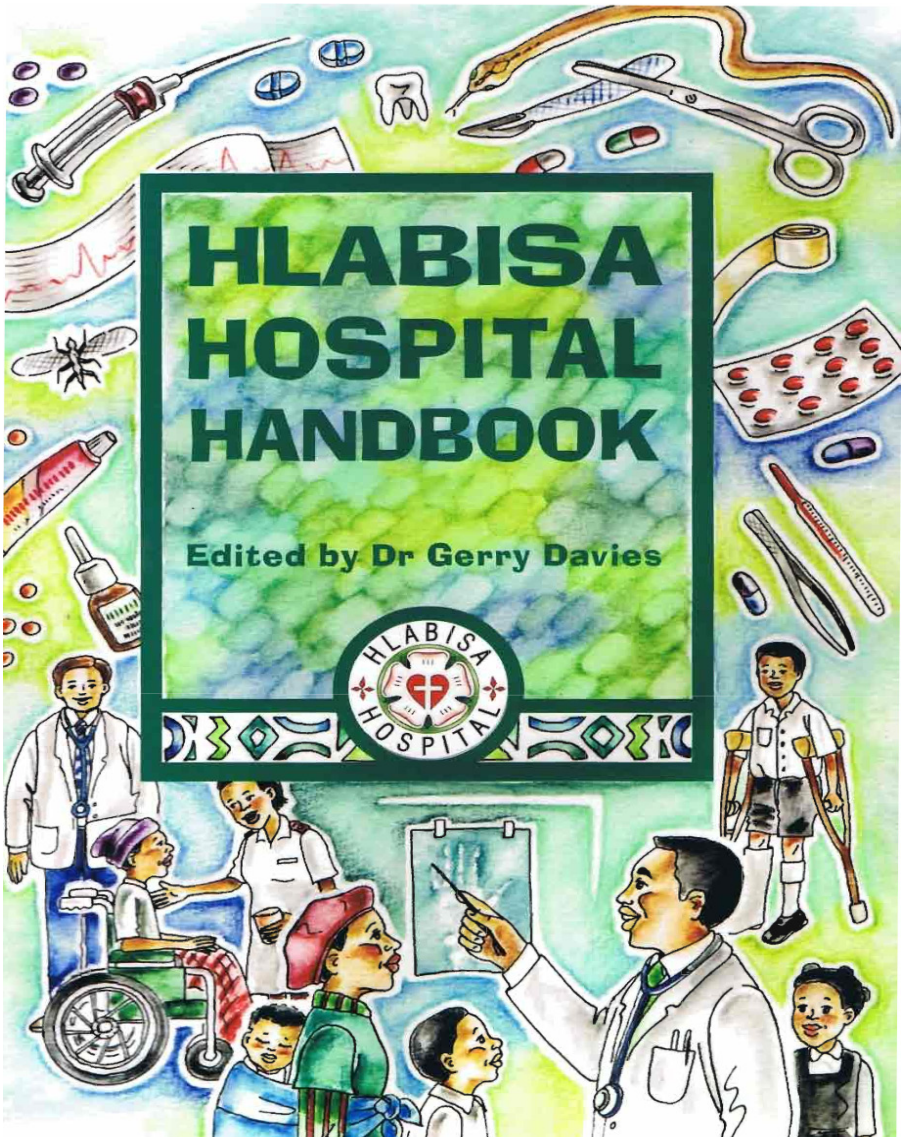
Hlabisa Hospital Handbook



Gerry Davies

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HLABISA HOSPITAL HANDBOOK

PROTOCOLS

SOUTH AFRICAN ACADEMY OF FAMILY PRACTICE / PRIMARY CARE

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.

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HLABISA HOSPITAL HANDBOOK

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Ubude abuphangwa

Dedicated to the memory of
Alma Buthelezi
and
Spence Alexander

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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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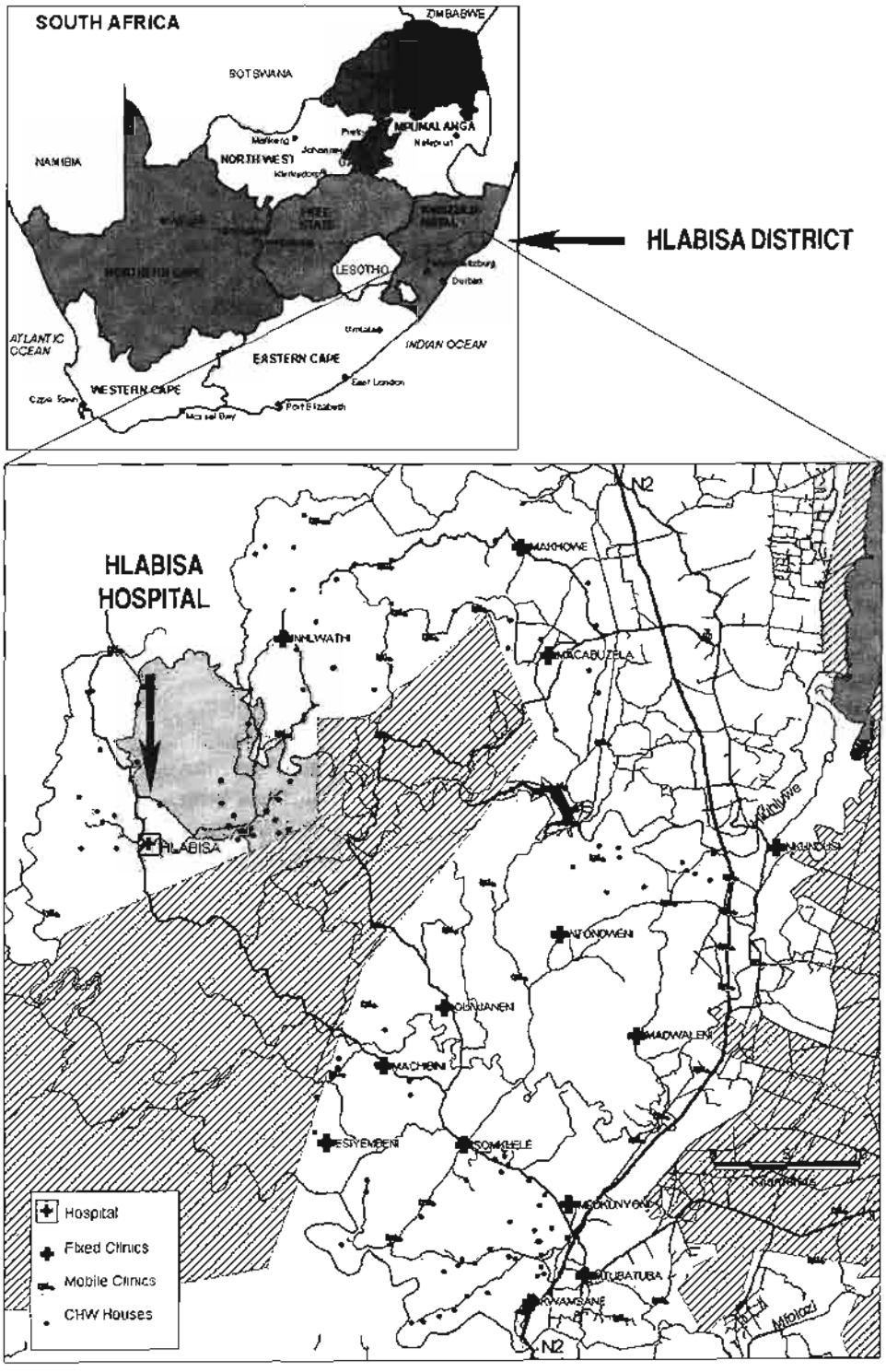


