and perianal disease are other serious if uncommon complications.

AMOEbic LIVER ABSSCESS

Few patients give a history of pre-existing or concomitant dysentery. The usual complaint is of right-sided hypochondrial or chest pain, usually for <2 weeks. Fever is present in 75% and is usually hectic/sustained (in contrast with colitis where fever is invariably absent).
Hepatic tenderness/swelling is present often with right basal chest signs. The presence of jaundice should suggest another diagnosis.

Polymorphonuclear leucocytosis is present in at least 80% and CXR may show a raised hemidiaphragm +/- pleuritis. The diagnosis is usually confirmed by US/S - a typical cystic structure will usually be present with transmission and shifting fluid inside of a similar echogenicity to the liver itself. They are usually single but rarely multiple. Do not look for amoebic cysts in the stool - they will be present in <50% - trophozoites may be identified in the 'fast drag' of an aspirate. Amoebic serology will be positive in 85%.

Treat with Metronidazole 800 mg t.d.s. for at least 3 and up to 10 days - this is the same high dose regime as for clostridia and dispenses with the need for a luminal amoebicide such as tetracycline.

We have followed a policy of selective aspiration for the following indications -
- Failure to respond to metronidazole (which may not penetrate a dense abscess wall, but the other concern here is to exclude a bacterial abscess)
- Raised hemidiaphragm (risk of rupture into the thorax with formation of a problematic amoebic empyema and hepato-bronchial fistula in up to 50%)
- Left lobe abscess (especially if adjacent to the pericardium)
- Pointing abnormally (rupture onto the abdominal wall may leave a nasty fistula and amoebic peritonitis carries a mortality of ≥20% even in the best hands).

There is little point re-scanning as this does not give any assessment of progress independently of the clinical picture and 1/3 of lesions take longer than six months to resolve completely.

AMOEBIC COLITIS

Generally a more chronic illness than bacillary dysentery and without fever. The best way to diagnose it is to do a warm, wet prep of stool straight from your glove +/- Esain counterstaining. The trophozoites typically contain ingested red cells. Even with this method, they are rarely seen in our lab, though cysts often are, and the widespread practice of giving Metronidazole for all cases of dysentery at clinic may have something to do with this. Management in confirmed cases (i.e. trophozoites seen, not just cysts) is Metronidazole 800 mg B I H for 5 days.

No cysticidal drug is currently generally available in South Africa and 90% of cyst passers will clear their infection by one year.

Further Reading
1) Adams E.B 1979 A Companion to Clinical Medicine in the Tropics and Sub-Tropics. Oxford University Press (references much of the original work by Powell et al. in Barbados)
2) Jackson TFGH 1995 'Amebiasis - resolution of a centuries old mystery and its impact on our understanding of the disease' C.M.E. 13 9 pp. 963
3) De le Rey Nel et al. 1999 'Indications for aspiration of amoebic liver abscesses' S.A.M.J. 75:373

SHIGELLOSIS

The African Shigella dysentery pandemic arrived in our district in 1994/5. The organism S. dysenteriae type I has supplanted other prevalent causes of dysentery in our district especially S. flexneri (>20% of isolates) and E. histolytica (<5%) - it is resistant to Ampicillin, Tetracycline, Chloramphenicol, Trimethoprim and Sulphonamides, and only demonstrates in vitro sensitivity to Nalidixic acid and Ceftriaxone. 150 cases were admitted to the paediatric ward during 1995 and the case fatality rate was 13%. Death was 3-4x more likely in children aged <2 years or who were malnourished, although it is important to remember that the disease is potentially serious in both adults and children. Shigella organisms can be isolated from the blood in ~10-15% of cases. Haemolytic-uraemic syndrome was a complication in 11% of admissions and vero-toxin producing Shigelllosis is a much more common cause of this disorder in black South African children at present than E.coli 0157.

Diagnosis

'Bacillary' dysentery is characterised by fever, blood and mucus-stained stools and colicky abdominal pain +/- tenesmus. The clinical presentation is so characteristic that further confirmation of the diagnosis is usually unnecessary but a highly positive faecal leucocyte test is typical of Shigella dysentery. Amebic trophozoites and campylobacters may also be recognised by an experienced microscopist on direct/ dark-field microscopy but are probably uncommon causes of dysentery in our district. Treatment decisions are virtually never made on the basis of culture in our hospital.

In all children and non-ambulant adults, check Hb, Pts, U&Es and ask about urine output. Be alert in the presence of complications. These include septicaemia, hypoglycaemia, myocarditis, encephalitis, pancreatitis and toxic dilatation/perforation in the acute phase. Children have tended to be hyponatraemic. Delayed complications include Haemolytic -uraemic syndrome, protein-losing enteropathy and reactive arthropathy.
Treatment
Non-severe
- Adults - Ciprofloxacin 500 mg b.d. 5 days p.o.
- Children - Nalidixic acid 15 mg/kg q.d.s. p.o. (Syrup available)
Severe
- Ceftriaxone 50 mg/kg/day slow i.v./i.m. b.d.
- In patients not improving after 48 hours, add Metronidazole 7.5 mg/kg 8 hrly

Haemolytic-uraemic syndrome - the crucial indicator in management is the urine output. Try to maintain >1 ml/kg/hr but be careful not to overhydrate. If the urine output falls whatever the aetiology cannot be restored with Furosemide 2 mg/kg stat i.v. bolus and/or Dopamine 3-5 µg/kg/min, then refer urgently for dialysis, as fluid overload can be stubborn and fatal in these children. Repeated transfusion may be required in the presence of continuing haemolysis and is indicated if Hb <6. Monitor BP and fluid status closely around the time of transfusions.

Encephalopathy - Check the Na +, B.P, Glucose. If all normal, presume Shigatoxin-related.

Further Reading

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PEPTIC ULCER DISEASE

Dyspepsia may affect up to 30% of rural Africans but the majority are not related to peptic ulceration. However, peptic ulcer disease can no longer be considered uncommon in black South Africans - the incidence of admission for this illness increased 12x at KEH VIII between 1950 and 1976. We have operated for four perforated D.U.s at our hospital since 1995 - in our experience this is a commoner complication than the pyloric stenosis traditionally attributed to peptic ulcer in rural Africa. The peak incidence is in the 3rd decade and sex ratio M:F 4:1.

Dyspepsia can be simply approached by distinguishing two symptom groups -
- **Reflex type** - complain of heartburn, acid braise and burning retrosternal discomfort made worse by lying down and large meals.
- **Ulcer type** - complain of epigastric pain often worse at night and relieved by antacids. The pain may be relieved or exacerbated by eating. In all cases do a stool microscopy and rule out use of NSAIDs and alcohol.

Management depends on the type of dyspepsia and the age of the patient (risk of malignancy).

**Dyspeptic symptoms for more than 2 weeks**

- 'Reflex' and under 45 years
  - Trial of Antacid
  - 10 ml t.d.s. after eating and at night
- Cimetidine 200mg nocte for 4 weeks

**Gastroscopy**

- Oesophagitis
- Duodenal/Gastric ulcer
- Gastritis (histological)

**Test for Helicobacter pylori**

- Omeprazole 20 mg b.i.d.

**Treat symptoms**

- Consider elimination therapy only if not controlled

Positive -
- Omeprazole 20mg q.d.
- Amoxillin 1g b.d.
- Metronidazole 400mg b.d
  (all for one week)

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*Helicobacter pylori* infection is much more common in Africa than Europe - 50% of African children acquire it by the faecal/oral route prior to age 10 years, ~75% of adults are infected. Metronidazole resistance is probably common in our district, especially in females. Chronic *H pylori* gastritis is the rule at gastroscopy in developing countries - it is found in 90% and eradication therapy seems not to
lead to an improvement in symptoms. In some studies it has been demonstrated to be a pre-malignant change. However, the incidence of gastric carcinoma is only 1-3 per 10^6 in black South Africans and the value of elimination therapy is unproven in this situation. Gastric ulcers are much less common than D.U.S but ~25% in RSA will be malignant so G.U.s should always be biopsied.

Further Reading
1) Louw JA et al. 1995 "Helicobacter Pylori eradication in the African setting with special reference to infection and duodenal ulcer recurrence" GUT 35:54
2) Segal et al. 1983 "Duodenal and gastric ulcer in Soweto" S.A.M.J 64(20):777
5) 1999 Diagnosis and Management of Dyspepsia - clinical guidelines S.A.M.J 80:897

POISONING

PARAFFIN

This is the commonest form of poisoning we see in children. Paraffin is a common fuel in many households and is often kept in unmarked drinks bottles. Fortunately it is irritant but has an unpleasant taste, so most poisonings involve small amounts. Due to its low surface tension it is readily aspirated and causes a chemical pneumonitis which may progress to haemorrhagic pulmonary oedema. Other common features are vomiting and drowsiness. Fever correlates with severe poisoning and almost never with secondary infection.

Pulmonary complications occur in up to 50% and are almost always apparent by 8 hrs. Respiratory distress and bronchospasm with a fever are the usual findings. CXR (does not need to be obtained routinely) may reveal perihilar or confluent infiltrates in a lobar distribution (rarely, pneumatoceles or pneumothorax) and a double gastric fluid level.

Do not give olive oil or milk and do not induce emesis unless it is clear on admission that the patient has ingested a massive dose (e.g. admitted in unresuscitable coma shortly after ingestion, when gastric lavage with a cuffed E.T. tube in place is indicated). Antibiotics should be prescribed only if fever persists and/or there is clinical deterioration after 24 hours. Patients with signs of pneumonitis should be managed supportively with oxygen and a short period of ventilation if required. Steroids are of no benefit.

PURGE NUT 'INHLAKUVA'

Small children sometimes ingest the seeds of *Jatropha curcas* (umbrella tree). These are black in colour and have a sweet, peanut-like taste. They contain curcin, a ricin-like poison that causes a severe gastroenteritis. The plant is commonly used by local traditional healers as a purgative. The clinical presentation is of abdominal pain, diarrhea, vomiting and sometimes dehydration. The condition is fortunately self-limiting and supportive treatment only is required.

JIMSON WEED 'ILLOYI'

*Datura stramonium* is a common weed containing parasympatholytic alkaloids including atropine and hyoscine. It is used by traditional healers as a wound dressing/poultice and may be smoked for chest complaints. Ingestion of medicinal preparations or food containing the plant (it may be mistaken for spinach) may result in a central anticholinergic syndrome characterised by confusion, altered conscious level, convulsions and features of atropinisation. It may be responsible for some cases of unexplained neurological/psychiatric episodes in our population. Neostigmine 1-2 mg i.v. repeated at 10 minute intervals according to the response may produce a dramatic response.

MUSHROOMS

The most common poisonous mushrooms in our area are *Amanita* spp. esp. *Phalloides* and *Chlorophyllum Molybdites* both of which may be mistaken for edible varieties. Poisoning by the *A. muscaria/pantherina* group (principal toxins - muscarine acid with low doses of muscarine) typically causes prompt dizziness, ataxia, hallucinations and sometimes convulsions. Frank muscarinic effects are uncommon. Supportive treatment only is required and fatalities are rare. *Chlorophyllum* poisoning is usually characterised by gastrointestinal disturbance within two-three hours and requires supportive care only.

Poisoning with *A. phalloides* and related species (principal toxins - amanitin cyclopellidase) is characterised by a delay in initial symptoms for 6-24 hours followed by profuse vomiting and choleatic diarrhea. A 48-72 hour remission then supervenes but is often followed by acute hepatic and renal necrosis which is fatal in 50-60% of cases. It is only in this phase that neurological symptoms may become prominent as a result of metabolic encephalopathy. Megadoses penicillin therapy is thought to oppose the effects of amanitins and doses of 1 Mg/kg/day should be given if poisoning is suspected.
PESTICIDES

Diazinon is the most widely used and freely available organophosphate pesticide in our area. It is available in preparations for clipping domestic animals and stock and is also used as a spray around farm compounds to control cockroaches. Paraquat is also in general use as a herbicide. Most of the other commonly used compounds e.g. Amitraz and Pyrethroids are essentially non-toxic to humans.

Mild Organophosphate poisoning is characterised by CNS stimulation and predominantly muscarinic effects e.g. nausea, vomiting, diarrhoea, sweating, miosis and hypersalivation. More severe poisoning involves CNS depression and nicotinic effects at neuromuscular junctions e.g. generalized weakness and fasciculations. Ganglion-blocking effects e.g. bradycardia and hypotension occur late. Atropine 0.02-0.05 mg/kg/hr i.v. should be given every 15 minutes until frank clinical atropinisation is apparent. This may involve doses of up to 5 mg initially and the dose should be tapered off slowly over 24 hours. Diazepam is effective to control convulsions. Oxime therapy is not available in our hospital and has not been shown to influence outcome.

An 'intermediate syndrome' of brainstem/CNS lesions with proximal peripheral neuropathy may develop 1-4 days after exposure and persist for 2-3 weeks.

Ingestion of paraquat causes corrosion of the gastrointestinal tract, hepatic and renal failure and progressive pulmonary fibrosis. Splashes of dilute solution may cause corneal and skin ulceration and inhalation (e.g. spraying without masks) may cause transient mucosal inflammation. Ingestion of significant amounts of solution carries a mortality of up to 60%. Since paraquat is virtually fully absorbed within 1 hour, gastric lavage/administration of activated charcoal even if carried out immediately on admission is unlikely to influence the outcome. The value of haemodialysis is doubtful since plasma levels of paraquat are low and it is rapidly distributed to other compartments.

IRON

Severe poisoning is only likely if more than one tablet/kg of Ferrous Sulphate has been ingested - abdominal x-ray can confirm the number since the tablets are radiopaque. The clinical features are biphase with initial abdominal pain, diarrhoea and vomiting of black fluid which subsides as the iron enters the reticuloendothelial system. In severe poisoning, this may be followed after 12-48 hours by shock, metabolic acidosis and signs of hepatic and renal failure. If there are clinical signs of shock or altered consciousness in the initial stages or if more than one tablet/kg has been ingested, give Desferrioxamine i.v. 2g adults /1g children, then 5mg/kg/hour for 12 hours.

POTASSIUM PERMANGANATE

This is another common childhood poisoning which seems benign in most cases. However, upper G I. erosion is described and rarely laryngo-/bronchospasm with bradycardia and hypotension. Hepatic and renal damage may occur. Treatment is supportive - give milk to drink and do not do gastric lavage.

ALCOHOL

Gastric lavage is of no value and most patients require supportive treatment only. However, keto- or lactic acidosis may occur and significant hypoglycaemia is a recognized complication in children - therefore start a 5% dextrose infusion and check the HGT. Add 50 ml 50% dextrose to the bag if it is low. Methanol may be present in small amounts in certain local brews but we have no way of confirming this - so in patients who are clinically acidoic give Sodium Bicarbonate 8.5% and titrate according to the response.

BATTERY ACID

Car batteries contain 28% Sulphuric acid. Strong acids tend to spare the esophagus but cause severe gastric lesions. Pass an e.g. tube but do not do a wash-out. Intubate if evidence of laryngeal involvement, as the situation is only likely to deteriorate. Resuscitate vigorously and refer immediately for gastroscopy/aparatomy if signs of perforation or haemorrhage. If the patient is stable, refer for endoscopy at - 5 days to assess the degree of damage and likelihood of stricture developing.

CANTHARIDIN

Some traditional healers are thought to make use of extracts of Mylabris ('blister') beetles in preparing aphrodisiacs. They contain high levels of cantharidin and poisoning is characterised by burning of the throat and genitals, vomiting and bloody diarrhoea. Haematuria and priapism may also occur. There is no antidote but vigorous resuscitation may be required.

Poisons information service St. Augustine's
Durban 0800 333444 (24 hour service)

Further Reading
2) Reed RP and Conradie 1997 'Epidemiology and clinical features of paratilin (sorusone) poisoning in rural African children' Ann trop. Med 17:49
Palliative care has been defined as the active, vital care of patients whose disease is no longer responsive to curative treatment. In resource-poor countries, a better description may be the care of patients for whom curative treatment is not available. It is often the only option available, but managing it well can mean the world of difference to the patient’s comfort and quality of life.

PAIN

The keys to good pain control are careful assessment, and where possible, diagnosis of the cause, use of the analgesic ladder and review of the effectiveness of the medication used.

Analgesic ladder -
(As per cancer pain relief programme WHO)
Non opioids: Paracetamol to max 1g/DOS
NSAIDS: Ibuprofen 200-800mg TDS
Weak opioids: Codeine, Codeine/Paracetamol mix, Valleran (children)
Strong opioids: Morphin, Pethidine

On each step, build up to the maximum dose of drug and frequency of that drug, and if the pain is not controlled, move up to the next step. Some classes can be mixed, i.e. NSAIDS and any of the others, and other drugs may be useful as analgesics e.g. carbamazepine.

Strong opioids in severe pain can be increased as necessary to control the pain without risk of overdose, but be aware of side effects – particularly those that may contribute to pain or discomfort e.g. constipation. If a dose of morphine is effective in relieving the pain, but doesn’t last 4 hours, a 50% increase in the dose should be made until pain relief is achieved.

PAIN NOT RESPONSIVE TO OPIOIDS:
BONE PAIN
NSAIDS can be particularly effective in bone pain, with or without a weak or strong opioid in addition.

NERVE PAIN
DYSAESTHESIA
Constant burning pain can be helped with tricyclics - in particular amitriptyline. Small doses are often effective - start at 10 mg at night and work up to a maximum of 100mg. Taking it at night also helps with the insomnia often related to pain. Remember, it may take about a week to reach effect. This can be useful with painful feet.
NEUROPATHIC/NEURALGIC PAIN

These are usually shooting in nature and can be eased by carbamazepine. Start at 100mg BD and increase as necessary.
Steroids can also be helpful in some nerve pain.

PAIN RELATED TO INFLAMMATION
Not surprisingly, these often respond to NSAIDS, for example headache due to meningitis or arthritic pain.

PAIN RELATED TO ORGANOMEGALLY
E.g. Liver pain from stretching of the capsule - may be relieved with steroids.

DIARRHOEA
- Codeine Phosphate
- Loperamide
- Stronger opioids as necessary i.e. morphine in severe cases (assuming treatment via the diarrhoea protocols has been tried and other treatable causes excluded).

NAUSEA AND VOMITING
- Metoclopramide - if vomiting is post food or medication, give antiemetic 1/2 hour prior to intake or in the case of tablets, if possible, give them with food.
- Alternative antiemetics include chlorpromazine or haloperidol due to their dopamine antagonistic effects.

BREATHELESSNESS
Relieve any obvious cause such as pleural or pericardial effusions or ascites and treat anaemia. Regular small doses of benzodiazepines may help to ease the panic sensation of breathlessness, or if necessary, regular short acting opioids e.g. morphine 2.5 (to the patient who can’t breathe), mg 4 hourly. Nebulised morphine solution, where available, can also be useful. Dissolve 5 mg morphine powder (i.e. for IV use) with 5 ml water and put in the nebuliser chamber.

COUGH
Codeine or even morphine can be helpful as cough suppressants. In resistant cases, nebulised local anaesthetic can work.

RAISED INTRACRANIAL PRESSURE
Steroids, in particular dexamethasone, may help by reducing associated cerebral oedema and in some cases shrinking the lesion. Start with 4 - 8 mg QDS, if there is to be a response, it is seen within a few days. If there is none, stop. If they respond maintain the dose for 5 - 7 days then start to slowly wean down.

CEREBRAL IRRITATION
Symptoms such as twitching, agitation or severe myalgia may be reduced with small regular doses of benzodiazepines; start with 2 mg TDS diazepam and increase as necessary. Be careful to determine irritability from fitting, which requires standard anti-epileptic treatment.
**CHOLERA**

Cholera is a water-borne diarrhoeal illness caused by low quality water supplies and poor sanitation. As I write, the District is in the middle of its second major outbreak in 10 years. The epidemic has affected the rest of the Province and appears to be spreading to the rest of the country.

**Cholera should be suspected when:**
- a patient > 5 years develops severe dehydration from acute watery diarrhoea (usually with vomiting)
- any patient > 2 years has acute watery diarrhoea in an area where there is an outbreak of cholera.

Up to 90% of people infected are asymptomatic. The incubation period is 2-3 days and most symptomatic patients have only a mild disease with loose stools 2-3 times per day for 5-7 days; this is clinically indistinguishable from any other form of diarrhoea. No treatment is necessary.

**Thirst in a patient with diarrhoea is a symptom dictating urgent treatment.** The patient has lost 2-3% of body fluid, and may also be vomiting. Treatment is with adequate quantities of oral fluid.

In the classic cholera case the onset of severe diarrhoea follows suddenly and shortly after a prodromal phase. Stool output rises rapidly, soon takes on the classic "rice water" appearance, and the patient can develop shock within a few hours. Untreated, the patient rapidly becomes dehydrated, haemo-concentrated and hypovolaemic. A metabolic acidosis develops. These and all the "complications" of cholera are a result of loss of fluid. Treatment is therefore fluid replacement.

At >5% fluid loss clinical dehydration starts to become obvious. The patient is in danger of hypovolaemic shock. If fluid loss exceeds 10% survival is unlikely. Treatment is urgent IV fluids.