HEALTH FACILITIES IN THE HLABISA DISTRICT

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 at Dukuduku and a large rural hinterland rising to 300+m above sea-level in the north west. 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 cane-cutters receive as little as R2.50 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 Richard's 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
January 2001.

Further reading

1) Couper I 1999 'Why doctors choose to work in rural hospitals' S.A.M.J 89 736

2) Reid, SJ 1999 'The procedural skills of rural doctors' *S.A.M.J* 86:769

ANAESTHESIA

PRE-OPERATIVE ASSESSMENT AND ORDERS

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 *patent i.v. line*. Blood for ectopics 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.O. 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 *Cimetidine 800 mg stat p.o + Sodium Citrate 0.3M 30 ml 30 minutes prior to surgery* to minimise the risk of aspiration, should general anaesthesia / paramedication be required. Patients for *spinal anaesthesia* should be pre-loaded with *1litre 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.

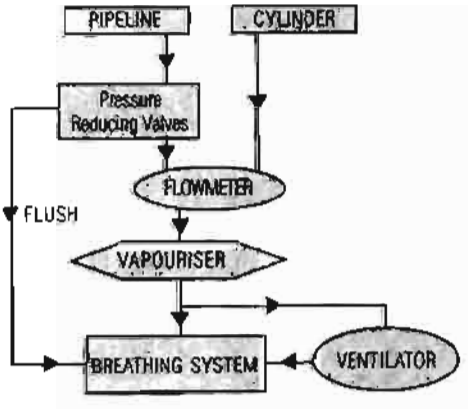
Further Reading
1) Carson et al. 1988 'Severity of anaemia and operative mortality and morbidity' *Lancet* i 727
2) Johnston JR 1982 'A field trial of cimetidine as the sole oral antacid in obstetric anaesthesia' *Anaesthesia* 37:33
3) Phillips et al.1993 'Pre-operative drinking does not affect gastric contents' *B J. Anaesth.* 70:6

EQUIPMENT AND SAFETY

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.D.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.

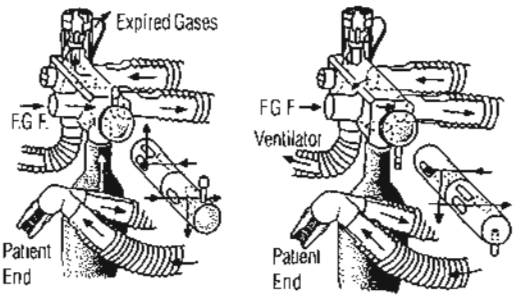
THE MACHINE

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 vapouriser 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 "spill" 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 D 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 off 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*.

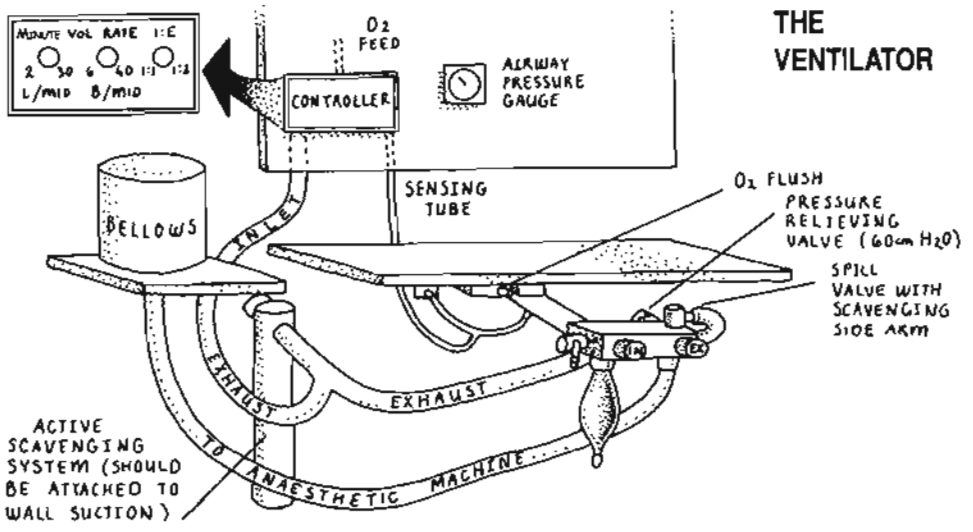
The *respiratory rate* should be set on the dial at *12 / min for an adult* and slightly higher for a child. The *required tidal volume is 10 ml / kg* - multiply this by the R.R. to obtain the *minute volume* (8-10 l / min for most adults) and *set this on the minute volume dial*. Set the *I:E ratio 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 bulk supply* outside the theatre - these should read ~ 400 kPa for gases and - 70 kPa for vacuum.
2) Disconnect *the O_2 (white) and N_2O (blue) pipelines* from the PMGV outlet on the ceiling behind the machine. Turn on the flowmeter - no flow should occur and the O_2 failure alarm will sound.



3) Turn *ON the O_2 cylinder* - there should be no leakage of gas at the back of the machine. The O_2 bobbin only should rise and the O_2 cylinder gauge should read full.
4) *Turn ON the N_2O cylinder* - there should be no leakage of gas at the back of the machine. The N_2O bobbin should rise and the N_2O cylinder gauge should read full.
5) *Turn OFF the O_2 cylinder* - BOTH bobbins should fall. The O_2 failure alarm should again sound.
6) Reconnect *the O_2 pipe* to the PMGV outlet - BOTH bobbins should rise, the O_2 failure alarm should be turned off.
7) *Turn OFF the N_2O cylinder* - the N_2O bobbin only should fall.
8) *Reconnect the N_2O pipe* to the PMGV outlet - the N_2O 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 O_2 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 (N.B. the power failure alarm is audible only).

Further test the high and low airway pressure alarms as follows :

- fit 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 1 l
- put finger tightly over the end
- the *"set volume not delivered" alarm* will sound and the bellows will re-expand before reaching the 1 l 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) Humphrey D 1986 'Towards simpler, safer and more efficient anaesthetic breathing systems' in M. Moles (ed.) *Excerpta Medica, Recent Advances in Anaesthesia, Critical and Immediate Care*, Hong Kong, 93-107
2) *Ohmeda 7000 Manual* (Ohmeda)

AIRWAY MANAGEMENT

Control of the airway is the FIRST PRIORITY in anaesthetics - 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 III/IVs (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 / instill 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% O_2 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, inflate 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 oesophgeal detector device or take 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 shallowly 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 Halothane and attempt intubation with a second dose of Suxamethonium if required. Be very cautious about a second attempt in non-emergency patients and cancel non-urgent 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 Suxamethonium. Try a nasopharyngeal airway / tube and release cricoid pressure. If all else fails do an emergency needle crico-throidotomy and set up for formal surgical cricothyroidotomy.

LARYNGEAL SPASM

Occurs most commonly during *induction* but may also occur after extubation, especially if mal-timed (i.e. when the patient is semi-anaesthetised). 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 laryngoscope (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 spill 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 Guedel airway as well to pre-empt this problem.

Further Reading
1) Kerr JH 1987 'Is the tube in the trachea ?' *B.M.J.* 295:400
2) Turnstall ME 1976 'Failed intubation drill' *Anaesthesia* 31:850
3) Wee MYK 1988 'The oesophageal detector device. Assessment of a new method to distinguish oesophageal from tracheal intubation' *Anaesthesia* 43:27

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 arrythmias and loss of airway control seem to occur quite infrequently.

Monitoring of patients during general or spinal anaesthesia is essential for spotting /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 O_2 dissociation curve lies at ~ 90% and that blood O_2 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 anaesthetic 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 /without 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".

STAGES OF ANAESTHESIA

STAGE	RESPIR-ATION	PUPILS	EYE REFLEXES	URT & RESPIRATORY REFLEXES
1. Analgesia	Regular Small Volume			
2 Excitement	Irregular		Eyelash absent	
3 Anaes-thesia *Plane I*	Regular Large Volume		Eyelid absent Conjunctival depressed	Pharyngeal & vomiting depressed
Plane II	Regular Large Volume		Corneal depressed	
Plane III	Regular becoming diaphragmatic Small volume			Laryngeal depressed
Plane IV	Irregular Diaphragmatic Small volume			Carinal depressed
4 Overdose	Apnoea			

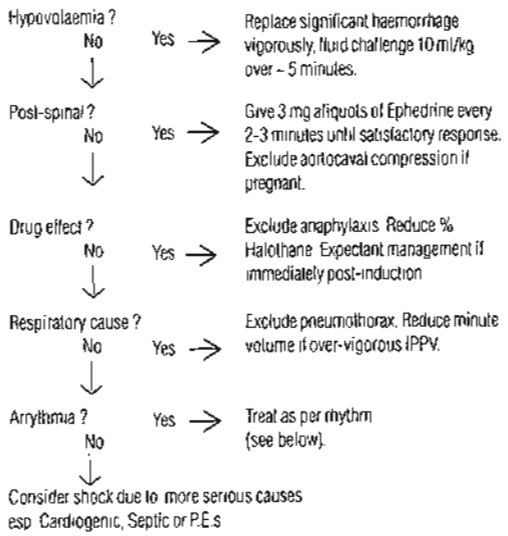
COMPLICATIONS DURING ANAESTHESIA

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 / 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 colloid 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.

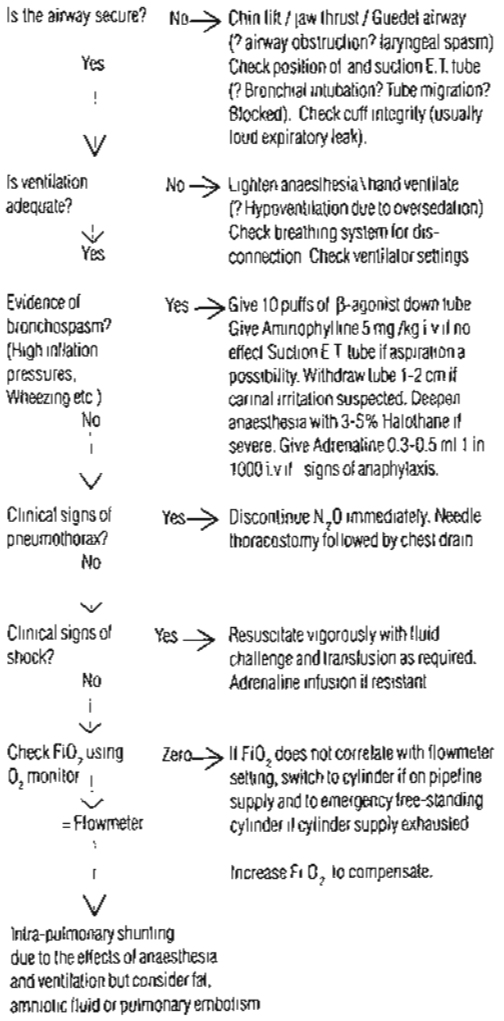


Hypertension intra-operatively is the norm under ketamine anaesthesia. In other circumstances, it usually reflects pre-existing hypertension, poorly controlled pre-op and/ or "light" anaesthesia. Hypercapnia should however be considered in patients undergoing mechanical ventilation / long procedures when CO_2 retention could occur.