Renal failure is a result of hypovolaemic shock. Metabolic acidosis is primarily a result of loss of bicarbonate in stool. Both can be corrected by adequate fluid replacement. Hypoglycaemia may occur in some patients, and since it carries with it a higher mortality, routine use of replacement fluids containing dextrose is recommended.

In children a fever and marked crowsness are often present, and coma, rare in adults, is not uncommon. Convulsions can occur which may respond to IV glucose.

Cholera is said to be more severe in pregnancy. In the last 3/12, maternal mortality may be as high as 50%. Most foetal deaths occur within 24 hours of onset of illness and early treatment does not appear to alter the outcome. Retained placenta after abortion is also common.

In all but the mildest cases fluid balance must be carefully monitored. If a cholera cot is not available, catheterise the patient front and back; we use a large Foley catheter rectally connected to a urine bag, which facilitates measurement and makes life easier for the nursing staff.

The type of IV fluid used is less important than the quantity. Ringer's Lactate is the fluid of choice; if available with pre-added dextrose, this is even better. In its absence N Saline is an acceptable alternative. Rehydration can and should be accomplished within 4 hours. As a guide, moderately and severely dehydrated patients require 50-60 and 90-120 mls/kg respectively plus replacement of ongoing losses. Antibiotics are indicated in patients with moderate or severe dehydration requiring IV fluid, especially if only N Saline is used as replacement fluid. A single dose of ciprofloxacin 1gm (20mgs/kg in children) should be given.

In addition, health education should be carried out at every opportunity. Patients and communities must be advised on the importance of water purification, good sanitation and scrupulous personal hygiene. Sedimentation, solar heating, addition of bleach and boiling are all methods of improving water quality at point of use.

**Further reading**

4. WHO 1993 *Guidelines for Cholera Control* (Good tables for fluid replacement)*.
Psychiatric services are poorly developed in rural South Africa, but the need in the community is great. A random cluster survey conducted in a rural community in KwaZulu-Natal using SRQ-20 and DSM-IV, found a prevalence of dysthymia of 7.3%, major depression 4.8%, generalised anxiety 3.7% and dysthymia/depression combined in a further 8.2%. Probably much of this morbidity is dealt with in the community by traditional healers. Few patients present to OPD with a complaint of depression alone, and if either occurs in the context of chronic physical illness, or presents with multiple somaticised complaints. Post-partum depression however, seems to be quite common in newly-delivered mothers: one community survey found it to be almost three times as common as in Europe. Practice at our hospital is dominated by florid acute psychoses often with very disturbed behaviour, which prompt families to bring the patient to hospital. Many of these episodes are short-lived and seem to be precipitated either by problems at home or heavy alcohol or cannabis use in men (cannabinoids are found in the urine of – 30% of black psychiatric in-patients, though they are not associated with any unique pattern of symptoms). In women they sometimes precede a calling to become a sangoma. True schizophrenia in the black population of South Africa seems to be associated more with aggressive/disruptive behaviour, irritability, sexual/fantastic delusions and self-neglect than in Europeans, and is thought to carry a poorer prognosis.

**ACUTE CONFUSION**

The priorities are:
- Keep calm and summon assistance
- Give adequate medication to control violent behaviour
- Exclude an organic cause
- Get a history from the relatives

**Assessment**
- Avoid confronting violent patients directly and make some attempt to allow them to air their grievances. However, severely disturbed patients, who constitute a danger either to themselves or the staff in OPD, should be sedated rapidly with Haloperidol 5-10 mg im/iv +/- Diazepam 10 mg im/po, repeated if necessary after thirty minutes to a maximum of 40 mg each per day. If the patient will accept oral medication, allow him/her to take it (same doses) and calm down in a quiet place. Reduce doses in the elderly/malnourished. Make sure Biperiden is available to deal with an acute dystonic reaction (5 mg im).
- Make a mental state examination of the patient as far as you can and carry out routine observations and a limited physical examination in OPD if the patient will allow it.
- Look for factors suggesting an organic basis for the confusion, especially alcohol and epilepsy. Particular points in the examination to pay attention to include:
  - Reduced and/or fluctuating conscious level
  - Abnormal attention and orientation
  - Incoherent speech
  - Abnormal involuntary movements
  - Visual rather than auditory hallucinations
  - Fever (typhoid, malaria)
  - Meningism
  - Ataxia/Nystagnus/VT palsies (Alcohol withdrawal)
  - Asterixis/Jaundice/Asches (hepatic encephalopathy)
  - Hypertension (hypertensive encephalopathy)
  - HGT (hypoglycaemia, often alcohol-related).

Routinely check the W&A and any other tests (Blood glucose, Kalquick, LP), dictated by your findings.

**Collateral History**

We rarely see the relatives again on the ward, so often this is the best opportunity! Important points include –
- Duration, form and content of symptoms
- Drug or alcohol use (Dagga, Ushwawa, Isigathala etc.)
- Precipitating psychosocial factors (quarrels, deaths etc.)
- Previous medical and psychiatric history, previous admissions and treatment
- Where and with whom does the patient live?
- Who is normally responsible for supervising medication at home and how adherent is the patient?
Admission

A period of medication-free observation would be ideal, but in practice patients requiring admission are often so disturbed or disruptive that psychotropics will be needed. Chlorpromazine 100 mg b.d. (ids if very agitated) will usually suffice, but also write a prn dose of 50-100 mg po/im up to 8 hrly. In elderly or frail patients start with a lower dose, e.g. 25 mg ids. Anticholinergic medication does not need to be given routinely. If the patient does develop troublesome extrapyramidal side-effects give Orphenadrine 50 mg ids.

Discharge

Usually the MO covering psychiatry will discharge the patient when their symptoms are controlled/resolved. Try to ensure that the relatives have been seen either on the ward or at a home visit by the psychiatry team.

Follow-up

Patients often need to continue medication and are given a date to attend their local clinic (preferably Monday or Friday mornings unless they need MO review) or OPD (Psychiatry nurse clinic Tuesday mornings). Fluphenazine decanoate is our first line maintenance depot injection, usually 25-50 mg im 2-4 weekly. Anticholinergics do not need to be given routinely. Those who need them can often be managed with Orphenadrine 50 mg ids for 3-4 days immediately after their injection, unless they have well-established tardive dyskinesia.

MENTAL HEALTH ACT

Patients being transferred to a psychiatric institution for further assessment, usually Madadeni Hospital, need to be admitted under the Mental Health Act 1973, unless they are willing and able to give properly informed consent. An application for admission on form 62/2 (available in OPD) should be completed by two medical officers and presented to the magistrate, who may then issue a reception order, which allows the patient to be detained in the appropriate institution for a maximum of 42 days.

DEPRESSION

Tricyclics are usually the first choice, starting at 50-75 mg nocte and increased over 1-2 weeks to 150 mg nocte. Doses as high as 300 mg may be necessary. However if response is poor, consider early use of fluoxetine 20-40 mg daily. Be wary about plans for discharge if there is a significant risk of suicide/self-harm, as tricyclics may take up to two weeks to work.

ALCOHOL PROBLEMS

Most local alcohol abusers seem to drink sporadically and tend to present with an acute psychosis immediately post-binge, rather than true delirium tremens or Wernicke's encephalopathy. However, if you suspect alcohol as a precipitant, start a 5% dextrose infusion with 100 mg thiamine added to each litre. Continue vitamin B complex 8 hrly orally + vitamin C 50 mg 12 hrly. Prescribe diazepam 20 mg stat po or 5-10 mg slow iv. Re-assess in two hours and give the same dose again 2 hrly until symptoms subside. If loaded orally further sedation may not be required as diazepam has such a long half life but if loaded iv continue 5-10 mg iv.d.s. for 2-3 days in reducing doses. Rehabilitation in the community has been mostly unsuccessful in our experience, since intensive support has been impossible to provide and we haven't found disulfiram to be a useful alternative.

Further Reading

2) Davies G 1984 Initiation of primary psychiatric care in Transkei 65:96-14
3) Ensmink K et al. 1992 “Expression of schizophrenia in black Xhosa-speaking and white English-speaking South Africans” 63:893
4) Solomon K et al. “Toxic cannabis psychosis is a valid entity” S.A.M.J. 78: 476
PAEDIATRICS
GASTROENTERITIS

Acute infective GE is a common cause of morbidity and mortality in South African children, with data from 1988 - 1995 suggesting that 43.5% of deaths in black children in the postneonatal period were attributed to GE. In the Hlabisa Hospital paediatric audit (1995 - 1996) acute GE was the primary diagnosis in 21% of inpatient deaths, with dysentery another 13%. Rotavirus has been demonstrated to be as common (~ 20% of cases) as bacterial pathogens in black SA children - Enteropathogenic/toxigenic E. coli, Campylobacter and Shigella Flexneri are the commonest bacterial isolates.

Remember that diarrhoea and vomiting can be the presenting features of parenteral infections (otitis media, pneumonia, meningitis, sepsis etc.) and surgical conditions (appendicitis, intussusception etc.)

Although GE can be a minor illness treated successfully by ORS in the community, patients presenting to the MO have usually failed this management. A further trial of supervised ORS in OPD may be appropriate but remember many of these children are dehydrated and metabolically deranged. Additional attention must be paid to the nutritional status and the use of enemas since these both contribute to fluid and metabolic disturbance.

The natural history of acute gastroenteritis is towards resolution within 5 days, if there has been no improvement at all within this period suspect complications. The development of chronic diarrhoea is an ominous development and nutritional rehabilitation is a critical aspect of management.

History
- DURATION: acute
  - prolonged - ie over 2 weeks
  - recurrent - consider
  1. cycle of undernutrition/recurrent GE/further malnutrition
  2. social re-exposure to pathogens - water source etc.
  3. HIV status
- STOOL: frequency (<8x/day associated with secretory diarrhoea and hypotraemia)
  - consistency
  - colour - blood/mucus (? dysentery - see separate protocol)

- FEEDING: type and volumes of food and fluids tolerated
- FEVER
- URINE: frequency and volume (mothers can reliably assess this)
- ENEMAS
- RTHC: weight - absolute and relative to previous immunisations

Assessment

HYDRATION STATUS:
A rapid but thorough examination with attention to nutritional and hydration status determine the management. Intravenous fluids will be required for all those who are:
- SHOCKED i.e. impalpable radial pulse or capillary filling time > 4 sec
- DROWSY or FLOPPY
- ABDOMINAL DISTENSION - ileus /obstruction
- ACIDOSIS - gasping / sighing respiration
- FAILED ORS - no improvement after 4 hours supervised
- FAILING FEEDS - resistant vomiting after 4 hours

ADMIT
- anyone requiring a drip
- any child
  - <1mth old
  - <3rd centile or Kwash
  - recurrent GE in 2nd episode in 1 month

THE KEY TO GOOD MANAGEMENT IS REGULAR CRITICAL REVIEW

Fluid Therapy

THE AIM IS TO COMPLETE REHYDRATION WITHIN ~ 4 HOURS

If shocked:
- RINGERS/NORMAL SALINE 30 ml/kg BOLUS IN OPD
- Intravenous line if cant get IV within 5 minutes.
- Give O₂
- Keep warm
- HGT stat and 2-4 hourly
- Review after 30 minutes, repeat 30ml/kg bolus if no change.
If not shocked or following above resuscitation:
- 1/2 Darrow's + Dextrose 15ml/kg/hr
- Review after 4hr
- Start oral fluids ASAP
- Use homemade Sugar-Salt Solution - teach mum to make

S.S.S. = 1 LITRE BOILED WATER + 8TEASPN SUGAR + 1/2 TEASPN SALT
- Give by cup/spoon, NEVER a bottle. Slow sips not cup/lis 15-25 ml/kg/hr - guided empirically by volume of stool/urine
- Continue breast (formula if no choice) after 4 hours or as soon as on oral fluids - give milk and then offer S.S.S. - do not water down the milk!

Antibiotics
- <1 month age - Penicillin and Gentamicin
- M malnourished / Immunocompromised - Ampicillin and Gentamicin
- Shocked (likely septicaemia) - Ampicillin and Gentamicin
- Dysentery - Nalidixic acid or Ceftazoline (see separate protocol), consider Erythromycin or Metronidazole if poor response (possible Campylobacter or Amoeba infection)
- Coexistent parenteral infection eg meningitis/pneumonia, urinary tract infection.

Electrolyte Imbalance
DO U+E SAME DAY ON ALL WHO NEED ADMISSION

A. Hyponatraemia:
Usually associated with malnutrition and hypoalbuminaemia. Central Pontine Myelolysis has not been described in acute hyponatraemia in children.
- If Na⁺ < 125 mmo/l/give Ringer's at 10ml/kg/hr - repeat U+E after 8hr Change to 1/2D+D when >125 mmo/l.

B. Hypernatraemia:
Usually late, well-nourished children on formula feeds. Implies significant intracellular hyperoncoticity and dehydration and giving isotonic solution risks rapid osmosis into cells and cerebral oedema. Suspect if rubbery skin or jellify/irritable with less dehydration than expected.
- If Na⁺ >150 mmo/l/give 1/2 D+D 1L + 48ml 8.4% NaHCO₃ at MAX 10ml/kg/hr.
Change to 1/2D+D after 24 hrs.
Repeat U+E's after 4 hours. Na⁺ should fall at ≥1 mmo/l/hr, bicarbonate may drop the K⁺.

C. Hypokalaemia:
Suspect with acidosis, severe hypotonia, abdominal distension/ieus.
- If K⁺ >4.0 mmo/l, or continuing diarrhoea, and taking orally it should self-correct but monitor and consider excess K⁺ loss eg oedema, vomiting etc.
- If K⁺ <4.0 but >2.5 mmo/l and drinking, give KCl 5ml of 15% solution orally TDS for 3/7 (i.e. the IV preparation) but dilute with at least equal vol. milk.
- If K⁺ <2.5 mmo/l correct IV i.e. 40 mmo/l KCl into 1L IV fluids.
REPEAT - IDEALLY AFTER 4 HOURS - K⁺ levels change rapidly both ways and may be the cause of sudden collapse and death even after a few days.

Nutritional Support
REFEEDING IS CRUCIAL TO AVOID CYCLE OF GE AND MALNUTRITION.

Continue milk as soon as back onto oral fluids. Start looas soon as tolerated. Avoid fat and fibre. Don't worry if the stools remain watery if the child is improving. Delay feeds for 48 hours in children who were admitted in shock due to the risk of gut ischaemia, but feed all others.
- VITAMINA: give to all - 100,000U if <1 yr; 200,000U if >1 yr. give orally if possible (unless documented dose within last 3mth).
- MALNUTRITION REGIME: malnourished but also consider if dramatic weight loss or underweight for age or prolonged or recurrent diarrhoea.
- MAGNESIUM SULPHATE: 0.5ml IM once weekly if diarrhoea over 1 week.
- FOLATE: 5mg OD 5/7 if diarrhoea over 1 week.

Further Reading
4) Ferrinho PO et al. 1989 "Diarrhoeal diseases and oral fluid therapy in the Delukuspan health ward" SAMJ 78:496
5) Wittenberg DF 1985 "Pediatric diarrhea: rehydration therapy rethought" S. A. M. J 85:555
CHRONIC DIARRHOEA

This is defined as 3 or more liquid stools per day persisting for 14 days or more. 5-10% of children with acute GE develop chronic diarrhoea which accounts for at least as many deaths as the former (4% of paediatric in-patient deaths in Hlabisa).

Risk factors include pre-existing malnutrition, less than 1 year of age, recent acute diarrhoea, immuno-suppression (including HIV), recent non-enteric infection (especially measles), previous persistent diarrhoea. Primary lactose intolerance is present in ≤50% of black children in Durban, but this seems to cause few clinical problems.

Despite such a clear definition it can sometimes be difficult deciding if a particular child has persistent diarrhoea, since the pattern described by the carer may not be reliable. We tend to admit and treat as acute GE for the first few days to get some idea of what's going on. If the diarrhoea continues profusely or a clearer story emerges then consider the measures below. Time scales are given as a guide only.

Prevention of Chronic Diarrhoea
STOP ACUTE DIARRHOEA BECOMING CHRONIC
- Use oral rehydration - vomiting stops earlier hence eating begins sooner.
- Food = Treatment: Do not stop breast feeding in children with diarrhoea.
- If formula fed, restart feeds as soon as back on oral fluids - give every third hour. Do not dilute milk.
- Air for normal diet in 3 days - encourage starchy foods e.g. porridge, mashed banana.
- Don't worry if food makes diarrhoea worse at first - it'll help once it gets absorbed.

Treatment of Chronic Diarrhoea
If diarrhoea lasts over 7 days:
- Fofetex 5mg DD 7/7
- MgSO4 0.5ml IM once weekly.
- Consider changing feeds as below:
  - Formula fed infants: change to Pelargon - If settles then discharge on Pelargon but review 2 weeks. If doesn't settle then stick test for reducing substances: If >0.5% pos change to Infalasoy. If -ve refer to Infalasoy then give breast milk cocktail.
  - Breast fed infant: Test for reducing substances - If result positive 2% or more then change to Infalasoy and get mum to express and discard (to maintain milk production with a view to recommencing breast milk later - within a week of the diarrhoea stopping, before discharge from hospital).
  - If less than 2% or negative continue breast milk.
    - If either group not improving give BOWEL COCKTAIL:
      Gentamicin 80mg BD orally 3 days. Cholestyramine 1g qds 3 days.
    - If above measures are not helping then start a semi-elemental formula (talk to dietician at NGZ).

Discharge
Do not discharge a breast fed baby until back on breast feeds. If stable on a Pelargon or Infalasoy then discharge on this formula and review in 2 weeks to consider reintroduction of lactose etc. If requiring semi-elemental formula then do not discharge without an appropriate food challenge.

Special points
- Ensure there are no parasites in the stool and consider an HIV test.
- If a child is still not responding then consider referral e.g. for TPN if HIV negative.
- If it's a difficult case talk to the paediatricians/dieticians for advice (Ngwelezane or King Edward Hospital).
- Special feeds - NAN = whey predominant, PELARGON = casein predominant acidified INFASOY = lactose free, ALFARE = semi-elemental whey hydrolysate.

Further Reading
1) Mofokate K et al. 1982 "Persistent and acute diarrhoea as the leading causes of child mortality in urban Gumbi-Bissau" 76 MSH 276 716
3) Waterberg DF and Mosia A 1991 "The practical significance of lactose malabsorption in institutionalised black children" S.A.M.J. 79 70
ACUTE RESPIRATORY INFECTIONS

Globally Acute Respiratory Infections (ARIs) are the main cause of death of children under the age of five and are a major cause of childhood morbidity, accounting for 28% of the paediatric medical admissions to Khulisa Hospital IN 1995 and about 13% of deaths under the age of 1 year in South Africa. Lung puncture studies show that 75% of serious lower respiratory tract infections are bacterial (streptococcus pneumoniae, haemophilus influenzae) but in RSA 3-18% of admissions are due to RSV. The advent of the HIV epidemic and vaccination against measles, pertussis and Hib have also changed the clinical picture in recent years.

Whilst prevention is critical to child health via improving nutrition, vaccination coverage and indoor air quality, in the short term the priority is to ensure that pneumonia is recognised early and treated before it becomes life threatening.

Assessment
- The WHO standard case management strategy relies on reproducible signs that can be elicited by all health care workers, particularly fast breathing, which is a much better predictor of ARI than any other clinical sign including auscultation.

<table>
<thead>
<tr>
<th>Severity</th>
<th>Criteria</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Cough, Fever</td>
<td>Paracetamol</td>
</tr>
<tr>
<td></td>
<td>Respiratory rate &lt;50/min</td>
<td></td>
</tr>
<tr>
<td>Moderate</td>
<td>Cough, Fever</td>
<td>Paracetamol</td>
</tr>
<tr>
<td></td>
<td>Respiratory rate 50-100/min</td>
<td>Amoxicillin</td>
</tr>
<tr>
<td>Severe</td>
<td>Cough, Fever</td>
<td>- See Below</td>
</tr>
<tr>
<td></td>
<td>Respiratory rate &gt;100/min</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Intercostal recession</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cyanosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unable to draw</td>
<td></td>
</tr>
</tbody>
</table>

Youth infants (severely malnourished)
- Infants under 2 months or severely malnourished should be moved up a class e.g. moderate to severe. They are more prone to severe infections and deteriorate rapidly, and are more difficult to diagnose, as they often do not cough but may have a variety of non-specific signs e.g. feeding, convulsions, abnormal breathing, etc. It may be difficult to distinguish pneumonia from septicaemia, URTI or meningitis. A low threshold for admission.
- Admit all children with severe pneumonia to ICU.