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 -



ARRYTHMIAS

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 arrythmias 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 supression 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 maneuvres should be tried with P.A.T., but if there is no response and in cases of paroxysmal A.F., give *Digoxin 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. Frank V.T. (v. uncommon in our patients) requires lignocaine 1.5 mg/kg stat 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 *erythematous 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) Hanning and Alexander-Williams 1995 'Pulse Oximetry - a practical review' *BMJ* 311:367

2) Zanik PE and Davis HS 1968 'Cardiac arrythmias during halothane anaesthesia' *Anaesth. Analg.* 47.299

3) Morris WR 1993 'Desaturation - diagnosis and treatment' *Balliere's Clinical Anaesthesiology* 7(2).215

4) Fisher MM and Boldo BA 1984 'Anaphylactoid reactions during anaesthesia' *Clin. Anaesthesiol* 2:677

5) Scott D.A. and Davies M.J. 1993 'Hypotension' *Balliere'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 some 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% (5mg / 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 = 0. 1 ml of 1:1000 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 arrythmias 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 20ml 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 ephedrine or an adrenaline infusion may be required to support the circulation temporarily.

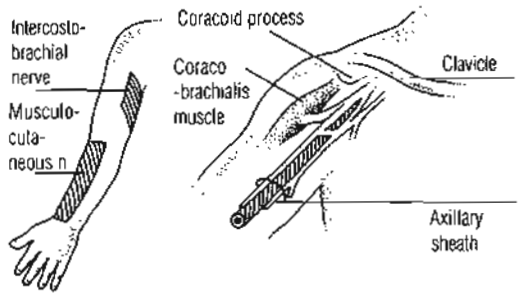
AXILLARY BLOCK

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

The principal pitfall is *poor blockade of the musculo-cutaneous* and *intercosto-brachial nerves* which leave the sheath proximally at the level of the coracoid 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 mls 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* (musculocutaneous 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



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

Further Reading
1) Lanz E et al. 1983 'The extent of blockade following various techniques of brachial plexus block' *Anaesth Analgesia* 62:55
2) Pere P et al. 1993 'Clinical and radiological comparison of perivascular and transarterial technique of axillary brachial plexus block' *B.J. Anaesth.* 70(3):276
3) Gormley WP 1996 'The effect of alkalinization of lignocaine on axillary brachial plexus anaesthesia' *Anaesthesia* 51(2):185

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.

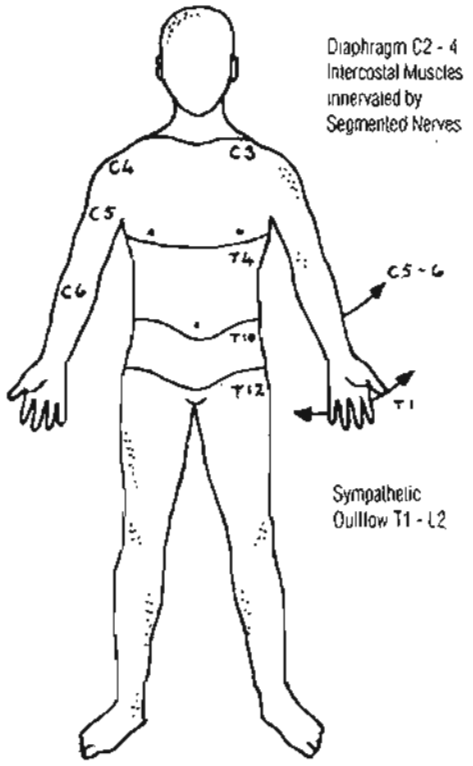
The contra-indications to spinal anaesthesia are :
- bleeding diathesis / platelets < 100
- hypovolaemia
- fixed cardiac output (e.g. severe mitral stenosis)
- local sepsis on the back
- raised intra-cranial pressure
- G.I. perforation (increased peristalsis due to unopposed parasympathetic action)
- 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 N/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 barbotage. *ONLY 0.5% Bupivacaine with 5% dextrose (s.g. 1. 024) should be used – 1. 5-2 0 mls (7.5 - 10 mg) for Caesarian Section, 1. 0-1.5 mls (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 /5° 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 T8-10* 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 *fast 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 T1or 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% Lignocaine* as follows - add 20 ml 2% Lignocaine to 80 ml sterile 0.9% Saline with 0.5 ml 1:1000 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 paraspinal haematoma or abscess.

Further Reading

1) Wilkinson D 1996 'Low spinal anaesthesia for Caesarian Section' Update in *Anaesthesia* 6:28

2) Van Bohaert LJ 1996 'Spinal Anaesthesia for Caesarian Section' Update in *Anaesthesia* 7:30

3) Inglis A et al. 1995 'Maternal position during induction of spinal anaesthesia for C/S. A comparison of right lateral and sitting positions' *Anaesthesia* 50(4):363

4) Chambers WA 1982 'Spinal anaesthesia with hyperbaric bupivacaine' effects of concentration and volume administered' *B.J. Anaesthesia* 54(1):75

GENERAL ANAESTHESIA

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 acheived until nystagmus and voluntary movement have been abolished*. Usually *children* should be pre-medicated with *atropine 0.02 mg/kg* to prevent *sialorrhoea* 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 mls 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). Neuromuscular 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 O_2 / N_2O 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. ectopics, 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.
   - *Suxamethonium* 1mg/kg i.v. bolus. Keep the mask sealed 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) *Alcuronium 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 - N_2O / O_2 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 alcuronium 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 N_2O 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).
   - Extubate.

Further Reading
1) White et al. 1982 'Ketamine -its pharmacology and therapeutic uses' *Anaesthesiology* 56:119
2) White P.F. 1988 'What's new in intravenous anaesthetics?' *Anaesthesiology clinics of N. America* 6 (2) 297
3) Kelcham D.W. 1990 'Where there is no anaesthesiologist : the many uses of Ketamine' *Trop Doc.* 20:163

OBSTETRICS & GYNAECOLOGY

MATERNITY

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* with/without the aid of a traditional birth attendant. Some use traditional medicines to facilitate labour (*isihlambezo, inembe, imbelekisane)* and afterwards (*ugobho)*. Some of the plants included in these preparations e.g. *clivia miniata* have powerful oxytocic effects. At home the cord may be cut with a razor blade but is traditionally cut with a reed (*umhlanga)* 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 3rd 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 10^5*. 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 pelves* 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 3rd trimester / first stage and *frequent 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 *caesarian section* for hospital/clinic deliveries is currently *~ 15 %*.

Only about *2.5%* of the Zulu population are *Rhesus negative* and the commonest Rhesus phenotype is *cDe* 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
1) Larsen JV et al. 1983 'The fate of mothers delivering at home in rural Kwazulu' *S.A.M.J.* 63:543
2) Stewart K.S et al. 1979 'Pelvic dimensions and the outcome of trial labour in Shona and Zulu primigravidas' *S.A.M.J.* 55:847
3) Larsen JV 1996 'The differences between caucasian and black obstetric patients in South Africa' *O&G Forum* p.36
4) Larsen J.V et al. 1996 'Maternal mortality in hospitals in Zululand July 1993 - June 1994' *S.A.M.J.* 86: 424
5) Mabina M.H. et al. 1997 'The effect of traditional herbal medicines on pregnancy outcome' *S.A.M.J.* 87. 1008

ANTENATAL

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:

- *Aproteinuric* - may be chronic hypertension (present in the first half of pregnancy), or gestational.These patients are at lower risk.
- *Proteinuric* - 1+ 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, abruption, 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 aproteinuric, U/Cr/Urate/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
   - Hydrallazine 25-50 mg 6 hrly
   - Nifedipine 10-30 mg 8 hrly
   - Prazosin 1-5 mg 6 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

Management

- Control B.P..
- Check U&Es, Cr, Urate and platelets twice weekly.
- Kick chart b.d..
- CTG twice weekly and anytime foetal movements poor.
- *<34 weeks* give decadron 6 mg i.m. 12 hrly x 4 doses.
- be conservative if
  - BP controlled
  - foetus well
  - renal function does not deteriorate

  Otherwise delivery is indicated - consider referral to a unit with neonatal ICU.
- *>34 weeks* (do amnio if unsure) deliver - by induction or by C/S if evidence of foetal compromise.
- Imminent eclampsia - characterised by restlessness, headache, hyperreflexia/clonus and hepatic pain/tenderness in the presence of deteriorating bloods. Give Nifedipine or Hydrallazine to control B.P. as required and give a loading dose of Magnesium Sulphate. Delivery should be urgent if the maternal condition does not stabilise, the gestation is <28 or >34 weeks or there is foetal distress and should certainly be achieved within 24 hours in all cases.

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 O_2 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 patellar 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 hydrallazine 6.25 mg i.m. in unconcious patients (but beware signs of cardiac failure). Repeat in 30 minutes if no response but be careful not to precipitate a hypotensive 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 H_2O). 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 feasibility of vaginal delivery and condition/viability of the foetus. Order Hb,U&Es/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 and if not already in labour induce on admission 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 laryngeal oedema (have 7.0 ET tube at hand)and the effects of magnesium sulphate on other drugs - reduce doses of muscle relaxants and opiates 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
1) Mwinyoglee et al. 1996 'Eclampsia at Ga-Rankuwa hospital' *S.A.M.J.* 86. 1536
2) Collaborative Eclampsia Trialists 1995 'Which anticonvulsant for women with eclampsia? Evidence from the Collaborative Eclampsia Trial' *Lancet* 345.1455
3) ECPPA Collaborative Group 1996 'ECPPA. Randomised trial of low dose aspirin for the prevention of maternal and foetal complications in high risk pregnant women' *Br J. Obs. Gyn.* 103: 39

ANTE-PARTUM HAEMORRHAGE

P.V. bleeding in the second half of pregnancy. Abruption 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 abruption, 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&Es, 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 abruption in a scarred uterus - always consider the possibility of 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 atonia or rupture or resistant coagulopathy.

Further Reading
1) AbdRabbo SA 1994 'Stepwise uterine devascularization: a novel technique for management of uncontrollable PPH with preservation of the uterus' *Am. J. Obst Gynae* 171:694
2) B-Lynch C et al. 1997 'The B-Lynch surgical technique for the control of massive post-partum haemorrhage: an alternative to hysterectomy? Five cases reported' *B. J. Obs Gyn.* 104:372
3) Heij HA 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 Obs.* 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 6hrly 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/Synto 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) Van Heerden J, Steyn DW 1996 'Management of premature rupture of membranes after 34 weeks gestation - early vs delayed induction of labour' *S.A.M.J.* 86:262
2) Owen J et al. 1993 'Randomized trial of prophylactic antibiotic therapy after pre-term amnion rupture' *Am. J. Obs Gyn.* 169:975

PRETERM LABOUR (WITH INTACT MEMBRANES)

- *Confirm the diagnosis* - 'pseudo-contractions' may result from U.T.I. True preterm labour may be precipitated by twins, abruption or chorioamnionitis. Do a P.V., diptest 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 tocolysis 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 titrated to the contractions and the maternal pulse (which must never exceed 120/min). Continue the infusion for 6 hours then reduce it to 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* - 6 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.