Management
- **OXYGEN THERAPY**
  - Oxygen is indicated if
    - central cyanosis
    - inability to drink
    - severe chest indrawing
    - ≥60 breaths/minute (if 2 months to 5 years)
    - grunting (if <2 months)
    - restlessness, if O₂ improves condition
- Preferably by NASAL CATHETER - an 8FG catheter with holes at the end inserted until the tip lies inside the back of the nose (same distance as between nostril and inner tip of eyebrow). The tip should not be visible when the child's mouth is open. This is safer (more efficient) than the nasopharyngeal catheter and more efficient than the safer nasal prongs.
  - Most can be treated with flows of 1 litre/min or less. Do NOT give more than 2 litres/min as there is a danger of gastric distension.
  - Continue 2-4 days usually depending on response

ANTIBIOTICS

>2 MONTHS
- **PO Amoxicillin** (non severe)
  - <1 year 10-15mg/kg 1ds
  - 1-5 years 125-250mg qds
  - >5 years 250-500mg tds (total 8 days)
- **IV Ampicillin** (severe)
  - <1yr 15-20mg/kg qds
  - 1-5 yrs 125-250 mg qds
  - >5 yrs 250-500 mg qds

Switch to amoxicillin when apyrexial for 24 hours. If very severe, or no response after 48 hours suspect staphylococcal pneumonia / tracheitis.
- **IV Chloramphenicol** then oral when tolerated and apyrexial 12.5-25mg/kg qds, max 500mg (total 10 days)

<2 MONTHS
- Suspect Group B strep and Gram negatives
  - Benzyl penicillin 20-25 mg/kg qds iv/im AND Gentamicin
  - <2.5 kg 2.5 mg/kg od, <3.5 kg 3.5 mg/kg od and gram negatives) >3.5 kg 5 mg/kg od (for 14 days)

IF HOSPITAL ACQUIRED
- IV/IM Gentamicin or Ceftriaxone (risk gram negatives)
- **Bronchodilator** if wheezy
  - <1 year Atrovent nebs 125mcg in 2 ml saline 1ds
  - >1 year Salbutamol nebs 1.25-2.5mg in 2ml saline 4-6 hourly >PRN
- Vitamin A
  <1 year 100,000 units,
  >1 year 200,000 units PO/IM stat
- Paracetamol if febrile
  2-12 months 60mg, <5 years 120mg, >5 years 500mg
  4-6 hourly, max 4 days
- Further investigations
  CXR if not initially indicated unless there is doubt
  about the diagnosis, poor response to initial therapy
  or complications are otherwise suspected. Consider
  a KalQuick test or thick film in season, since respiratory
  distress due to severe malaria can be hard to
distinguish from pneumonia.

IMMEDIATE COMPLICATIONS
  - If there is a poor response in the initial phase after
    changing to chloroquine consider:
    - foreign body aspiration (collapse on CXR, needs
      bronchoscopy)
    - effusion/empyema which should be drained by tube
      or referred for thoracotomy if refractory
    - PCP which accounts for ~10% of acute severe
      pneumonias in high HIV prevalence areas of Africa.
      The typical features are a child < 6/12 with severe
      hypoxia and clinically clear chest. Co-Trimoxazole
      is the treatment of choice.
    - acute tuberculous bronchopneumonia with
      characteristic lymphadenopathy on CXR.

DELAYED COMPLICATIONS
  - 70% of children with chronic lung disease will be
    HIV-positive and/or malnourished while bronchiectasis
due to pertussis, measles etc. is probably declining
to successful vaccination. In this group, tuberculosis is
about as common as lymphoid interstitial pneumonitis
which is associated with clubbing, parotid enlargement
and a reticulonodular pattern on CXR. If a trial of TB
therapy fails, the trial of steroids is usually indicated if HIV
status is known.

Further Reading
1) WHO. Acute respiratory infections in children. Case management
   in small hospitals in developing countries. A manual for doctors
   and other health workers. WHO ARI 50
2) Graham S et al. 2000 “Clinical presentation and outcome of
   pneumonia in HIV-infected children” Lancet 355: 369
3) Juema PM et al. 1998 “Persistent and chronic lung disease in HIV-1
   infected and uninfected African children” AIDS 12: 1185
   virus infections in South African children” S.A.M.J. 89: 937
5) van Schrindt YE et al. 1991 “Acute respiratory infections as an
   important cause of childhood death in South Africa” S.A.M.J. 80: 79
6) Redd SC et al. 1994 Clinical signs of pneumonia in children among
   a hospital outpatients’ department in Lesotho” Bull. W.H.O. 72: 113
7) Ongango FE et al. 1993 “Hypoxaemia in young Kenyan children with
   acute lower respiratory infection” B.M.J. 306: 512

H. influenzae B epiglottitis has been an extremely rare
condition in South African series and this might reflect a
lack of LPS in SA strains. Upper airway obstruction is
however a particular problem in malnourished/measles
patients with bacterial tracheitis. As many as 1 in 20
children with acute laryngotracheobronchitis will require
intubation.

Diagnosis
Viral croup is by far the commonest form. A confident
diagnosis of this condition can be made if:
   - a previously well child under 2 years of age gradually
     acquires a progressive inspiratory obstruction before
     cough
   - start a day or two after the onset of an URTI
   - mild fever (fever < 38.0°C) may be present
   - the child is well, apart from the respiratory obstruction

Features suggesting another cause:
   - dramatic onset of severe obstruction (foreign body)
   - incomplete immunisation (diphtheria)
   - dysphagia or if the patient prefers a sitting position
     (epiglottitis, retropharyngeal abscess)
   - systemic toxicity e.g. with rash (Spilke tracheitis)

Check the CXR for an inhaled radio-opaque FB/segmental
hyperinflation and look at the upper airway silhouette.

ASSESSMENT OF SEVERITY

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>INSPIRATORY Obstruction only</td>
<td>Observe in OPD/home if favourable conditions</td>
</tr>
<tr>
<td>II</td>
<td>INSPIRATORY + PASSIVE EXPIRATORY Obstruction</td>
<td>Admit</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nebulised Adrenaline* hourly</td>
</tr>
<tr>
<td>III</td>
<td>INSPIRATORY + ACTIVE EXPIRATORY Obstruction</td>
<td>Admit</td>
</tr>
<tr>
<td></td>
<td>(i.e. visible / palpable abdominal</td>
<td>Continuous nebulaled Adrenaline* in 1 hour,</td>
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<tr>
<td></td>
<td>contraction of palpable</td>
<td>consider intubation and transfer</td>
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<tr>
<td></td>
<td>pulsus paradoxalis)</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>RESPIRATORY FAILURE</td>
<td>Continuous nebulaled Adrenaline* intubate and</td>
</tr>
<tr>
<td></td>
<td></td>
<td>transfer for ventilation</td>
</tr>
</tbody>
</table>

*Adrenaline/nebulisers = 2 ml 1:1000 in 2 ml 0.9% Saline

Note: Stridor may become so much as the obstruction
becomes more severe.
- Give humidified $\text{O}_2$ by mask (not tube).
- Keep child comfortable and avoid painful procedures as crying exacerbates laryngeal oedema. Feed small amounts as often as tolerated.
- Give Dexamethasone 0.15 mg/kg stat i.v./p.o. as a single dose to all admissions. The i.v. preparation can be used as a nebuliser if the patient is in extremis or demonstrates tachypnoea to adrenaline.
- Give Penicillin & Chloramphenicol if - acute onset, fever >38.0°C, systemically unwell, older child, concomitant pneumonia.
- If intubation is required, the best method is to induce with ketamine 2 mg/kg maintaining spontaneous respirations with 100% $\text{O}_2$ throughout. Spray the pharynx and cords with lignocaine and pass a flexible ETT (2.5 mm if <12, 3.0 mm if 6-12 and 3.5 mm if 12-3 years). If the tube won’t pass easily with gentle pressure don’t force it, use the next smaller size. Magill’s forceps may be useful to direct the tip of the tube but avoid the temptation to push it in forcefully. Ensure that spontaneous respiration is re-established - arrange for transfer in an ambulance with skilled paramedical staff and a transport ventilator.

Further Reading

2) van der Walt D et al. 1989: ‘Steady state growth of Lactobacillus: A meta analyzer of the evidence from randomised trials’. Paediatrics 83: 582.

MALNUTRITION

This is a serious problem in our health ward. 50% of children in our area are below the 10th centile, given on American growth charts. 21% of all paediatric admissions in 1995-6 had signs of severe malnutrition, though this was not always par se the reason for admission. A further 22% were classified as ‘underweight for age’. These ill children are however only the tip of the iceberg - community surveys have also demonstrated a high prevalence of stunting in well children in our area. The staple cereal maize, though high in carbohydrate, contains poor quality protein which lacks tryptophan and lysine and is poorly absorbed. It is also bulky and repeated feeds during the day are necessary to supply the required daily calories.

Every mother should be advised to supplement her children’s porridge with vegetable oil, peanut butter, sour milk (amasi) or beans.

Every child attending under-5s clinics should have an up-to-date Road-to-Health card with serial weights from each attendance - however it is important that growth monitoring does not become an end in itself. Children clearly littering need some sort of assessment and intervention! The causes of malnutrition are complex and will often lie outside a strictly medical remit. However a common scenario is a cycle of repeated infection and worsening nutrition, especially during the weaning period.

Our community nutrition programme is attempting to address broader issues such as maternal education and food security.

The decision to admit to hospital is usually determined by co-existing illnesses and/or unambiguous signs of kwashiorkor or marasmus, but some children are admitted when U.W.F.A. where intervention in the community has failed, if they are a TB contact or social circumstances are suspect. The Nutrition Office is best placed to make this assessment in borderline cases and will arrange for any follow-up necessary in the community.

The case fatality rate for severe malnutrition on the ward is ~20% despite intensive intervention. This reflects the fact that ~30% of marasms and 10% of kwashis are HIV +ve and highly susceptible to gram negative septicaemia, often from co-existing and underdeclared UTIs.

<table>
<thead>
<tr>
<th>Weight for age</th>
<th>Oedema present</th>
<th>Oedema absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>(% of expected)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50-60</td>
<td>KWASHIORKOR</td>
<td>U.W.F.A.</td>
</tr>
<tr>
<td>&lt;60</td>
<td>MARASMIC-KWASH</td>
<td>MARASMUS</td>
</tr>
</tbody>
</table>

The Wellcome Classification of severe protein-energy malnutrition (NB: the expected weight = 50th centile for the child’s age)
KWASHIORKOR

This is often an acute presentation on a background of chronic undernutrition, typically characterised by apathy, oedema and weight loss, often with thin, gingery hair and characteristic dermatitis. Hepatomegaly from triglyceride deposition is usual and diarrhoea can result from malnutrition itself. The child seems uninterested in food.

Every child with signs of kwashiorkor should be admitted (if possible to ICU). These children are very ill and severely immunocompromised. They have an increased total body Na⁺ and a decreased total body K⁺ which leads to cardiovascular instability, oedema and paralytic ileus/gastric stasis. However they may also be hypovolaemic due to hypalbuminaemia. They cannot thermoregulate and rapidly become hypothermic. Infection is present in most but usually clinically silent.

MARASmus

These patients demonstrate severe muscle wasting (the arm circumference will be <12.5 cm), hair/skin changes are absent and they are often hungry.

The two clinical syndromes are not always distinct, and some marasmic children admitted with an intercurrent infection rapidly develop signs of kwashiorkor on the ward.

ON ADMISSION
- O.R.S. +/- breast milk only p.o. 2 hrly for 24 hours.
- Keep child warm, check temperature and HbTs hourly.
- Check Hb, U&Es and diuretic urine.
- If shocked give 20ml/kg Ringer’s or blood if severely anaemic. Children requiring continued i.v. fluids should also be given Calcium gluconate 4 mg/kg/day while on i.v.i. - try to avoid this wherever possible and use 1/2 D₄₀ for maintenance.
- i.v. access required for antibiotics. Give O.R.S. 4mLs/kg/hr only to mildly dehydrated children but more severely dehydrated children should receive 1/2D₄₀ at 5-10 mls/hr and be reassessed after 4 hours.

Stat doses on day of admission
- Measles immunisation (if not documented, in case there is an outbreak on the ward).
- Albendazole ii tabs stat.
- Folate 5 mg stat.
- Vitamin A 100,000 i.u. stat i.m. (<1 year) or 200,000 i.u. (1-5 years) - repeat day 2.
- Vitamin B₂ Co 1 amp stat i.m.
- Vitamin K 5 mg stat i.m.

Start
- Ampicillin 25mg/kg + Gentamicin 5mg/kg daily i.v. for 5 days.
- Oral Potassium syrup 5-7 mmol/kg/day (continue until oedema resolved)
- Magnesium hydroxide 0.5 mmol/kg/day i.m. for 7 days
- Folate 5 mg daily
- Vitamin K 5 mg i.m./p.o.
- For 14 days Zinc Sulphate tablets 15 mg tablets
- Vitamin B₂ Co 1 lab. daily p.o.
- Vitamin C 1 tab daily
- Multivitamin syrup 10 ml daily
- Ferrous Sulphate 3 mg/kg/day as soon as gaining weight and apyrexial (NOT before - increased mortality from infection)

Feeding
- Give clear fluids only for 48 hours if no significant abdominal distension (ileus, risk of aspiration)
- Otherwise EBM/full strength formula 100 ml/kg/day with similar volume of clear fluids immediately after each feed. N.G. feeding is frequently required due to anorexia.
- Start high energy milk after 5-7 days.
- Poor prognostic signs are hypothermia, hypovolaemia, Albumin <16, Jaundice, Bleeding, purpura.
- Oedema is not in itself a prognostic indicator so Albumin infusion is rarely indicated for this reason alone.

High Energy Milk (F-100)

28 Scoops* dried whole milk (110g)
7 Scoops sugar (50g)
4 Scoops oil (30g)
*Use only a blue scoop

Make into a paste and slowly add cooled, but warm, boiled water to make up litre.

Further Reading
2) Macnabue UE 1994 Protein-Energy malnutrition - results of rehabilitation Supplement: SA M.J July
Road to Health Chart

**IMPORTANT**: Always take this card with you when you visit any health clinic, doctor or hospital, and present the card on school entry.

**Child's name**

- **Gender**: boy / girl

**Date of birth**

- **Day**: ________
- **Month**: ________
- **Year**: ________

**Place of birth**

- **Birth weight**: ________
- **Birth length**: ________
- **Birth head circumference**: ________

**Problems during pregnancy / birth / neonatally**

**APGAR**

- **1 min**: ________
- **5 min**: ________

**Gestational age (weeks)**

**Serology**

**Mother's Name**

- **Literate**: ________
- **Illiterate**: ________

**Father's Name**

**Constitution not the mother**

**Where does the child live?**

**How many children has the mother had?**

**Number born**: ________

**Number alive now**: ________

**SPECIAL NEEDS (circle if answer becomes YES)**

- Was the baby less than 2.5kg at birth [ ]
- Is this baby a twin [ ]
- Is this baby bottle fed [ ]
- Does the mother need more family support [ ]
- Are any brothers or sisters overweight [ ]
- Are there any other reasons for taking extra care for example tuberculosis, single parent etc.

**IMMUNISATIONS**

**PRIMARY BOOSTERS**

**Date given**

**Signature**

**Date given**

**Signature**

**BCG**

1. [ ]
2. [ ]
3. [ ]

**DTP**

1. [ ]
2. [ ]
3. [ ]
4. [ ]

**Measles**

1. [ ]

**Other**

1. [ ]
2. [ ]
3. [ ]

**Vit A**

1. [ ]
2. [ ]

**Vision screening (6 - 8 yrs)**

**Hearing screen (7 to 9 months)**

**Result L**: ________
**Result R**: ________

**CARD GIVEN AND MOTHER TAUGHT BY**

**ORAL REHYD. Taught**

**RAJON DATES Used**

**TUBERCULOSIS SCREENING**

**Heal/Mantoux/Hide**

**Date**: ________
**Grade**: ________

**TB contact**: ________
**TB notified**: ________
MEASLES

We have seen few cases of measles in our district since 1994. 10,000+ cases/year were notified until 1990 when mass immunisation campaigns began, but large outbreaks have continued to occur in recent years in RSA and many cases occur in previously vaccinated individuals. Annual mass National Immunisation Days will continue in the near future in an effort to push up the vaccination rate to the required 95%, but eradication seems unlikely to be achieved in the near future since the virus has such a high infectivity. The disease carries a 5-15% case fatality rate in Africa and may cause persistent immunosuppression for as long as two years afterwards.

Diagnosis
- The incubation period is 10-14 days with fever, coryza and conjunctivitis.
- Koplik's spots (small red spots on the buccal mucosa with white centres like salt grains) appear 2 days prior to the appearance of the rash.
- The rash is typically a maculopapular erythema spreading centrifugally from the head and neck, lasting by the third day and desquamating with resolution of fever.

Management
Many patients can be managed at home as most are non-infectious by the time the rash appears.
- Give Vitamin A 100,000 (under 1s) or 200,000 i.u. orally at least once to all cases. Ideally this should be repeated the following day and at 4-6 weeks.

Admit
- Children under 1 year
- Signs of malnutrition
- Complications (fever after the third day, evident pneumonia, gastroenteritis or encephalitis)

Treatment
- Anoxyclillin 125-250 mg p.o. 6 hrly for 7 days
- Chloramphenicol eye ointment 6 hrly for 5 days to all admissions.

Problems on the ward
- Pneumonia and Laryngotracheobronchitis - Gentamicin 5 mg/kg i.v once daily for 1 week + Clexacillin 125-250 mg 6hrly i.v.
- Diarrhoea - usual supportive care + Metronidazole 200 mg p.o. for 5 days all/prior to discharge.
- Corneal ulceration - commonly due to malnutrition/vitamin A deficiency but consider topical Acyclovir if oral ulceration as well. In HIV +ve children Pseudomonas keratitis is a common cause and requires Gentamicin drops 1-2 hrly.
- Encephalitis - supportive management and control of convulsions are the only effective interventions.
- Try to ensure good nutrition in the month after recovery.

Further Reading
1) Hussey GD & Klein M 1990 'A randomized controlled trial of vitamin A in children with severe measles' N E J M 323 160
2) Warnell HC et al. 1979 'Severe orofacial herpes of mouth and eye following measles' T R S T M H 73 95
5) Hussey GD 1986 'Clinical problems in measles case management' Ann Trop Paediatr 16 301

MENINGITIS

Bacterial meningitis carries a case fatality rate of 20% on our paediatric ward. The commonest isolates in KwaZulu-Natal are probably H.influenzae, S.pneumoniae and N. meningitidis (in that order) and studies of pneumococcal meningitis in Ubonombo have demonstrated very few cases of intermediate penicillin resistance. The commonest identified causes of 'aseptic' (Gram stain negative) meningitis in South African children are tuberculosis (~20%), enteroviruses (~15%) and mumps virus (~5%). TB is now the commonest form of meningitis attributable to any single pathogen in Cape Town. However, quite a large proportion of gram stain negative, bacterial culture positive cases occur, probably related to recent antibiotic administration in the community for associated parameningeal infections.

Diagnosis
Suspect the diagnosis in any patient with acute onset of non-localising neurological signs. The history is usually <24 hours in classical bacterial meningitis - suspect another diagnosis if longer. Meningeal signs will often be absent in infants or unconscious patients. Check the ears and sinuses for evidence of parameningeal infection. L.P. is also indicated in any child <18/12 with febrile
convulsions of the older child with more than two such seizures during a single illness.

Lumbar puncture is almost always safe when the fontanelle is open - cerebral herniation does uncommonly occur in older children in the absence of localising signs but is poorly predicted by clinical or CT findings (papilloedema excepted). However, dextrose lumbar puncture pending resuscitation in shocked/dehydrated patients.

<table>
<thead>
<tr>
<th>Cell Type</th>
<th>Normal</th>
<th>Bacterial</th>
<th>Tuberculous</th>
<th>Viral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cells</td>
<td>0-5/mm³</td>
<td>&gt;1000/mm³</td>
<td>&lt;50/mm³</td>
<td>&lt;50/mm³</td>
</tr>
<tr>
<td>Protein</td>
<td>≤0.05 g/l</td>
<td>&gt;1.5 g/l</td>
<td>1.5 g/l</td>
<td>&lt;1 g/l</td>
</tr>
<tr>
<td>Glucose</td>
<td>≥90% of meals</td>
<td>&lt;50% of meals</td>
<td>&lt;50% of meals</td>
<td>≥90% of meals</td>
</tr>
</tbody>
</table>

Gram stain has a sensitivity of <60% vs. culture in the presence of prior antibiotic treatment. It may be slightly more sensitive than India ink staining at detecting cryptococci.

A "traumatic tap" may cause confusion about the diagnosis - Subarachnoid bleeding is excluded if the CSF clears gradually during the tap and there is no xanthochromia. Add two WBCs and 0.1 g/l protein to the normal values for each 1000 red cells present.

Management
- Give O₂ and routine care of the unconscious patient (nurse on side, turn 2 hrly). Monitor the response to treatment with assessments of tone, pupils and head circumference 1-2×/day.
- Give a stat. dose of Dexamethasone 0.1 mg/kg i.v. immediately prior to starting antibiotics. If the patient is unconscious or gram stain shows Gram -ve cocci-bacilli in a conscious patient continue with 0.1 mg/kg 6hrly for 3 days.
- Start Ampicillin 100 mg/kg 6 hrly and Chloramphenicol 25 mg/kg 6 hrly i.v. while waiting for LP results. If the gram stain shows Gram -ve cocci/gram -ve diplococci change to Penicillin 1-2 M 6 hrly. If the LP shows Gram -ve cocci-bacilli or no organisms in the presence of pleocytosis and reduced glucose continue Chloramphenicol but reduce the dose to 12.5 mg/kg 6 hrly after 48 hours. Continue antibiotics for 10 days (14 days if <2 months). However, if the diagnosis was circumstantial 5-7 days will suffice.
- Restrict fluids to no more than 2 ml/kg/hr i.v. and check U&S - SIADH is common and over-hydration may precipitate cerebral oedema.
- Control convulsions with bolus Diazepam 0.3 mg/kg i.v. p.r.n. and a loading dose of Phenobarbitone 20 mg/kg slow i.v. if not well controlled. If convulsions continue give 5 mg/kg maintenance doses daily.
- If there is no significant change after 48-72 hours - Repeat the Na⁺ to exclude significant hyponatraemia.
- Do a cranial US/S if the fontanelle is still open.
- Repeat the L.P. (if there are no contra-indications)
- If there is a persistent pleocytosis and gram stain with no localising signs and normal US/S, consider changing to Ceftriaxone.
- If the findings are now more consistent with TBM, start TB treatment (HRZ + Ethambutol for children).
- (In practice some children are started on both Ceftriaxone and TB treatment without LP prior to referral for CT - this may show significant basal enhancement and allow the former to be withdrawn).
- If there are evolving localising signs, increasing head circumference or evidence of enlarged ventricles or subdural collection on US/S refer for CT and drainage/shunting at KEH VIII.
- Patients with a parameningeal focus should always be referred early for C.T. scan as the risk of subdural encephalomyelitis is high in this group.

Further Reading
3) Raja R et al. 1995 "Cranial tomography in pyogenic meningitis in neonates and infants" J. Hosp. Infect. 41 163
5) Renwick S et al. 1993 "Cerebral hypoxia during bacterial meningitis in children" BMJ 306 953

Otitis Externa
Infected ear canal with normal drum? -50% fungal. Post-auralic L/S may mimic acute mastoiditis but the ear is not usually displaced downwards.
- Dry mopping, Acetic acid 2% in alcohol on a wick changed four times a day.
- Cloxacillin p.o. if frank furunculosis visible on otopscopy or aural cellulitis.
OTITIS MEDIA
Most of our cases present late, usually with long-established chronic supplicative disease and wet perforations. Always check the ears in children presenting with intracranial sepsis - many cases have a parameningeal focus!
- Acute - normally caused by S. pneumoniae but ~ 20-30% incidence of H. influenzae and M. catarrhalis in the under-5s. (Mostly non-β lactamase elaborating).
  Give paracetamol and Amoxicillin p.o. Myringotomy in the acute phase is almost never required but may be indicated if otorrhea is present.
- Chronic - dry mopping +/- suction with a syringe and 5FG catheter. Never plug an ear with otitis media and advise the patient to prevent water getting int. Give 1% acetic acid in saline drops at least 4 x/day. Systemic antibiotics are of no value.
- Assess for complications - Refer early if attic or marginal perforation, evidence of mastoiditis or offensive/sarosanguinous discharge suggesting tuberculosis/cholesteatoma. The last two especially are associated with cranial nerve (mostly VIII) palsies.
- Patients with central perforations should be referred for assessment for tympano/ossiculoplasty when the ear is dry. This may help to prevent the infection re-occurring even if audiometry is relatively favourable.
- Middle ear effusion (chronic suppurative otitis media) is rarely picked up in black South African children but should be referred for insertion of grommets if unresolved after 3 months, and affecting hearing or language development.

Further Reading
4) Liebowitz LB et al. 1987. 'Minocycline susceptibility of upper respiratory tract pathogens to 13 oral antimicrobial agents' S.A.M.J. 72:385

GLOMERULONEPHRITIS
We see both nephrotic and acute nephritic presentations quite commonly in local children.

ACUTE NEPHRITIC SYNDROME
This is due to post-streptococcal glomerulonephritis in 95% which results from a recent impetigo/erythema in almost all cases. The disease seems to occur in better-nourished children who are able to mount a stronger hypersensitivity response to streptococcal antigens. It is uncommon under the age of two years. The history is usually of puffy face/swelling of the body and dark urine but haematuria may only be microscopic. Be aware that hypertensive encephalopathy or pulmonary oedema may occur in children with mild peripheral oedema and preserved renal function. Such complications may occur in <15%.
- The diagnosis is confirmed by the finding of red cells casts on urine microscopy and a tive A.S.O. titre (1:160 or greater in 90%+). Throat and skin swabs have a low sensitivity (~30%).
- Order 4 hrly abs inc B.P., CXR, U&E's/Cr, Albumin/Total Protein. Make sure accurate fluid charts are kept, weigh and diuretic urine daily.
- Restrict fluids on admission to 15 ml/kg/24 hrs and thereafter to the previous days output.
- Low-salt diet (NO Cheesy puff(s)). Low protein diet if urea >20 mmol/L. However, don't try to enforce bed rest.
- Penicillin VK 250 mg 6 hrly x 10 days (eliminates the infection but probably has no effect on the outcome).
- Control hypertension - confirm the readings yourself. If no 7 or 12 cm cuff is available, use an adult 20 cm cuff around the leg. This is equivalent to brachial B.P. +10 mm Hg but the large cuff will tend to underestimate the B.P. of a small child. The 90th percentile for a 5 year old is 110/70
- Give Frusemide 1-2 mg/kg p.o. 8 hrly if diastolic >90 mm Hg.
- Give Nifedipine 5 mg sublingually if diastolic >110 mm Hg.
- Pulmonary oedema -
  - Nurse upright, O2 3-6 l/min by mask.
  - Morphine 0.1 mg/kg i.v.
- Frusemide 2 mg/kg i.v. over 5-10 minutes.
- Repeat 4 mg/kg if no prompt response.
- Dopamine 3-5 mg/kg/min can also be used.
- Emergency peritoneal dialysis and/or refer if no improvement.

The prognosis of PSGN is excellent - most children undergo a quick diuresis and recover within seven days. Mild persistent haematuria/proteinuria in the first few weeks is common but always check the U&Es prior to discharge as this helps to exclude the rare complication of rapidly progressive GN. There is thought to be no long-term effect on renal function in the uncomplicated case though microscopic haematuria may persist for ≤18 months and be exacerbated in the presence of intercurrent infections.

Further Reading

NEPHROTIC SYNDROME

Defined as Oedema, Proteinuria (>50 mg/kg/24 hours) and Albumin <25 g/l. The commonest cause (~40% of cases) is HBsAg +ve membranous glomerulonephritis. Only ~13% have histological changes consistent with Minimal Change disease and of these only half are steroid-responsive. Schistosomiasis may be found to co-exist with nephrotic syndrome but is probably not causally related. Syphilis is a rare cause in children.

- The diagnosis is in practice confirmed by 2-4+ proteinuria on dipstick with a urine microscopy that shows no signs of UTI. The simplest way to quantify this is to do an early morning specimen for protein/creatinine ratio. In nephrotics the ratio is >200 mg protein/mmol creatinine (normal <20); Check U&Es/CT, Albumin and send blood for Hepatitis B and syphilis serology.
- Order daily weight, B.P. and urinalysis. Accurate fluid charts are helpful.
- Low salt diet.
- Give Hydrochlorothiazide 1mg/kg + Spironolactone 1mg/kg daily. To control oedema try not to fluid restrict as there is a risk of precipitating hypovolaemia in these children. Give 20% Human Albumin 5 ml/kg over at least 2 hours with Frusemide 1 mg/kg cover for resistant oedema.

- Give Penicillin VK 250 mg b.d. in the initial stages as nephrotics are very prone to bacterial infection esp. Pneumococcaemia. Be on the lookout for infection especially UTI and periostitis.
- HBsAg/HBeAg has a +ve predictive value for membranous GN of 90/98% respectively. There is a spontaneous remission rate in this group of 30% over 5 years and they should be followed up on diuretic therapy only +/- analapril for persistent heavy proteinuria. HBsAg +ve patients should be given a trial of steroids (2 mg/kg/day max. 60 mg for 6 weeks then 0.5 mg/kg/day for a further 5 weeks) if there is no response, refer for assessment and biopsy. Treat any other specific precipitating infection (esp. Syphilis and schistosomiasis).

Further Reading

TUBERCULOSIS IN CHILDREN

In a country like RSA where the annual rate of infection is estimated at ≤2%, as many as one child in five will have acquired T.B. infection by the age of ten years. Probably 10% will at some stage of their life develop T.B. disease and this is both most likely and most severe in the under 5s and teenagers. Diagnosis is difficult as less than 30% can be demonstrated to be culture +ve on smear of sputum or gastric aspirate (the latter requiring special alkaline media). The approach is therefore one of ‘circumstantial diagnosis’, in most cases though a significant number of older children will be able to produce sputum for smear and children with scrofuloderma may have a +ve smear from a sinus or +ve culture/histology on lymph node biopsy. However the appearance of the latter is so characteristic that a trial of T.B. treatment is often justified.

We have found it useful to apply the WHO provisional guidelines for diagnosis rather than any particular scoring system -
### A. Suspect Tuberculosis

1. An ill child with a history of contact with a confirmed case of pulmonary T.B.
2. Any child:
   2.1 Not regaining normal health after measles/pertussis
   2.2 With loss of weight, cough and wheeze not responding to antibiotic therapy for respiratory disease
   2.3 With painless swelling in a group of superficial LNs

* For malnourished children this is defined as wt gain of >10% in one month.
* Mycobacterium bovis and other MOTTs are very uncommon in our practice.

### B. Probable Tuberculosis

A suspect case and any of the following:
1. Positive tuberculin test
2. Suggestive CXR
3. Suggestive histology
4. Favourable response to T.B. treatment

### C. Confirmed Tuberculosis

1. Detection by microscopy or culture of tubercle bacilli from secretions or tissues, or
2. The identification of the tubercle bacilli as Mycobacterium tuberculosis by culture characteristics

There are a few clinical provisos to add to this:
- In practice the specificity and sensitivity of symptoms and signs are low but the most common symptoms are cough, fever, wheezing and failure to gain weight. Other causes of childhood wheezing may be confused including asthma, viral infections and P.I.E. from wandering Ascaris larvae. Pertussis is another important cause of chronic cough.
- A definite history of close contact (sleeps in the same room, breastfeeding) with a tuberculous adult carries a 5% risk of active disease in the under 5s of ~20% in some studies.
- Good quality CXRs are required - they should be taken at the beginning of expiration (i.e. as a child starts to cry) and at least six intercostal spaces should be visible. High (~100) KV films with a short exposure time (1/100) are useful to demonstrate mediastinal lymph nodes and decrease blurring from movement. The common findings are segmental air-trapping or atelectasis, alveolar/interstitial infiltrates and pleural effusion.
- 2nd immunodeficiencies esp. HIV, malnutrition and concomitant infections may cause a false negative skin test and prior BCG vaccination a false positive, though at least 80% who received it as infants will be non-reactors by age 5 (look for the BCG scar on the right arm - almost all children in our health ward have it. The Japanese immunising tool leaves a characteristic square pattern). The Tine test is less reliable than a formal Mantoux but can be semiquantitatively graded according to the following method. It is not designed to draw blood!

#### GRADING OF THE TINE TEST

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No palpable reaction/papules &lt;2mm</td>
<td>Negative</td>
</tr>
<tr>
<td>1</td>
<td>One or more papules &gt;2mm</td>
<td>Doubtful</td>
</tr>
<tr>
<td>II</td>
<td>Coalescence of two or more papules but no ring</td>
<td>Positive</td>
</tr>
<tr>
<td>II+</td>
<td>Ring of induration</td>
<td>Positive</td>
</tr>
<tr>
<td>III</td>
<td>Smooth dome shaped swelling</td>
<td>Positive</td>
</tr>
<tr>
<td>IV</td>
<td>Ulcerated/Vesiculated</td>
<td>Treatment mandatory</td>
</tr>
</tbody>
</table>

**Isoniazid prophylaxis** has a very limited role in our district. It is demonstrably effective but a ‘mopping up’ strategy for low-prevalence countries. Treating one spumt positive adult probably prevents 20 new infections but it is necessary to give ~40 children prophylaxis to prevent one case. It is presently our policy to give INH prophylaxis only to breast-feeding infants of AFB+ve mothers where the benefits are clear and the risk of severe disease high.

**BCG** is administered to all children in RSA at birth and is most useful in preventing severe forms of T.B. especially T.B. meningitis.
Children are treated essentially the same way as adults and it is our policy to give six months treatment to all non-severe cases. Spinal T.B. requires twelve and meningal T.B. eighteen months of treatment. However, Ethambutol is never prescribed to children due to the risk of optic neuritis so Ethionamide is substituted instead (see T.B. treatment chart).

Further Reading
4) Colditz GA et al. 1994 'Efficacy of BCG vaccine in the prevention of tuberculosis: meta-analysis of the published literature'. JAMA. 271 (9) 688

**PAEDIATRIC HIV INFECTION**

The paediatric ward was one of the first in the hospital to show the impact of the HIV epidemic. In 1997 a survey found 26% of all admissions to be HIV-infected. These children were more likely to have malnutrition and/or to have been previously admitted and less likely to respond to antibiotic therapy for intercurrent infections. In-patient mortality was significantly increased (21 vs. 7%). Follow-up studies elsewhere in Sub-Saharan Africa suggest a mortality of 60% at 5 years.

WHO/CDC CLINICAL CASE DEFINITION (1986)

**MAJOR SIGNS**
- Weight loss/FTT
- Chronic Fever >1 month
- Chronic Diarrhoea >1 month

**MINOR SIGNS**
- Generalised lymphadenopathy
- Oral candidiasis
- Repeated common infections
- Persistent cough >1 month
- Generalised dermatitis
- Confirmed maternal infection

The clinical case definition was developed for epidemiological purposes where HIV testing is not widely available. The presence of at least two major and two minor signs is suggestive of AIDS but sensitivity and specificity are poor if applied to individual patients. Other useful clinical indicators are oral candidiasis refractory to therapy, shingles and chronic parotid swelling.

- HIV serology in children is complicated by the presence of passively transferred maternal antibodies for up to 18 months. Therefore supplementary methods are used in addition - p24 antigen up to 6 months (only ~20% sensitive) and IgG3 thereafter. However, these tests are costly, relatively insensitive and have little influence on case management. Clinical follow-up and repeat HIV ELISA at 18 months is therefore often the simplest policy.

- In-patient problems are similar to those in uninfected children (malnutrition, pneumonia, tuberculosis and gastroenteritis/chronic diarrhoea) but with exaggerated/refractory manifestations and increased mortality. More specific manifestations include encephalopathy (microcephaly, developmental regression and UMN signs), shingles and Pneumocystis pneumonia. Prompt and appropriate treatment of these problems in the community is critical.

- Attention to nutrition including vitamin A status is crucial. Breast-feeding should be encouraged for almost all children for at least six months unless the mother has access to secure supplies of both safe water and formula and can demonstrate competence with formula preparation.

- Immunisations can be given as usual unless very sick from birth, when BCG should be withheld. A second dose of measles vaccine is recommended due to sub-optimal responses in HIV-positive children.

- Thrice weekly co-trimoxazole should be given after the first month as prophylaxis against both bacterial and pneumocystis infection. Consider isoniazid prophylaxis if smear positive T.B. contact.

Further Reading
4) Fanni W et al. 1998 'Vitamin A supplements and mortality among HIV positive and negative children in Tanzania'. Abstract 42531 12th World AIDS Conference
5) Schaal R et al. 1995 'Mortality in a cohort of children born to HIV infected women from Durban South Africa'. S.A.M.I. 99:645
IMMUNISATIONS

Currently only 76% of children in our health ward receive all doses of vaccines recommended by the W.H.O. Expanded Programme on Immunisation by the age of one year. However, in general, we see few cases of the traditional 'vaccine-preventable' diseases in our district.

~77% of mothers receive tetanus toxoid during pregnancy but we still see occasional cases of neonatal tetanus from unvaccinated mothers/home births. South Africa is currently in the eradication phase of poliomyelitis control and surveillance of all acute flaccid paralyses is ongoing. Very few cases of pertussis have been reported from KwaZulu-Natal in the past few years.

Hepatitis B immunisation was added to the E.P.I. in 1995 - there is intense transmission of the virus between the under-5s in Southern Africa which results in a high carrier rate and carcinogenesis in later life.

"If a child is well enough to go home, that child is well enough to go home immunised." There are virtually no contra-indications to immunising a child at a clinic/OPD visit and such an opportunity should never be missed. The only absolute contra-indication is a history of previous severe reaction to a vaccine. Febrile illness and diarrhoea are NOT contra-indications! The administration of live attenuated vaccines (especially Measles and B.C.G.) to H.I.V. +ve children carries a small risk of disseminated infection, but, is not thought to be dangerous if the child does not have clinical AIDS

IMMUNISATION SCHEDULE

<table>
<thead>
<tr>
<th>Age</th>
<th>Vaccine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>TOPV, BCG*</td>
</tr>
<tr>
<td>6 weeks</td>
<td>TOPV, DTP, Hep B Hlb</td>
</tr>
<tr>
<td>10 weeks</td>
<td>TOPV, DTP, Hep B Hlb</td>
</tr>
<tr>
<td>14 weeks</td>
<td>TOPV*, DTP, Hep B Hlb</td>
</tr>
<tr>
<td>9 months</td>
<td>Measles</td>
</tr>
<tr>
<td>18 months</td>
<td>TOPV, DTP, Measles</td>
</tr>
<tr>
<td>5 years</td>
<td>TOPV, DT</td>
</tr>
</tbody>
</table>

* Repeat once if no scar visible at next visit.

* Give an extra dose at 18 weeks if any of the primary series given during an episode of diarrhoea.

CATCH-UP IN UNIMMUNISED CHILDREN

- 0-5 weeks - TOPV and BCG, followed at 6 weeks by remaining schedule.
- 6 weeks - 9 months - BCG, TOPV, DPT, Hep B at the same time, following by all outstanding doses at the normal time intervals.
- 9 months - 2 years - TOPV, Measles, DPT and Hep B at the same time. All outstanding doses are then given at normal time intervals.
- 2-10 years - Give TOPV, Measles, DT at the same time. All outstanding doses are then given at normal time intervals.

INDIVIDUAL VACCINES

- **BCG (Bacille Calmette-Guérin)** - Live bacterial vaccine 0.03 ml by multiple percutaneous puncture (Japanese immunising tool) of the right upper arm. Do not use antiseptic or alcohol swabs to prepare the site - you will kill the bacilli! If no scar is visible at 3 months of age, repeat.

- **TOPV (Trivalent Oral Polio Vaccine)** - Live virus vaccine 3 drops on the tongue. Breast-feeding only has an effect in the neonatal period when colostrum may interfere with immunisation. Therefore, feeds should be withheld for 2 hours before and after this first dose only. If the child spits it out, repeat immediately. If any of the doses are given during a diarrhoeal episode give an extra dose at 18 weeks. Give the vaccine even if there is a history of poliomyelitis since the child will only be immune to the specific infecting strain and not the other two.

- **DPT (Diphtheria, Pertussis and Tetanus)** - Combined toxoid and killed bacterial vaccine. 0.5 ml intramuscularly. Febrile reactions and pain/swelling at the injection site may occur.

- **Measles** - Schwarz strain live virus vaccine. 0.5 ml intramuscularly. A mild morbilliform rash and fever may occur up to a week after immunisation. Vaccination should be avoided in active T.B. but must be given to children with malnutrition.

- **Hepatitis B** - Plasma derived HBsAg vaccine (Korean) 0.5 ml intramuscularly. Fever and pain at the injection site may occur up to 24 hours.

- **Hib** (Haemophilus Influenzae).

The preferred sites for Intramuscular injections are the lateral thigh in children <1 year and the deltoid in children >1 year.
THE COLD CHAIN
This is essential to a successful immunisation strategy.
- No vaccine should ever be exposed to direct sunlight or heat.
- Keep the live virus vaccines (Measles and Polio) in the freezer compartment (which should be c0C).
- Keep the other vaccines (DPT and BCG) in the refrigerator compartment and do not allow them to freeze. If you suspect that they have frozen, do a shake test - take the vaccines out of the fridge, shake once and leave in a cool place away from sunlight. If after 15-30 minutes an obvious sediment has formed in a vial discard it.
- Make sure the ambient temperature in the refrigerator compartment is 4 - 8°C. Do not store vaccines in the doors as the temperature here is usually >8°C. Food and drink should not be stored in the vaccine fridge and ideally the door should not be opened more than 2-3 times each day.