UTI

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 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 approachs 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 hrly for 14 days (much less effective)

Further Reading

1) Wilkinson D et al 1997 'Epidemiology of syphilis in pregnancy in rural South Africa. Opportunities for control' *Trop. Med. Int. Health* 2(1):57
2) Rolfs RT 1995 'Treatment of Syphilis 1993' *Chn.Inf. Dis.* 20 (Suppl 1): S23

ANAEMIA

- Admit if Hb<7 or <9 close to term
- Check stools for parasites and urine for bilharzia.
- Give ferrous sulphate 200 mg 8 hrly 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 <8 g prior to C/S)

POLYHYDRAMNIOS

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

- Check the blood glucose and Rhesus 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. *Gastroschisis* and *anencephaly* are also quite easy to visualise on US/S - these last three can be confirmed by a *maternal serum α-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 hrly* 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+cm when labour ensues. Examine the child carefully and always pass an n.g. tube to check that the oesophagus is patent.

Further Reading

Kirshon B et al 1990 'Indomethacin therapy in the treatment of symptomatic polyhydramnios' *Obs Gyn.* 75 (2):262

INTRAUTERINE DEATH

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

Induce if the patient requests this, except in the case of grandmultiparae 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-GTT). 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 doses to achieve a blood glucose <7 mmol/l at all times. Some mothers with mild diabetes can be managed on glibenclamide 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 of still 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 hrly - adjust the dose of insulin according to the

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

Further Reading
1) Coetzee EJ & Jackson WA 1984 'Oral hypoglycaemics in the first trimester and foetal outcome' *S.A.M.J.* 65(16):635
2) Kyos SL et al. 1993 'Insulin-requiring diabetes in pregnancy. a randomized trial of active induction of labour and expectant management' *Am J. Obs Gyn.* 169(3).611
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, HIV, Glucose. Assess the cervix.

All patients

- Folate 5 mg daily,vitamin C 100 mg, Slow Mg $^{2+}$ I 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

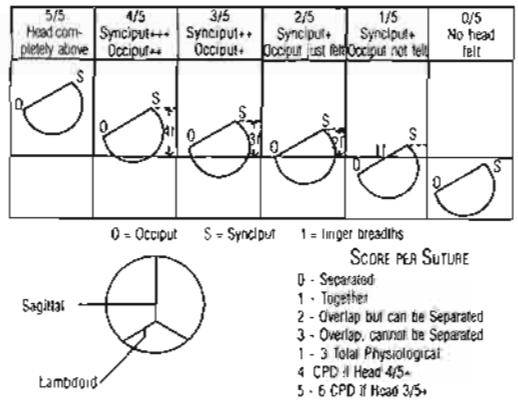
Review the antenatal and inpatient records before looking at the partogram. Take account of the antenatal risk factors and *confirm that the partogram is correctly filled in.*

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 *fifths palpable* per abdominum.

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 6) 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 11 cm long!) The *intertuberous 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 insurmountable *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) Lennox CE and Kwast BE 1995 'The partograph in community obstetrics' *Tropical Doctor* 25:56
2) Thacker SB et al. 1995 'Efficacy and safety of intrapartum electronic foetal monitoring: an update' *Obstet Gynaecol* 86:613
3) Khatree MH and Mokgokong ET 1979 'The significance of meconium staining of liquor amnii during labour' *S.A.M.J.* 56:1099

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 *C/S 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.

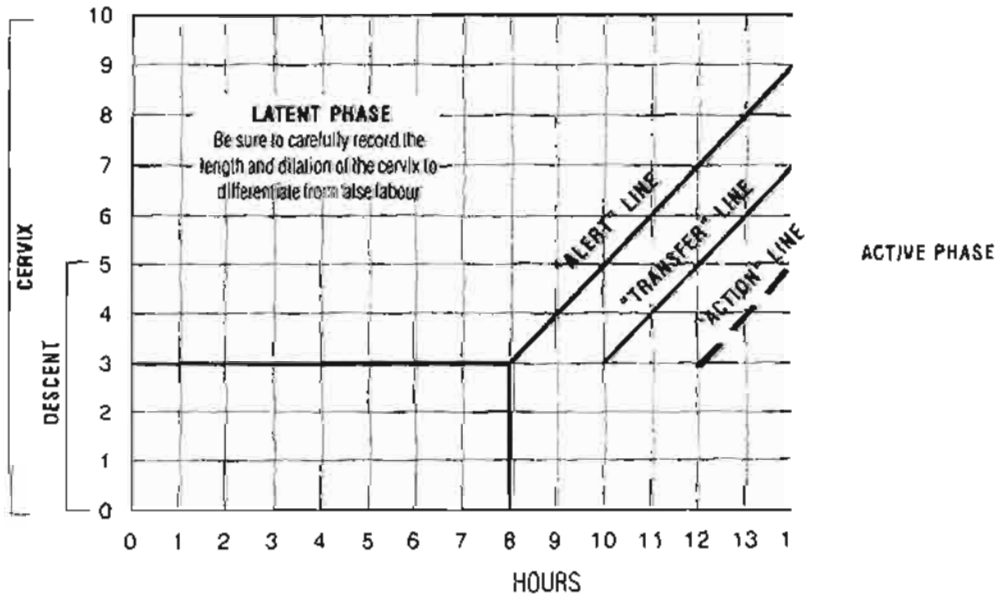
Be sure to examine the mother *2 hrly to 8 cm and hourly thereafter*. No mother should be allowed more than *thirty minutes in 2nd stage* before steps are taken to expedite the delivery

GUIDE TO MODE OF DELIVERY WITH CEPHALIC PRESENTATION AND LIVE FOETUS

Vertex

Station of head	Moulding Score	Foetal Distress	Mode of delivery
0	0-4	+/-	Vacuum
2/5	0-4	+/-	Vacuum
0-2/5	5-6	0	Trial of vacuum
0-2/5 & funnel pelvis	5-6	+	Symphisiotomy +/- VE
3/5 & head descends with contractions	0-3	0	Trial of vacuum
3/5 & no descent	0-3	+	C/S
3/5	4-6	+/-	C/S
4/5	1-6	+/-	C/S

Brow or face C/S



INDUCTION AND AUGMENTATION OF LABOUR

Never attempt to induce labour with Pgs or Syntocinon in *grande 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.

- PO/P1 - PgE[1] (Misoprostol) 50 µg (1/4 tablets) once daily
- P2/P3 - PgE[2] (Dinoprostone)1 mg (2 tablets) 8 hourly
- P4 - PgE[2] 0.5 mg 12 hourly

Repeat CTG 1-2 hours after insertion.