Further reading
2) Schoub BD et al. 1996 'The winter 1996 mass immunisation campaign - is it the best strategy for South Africa at this time?' S.A.M.J. 86:1126
3) Ministry of Health 1995 'Immunisation that works! The vaccinators manual of the expanded programme on immunisation in South Africa'

TEN STEPS TO SUCCESSFUL BREASTFEEDING
Every facility providing maternity services and care for newborn infants should:
1. Have a written breastfeeding policy that is routinely communicated to all health-care staff.
2. Train all health care staff in the skills necessary to implement this policy.
3. Inform all pregnant women about the benefits and management of breastfeeding.
4. Help mothers initiate breastfeeding immediately, at the latest within half an hour of birth.
5. Show mothers how to breastfeed, and how to maintain lactation even if they should be separated from their infants.
6. Give newborn infants no food or drink other than breast milk, unless medically indicated.
7. Practice rooming-in: allow (encourage) mothers and babies to remain together 24 hours a day from birth.
8. Encourage natural breastfeeding, i.e. frequently and on demand.
9. Give no artificial teats or pacifiers (also called dummies or soothers) to breast-feeding infants. Do not encourage the use of nipple shields either.
10. Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from the hospital or clinic.

Hlabisa Hospital received Baby Friendly Hospital Initiative (BFHI) accreditation in March 2000, after being assessed in October 1999. This means that the hospital practices are in compliance with the Ten Steps as indicated below in the UNICEF/WHO joint statement of 1989. It also indicates that the hospital observes the Code for the Marketing of Breastmilk Substitutes. Specifically, no sales representatives for breastmilk substitutes or bottle-feeding are permitted in this hospital; no posters or other promotional material are allowed, and breastmilk substitutes are given only on a doctor’s prescription for medical reasons.
NURSERY
ASSESSMENT:
Check: colour, heart rate and respiratory effort

<table>
<thead>
<tr>
<th>COLOUR</th>
<th>HEART RATE</th>
<th>RESPIRATORY</th>
<th>ACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>PINK</td>
<td>&gt;100/min</td>
<td>good</td>
<td>No suction!</td>
</tr>
<tr>
<td></td>
<td>&gt;80/min</td>
<td>gasping</td>
<td>gentle suction O₂ by mask</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>keep warm</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>no response</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>monitor</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>hand ventilate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>no response</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>chest compressions</td>
</tr>
<tr>
<td>BLUE</td>
<td>&lt;80/min</td>
<td>apnoeic</td>
<td>gentle suction O₂ by mask</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>intubate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>hand ventilate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>chest compressions</td>
</tr>
<tr>
<td>WHITE</td>
<td>0/min</td>
<td>apnoeic</td>
<td>intubate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>hand ventilate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>chest compressions</td>
</tr>
</tbody>
</table>

Make sure the resuscitator has been turned on/warmed up before the baby is delivered. Note the time. Always assess the response after each step before proceeding to the next!

TEMPERATURE
- keep baby warm and DRY. A towel is much better than a linen saver!

SUCTION
- healthy babies do NOT need it
- mouth first, then nares, gently (neonates are obligate nasal breathers)
- DO NOT suction the deep pharynx - this can cause bradycardia/laryngospasm
- once or twice only - do not keep repeating it.

OXYGEN
- 100% @ 4L/MIN is fine for short periods. Water column at 20 cm H₂O helps to prevent barotrauma from over-enthusiastic ventilation.

MASK/AIRWAY
- Use the Samson resuscitator mask
- A Guedel airway is not usually necessary
- Do not hyperextend the neck

TRACHEAL INTUBATION
- pre-oxygenate
- ETT sizes <30 wks/1kg 2.5 30wks/1kg 3.0

- Introducer - make sure the wire is completely inside tube so it won’t perforate the airway!
- Intubate via the mouth, as this is most rapid
- pass only 7 - 8 cm tube down and secure
- first inflation - sustained for 2 - 3 seconds
- continue at 30 - 40 breaths/min

CARDIAC COMPRESSION
- HR 120/min i.e. 3 compressions : 2 ventilation
- cup both hands under baby’s back, one either side, thumbs on sternum and compress thorax

DRUGS
If no increase in heart rate/pale or blue with O₂/CPR Peripheral i.v. access usually possible, if not insert an umbilical vein catheter. Give the initial boluses via the ET tube if venous access delayed. Remember to flush the line between boluses!

1. ADRENALINE 0.1ml/kg of 1:10 000 i.v. (ie 1ml of 1:1000 + 9ml sterile water/saline) or 0.1mg/kg of 1:1000 via ET tube. Repeat every 3 mins, and increase the dose to 0.1ml/kg of 1:1000 i.v. if resuscitation is prolonged
2. FLUIDS - if fetal haemorrhage and shock give haemaccel/O -ve blood 10-20ml/kg over 10mins. Once perfusion restored Neotyle 5 ml/kg/hr maintenance
3. CHECK HGT - give 3-5 ml/kg/hr Neotyle i.v. if low (Neotyle contains 10% dextrose, don’t give bolus injections of 50% dextrose as they are very hypertonic)
4. NALOXONE - 200 μg (1 amp. Narcan Neonatal) i.v
ONLY if mother had opiate within 6 hrs of delivery (may need 2nd dose according to response)
5. ATROPINE: 0.3mg if < 3.5 kg, 0.6mg if > 3.5 kg. ONLY if persistent bradycardia in the presence of an otherwise satisfactory response.
6. SODIUM BICARBONATE - 4.2% (5mls 8.5% Sodium bicarbonate + 5mls sterile water/saline), 2mls/kg i.v. slowly (2 ml/min). ONLY if prolonged resuscitation and/or acidosis.

WHEN TO STOP
- No sign of life after 10 mins
- If HR responds but no spontaneous respiration after 30 mins.

IF POOR INITIAL RESPONSE
- Technical fail - 702 connected
- ETT in trachea/too far down one bronchus/blocked
- If in doubt - remove and replace
- Pneumothorax
- Diaphragmatic hernia
- Congenital heart disease

FOLLOWING RESUSCITATION
- GENERAL:
  - Incubator
  - O2 (if required)
  - Vitamin K 1mg
  - Check HGT 3-4 hourly
  - Continue 10% dextrose infusion if necessary
  - Feed - via NGT initially, i.v.i if very ill
  - If prem - nurse prone
- RESPS: note colour, rate, regularly, deep/shallow (risk pulmonary hypertension)
- CARDIOVASC: HR, peripheral perfusion (?failure)
- ABDOMEN: vomiting, distension (NEC)
- RENAL: oligoanuria (renal failure and sign of hypoxic organ damage)
- CNS: convulsions, tone, fontanelle, feeding ability (hypoxic ischaemic encephalopathy, IVH)

We still record the Apgar Score at 1 and 5 mins, more as a guide to prognosis than an aid to resuscitation.

THE APGAR SCORE

<table>
<thead>
<tr>
<th>SIGN</th>
<th>SCORE 1</th>
<th>SCORE 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Below 100/mins</td>
<td>Above 100/mins</td>
</tr>
<tr>
<td>Respiratory effort</td>
<td>Weak</td>
<td>Good, cries</td>
</tr>
<tr>
<td>Muscle tone</td>
<td>Some flexion</td>
<td>Well flexed</td>
</tr>
<tr>
<td>Reflex irritability</td>
<td>Grimace</td>
<td>Cough / sneeze</td>
</tr>
<tr>
<td>Colour</td>
<td>Pink body, blue limbs</td>
<td>Completely pink</td>
</tr>
</tbody>
</table>

Prem and L.B.W babies account for 80% of admissions. 95% of neonatal deaths in our hospital occur in babies < 2.5 kg.

Principles of care in the nursery
The following help minimise mortality:
- Minimal handling: The baby must be handled only when absolutely necessary. The more sick the baby is, the more rest he needs.
- Only the mother must handle her baby. Other mothers, nurses and doctors must keep hands off as far as possible. This reduces cross-infection.
- Cleanliness: Keep hands and equipment clean - hands should be washed with Hibitane between examinations.

Supportive care
- WARMTH: all babies must be kept warm. If cold wrap him/her up and either "kangaroo" next to mother's
skin, or put in incubator. Quickly! Or respiratory distress and hypoglycaemia will ensue.

- **OXYGEN**: If needed, is needed via a headbox and must go through an humidifier.
- **BLOOD SUGAR**: Small and/or sick babies are at risk of HYPOGLYCAEMIA and need glucose either as milk or i.v. neotyle.
- **SEPSIS**: Any baby that is ‘not well’ or has been born pre-term may be septic. Look for specific causes and cover for sepsis if at risk.

**Routine care**
- At birth, Dry, note APGAR, examine and give to mum to breast feed.
- Vitamin K
- povidone iodine/chloramphenicol eye ointment
- mercurichrome to cord
- chart weight and head circumference
- bath once stable/warm
- note scalp colour, meconium (green) day 1 ->yellow day 5 urine colour; 6 wet nappies/day = enough breast milk
- Nurse sick babies prone.

Further Reading
1) Wilkinson G et al. 1996 'Impact of prematurity on admissions to the neonatal nursery of a rural Southern African rural hospital' J Trop Paeds 45:76
2) Simkins C 1999 'Kangaroo mother care' J Trop Paeds 45:192

**FEEDING SICK/ HIGH RISK BABIES**

**ROUTINE FEEDS**
Babies should be sucking at 35wks. before this use expressed breast milk (EBM) via ng tube.

Feed frequency

| < 1.5 kg | 1-2 hrly |
| 1.5 - 1.8 kg | 3 hrly |
| 1.8 - 2.0 kg | 4 hrly |

Pre-term babies lose more water, electrolytes, energy & risk aspiration during feeds.

The first feed must be within two hours of birth to maintain glucose levels.

Breast milk is best for all babies - pre-terms too! If they must have artificial then NAN if >1.5kg, PRENAN<1.5kg

**HIGH RISK/SICK BABIES**
If ill / <1.5 kg - ivf for the first 24 - 48 hours until
- meconium passed
- abdomen soft and not distended

Then give EBM (or PRENAN) via the NGT as below:

<table>
<thead>
<tr>
<th>DAY</th>
<th>ML/KG/DAY</th>
<th>MLK</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>75</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>25</td>
</tr>
<tr>
<td>3</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>75</td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>100</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>150</td>
</tr>
</tbody>
</table>

If abdo distends/vomiting/2mL/kg aspirated -> STOP EBM! - We have had too many cases of necrotising enterocolitis from over-feeding.

**INTRAVENOUS FLUIDS**
If babies are too ill to tolerate milk for >72 hrs they need TPN which we can’t give. Referral attempts usually fail and they can survive for up to a week on ivf alone. Neonates have much higher fluid requirements than adults due to large insensible and obligatory renal losses. Requirements are higher in pre-term infants and increase daily until day 4.

<table>
<thead>
<tr>
<th>DAY</th>
<th>1 - 60</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>90</td>
</tr>
<tr>
<td>3</td>
<td>120</td>
</tr>
<tr>
<td>4</td>
<td>150 mL/kg/day</td>
</tr>
</tbody>
</table>

Add 25-30 mL/day if under phototherapy or diarrhoea.

Weigh daily - loss of >10% birth weight -> increase needed

- Use Neotyle for maintenance (K+ 15 mmol/L).
- 1/2 Darrow's + 5% Dextrose is preferred for replacement in diarrhoea (K+ 17.5 mmol/L).
- 0.45 - 0.9% Saline is preferred for replacement of sweat or gastric losses (No K+).

**ENERGY REQUIREMENTS**
All infants should have their weight charted daily. Sufficient calories are provided by 150 mL/kg/day of breast milk (~440 kJ/kg/day). The best measure of sufficiency is weight gain. Healthy babies regain birth weight by day 10, and should gain ~30g/day. If not increase feeds ≤200 mL/kg/day; and give 0.5mL sunflower oil alternate feeds, or 1 extra scoop of PRENAN or NANN to 100mL formula.

**SODIUM REQUIREMENTS**
Normal requirements are 1-3 mmol/day. Very L.B.W babies (<1.5 kg) need 4-5mmol/day - mix 2.5mLs of 8.5% sod bic + 2.5mLs water and add to 100mLs of breast milk (or 1mL of 4% - if available - to 20 mLs).
HYPOTHERMIA

- Rectal Temperature <35.5°C.
- Pre-term, U.W.F.A. and wasted infants at high risk.
- Thermoregulation adequate out of the incubator in most >1.75 kg.
- The temperature response to infection in neonates is unpredictable, and may result in hypothermia or hyperthermia instability, as well as fever.
- Signs - cold, oedematous, cyanosed infant? with respiratory distress and bleeding.
- Prevent hypothermia by ensuring that babies are dry and well wrapped up, with a hat if necessary.
- Treat in incubator or under radiant warmer at 37°C.
- Give O₂, add 10 ml 4% Sodium Bicarbonate to the drop and check HGT.
- Consider i.v. antibiotics if there is a suspicion of infection.

ROUTINE INCUBATOR TEMPERATURES (°C)

<table>
<thead>
<tr>
<th>Birth weight (kg)</th>
<th>Days</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 5 10 15 20 25 30</td>
</tr>
<tr>
<td>1</td>
<td>35.5 35 35 34.5 34 33.5 33</td>
</tr>
<tr>
<td>1.5</td>
<td>35 34 33.5 33.5 33 32.5 32.5</td>
</tr>
<tr>
<td>2</td>
<td>34 33 32.5 32.5 32 32 32</td>
</tr>
<tr>
<td>2.5</td>
<td>33.5 32.5 32 31 31 31 31</td>
</tr>
<tr>
<td>3</td>
<td>33 32 31 30 30 30 30</td>
</tr>
</tbody>
</table>

RESPIRATORY DISTRESS

- Tachypnoea (RR>60), intercostal recession, nasal flaring, central cyanosis, grunting, apnoeas

Though there is a high incidence of prematurity in black South African neonates they demonstrate a relatively low incidence of the respiratory distress syndrome and Hyaline Membrane disease is virtually unknown >33w gestation. Infection probably accounts for 40% of clinically identified cases of respiratory distress in infants of late gestation. Attention to the first four especially helps to avert pulmonary vasoconstriction and consequent R-L shunting via the Foramen ovale and ductus arteriosus.

Supportive management includes:
- Warmth, minimal handling, energy (i.v. Neolyte)
- Observations: RR, recession, grunting, cyanosis, HR, temp
- O₂, until tongue pink. 1-6 l/min via mask/tube & reservoir =100%
- 2-3 l/min via headbox/incubator =30-40%
- Ensure humidifier is always attached
- Reduce to minimum necessary a.s.a.p.
- CXR
- <34 wks: aminophylline

HYALINE MEMBRANE DISEASE

Pre-term/diabetic mum, negative amnio prior to delivery and on gastric aspirate. Early and increasing respiratory distress + inc O₂ requirements in 1st 48 hrs mortality approaches 100% in peripheral hospitals.
- O/E: infant inactive, peripheral oedema
- CXR: small lungs, reticulo-granular infiltrates, air bronchograms.

Management:
- Fluid restrict
  - Day 1 & 2: 60ml/kg
  - Day 3 & 4: 90
  - Day 5 & 6: 120
  - Day 7 & 8: 150
- Antibiotics: pen & gent iv/im (especially if intubated)
- Surfactant available at referral hospitals
- Look for and treat other associated complications of asphyxia prematurity.

NORMAL RANGES FOR NEONATES

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb</td>
<td>14-21.5</td>
</tr>
<tr>
<td>Hct</td>
<td>33.6-68.4%</td>
</tr>
<tr>
<td>MCV</td>
<td>100-140</td>
</tr>
<tr>
<td>PLT</td>
<td>150-400</td>
</tr>
<tr>
<td>WCC</td>
<td>9-35</td>
</tr>
<tr>
<td>Na</td>
<td>132-142</td>
</tr>
<tr>
<td>K</td>
<td>5-7.5</td>
</tr>
<tr>
<td>Cl</td>
<td>96-106</td>
</tr>
<tr>
<td>Ur</td>
<td>1.6-4.9</td>
</tr>
<tr>
<td>Cr</td>
<td>20-60</td>
</tr>
<tr>
<td>Ca</td>
<td>1.7-2.5</td>
</tr>
<tr>
<td>Mg</td>
<td>0.7-1.0</td>
</tr>
<tr>
<td>CSF</td>
<td></td>
</tr>
<tr>
<td>Leucocytes</td>
<td>10x10^9/l</td>
</tr>
<tr>
<td>Protein</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td></td>
</tr>
</tbody>
</table>

LEUCOS <5:75% lymph
TRANSIENT TACHYPNOEA / WET LUNG SYNDROME

Pulmonary maladaptation associated with CS, prenatal/intrapartum asphyxia, maternal sedation, polyhydrarnia. May be term or pre-term. Amino/gastric aspirate +ve. Excellent prognosis. By definition, significant improvement within 24 hours.

**Diagnosis:** early respiratory distress with a hyperinflated chest.

**CXR:** (after 6 hrs) - hyperinflated, increased perihilar vascular markings, clear peripheral fields.

**Management:** _O₂_ requirements up to 50% initially but rapidly falling over 1st 72 hours.

MECONIUM ASPIRATION

Term/post, rarely pre-term. Meconium - stained liquor and meconium is suctioned from ETT/beyond cords. Chemical pneumonitis and airway plugging result. Mortality ~12% according to associated asphyxia

**Diagnosis:** immediate respiratory distress in the presence of the above

**CXR:** paradoxical CXR appearances with both air-trapping and segmental collapse

**Management:**
- Stomach washout with 50% NaClime + 50% Sod bic. with ET in-situ (to prevent further aspiration).
- Pen, Gent i.v.i.m.
- Watch for associated meconium gastritis and pneumothorax/mediastinum.

PNEUMONIA

Usually arises a few days post-delivery but may be early following maternal chorioamnionitis (offensive liquor and fever). The usual organisms are Group B Streptococci and Gram negative bacilli (esp. E.coli).

**Diagnosis:** Pyrexia/hyperthermia, Gastric aspirate: pus cells, bacteria (only of value before first feed)

**CXR:** collapse/consolidation

**Management:** Amoxicillin 50 mg/kg 6-8 hrly + Gentamicin 5 mg/kg q.d. i.v. or Procaine penicillin 50,000 i.u/kg + Gentamicin 5 mg/kg both once daily i.m. (if i.v. access a problem)

PNEUMOTHORAX

May be associated with H.M.D. and Meconium Aspiration

**Diagnosis:** sudden collapse, increased _O₂_ needs, decreased _air entry/breath sounds, palpable liver (if right) decreased heart sounds transillumination _O₂_ affected side brighter/CXR

**Management:**
- mild RD/no cyanosis — >headbox _O₂_ + obs.
- chest drain if more severe (cannula in axilla, attach to tubing from giving set, other end of which is placed through hole made in lid of specimen pot. Have water in pot to cover end of tube and cut another length of tube to feed out of pot through another hole in lid.)

HEART FAILURE

Due to: PDA, congenital malformations (VSD the most common, ASD and Coarctation also), excess i.v.i., hypoxia, anemia.

**Diagnosis:**
- Tachycardia > 160/min
- Tachypnoea > 60/min
- Hepatomegaly > 2cm below costal margin
- Murmur
  - Continuous, under L clavicle => PDA (venous hum sounds like a PDA but disappears on head movement or compression of the jugular veins)
  - Ejection systolic, fixed splitting of _S₂_ — ASD
  - _Pan-systolic_ L. sternal edge => VSD
  - Short systolic, heard at the back => Coarctation
- Bounding/absent pulses
- _O₂_- resistant cyanosis due to large R—>L shunt (probable tetralogy)

**Management:**
- Fluid restrict to 60 ml/kg/day.
- Frusemide 1mg/kg i.v. slat.
- Digoxin 0.05 mg/kg/day divided into 4 doses over 24 hours then 1/4 of this dose daily.
- Transluce with packed cells if PCV<30/Hb<10. correct hypoglycaemia and acidosis.
- If _O₂_-resistant cyanosis, stop _O₂_ and give _P_{O₂}30-60_ μg/kg/hourly p.o (dissolve 500 μg tablet in 10 ml sterile water, 1 ml = 50μg) to maintain the DA prior to transfer for surgery.

DIAPHRAGMATIC HERNIA

**Diagnosis:** scaphoid abdomen/CXR

**Management:**
- NGT + aspirate regularly
- NPO
- iv pen + gent
- Transfer

APNEIC ATTACKS

= stopping breathing long enough to cause cyanosis/bradycardia/palor
Do not confuse with normal periodic breathing which occurs in preterm + term infants - frequent short pauses (<20s) in resps which do not cause cyanosis etc.

Due to:
- immaturity (<34wks, after 48hrs, after feed)
- infection (pneumonia, septicaemia, meningitis, NEC)
- CNS - asphyxia, I&L, drugs (pethidine), PVH
- GI - reflux, perforation, large feeds, vomiting
- CVS - hypovolaemia, anaemia+/+ PDA, CCF, hypovolaemia (APH)
  - metabolic - hypoxia/ hypothermia
  - hypo-glycaemia / calcaemia / ataxia
  - hypernatraemia
- increased ambient temperature

Treatment:
- Oxygen - headbox
- Transfusion - 10ml/kg if Hb<10
- If immature (<36wks) - aminophylline : loading dose 5mg/kg than 2mg/kg I&Ls (1mg/kg I&Ls if < 1.5 kgs
  - septic screen + ivABx
  - Treat other identified causes.

AIRWAY OBSTRUCTION

Due to cheanal atresia -> stridor + cyanosis when asleep but pink when awake and mouth open.

Ox: polyhydramnios, failure to pass n.g. tube per rectum post-delivery (blocked nose - saline nose drops).