Once the cervix is *3 cm dilated and well-effaced*, Amniotomy + 2u/l Oxytocin infusion 10 -60 drops/min should be started.

Induction of labour in P5 and upwards may present problems - useful methods are nipple stimulation 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./ Malpresentation 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 Syntocinon 2u/l starting at 10 drops/min, increasing every 30 minutes to max 60 drops/min.
- Change to 10 u/l 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 Syntocinon 2u/l *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 hexoprenaline 5-10 mg slowly i.v. If foetal distress persists C/S will be necessary if vaginal delivery is not imminent.

Further Reading
Merrell DA et al. 1995 'Induction of labour with intravaginal misoprostol in the second and third trimesters of pregnancy' *S.A M.J* 85(10):1088

OTHER INTRAPARTUM PROBLEMS

BREECH

We attempt *ECV at term* for all breechs. Vaginal delivery for all singleton breechs 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 almost all singleton breechs*, preferably electively.

However, you may be called upon to deliver a *fait accompli breech in labour* or one of *twins*, or the uncommon case of a *multip with a well-tried pelvis* having an elective vaginal delivery. Progress must be *perfect* and augmentation is never appropriate. Familiarise yourself with the mechanism of breech delivery, especially Lovset's and Mauriceau-Smellie-Veit manoeuvres.

TWINS

The big cause of perinatal mortality with twins is *premature labour*. Even term twins are smaller than singletons, and *trial of labour is appropriate even with a scarred uterus*, if the first twin is cephalic (85% of cases). Breechs 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.

Syntocinon 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 lie/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 20 u/l Syntocinon 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 footling 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. *CPD 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 Manoeuvre* - 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 Syntometrine 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/l, 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 20u/l*
- *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' speculae handy. Exclude

- *retained products* (remove manually or with blunt currette/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* (sulure)
- *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 foetal 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 breast-feeding.

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 hrly 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 caesarian 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 risks of transmission probably outweigh the benefits conferred. Some mothers who have secure 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

1) Leroy V et al. 1998 'International multicentre pooled analysis of late postnatal mother-to-child transmission of HIV-1 infection' *Lancet* 352:597

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 effects of vitamin supplements on pregnancy outcomes and T cell counts in HIV-1 infected women in Tanzania' *Lancet* 351:1477

4) Bobat R et al. 1996 'Determinants of mother-to-child transmission of human immunodeficiency virus type 1 infection in a cohort from Durban, South Africa' *Paed. Inf. Dis. Journal* 15 604

5) Biggar RJ et al 1996 'Perinatal intervention trial in Africa: effect of a birth canal cleansing intervention to prevent HIV transmission' *Lancet* 347 : 1647

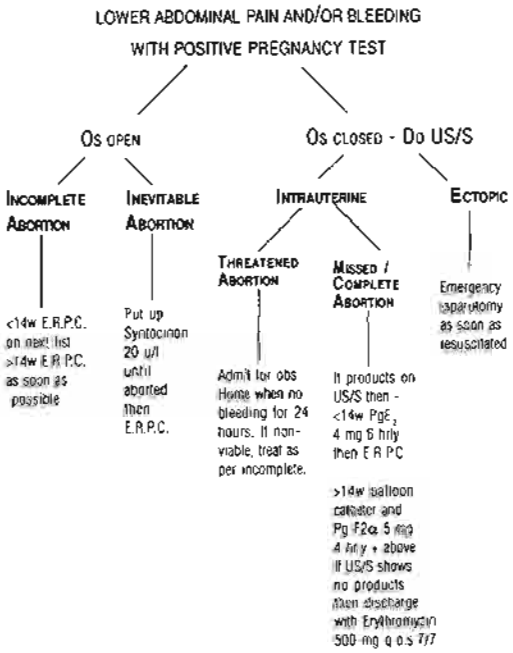
ABORTION

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 1996 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 & Flagyl 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.



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

ECTOPIC PREGNANCY

A positive pregancy 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 lucent 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 Ngwelezane but we have only done one unnecessary laparotomy in the last four years using β-HCG and US/S findings alone

Further Reading
1) Rees H et al. 1997 'The epidemiology of incomplete abortion in South Africa' *S.A.M.J.* 87:432
2) De Jonge E.T.M 1994 'Is ward evacuation for uncomplicated incomplete abortion under systemic analgesia safe and effective?' *S.A.M.J.* 84:481
3) Pedersen JF 1980 'Ultrasonic scanning in ectopic pregnancy' *B.J. Radiology* 53:625

AMENORRHOEA

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

1° 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.

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 galactorrhoea. 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 galactorrhoea. If the patient is concerned about her fertility, also arrange a provera test at the first visit. Give 5 mg t.d.s. for 5 days. A withdrawl bleed signifies a well-oestrogenised endometrium and probable supra-ovarian cause.

In patients with frank galactorrhoea, 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 Prempak-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
1) Delvoye P 1980 'Breastfeeding and post-partum amenorrhoea in central Africa 2 Prolactin and post-partum amenorrhoea' *J. Trop. Paeds.* 26:184
2) Crosignani RG 1996 'A practical guide to the diagnosis and management of amenorrhoea' *Drugs* 52:671
3) Gray RH 1981 'Vaginal bleeding disturbances associated with the discontinuation of long-acting injectable contraceptives' Br J. Obs. Gyn. 88:317

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 Ngwz show evidence of tubal damage on HSG and ~40% are found to be anovulatory (some women have both problems). Only ~5% 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 *migrant 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 volume1-4ml. 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/ Day21 progesterone + TFTs (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* 65:854

3) Desgrees De Lou A et al. 1999 'Impaired fertility in HIV-1 infected pregnant women : a clinic-based survey in Abidjan' *Cote d'Ivoire AIDS* 13:517

4) Chigumadzi PT et al. 1998 'Infertility profile at King Edward VIII Hospital , Durban , South Africa' *Trop. Doc.* 28:168

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 florid 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 >10,0*
- *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 t.d.s. for 1 week

SEVERE CASES

(defined as *Fever >38°C, Pulse >100, peritonism or tender adnexal mass*)

- Ampicillin 1g 6-hourly i.v.
- Metronidazole 1g b.d. p.r.
- 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 Gynaecol.* 61 (1) 113

2) Landers DV and Sweet RL 1985 'Current trends in the diagnosis and treatment of tubo-ovarian abscess' *Am J. Obstet. Gynaecol.* 151:1098

3) Kamenga et al 1995 'The impact of HIV infection on Pelvic inflammatory disease: a case-control study in Abidjan, Ivory Coast' *Am J. Obs Gynae.* 172(3).919

4) Dodgson MG 1994 'Antibiotic regimens for treating acute Pelvic Inflammatory Disease : an evaluation' *J. of Reprod. Med.* 39:285

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 35s would be required, and we are already struggling to implement effective primary health care 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-caulery and podaphyllin or beladine paint. 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 *atypical lesions should always be biopsied*.