Further Reading:

NEONATAL SEPSIS

Any 'ill' baby should be considered to have an infection as the signs may be very non-specific. The risk of infection is at least 3x in premature/LBW infants, especially those who required vacuum extraction/resuscitation. Maternal chorioamnionitis is another significant risk factor. The common causes of early neonatal sepsis (1st week) are Group B Streptococci, E coli, other gram negative bacilli esp. Klebsiella spp., and Staphylococci. In the first week meningitis will be present in 30% of cases. Syphilis is the major chronic intrauterine infection in our practice though we have also seen cases of congenital rubella and the prevalence of +ve Toxoplasma serology in our antenatal population is probably ~50%. Both N gonorrhoea and C trachomatis cause neonatal conjunctivitis and the latter may also result in a delayed pneumonia at 4+ weeks.

Diagnosis

GENERAL SYMPTOMS / SIGNS:
- lethargy
- no increase / decrease in weight
- pallor
- poor feeding
- vomiting
- hypothermia
- jaundice
- purpura (TCP/DIC)
- apnoea / shock
- temperature instability
- hypo-glycaemia
- hypotonia oedema acalana

SIGNS OF LOCAL INFECTION:
- smelly cord
- meningitis (fits)
- pneumonia
- abdominal distension and blood pr (NEC)

Abnormal Absolute Neutrophil count and ratio of band forms >12-16% are predictive of infection.

<table>
<thead>
<tr>
<th>Age</th>
<th>Total Neutrophil count</th>
<th>% Band forms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>1.8-6.0</td>
<td>&lt;16</td>
</tr>
<tr>
<td>24 hours</td>
<td>7.2-12.6</td>
<td>&lt;16</td>
</tr>
<tr>
<td>48 hours</td>
<td>3.6-8.1</td>
<td>&lt;13</td>
</tr>
<tr>
<td>5 days</td>
<td>1.8-5.4</td>
<td>&lt;12</td>
</tr>
</tbody>
</table>

- Gram stain of gastric aspirate prior to first feed may also be useful. >5 WBCs per high power field is suggestive of aspiration of infected liquor. Gram +ve cocci in pairs are usually Group B Streptococci.

- Lumbar Puncture should always be considered on sick neonates with no obvious focus of infection. The normal values are for the first week are:
  - Protein 0.25 - 1.0 g/l
  - Glucose 2.2-3.3 mmol/l
  - Polymorphs 0-10/mm³
  - Lymphocytes 0-10/mm³

- Urine microscopy.

Management

Supportive: ie O₂, iv Neotyle/ NG feeds) as required. Monitor HGTs and temperature closely.

Antibiotics:
- Ampicillin: 62.5 mg q6s po 1 week for mild/ non-specific infections
- Benzylpenicillin: 100 000 U/kg/day divided into two doses im/iv bd
Gentamycin: <1kg: 2.5mg/kg/day iv/im once daily
<2.5kg: 3.5mg/kg/day
>2.5kg: 4.5mg/kg/day

Erythromycin: 62.5mg qds po/pers n.g.i.
in respiratory infections not responding
to the above

Ceftriaxone: 60mg/kg/day iv/im

SPECIFIC INFECTIONS

CHORIOAMNIONITIS

Infection of the amniotic fluid probably occurs in
<4% of pregnancies and may increase the perinatal
mortality by as much as 4x. The most significant risk
factor is prolonged pre-labour rupture of membranes.
At least 30% will already have amniotic fluid cultures at
the time of rupture. The infection is invariably
polymicrobial but addition of anaerobic cover seems to
confers no benefits.

Give Ampicillin and Gentamicin for 5 days to all infants
<37w delivered of mothers with PROM and/or clinical
evidence of Chorioamnionitis. Term infants are at lower
risk - manage expectantly if well and satisfactory septic
criteria.

SEPTICAEMIA/MENINGITIS

Signs of meningitis are subtle in neonates (irritability,
seizures, staring, high-pitched cry) so have a low-
threshold for L.P in a possibly septicemic infant. There
is a high incidence of verticillitis and complications in
neonatal meningitis - overall mortality 50%, 50% of
survivors have brain damage. Use the higher doses of
Penicillin and Ceftriaxone in meningitis to ensure CSF
penetration.

Give Benzylpenicillin 50,000-100,000 i.u./kg/day in
2 divided doses + Gentamicin 7.5 mg/kg/day o.d. i.v.
(intralcal administration is of no benefit).

Consider changing to Ceftriaxone 50-100 mg/kg/
day o.d. if Gram stain shows Gram -ve bacilli and/or there
is a poor initial response to Pen + Gent.

Give Phenobarbitone 20 mg/kg iv/im ss prophylaxis
vs. seizures - then 5mg/kg po od.

NECROTISING ENTEROCOLITIS

Necrosis usually at the terminal ileum and proximal
colon, associated with prenatal hypoxia and infection.
May be related to premature introduction of feeds.

Diagnosis:
- septicaemia/shock
- tender, distended abdomen
- vomiting (+/- bile)
- blood in stool (-75% occult)
- AXR - excludes volvulus/obstruction, intramural gas
  is diagnostic, check for free peritoneal gas indicating
  perforation

Complications: perforation, haemorrhage, sepsicaemia,
malabsorption, strictures.

Management:
- N.P.O., N.G.T., I.V.I., resuscitate shock with F.D.P.
  according to response
- Daily abdominal girth measurements
- Benzylpenicillin + Gentamicin
- Reinroduce feeds slowly (when bowel sounds present/
  abdo normal)
- If perforated or in need of TPN (ie NPO for >3days)
  refer.

NEONATAL CONJUNCTIVITIS

The common causes are Chlamydia, N. gonorrhoeae
and Staphylococcus (in that order). Gonococcal
conjunctivitis quickly progresses to corneal perforation
and pannus on the neonates and clinical findings do
not discriminate well between organisms, so ALWAYS
GRAM STAIN the discharge!

Management:
- In all cases bathe eye with sterile water/saline 2hrly. If
  very purulent, irrigate with bag and giving set.
- Chloramphenicol 1% ointment 4-6 hrly (active vs.
  chlamydia and staph).
  	Ceftriaxone 25-50 mg/kg stat i.m.
  	Erythromycin 62.5 mg qds po fo 7 days.
  	Teal both parents for STDs.

OMPHALITIS AND NEONATAL TETANUS

The normal umbilical cord is soft and white
immediately post-partum. It becomes brown and dry over
a few days before sloughing within 2 weeks. An infected
cord is smelly, persistently wet and has a periulcerative
flare. Portal phlebitis, peritonitis and septicaemia may
follow. The usual causes are Staphylococcus, Streptococci
and Clostridium tetani leads to neonatal tetanus. Almost
all cases of tetanus that we see in our hospital follow delivery at home where cord
care has been poor and the mother has not been given
toxoid during pregnancy. Signs include poor suck
trismus, listing, apnoeas and spasms, which occur in
full consciousness and often with continued crying.
Severe cases are identified by an incubation period of <5
days and a prepatent period (time from first onset of
symptoms to first spasm) < 48 hours with 12+ spasms/day. The mean duration of ventilation at KEH is 23 days and the overall mortality 22%.

**Management:**

**OMPHALITIS:**
- Routine cord care: Surgical spirit 6 h every 5 hours. Do not cover!
- Established infection - Surgical spirit 3 h every 5 hours + Penicillin and Gentamicin i.v.

**NEONATAL TETANUS:**
- clear airway, O₂
- no stimulation
- NGT - 30 mins after sedation
- fluids: EBM - small frequent feeds (decrease risk aspiration/regurgitation)
- suctioning & turning after each dose sedation
- Monitor O₂ saturations and pulse and record during spasms
- Diazepam 1 mg/kg iv/pr till spasms stop and then 0.25-1 mg/kg 4 h every
- Chlorpromazine 12.5 mg i.m. 4-6 h if spasms not controlled
- Penicillin 25,000 u/kg 6 h
- Human tetanus immunoglobulin 500 u i.m.
- Tetanus toxoid 0.5 ml stat i.m. (an attack of tetanus does not confer immunity and a primary series of three doses of toxoid is indicated)
- Refer all severe cases to Ngwz/KEH as ventilation is usually required

**CONGENITAL SYPHILIS**

Risk of congenital infection 50% but only 10% of babies of untreated WR +ve mothers have clinical signs of Syphilis at birth. These include:

- LBW
- Syphilitic pemphigus (hands & feet)
- Heavy greasy placenta > 1/5
- Anaemia
- of infant's birthweight
- Thrombocytopenia/parotitis
- Hepatosplenomegaly
- Nasal discharge (blood-stained)
- Lymphadenopathy
- Metaphyseal 1 bone X-Rs
- Condyloma lata (esp. mouth) Meningo-encephalitis

Such a baby, with any of these signs, should be treated for congenital syphilis.

- Spirochaetes may be visible on dark-field microscopy of nasal discharge or skin scrapes. The only definitive serological test is a +ve TFA-Abs IgM but even this may be negative at birth if the infection was acquired late in pregnancy. A +ve RPR on the baby at birth may only signify passive immunisation from the mother.

Therefore management should be as follows -

**SKIN INFECTIONS**
- Bullous impetigo
- Pus filled blisters in the umbilicus / nappy area caused by staphylococci (aspire gram stain diagnostic)

**Treatment**
- wash - Chlorhexidine bd for 5 days, uncover cord - no nappy!

Treatment cord infection - prevent spread! If unwell — i.v. Aox

- Erythema toxicum:
  - day 2-3 - red blotches / yellow papules over face & chest
  - disappear in around 1 week
  - baby well, no treatment needed
  - (Dx: eosinophils in pusules)

- Morbilliform rash -
  - raised red velvety rash especially around skin creases in nappy area caused by Candida albicans.

**Treatment:** mycostatin cream, uncover, nurse prone for 48hrs - if no help try mycostatin drops.

- Nappy rash (spares creases). Use zinc and castor oil cream as a barrier.

- Sweat rash - small clear blisters, fine rash on forehead, chest. Washing is the best treatment!

- Pustular melanosis: blisters -> burst -> pigmented skin. No treatment required.

**CANDIDIASIS**

**MILD:** Small areas, sucking well, no treatment.

**SEVERE:** big areas, red, poor feeding etc.

Rx: mycostatin 1 ml after feeds for 7 days & on mum's nipples/vagina (cream)
Further Reading:
1) Monroe et al. 1979 'The neonatal blood count in health and disease I: reference values for neutrophilic cells' J.Perinatal Med. 5:39
2) Milbery NC et al. 1991 'Anaerobic coverage for intra-amniotic infection: maternal and perinatal impact' Am J Perinatal 8:333
4) Lagier M et al. 1986 'Single dose therapy of gonococcal ophthalmitis neonatorum with ceftriaxone' N.E.J.M. 315(22):1392

**HYPOGLYCAEMIA**

- Blood glucose < 2.6 mmol/l

**HYPOGLYCAEMIA CAUSES BRAIN DAMAGE - PREVENT IT!**

At risk: pre-term, UWFA, wasted stressed, liver damage, resp. distress, hypothermic, diabetic mum, OWFA, polycytheamic.

Symptoms: none or:
- decreased brain function: lethargy, hypotonic, weak cry, apnoea, absent moro, cyanosis increased brain function: jittery, staring, making lists, abnormal eye movements, convulsions, sweating.

**TREATMENT**

HGT < 1:
- 1ml/kg 25% dextrose + start infusion of neotyle at 10ml/kg/h
- repeat HGT 15 mins
- < 2 gram

HGT = 2:
- milk feed + dextrose mixed (0.5ml/kg of 25% dextrose) + start iv neotyle - 10ml/kg/h
- repeat HGT 15 mins
- < 2 or = 2 gram

4 - 7 (normal):
- milk feeds HGT hourly for 3hrs, 3hrly once stable
- Rx as < 2 gram

HGT > 4:
- milk feed (10ml/kg)
- or glucose water (5%) iv 10ml/kg
- or milk feed + tsp sugar
- repeat HGT 15 mins
- = 2 or < 2 gram

Repeat milk feed + dextrose mix
- repeat HGT 15 mins
- < 2 or = 2 gram

Rx as < 2 gram

Sick infants should have - IV - 10% glucose (neonatalry) monitor
- HGT 4-hourly
- feed hourly to start with in at risk groups prems: 60ml/kg/24h SFD/post-mature/diabetic mum / symptomatic: 90ml/kg/24h HMD: add 1ml of 50% glucose to 90ml neotyle

**25% dextrose = 5ml dextrose + 5ml sterile water**

Last resort if can't get iv, 5ml 50% dextrose: + add to 10ml/kg milk via NGT/po

Repeated hypoglycaemia —-> metabolic cause —> refer HGT monitoring; should be as above until on 10ml/kg/day milk feeds (teach mum to do it if possible)

**HYPERGLYCAEMIA = > 9.7 mmol/l**

Due to: neonatalry over-infusion, PVH, stress reaction, often to infection.

Causes: polyuria, dehydration, PVH Rx 5% dextrose, milk feeds, if >20mmol/l despit this then infuse insulin at 0.05 - 0.1 Units/kg/hr & strictly monitor the HGT to detect hyperglycaemia hourly. Stop infusion once HGTs normal but continue to monitor HGTs hourly until stable for 3 hrs then go to 3hrly measurements. The very sick baby may be very sensitive to insulin and the blood sugar may fall within 15-20 mins of the insulin injection.
**THE SMALL NEONATE**

All babies <2.5kg are low birth weight (LBW). They may be Pre-term or Under-weight-for-(gestational)-age. There are important clinical differences between the two groups -

<table>
<thead>
<tr>
<th></th>
<th>PRE-TERM</th>
<th>UWFA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nutrition</td>
<td>adequate</td>
<td>poor</td>
</tr>
<tr>
<td>Mental</td>
<td>dull</td>
<td>alert/hungry</td>
</tr>
<tr>
<td>Suck</td>
<td>no</td>
<td>yes</td>
</tr>
<tr>
<td>Skin</td>
<td>thin</td>
<td>crinkly</td>
</tr>
<tr>
<td>Oedema</td>
<td>yes</td>
<td>no</td>
</tr>
</tbody>
</table>

and each has different problems -

<table>
<thead>
<tr>
<th></th>
<th>PRE-TERM</th>
<th>UWFA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypothermia</td>
<td>yes++</td>
<td>yes</td>
</tr>
<tr>
<td>Hypoglycaemia</td>
<td>yes</td>
<td>yes++</td>
</tr>
<tr>
<td>Asphyxia</td>
<td>yes</td>
<td>yes++</td>
</tr>
<tr>
<td>RDS</td>
<td>HMD/PDA</td>
<td>Meconium</td>
</tr>
<tr>
<td>Apnoea</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>Infection</td>
<td>yes++</td>
<td>yes</td>
</tr>
<tr>
<td>Hb</td>
<td>high</td>
<td>low</td>
</tr>
</tbody>
</table>

Source and Further Reading

**SPLEN SCORE**

Splen score provides an assessment of gestational age. It should be done on all sick babies.

*Underweight for Gestational Age (UWGA) and prem babies have different problems so it helps to decide which he/she is, and therefore what problems are more likely.*

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>SIGN</td>
<td>smooth, pink, thin, low iron, light</td>
<td>iron deficient, brown, low vision</td>
<td>jaundice, brown</td>
<td>yellowing</td>
</tr>
<tr>
<td>PLANTAR</td>
<td>anterior of sole</td>
<td>20 of sole</td>
<td>whole</td>
<td>sole</td>
</tr>
<tr>
<td>CREASES</td>
<td>anteromedial</td>
<td>medial</td>
<td>lateral</td>
<td>lateral</td>
</tr>
<tr>
<td>LABIA /</td>
<td>anterior of pubis</td>
<td>pubis</td>
<td>pubis</td>
<td>pubis</td>
</tr>
<tr>
<td>TESTES</td>
<td>anteromedial</td>
<td>medial</td>
<td>lateral</td>
<td>lateral</td>
</tr>
<tr>
<td>EAR</td>
<td>slow</td>
<td>rapid</td>
<td>recall</td>
<td>instant recall</td>
</tr>
<tr>
<td>NIPPLE</td>
<td>flat, bed &lt;5mm</td>
<td>bed =5mm</td>
<td>bed =5mm</td>
<td>bed =5mm</td>
</tr>
</tbody>
</table>

Add the scores of the 5 factors together and add the result to 30 to obtain the gestational age (if >30w)
If <30w use the Ballard system. In our nursery a SPLEN of 30w carries a mortality of 70% whereas a SPLEN of 31-36 carries a 9% mortality.
BALLARD SCORE
THE BALLARD SCORING METHOD
Each separate sign is given a score after examining that sign on the infant. These separate scores are then added together to give a total score. From the total score, the estimated gestational age can be read off the table.
Source: Perinatal Education Programme: Introduction and Overview - Newborn Care, p.5.

<table>
<thead>
<tr>
<th></th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Posture</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Square Window (wrist)</td>
<td>90°</td>
<td>60°</td>
<td>45°</td>
<td>30°</td>
<td>0°</td>
<td></td>
</tr>
<tr>
<td>Arm Recoil</td>
<td>180°</td>
<td>100°</td>
<td>90°</td>
<td>100°</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Popliteal Angle</td>
<td>180°</td>
<td>160°</td>
<td>130°</td>
<td>110°</td>
<td>90°</td>
<td></td>
</tr>
<tr>
<td>Scorf Sign</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heel to Ear</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Skin</th>
<th>gelatinous, red, translucent</th>
<th>smooth, pink, visible veins</th>
<th>superficial peeling, fur rash over veins</th>
<th>cracking of skin over veins</th>
<th>promentum deep cracking, no vessels</th>
<th>leathery cracked, wrinkled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lonugo</td>
<td>none</td>
<td>abundant</td>
<td>thinning</td>
<td>bold, areas</td>
<td>mostly, bold</td>
<td></td>
</tr>
<tr>
<td>Plantar Crosses</td>
<td>no crease</td>
<td>faint red marks</td>
<td>anterior transverse crease</td>
<td>crease, 1/3</td>
<td>crease, cover, entire sole</td>
<td></td>
</tr>
<tr>
<td>Breast</td>
<td>barely percept.</td>
<td>flat areola no bud</td>
<td>stippled areola 1-2mm bud</td>
<td>raised grepolo 3-4mm bud</td>
<td>full grepolo 5-10mm bud</td>
<td></td>
</tr>
<tr>
<td>Ear</td>
<td>pinna flat, stapes folded</td>
<td>s. curved pinna, soft</td>
<td>wall-curved pinna, soft</td>
<td>well-defined crease</td>
<td>thick cartilage, ear, stiff</td>
<td></td>
</tr>
<tr>
<td>Genitals</td>
<td>scrotum empty no rugae</td>
<td></td>
<td>tastes descending, few rugae</td>
<td>tastes down, good</td>
<td>tastes pendulous, deep rugae</td>
<td></td>
</tr>
<tr>
<td></td>
<td>male</td>
<td></td>
<td>large phimora equally prominent</td>
<td>phimora large, minora</td>
<td>phimora minora</td>
<td></td>
</tr>
<tr>
<td></td>
<td>female</td>
<td></td>
<td>prominent clitorus &amp; labia minora</td>
<td>clitorus &amp; labia minora</td>
<td>clitorus &amp; labia minora, completely covered</td>
<td></td>
</tr>
</tbody>
</table>

**BALLARD SCORE CHARTS**

**BALLARD TABLE**

Score Wks.
5  26
10 28
15 30
20 32
25 34
30 36
35 38
40 40
45 42
50 44

Source and Further Reading
FITS

Importance
- Continuing fits can damage the brain
- Fits in the newborn usually indicate serious underlying illness. This must be looked for and treated.

Recognising a fit
- Subtle signs:
  - sustained deviation of the eyes
  - drooling or lip smacking
  - apnoea
  - ‘cycling’ movement of the legs
  - sustained posturing of a limb
- Other signs:
  - generalised tonic posture (sustained stiffening of body and limbs, backwards extension of neck
  - clonic (rhythmic jerking - one limb, whole body, or moving from limb to limb)
NB some babies have jittery movements which are not. These may indicate low glucose or calcium or have no identifiable cause.

JITTERS FITS

Eye deviation or other eyesigns
Responss to stimulation
Dominant movement
Movements stop when limb gently flexed

No
Yes
No
Tremor
Rhythmic jerking
Yes
No

* both directions of movement are of equal size and duration
** movement has a fast and slow component.

Causes
- Common:
  - perinatal asphyxia (causes fits because of hypoxia, electrolyte disturbance, hypoglycaemia, low BP, intracranial bleed)
  - low glucose (<2.2 mmol/l)
  - low calcium (<1.7 mmol/l)
  - low magnesium (<4.0 mmol/l)
  - high sodium (>150 mmol/l)
  - low sodium (<120 mmol/l)
  - meningitis
  - intra-cranial haemorrhage
- Less common:
  - high bilirubin (kernicterus)
  - polycythaemia (HCT >70%)
  - structural brain abnormality
  - inborn error of metabolism

Treatment
- Clear airway
- Give O₂ - bag and mask if not breathing or still cyanosed in oxygen
- Check HGT immediately; if <or =2 treat as below
- Emptly stomach with NGT, nurse prone
- Keep warm
- Give an anti-convulsant:
  a) Phenoobarbitone 20mg/kg iv over 5mins
  b) If still fitting after 10 mins give further phenobarb 10mg/kg iv
  c) Diazepam 0.2mg/kg iv
  d) Phenytin 15mg/kg iv over 20 mins (diazepam often causes apnoea)
- If venous access is impossible, give phenobarb im and diazepam pr
- Think WHY - review infants notes with above causes in mind

Investigation
- Urgent: ALL OF THESE MUST BE DONE
  - glucose
  - calcium
  - sodium
  - potassium
  - chloride
  - AST
  - HCT (on FBC)
  - BR
  - LP (when fit stopped)
- Less urgent:
  - clotting studies (urgent if sepsis likely)
TREATING SPECIFIC CAUSES

HYPOGLYCAEMIA
- If HGT < 1.7 mmol/l and infant is fitting: 2ml/kg 10% dextrose/neonate electrolyte immediately and continue with infusion of this @ 6ml/kg/hr for one hour or until HGT > 4
- Then reduce the rate to 4ml/kg until HGT stable
- Check HGT's hourly until > 4 and before any change in rate of transfusion.

HYPOCALCAEMIA
- If Ca < 1.7 mmol/l and infant is fitting: 0.5ml/kg 10% Calcium gluconate diluted 1:4 with 10% dextrose or water for injections over 10 - 15 mins.
- NB highly irritant if extravasates. Do not mix with bicarb/phosphate.

POLYCYTHAEMIA
If HCT > 75% and fitting do 20ml/kg dilutional exchange transfusion replacing 20ml/kg blood with FDP over 30 mins.

SUSPECTED OR CONFIRMED MENINGITIS
Benzylopenicillin (200,000u/kg/d) and gentamicin for 14 days or cefotaxime (100mg/kg/d)

ASPHYXIA
(Hypoxic Ischaemic Encephalopathy)
1. Fluids - 2/3 maintenance. Increase glucose concentration to maintain HGT if necessary
2. Relative mild hypothermia is probably protective
3. Control fits. Don't need prophylaxis.

Maintenance
Continuing maintenance anti-convulsants necessary for: severe birth asphyxia
- meningitis
- recurrent or difficult to control fits
- persisting neurological abnormality
- structural brain abnormality
otherwise usually not necessary
Phenobarbitalone maintenance dose = 5mg/kg/d (od).

NEONATAL JAUNDICE

Jaundice can be difficult to pick up in neonates - the tip of the nose and feet are often a better guide than the sclerae. Up to 50% of infants become jaundiced around the third day of life. Foetal Hb falls from 20 g/dl at term to ~14 g/dl in the first week and this increased red cell breakdown exceeds the capacity of hepatic conjugation, resulting in unconjugated hyperbilirubinemia. This physiological jaundice only requires treatment if severe.

- Examine the child carefully for evidence of infection, bleeding, cephalhaematoma, hepatomegaly etc
- Check Hb, Blood group (child and mother if not known), Direct Coombs's test, Bilirubin (total and conjugated) and urine for reducing substances (galactosaemia). Consider blood and urine cultures if infection is suspected
- A useful rule of thumb is that exchange transfusion will be required at (Gestation in weeks x 10) μmol/l and phototherapy at (exchange level - 100) μmol/l although reference charts according to weight and age are more accurate
- Jaundice evident in the first 24-36 hours suggests haemolytic disease of the newborn, generally from ABO incompatibility in our patients. Exchange transfusion will often be required, especially if the rate of rise > 85 μmol/l per day, so discuss early with Addington. Anaemia due to continuing haemolysis may be a problem as late as 6 months so Hb should be monitored monthly and transfusion is indicated for symptoms or Hb < 7.5 g/dl.
- Jaundice persisting beyond 14 days in premature and 21 days in term infants requires further evaluation. The commonest cause is breast milk jaundice - the conjugated bilirubin is typically < 10% total and the prognosis excellent. If > 20% Conjugated bilirubin more serious disorders should be considered (neonatal hepatitis, biliary atresia etc.) - start IV Ampicillin and Gentamicin, check TSH and refer to KEH for further investigations.
CONGENITAL TAIPEES EQUINO VARUS

Congenital talipes equino-varus is the commonest congenital foot deformity in all populations but is very prevalent locally, possibly 4/1000. 2/3 occur in males, 50% bilateral.

**Diagnosis**
The characteristic deformity includes –
- plantar flexion of ankle.
- inversion/varus of the hindfoot.
- adduction of the midfoot (“bean-shaped foot”).
- pes cavus especially 1st two metatarsals.
- The true “resistant” club foot is identified by resistance to passive correction, a short foot with a “tucked-in” small heel, dorsiflexed big toe and thin wasted-looking calf.
- Exclude obvious causes e.g. cerebral palsy, spina bifida/meningomyelocele and arthrogryposis (signified by other joint contractures).

**Management**
Very mild deformities may be treated with strapping (use traction kit strapping, not istoyplast, and use Benzoin tincture to make sure it sticks). However if response is poor, refer early for assessment at Ngwelezane.

Refer to Ngwelezane as early as possible for assessment of clinically severe deformities. Conservative management with weekly POPs for the first month then 2 weekly is pursued for 3 months. Residual equinus alone can be corrected 20-30° by an Achilles tenectomy at this time. If deformity remains severe, then further surgery will be required.

If conservative methods fail then a posteromedial release of soft tissues is carried out at six months and the foot maintained in POP for another 8 weeks.

Late-diagnosed deformities not responding well to the above methods can undergo a triple (Dunn’s) arthrodesis in adolescence.
SOCIAL SECURITY

Part of our duty as government medical officers is to furnish medical reports with respect to various social security grants provided for by the Social Assistance Act of 1992. This Act introduced state pensions for men over the age of 65 and women over the age of 60 years. It also recognised three grants which concern us. Applications for these by out-patients are dealt with at a forti-nightly clinic with the hospital social worker in O.P.D - applications by patients on the ward are usually dealt with by the doctor concerned.

DISABILITY GRANTS

People are eligible for such grants only if:
- They are a South African citizen (i.e. they have an I.D. book)
- They/or their spouse does not have the means to support the patient
- They are over 18 years or are a full-time student under the age of 21 years
- They qualify according to a means test (proof of income/assets - or lack of them must be submitted to the magistrate)
- A report by a medical officer certifies that the patient is unable to support his/herself
- The disability is likely to continue for six months or longer
- The applicants do not refuse to accept employment within their capabilities (an assessment of which is asked for on the form).

Assessment of disability is inevitably somewhat subjective - it is important to take into account the patient's educational attainments and suitability for various kinds of work (e.g. clerical work or labouring). Some assessment of activities of daily living can be helpful. In general, our policy has been to restrict recommendation of grants to the following cases:
- Amputees, paraplegics, some less severe limb injuries
- 'Mentally retarded' patients and severe psychiatric morbidity
- 'Destroyed lungs' from T.B. or occupational lung disease (which may also qualify for Workmen's Compensation - see details below)
- Symptomatic/debilitating H.I.V. infection
- Incurable/advanced malignancy
- Very poorly controlled chronic medical conditions (esp. asthma and epilepsy, rarely hypertension)
- Partially sighted with poor navigational vision

Though many cases are much more difficult to assess than these and for some families grants are the only source of income, it should be borne in mind that section 12 of the Act (False Representations) provides for prosecution in cases where the recipient is not considered to be entitled. The patient goes to the magistrate after the medical report is filled in, has fingerprints taken and fills in the application form, which is submitted to the Director-General of Social Services in Pretoria.

A Grant-in-aid may be given to the attendants of a patient in receipt of a disability grant who requires full-time attendance at home. A Maintenance Grant may be paid to a parent looking after a child whose spouse/partner has deserted/died, is not legally responsible for the child, or is in receipt of a disability grant.

A Care-dependency Grant is provided for a parent if the child is under 18 and requires constant care. The child is evaluated for a special school at the age of six years by an appointee of the local education authority. A Foster-care Grant may be paid to foster parents designated by the social worker in cases where children have been abandoned or neglected by their parents or where both parents have died.

WORKMEN'S COMPENSATION

This provides for by the Occupational Diseases in Mines and Works Act and the Compensation for Occupational Injuries and Diseases Act. The conditions specified are:
- Pneumoconiosis
- Silicosis
- C.O.A.D.
- Platinosis (occupational asthma)
- Asbestosis and related cancers
- Systemic Sclerosis

T.B. itself is only compensable if the patient has worked at least 200 shifts in a mine and left less than a year ago, and >10% loss of lung function is demonstrable on lung function testing. All miners are entitled to a free medical examination at the Johannesburg Medical Bureau for Occupational Diseases twice a year. The law stipulates that their transport costs to the bureau must be refunded. It is possible for M.O.s to carry out the examinations themselves and fill out forms G.W. 24/56 and annex. Lung function data are not essential.

The forms should be forwarded to George Mibonde P.O. Box 4584, Johannesburg 2000
For further information, phone 011 409 6322
SEXUALLY ABUSED CHILD

The S.A.P.S. Child Protection Unit dealt with 22,000 cases of suspected child abuse in R.S.A. during 1994 (a 35% increase over previous years) - our local unit in Richard's Bay is currently seeing 65 new cases each month in the region between Ingwavuma and the Tugela River. This is widely acknowledged as probably being the tip of the iceberg. Child abuse is defined as 'involvement of a child with or without the child's consent with an adult inside/outside the family designed for the gratification of the adult. Sexual abuse is involved in about 11% of cases and in such cases 65% can be shown to harbour STDs. In our setting ~50% of abusers are not family members, usually a neighbour.

Diagnosis

Presentations are varied and may be subtle -

- a parent, relative or teacher may come with a clear history.
- the child may report it in an indirect way to a career (e.g. nurse, neighbour), often hoping to alert authority.
- inappropriate play/precociously detailed understanding of sex.
- other behavioural disturbances e.g. regression, enuresis, sleep disturbance, abdominal pain, running away.
- children who want to stay in hospital.
- unexplained/suspicious genital or perianal symptoms or signs.

In the 'secrecy phase' after frank sexual contact has occurred, the child has often been threatened with death or serious injury by the perpetrator should she report him. Disclosure may be followed by a 'suppression phase' in which the perpetrator will put pressure on the child to withdraw the allegations, possibly with promises of change etc. The mother in such a situation may be unable to choose between her partner and her child and may even pressure the child herself to 'back-down'. The child may be afraid to speak out against authority, feel responsible/shamed and worried about losing the love of one or both parents. For all these reasons, an older nursing colleague who is ready to have prolonged input with the child is vital to successful management and prevention of continued abuse.

Examination

Potentially abused children may be (but are not always) sensitive to being examined by a stranger and examination under anaesthesia should be resorted to if this seems likely. Girls are best examined in the supine knee-chest ('frog-legged') position and boys in the left lateral especially if awake - the knee-chest position may be threatening for children abused in this way. Useful instruments for such an examination are small thumb forceps and nasal speculae. Look for -

- Vulval wounds or erythema/tenderness. Frank offensive discharge. Obvious ulcers/warts.
- Vaginal lacerations are usually vertical but large transverse lacerations of the posterior fornix can occur which may bleed heavily and for penetrate the perineal cavity.
- Lacerations of the hymen. The hymenal ring lies at the junction of the vaginal orifice and the vestibule enclosed by the labia minora. In the virgin, the hymen is a very thin membrane that closes the vaginal orifice but often has one or two small perforations in it <0.5 mm in diameter. Lacerations usually occur on the posterior half of the hymen between the 3 and 6 o'clock positions. In the chronically abused child, the lacerations may have healed and appear as lateral scarring of the hymen or redundant tags similar to the carunculae myrtiformes seen in post-pubertal women. The vaginal orifice may be distorted in shape by the scarring.
- Anal damage. In the acute stages, bruising, lacerations and sphincter spasm are to be expected, though there may be no signs if penetration was slow and well-lubricated. Chronic abuse may be associated with loss of sphincter control, reflex relaxation in response to stroking the peri-anal skin and skin tags/fissures often at the 6 o'clock position.

Management

- Take swabs for bacteriology and identification of spermatozoa if the last episode of abuse occurred less than 48 hours before or there is a distinct occur of semen. Biopsy and swab any ulcers present. Order a wet prep and gram stain of anal/vaginal swabs in the hospital laboratory. All other specimens should be handed as soon as possible to the C.P.U. in Richard's Bay.
- Take blood lor H.I.V. and W.R. Repeat these and, if appropriate, a Pap smear at 3 months.
- Give Erythromycin and Metronidazole in all cases of acute abuse.
- The J88 form carefully filled in should be processed by the local police station. It should include the child’s account of the incidents. A photocopy should be kept in the notes.

The social worker should be involved immediately. It seems likely that a child is suffering abuse. She should apply to the police for a case to be opened and will interview the mother/carer. In difficult cases she can arrange for the child to be removed to a place of safety without the immediate approval of a magistrate under Section 12 of the Child Care Act (1983). This order may be confirmed and arrangements made for the further care of the child within the next 24 hours at the children’s court in Mthubatuba. After discharge the social worker will conduct a home visit to ascertain the child’s continuing welfare and protection.

Many cases do not and probably should not come to trial due to the potential trauma to family and child. Sexual abuse of children is not specifically recognised in criminal law so cases are heard as rape, incest or sexual molestation (where penetration is deemed not to have occurred). In cases where the abuser is a juvenile and the situation can be handled satisfactorily by the family itself this is the usual course of action. Further proceedings may however be necessary where the abuser is an adult and malicious intent involved. All cases of child abuse should however be reported to the magistrate in his capacity as Commissioner of child welfare, even if charges are not contemplated.

Furthem Reading

**ASSAULT**

In cases of assault where a criminal prosecution is contemplated, you will be asked to fill out a form J88 at the request of the attending officer (although substituting ‘Hlabisa Police’ is considered acceptable). The purpose of the form is to corroborate/cast doubt upon the assaulted party’s statement with the available medical evidence. In the case of a serious Offence against the Person (attempted murder, manslaughter, grievous bodily harm etc.) you may well be called upon to testify by the Public Prosecutor/Defence attorney and attention to detail could be important.

Be careful to write ‘alleged’ etc. when writing down the patient’s description of the incident.

You are expected to detail the ‘nature, position, extent of the injuries’ together with the ‘probable date and manner of causation’ and ‘any apparent discrepancy between any statement made by the person and the conditions actually found on examination’.

- BRUISES - are the result of rupture of small blood vessels and extravasation of blood into the soft tissues. The size of the bruise in itself is an unreliable indication of the size/shape of instrument or force used since bleeding may continue/spread after the time of injury, especially in women and elderly people. The typical pattern of colour change with time is purple/blue >brown >green >yellow. The more advanced colour changes can establish only that the bruise is a week or more old and more precise dating is impossible.

- ABRASIONS - are the result of removal of the superficial layers of skin from a rough surface striking the skin tangentially. The direction of the object across the surface of the skin may be inferred by partially detached layers of skin at the margin where the object left. Abrasions may reproduce the surface of the wounding object quite faithfully and retain it until healing occurs.

- LACERATIONS - are produced by blunt trauma of sufficient severity to cause splitting of the skin. In the case of a blunt instrument they usually occur in areas of soft tissue closely overlying bone. The margins of the wound are irregular and the surrounding tissue often bruised/crushed.

- Fibrous and uninjured neurovascular tissue may traverse the depths of the wound and bleeding may be
less than would be expected for the size of the wound. Lacerations do not reproduce the shape of the wounding object accurately.

- **CUTS** - are incised wounds caused by a sharp instrument. The skin adjacent to the wound is uninjured and the edges are straight. There is often considerable bleeding. The direction of the incision may be inferred from the fact that the wound is usually deeper at the end where the cut starts. It is impossible to infer the shape of the instrument from a cut.

- **STABS** - do give some indication of the minimum size and shape of the weapon involved.

- **GUNSHOT WOUNDS** - Entry wounds from rifled weapons (pistol, rifle) are generally clean-cut wounds slightly smaller than the diameter of the bullet. "Contact injuries" when the gun is pressed against the skin are accompanied by bruising/tearing of the skin or a circular bruise next to the wound caused by recoil of the barrel. Close range entry wounds (<1 m) are often characterised by a ring of soot/powder and hair singeing over the surrounding skin. Longer range wounds typically have a 1.5 mm band of abrasion around the hole and a characteristic 'grease collar' derived from the preservative that the bullets are packed in. Thus, it is impossible to infer the range at which a bullet was fired at any distance greater than 1m.

Exit wounds are usually larger than entry wounds, have irregular, everted edges and no soiling by gunpowder products.

Shot-gun wounds vary according to the range. At <1 m there will be a 2-3 cm round hole with a soot ring. There is considerable tissue destruction and wadding/casing from the cartridge will be driven deep into the wound. At 1-3.5 m the central wound will be surrounded by a ring of satellite 3mm wounds from diverging pellets. Debris from the cartridge may be evident in the wound. At >3.5 m the wound consists of a cloud of small pellet holes. The pellets themselves are usually lying very superficially in the skin.

Under 'Remarks' state whether you think the injuries you have described are consistent with the mode of wounding described by the patient or not and why.

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**RAPE**

Violence against women is common in Hlabisa. 23,318 rapes were reported in RSA in 1993 and these probably constitute 3% of the real total. Less than 5% of charges are found by the courts to be unbounded but <10% result in a conviction. Securing a conviction requires diligent collection of the medical evidence, though this will not usually stand alone. ~10% of raped women demonstrate genital injury and 30% other injuries. A successful prosecution rests on demonstrating that sexual intercourse probably occurred and that force or threat was involved, implying lack of consent from the victim.

Choose a suitably private environment where at all possible. Ask about the details of the alleged incident in as sensitive a manner as possible. Particularly enquire about the victim's behaviour after the incident viz. has she.doused, changed her clothes etc. and about her last menstrual period. Obtain verbal consent to carry out an examination.

Examine the whole body for wounds especially bruising/abrasions on the thighs, arms/back of the shoulders and face/neck from forcible restraint. Oval bruises from bites may be present on the neck and breasts.

Examination of the genitalia should pay particular attention to the following:

- Matting of the pubic hair by semen, presence of assailant's pubic hairs in that of the victim.
- Swelling, redness, tenderness and wounds of the vulva and vagina. Dilatation of the vagina (assessed by ability to accept 1, 2 or 3 fingers) does not constitute proof of intercourse in a virgin.
- Condition of the hymen (generally not helpful unless a fresh tear with bleeding and tenderness is present. It may be entirely absent in older women and conversely be elastic enough to remain intact despite intercourse in virgins, especially if tampons are being used.)

Carry out a speculum examination. Take a High Vaginal Swab lor bacteriology in the hospital lab and a second plain damp swab lor detection of spermatozoa (these may persist in the vagina for 5 days and in the cervix for up to 2 weeks). This should be smeared onto a clean glass slide, allowed to air-dry, covered with a second slide and taped together. Any semen stains on the patient's clothes should also be cut out. These specimens are placed into a sealed bag in the presence of a witness and
labelled/signed by the doctor concerned to allow later identification in court. These are supposed to be dispatched immediately by the police officer concerned to the State Forensic Laboratory in Pretoria. Demonstration of spermatozoa in hospital laboratories is not admissible as evidence.

Take blood for W.R. and H.I.V. serology and urine for a pregnancy test (These may be essential for later litigation/therapeutic abortion.) Give

- Ciprofloxacin 250 mg stat p.o.
- Metronidazole 2g stat p.o.
- Erythromycin 500 mg q.d.s p.o. for 10 days
- Ondanestadine 2 tabs stat and 2 tabs 12 hours later.
(Repeat dose with anti-emetic if patient vomits the first)

Allow time for counselling and arrange to see the patient again one week after her next period is due to repeat the pregnancy test and organize a termination if necessary. Repeat the W.R. at this visit if initially negative and H.I.V. serology at 3 months.

Further Reading
2) London et al. 1996 'Reply to the above' S.A.M.J 86:844

DEATH CERTIFICATION

At the present time - 45% of deaths in the Hlabisa magistrature are not formally registered. Registration is the duty of the magistrate, who fills in a handwritten Death Register form (form BI-7) and issues a temporary death certificate. Official death certificates (form BI-5) are issued by the Department of Home affairs in Mthatha but not all families can afford the time, money or effort to obtain one and there is no legal requirement that they do so. The deceased may not always have an I.D. book and a substantial number are buried at home.

Deaths at the hospital are notified to the magistrate/home affairs by a medical certificate in respect of death (BI-12) filled in by the doctor involved with the case. There are no specific exclusions at the present time as to acceptable modes/causes of death as long as what is given could reasonably be construed as such. ~ 10% of patients dying in the hospital are released without certification, since the family do not need a death certificate for pension or insurance claims (mostly elderly people and newborns/young children). Some corpses are not collected by their relatives - these are released from the mortuary after one month and a pauper's burial is arranged near the hospital.

Deaths in the community are recorded by C.H.W.s informally (since they invariably attend the washing of the corpse and burial), and until April 1996 could be directly registered with the magistrate by means of an induna's affidavit as to the cause of death stated by a relative of the deceased. This was sworn in front of the inkosi and signed by him. Since the withdrawal of this system, many deaths in the community without an I.D. book have had to undergo post-mortem in order to obtain a handwritten death certificate (BI-20). A BI-12 is filled in and forwarded to Pretoria with the deceased's fingerprints for confirmation. Deaths in the community, unattended by a medical practitioner but with an I.D. book still present something of a problem but a BI-12 may be issued at the discretion of a medical practitioner if there seems little doubt as to the cause of death and an induna's affidavit is available.

Stillbirths cannot be registered so a handwritten BI-20 is issued.

Unnatural deaths (Assaults, MVAs, suspected poisoning etc.) are registered with the police, who enter the case on either their Inquest Register for non-criminal cases (e.g. suicide, lightning strikes) or Crime Register. They issue a Proof of notice of death (BI-1577) immediately. A post-mortem by the District Surgeon is arranged and a form Z POL 181 (form 2) is issued, which can be used by the magistrate to issue a temporary death certificate (BI-7). Hlabisa police station handled about 20 such cases in 1996.

If you are called upon to examine a patient brought in dead or dead on arrival your observations may later be of use in cases of unnatural death. Aside from the usual signs of death (absence of heart or breath sounds for 5 minutes) certain features may help in establishing the time/mode of death:

- Rectal temperature falls by 0.9 °C/hour after death until it equilibrates with the ambient temperature. To obtain an estimate of the time since death divide the number of degrees less than 37 °C by 0.9°C/hr. The rate of fall may be slowed in fat or well-clothed corpses and accelerated in thin ones and so this method can be considered only an approximation.

- Hypostasis (dependent drainage of blood) is not normally apparent for at least 4-6 hours and when fully established usually places the time since death at >12 hours.
- Rigor mortis (muscle stiffness due to gelling of protein) is usually established by 6 hours, affecting the jaw and extremity muscles first and the whole body by 12 hours.

- Putrefaction usually begins over the abdominal wall followed by marbling of the superficial veins and blistering of the skin. Its presence normally establishes the time since death at >48 hours.

Take note of any suggestive signs of recent trauma or injury. Urine and blood may be taken for toxicology examination but poisoning has historically been a virtually impossible diagnosis to prove in our magistrature.

The Inquests Act of 1959 provides for the holding of inquests and post-mortem examinations in cases of death believed to be due to unnatural causes. Such deaths include:

- those which are likely to entail criminal proceedings
- deaths under anaesthesia
- deaths in police custody

If the cause of a death in the community is obvious from external examination and criminal proceedings against any person will definitely not follow (this should ideally be confirmed with a police officer), a Bl-12 can be issued by the doctor concerned stating the likely cause. If this is clearly not the case, the matter must be taken up with the District Surgeon (Dr S.J. Swanepoel 035 5901100). We do not carry out post-mortem for this or any other purpose on site at present but guidelines are available (D.N.H.P.D. document "Instructions to medical practitioners for the performance of medico-legal post-mortem examinations"). In practice it has been difficult to get consent from families for diagnostic post-mortems in our area.
COMPARTMENTS OF THE ABDOMEN IN ZULU
To convert any of the listed complaints into a question replace the 1st person prefix ngi- with the 2nd person prefix u- +/- suffix na? for emphasis.

Ngilimele - I have sustained an injury
Ngicopha - I am bleeding
Ngiyashisa - I have a fever/feel hot
Ngiyajuluka - I am sweating
Ngikwhelethele - I am coughing
Zinjani izikwhelethele - What is the sputum like?
Zimhlophe - white
Zilimi - green
Zinegazi - blood-stained
Ngicophile emziwimbeni - I am losing weight
Ngiphelilele emzali - I am losing strength
Ngiyaphadzawula/ Ngayagodola - I am shivering/having chills
Nginezuluwana - I am feeling dizzy
Nginezoxaba - I have a wound

Ngaphukile - I have a fracture
Nginezummba - I have an abscess
Ngiyaphuka - I have a rash
Ngiphuzukile - I have a bruise
Ngiphudu - I have diarrhoea (adults)
Uyakapala - She has diarrhoea (children)

Ukaka kanga ngelangana? - How many times a day do you pass stools?
Uyakanga ngaphandla? - How many times a day do you pass stools?
Anjani amakaka? - What are your stools like?

Ngiyaphakalaza / hlana - I am vomiting
Isisu sikumbele - my abdomen is distended / I am constipated
Angisitini - I am not passing any llasus

Ngadluwe yinangana - I have missed a period
Ngiphuma isisu - I had a miscarriage
Ngitwana ukukuhlola - I would like to examine you
Ngeke ngikulimaze - I won't hurt you
Khumula - Take off your clothes
Gqaka - Put on your clothes
Lala ngomhlanezo / ngohlangothi - Lie on your back/side
Khuphuka / phakama - Sit up

There is no exact equivalent of 'please' in Zulu but requests may be made more polite sounding by the use of the construction ngicela ukuthi - (I request you to) or by adding the suffix ke to the listed commands.

Yahla - Lie flat
Phenduka - Roll over
Phethumela - Breathe in and out
Bamba U moyo - Hold your breath
Khupha U moyo - Breathe out
Donza U moyo - Breathe in
Kharhuma - Open wide
Yiti 'aah' - Say 'aah'
Gosha - Open your legs wide
Yena kanje / ngapha - Do it like this (When all else fails)
Khulukhale / Phambisa - Relax
Unganyakazi - Stay still
Ungakhathazeki - Don't worry
Ngikhombise laPho kubuhlulu kekhona - Show me where it hurts.
Unguthile uma uzwa ubuhlungu - Tell me when you feel pain.
Uzolinza - You're going to be well.
Sizofihla igazi - We will take a blood test.
Kufanele uzolinza imiphumele - You must wait for the results.
Sizofihla umchama wakhe - We'll take a urine test.

You must' is usually expressed by the construction 'Kufanele' or 'Kumenele' followed by a verb or infinitive. 'I need' is expressed in most circumstances by 'ngidinga' plus an object or infinitive.

Uzolashwa - You will be treated.
Nanku umuthi wakhe - Here is your medicine.
Phuza amaphilisi zonke uzinsuku - Take the tablets every day.
Ungawegisi lo multi - Don't take more than is prescribed.
Ekenseni - In the morning.
Emini - At lunchtime.
Nhambana - In the afternoon.
Ehutuku - At night.
Phuza lo multi... - Take this medicine...
Kabili ngosuku - Twice a day.
Kalhafu ngosuku - Three times a day.
Kane ngosuku - Four times a day.
Phuza umuthi/gwenya amaphilisi ungakadli - Take the medicine/tablets before meals.
Phuza umuthi/gwenya amaphilisi usudileile - Take the medicine/tablets after meals.
Phuza umuthi/gwenya amaphilisi uma uzwa ubuhlungu - Take the medicine/tablets when you feel pain.
Consisela lo multi endlebeni - Put these drops in your ears.
Kufanele uncipha - You must lose weight.
Gqoka/sebenzisa ikhondom/Izazi - Wear/use a condom.
Ngicela ubuye nthlaka - 14 February. I would like you to come back on 14 February.
Khumbula usuku lokubyelela esibhledlela - Remember your next appointment.
Ungalukhothila - Don't forget (the date).

Uzolimzwelwa - You need an operation.
Sizokuthonga imxibha - We will stitch your wound.
Ngizochasiza - I will explain.
Ngidinga imvume yakhe - I need your consent.

Sizokhipha izitishi ngomhlahla - 2 May/ezinsukwini ezinhlanu - We will take the stitches out on 2 May/in 5 days.
Kusobala? - Is it clear?
Unembumvelo? - Do you have any questions?

PARTS OF THE BODY:

umziomba - body
ikhandu - head
isimongo/isunzi - forehead
ubuso - face
ishiya - eyebrow
ikhala - nose
ishitali - cheek
umlando - mouth
udebe - lip
ulimi - tongue
umfuthathi - jaw
isilevu - chin
i/amaziyana - tooth/teeth
intama - neck
i/amahloni - shoulder/s
i/mfizangalo - arm/s
i/zindoloflwane - elbow/s
i/mikheni - forearm/s
i/zingane - butlock/s
i/amathungu - thigh
i/amakhombe - shoulder/s
i/milimane - leg/s
i/amadolo - kneel/s
i/amagakala - ankle/s
u/zinyawo - foot/toel
u/zinyawo - foot/toel
u/zingane - toe/s
i/zingane - toe/s
i/mqanda - hand/s
i/mqanda - hand/s
i/imunye - finger/s
i/sibindi - liver
i/buchopho - brain
i/sibeletho - uterus

SELECTED DISEASES:

Wake waphathwa yini? - Have you ever suffered from?
Uphando - alcoholism.
Umlutshi wegazi/t'high-high' - hypertension.
Isilo seechansi - sexually transmitted disease.
Udrope - gonorrhoea.
Isimungumwane - measles.
Isiluba sunonya - asthma.
Ingubhunjwa - chicken pox.
Ingubhunjwa - chicken pox.
Isangankonko - whooping cough.
Uqhubhoo o Umalavuma - malaria.
Isilepero - leprosy.
Isichenene - schistosomiasis.
Isisuzi seeqazi - dysentery.
ITB - TB.

Ikufana kwabantu, e.g. lata - Zulu diseases often with no exact European equivalent.
GASTROINTESTINAL SYSTEM
Drug:
- Magnesium Hydroxide Mixture
- Aluminum Hydroxide 250 mg/Magnesium Trisilicate 500 mg tabs
- Cimetidine tablets 400 mg
- Omeprazole 20 mg capsules
- Metoclopramide tablets 10 mg
- Senna tablets 7 mg
- Liquid Paraffin
- Phosphate enema
- Cholestyramine powder
- Amethocaine 1% ointment

ENDOCRINE SYSTEM
Drug:
- Glibenclamide 5 mg tablets
- Metformin 500 mg tablets
- Actrapid 100 U/ml vial
- Actraphane 100 U/ml
- Prednisolone 5 mg tablets
- Hydrocortisone 100 mg/2 ml vials
- Dexamethasone 500 µg tablets
- Methylprednisolone sodium succinate 500 mg injection
- Thyroxine 50 µg tablets
- Carbimazole 5 mg tablets

RESPIRATORY SYSTEM
Drug:
- Salbutamol 100 µg inhaler (300 doses)
- Salbutamol nebuliser 5 mg/ml
- Salbutamol syrup 0.4 mg/ml
- Aminophylline 25 mg/ml injection
- Beclomethasone 100 µg and 50 µg inhaler (200 doses)
- Pseudoephedrine 0.25/2 ml & 0.5/2 ml
- Saline nose drops
- Compound Benoin Tincture
- Simple Linoleum
- Theophylline syrup 25 mg/5 ml
- Theophylline 250 mg Sustained release tablets

CARdiovascular SYSTEM
Drug:
- Hydrochlorothiazide 25 mg tablets
- Hydrochlorothiazide 25 mg/Triamterene 50 mg tablets
- Frusenide 40 mg tablets
- Frusenide 20 mg injection
- Reserpine 0.25 mg tablets
- Atenolol 50 mg tablets
- Hydralazine 25 mg tablets and 25 mg/ml ampoules
- Methyl Dopa 250 mg tablets
- Enalapril 5 mg tablets
- Nifedipine 10 mg capsules
- Digoxin 0.25 mg tablets

ANTI-INFECTIVES
Drug:
- Phenoxymethyl penicillin 250 mg tablets
- Benzylpenicillin 1 MU and 5 MU vials
- Procaine penicillin G 10/1 ml vials
- Benzathine penicillin 2.4 MU vial
- Ampicillin injection 500 mg vial
- Amoxycillin 250 mg capsules and 125 mg/5 ml syrup
- Ciprofloxacin 250 mg capsules and 500 mg vial for injection
- Chloramphenicol 250 mg capsules and 1 g vial for injection
- Co-trimoxazole 80/400 mg tablets and 40/200 mg/ml suspension
- Erythromycin 250 mg tablets
- Metronidazole 400 mg tablets and 1 g suppositories
- Doxycycline 100 mg capsules
- Ciprofloxacin 250 mg tablets
- Metildox acid 500 mg tablets and 250 mg/5 ml syrup
- Nitrofurantoin 50 mg capsules
- Gentamicin 40 mg/ml ampoules for injection
- Ceftriaxone 1 g vials
- Cefazolin 500 mg vials for injection
- Keloconazole 200 mg tablets
- Fluconazole 50 mg capsule
- Cefipime 500 mg vaginal tablets
- Fansidar (Sulfadoxine 500 mg/Pyrimetamine 25
ANTI-INFECTIVES cont.
mg tablets
Quinine Sulphate 300 mg tablets and Quinine
Dihydrochloride 300 mg/ml injection
Zidovudine 100 mg capsules
Praziquantel 600 mg tablets
Piperazine 750 mg/5 ml elixir
Niclosamide 500 mg tablets
Alendazole 200 mg tablets
Nystatin suspension 100,000 u/ml

TUBERCULOSIS DRUGS
Drug:
Rifampicin 150 mg, 450 mg and 600 mg tablets
Isoniazid 200 mg and 300 mg tablets
Pyrazinamide 500 mg tablets
Ethambutol 400 mg tablets
Ethionamide 250 mg tablets
Streptomycin 1 g vial for injection

ANAESTHETIC AND RESUSCITATION
DRUGS
Drug:
Ketamine 50 mg/ml injection
Diazepam 10 mg/2 ml injection
Thiopentone 500 mg vials
Suxamethonium 100 mg/2 ml vials
Alcuronium 10 mg/2 ml
Neostigmine 2.5 mg/ml ampoules
Atropine 0.5 mg/ml ampoules
Halothane
Adrenaline 1 mg/ml ampoules
Ephedrine 50 mg/ml for injection
Lignocaine 1% 25 ml vials for injection
Bupivacaine 0.5% 10 ml ampoule for injection
Hyperbaric Bupivacaine 0.5% 4 ml ampoule for injection
Calcium gluconate 10% injection
Sodium Bicarbonate 4% and 8.5% 50 ml ampoules for injection
Naloxone 0.02 mg/ml and 0.4 mg/ml ampoules

OBSTETRICS AND GYNAECOLOGY/ FAMlLY PLAnNING
Drug:
Oxytocin 1 u/ml and 10 u/ml vials
Oxytocin 5 u + Ergometrine 500 µg/1 ml
Hexoprenaline 5 µg/2 ml ampoules and 25 µg/10 ml infusion
Dinoprostone 0.5 mg tablets
Misoprostol 0.2 mg tablets
Ergometrine 0.5 mg/1 ml injection
Dinoprostone 5 mg/1 ml ampoules for injection
Medroxyprogesterone Acetate 150 mg/1 ml vial
Norethisterone enanthate 150 mg/1 ml vial
Triphasil 28s
Nordette 28s
Ovral 28s
Microlval (Levomogestrel 30 µg) 28s
Medroxyprogesterone acetate 5 mg tablets
Norethisterone 5 mg tablets
Premprak-N 0.625/5 mg and 1.25/5 mg 28s
Cyproterone acetate 100 mg tablets
Magnesium Sulphate 50% Injection 2 ml ampoules
Methenamic acid 250 mg capsules
Vitamin K 1 mg ampoules

ANTICONVULSANTS AND
PSYCHOTROPICS
Drug:
Phenytoin 100 mg capsules, 125 mg/5 ml suspension
and 250 mg/5 ml ampoules for injection
Carbamazepine 200 mg Sustained release tablets and syrup 100 mg/5 ml
Phenobarbital 200 mg/1 ml ampoules for injection
and 60 mg tablets
Valproate 200 mg / 300 mg capsules
Chlorpromazine 100 mg tablets and 50 mg/2 ml 
ampoule for injection
Trifluoperazine 5 mg tablets
Thioridazine 50 mg tablets
Clozapine 40 mg/4 ml injection
Haloperidol 20 mg/2 ml
Promethazine 50 mg/2 ml ampoules for injection and 25 mg tablets
Fluphenazine decanoate injection 25 mg/1 ml vials for injection
Zopiclone 5 mg tablets
Orphenadrine 50 mg tablets
Amtriptyline 25 mg tablets
Fluoxetine 20 mg tablets
Lithium carbonate 250 mg tablets
Diazepam 5 mg tablets
Trimeprazine 6 mg/ml syrup

ANALGESICS

Drug:
Paracetamol 500 mg tablets and 120 mg/5 ml aixir
Paracetamol 500 mg + 30 mg Codeine Combination tablets
Aspirin 300 mg tablets
Ibuprofen 200 mg tablets
Diclofenac 75 mg/3 ml ampoules for injection and 25 mg tablets
Indomethacin 25 mg capsules
Morphine Sulphate 10 mg and 30 mg Sustained release tablets
Morphine Sulphate 15 mg/ml ampoules for injection
Pethidine 25 mg/ml, 50 mg/ml and 100 mg/2 ml
Tilidine 50 mg/0.5 ml drops
Codeine phosphate 30 mg tablets

DERMATOLOGICALS

Drug:
Emulsifying ointment 500g jar
Zinc oxide paste
Basic lotion
Basic powder
Potassium Permanganate
Sodium Thiosulphate
Benzyloperoxide 5% gel
Gentian violet 0.5% solution
Povidone-Iodine ointment 500g jar
Whitfield's ointment
Clo-trimazole 1% cream
Selenium Sulphide 2.5% shampoo 50 ml
Griseofulvin 500 mg tablets
Benzy benzate 25% application 100 ml bottles
Benzene hexachloride lotion and shampoo 100 ml
Thiabendazole 17.6% solution 7.5 ml bottles
Calamine lotion
Hydrocortisone 1% cream
Clodiamed 0.05% ointment

OPTHALMOLOGICALS

Drug:
Hydroxypropylmethylcellulose/dextran 70 drops
Antazoline 0.05% drops
Dexamethasone 0.1%
Betamethasone 0.1%
Chloramphenicol 0.5% drops and 1% ointment
Gentamicin 0.3% drops
Aцикловир 3% ointment
Timolol 0.25% - 0.5% drops
Levobunolol 0.5% drops
Pilocarpine 1%, 2% & 4% drops
Acetazolamide 250 mg tablets
Cyclopentolate 1% drops
Atropine 1% drops
Pheylephrine 10% drops
Oxybuprocaine 0.4% drops

EAR, NOSE AND THROAT

Drug:
Acetic acid 1% ear drops
0.9% Saline nose drops
Glycopyrinate ear drops
Beclomethasone 50 mg nasal spray
Cetylpyridinium 5mg/10 ml mouthwash

MISCELLANEOUS

Drug:
Vitamin A 50,000 i u. tablets and 100,000 i u./2 ml ampoules for injection
Vitamin B Complex tablets
Nicotinamide 100 mg tablets
Tiamine 1000 mg/10 ml vials for injection
Pyridoxine 25 mg tablets
Vitamin B 12 injection 1 mg/ml
Multivitamin tablets
Ferrous Sulphate 200 mg tablets and Ferrous Glycinate (40 mg Iron/5 ml)
Ferrous Fumarate 200 mg /Folate 100 μg tablets
Folic acid 5 mg tablets
Ipecacuanha tincture
Activated charcoal
Desferrioxamine 500 mg vials for injection
Acetylcysteine 200 mg/ml injection
Tranexamic acid 100mg/ml ampoules for injection
MISCELLANEOUS cont.
Allopurinol 100 mg tablets
Omnipaque 380 mg/ml
Barium powder

I.V. FLUIDS
0.9% Saline
Modified Ringer’s Lactate
Half-strength Darrow’s solution with 5% dextrose
Dextrose 5%
Neolyte
Paediatric maintenance solution
Haemaccel
Human albumin 20% solution
Freeze dried plasma
Blood

VACCINES AND IMMUNOGLOBULINS
Drug:
BCG & diluent
DPT
DT
Tetanus toxoid
HBV
Measles & diluent
Polio
Rabies & diluent
Rabies immunoglobulin
Tetanus immunoglobulin
Hib
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The Hlabisa Hospital Handbook is an essential desktop reference book for every community service doctor, medical officer, or medical student who serves in a rural district hospital in South Africa. Generalist doctors in rural hospitals are called upon to deal with a vast spectrum of disease and pathology, which can be overwhelming to the recent graduate or newcomer from overseas, who is “thrown in at the deep end” with little or no supervision. Local clinical management protocols have not been compiled in many institutions, and medical staff make their own decisions without standardized treatment guidelines. Standard medical textbooks often suggest investigations and management plans that are unavailable in rural areas. The Handbook aims to fill this gap, and provide simple and practical guidelines for the commonest clinical conditions in tropical southern Africa.

Written specifically for the context of Hlabisa Hospital in KwaZulu-Natal, the Handbook’s approach in management to a wide range of conditions nevertheless is applicable to similar rural hospitals in other regions of the country. The author, a physician from the UK with four years experience in rural practice, has included all the clinical management protocols that he would have liked to have known when he started at Hlabisa, including a section on the Zulu language. He has drawn on the accumulated experience of a wide range of colleagues, and in this way has extended the scope of the material. This book is an extremely valuable resource for young doctors, and provides a baseline that can be adapted for use in district hospitals in other contexts.

In the deep end, the Hlabisa Handbook is a lifeline.

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