The preferred treatment for women is *electro-caulery*. 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 O.P.D./on ward. No more than 0.5 ml should be used at a single application and it should not be left on for longer than 6 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 *secondarily infected* growths are seen. Treatment is of the secondary infection (Flagyl 2g stat + Erythromycin 500mg qds 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 squamo-columnar junctions at different sites.) The only important differential of a hard, deformed cervix with contact bleeding is a bilharzioma which may respond to praziquantel and is uncommon.

- *CIN 1* - treat for PID 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 VIII (Gynae Oncology Clinic) for assessment. Do not send cases with renal tract involvement or evident distant metastases.

Further Reading

1) Heysteck MJ et al. 1995 'Screening for cervical neoplasia in Mamelodi - lessons from an unscreened population' *S.A.M.J.* 85 1180

2) McCoy D and Barron P 1996 'Cytological screening for cervical cancer - what are the opportunity costs?' (Opinion) *S.A.M.J.* 86:935

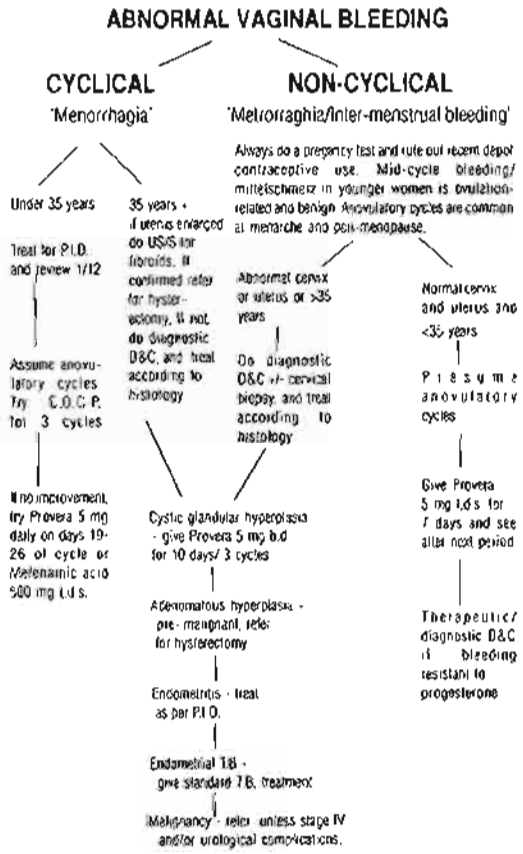
3) Stone K et al 1990 'Treatment of external genital warts: a randomized clinical trial comparing podophyllin, cryotherapy and electrodessication' *Genitourin. Med.* 66:16

4) O'Farrell N 1989 'Sexually transmitted pathogens in pregnant women in a rural South African community' *Genitourinary Medicine* 65:276

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 *endometriosis is rare*. Incidence rates for *Carcinoma of the cervix* in black women in South Africa are among the highest in the world (43+ per 10^5 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.



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 gynae examination.

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

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 Oestradiol 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 *Norethisterone 5-10 mg t.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. After that check the urine every 3 months for 2 years.

Further Reading

1) Bayer S and DeCherney AH 1993 'Clinical manifestations and therapy of dysfunctional uterine bleeding' *JAMA* 269:1823
2) Hall et al. 1987 'Control of menorrhagia by the cyclo-oxygenase inhibitors naproxen sodium and mefenamic acid' *Br. J. Obs. Gyn.* 94:554
3) Luiz DA et al. 1986 'Aetiology of postmenopausal bleeding in black women in Durban' *S.A.M.J.* 69:674

FAMILY PLANNING

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 conceive in the first place.

FAMILY PLANNING FOR WOMEN

INJECTABLE CONTRACEPTIVES

We routinely use Medroxyprogesterone acetate ("Depo-Provera") or Norethisterone Oenanthate ("Noristerat") 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 Noristerat 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. Heavy pv bleeding can be treated with Ethinyloestradiol 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.
2. Slow return to fertility (average is 8 months after the last injection).
3. 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.

Triphasil® or Nordette® are combined oral contraceptive pills suitable for non-lactating non-smoking women up to the menopause with no history of hypertension, 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 unreceptive 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.thrombo-embolism,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 T.Ls 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 Metoclopramide 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 : norethisterone enanthate given in the dosage regimens and depot Medroxyprogesterone acetate. Final report' *Contraception* 28:1

2) Buga G.A et al 1996 'Sexual behaviour, contraceptive practice and reproductive health among school adolescents in rural Transkei' *S.A.M.J.* 86:523

3) Peterson H B. et al 1996 'The risk of pregnancy after tubal sterilization: findings from the U.S. Collaborative Review of Sterilization' *Am. J. Obs. Gyn* 174 : 1161

4) Wang CC et al. 1999 'Risk of HIV infection in oral contraceptive pill users: a meta-analysis' *J. Acquir. Immune Defic. Syndr.* 21:428

SURGERY

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), which is 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/toileting 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 (esp. 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 ragged 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 underculling of tissues to acheive 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 fibrinous crust and a *dry gauze dressing* for the first few days is usually all that is required. *A clean wound should be disturbed 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 (tulle 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-epithelisation.

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% aqeous 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.

Aserbine 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/castor 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) +/- 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
1) Singer AJ 1997 'Evaluation and management of traumatic lacerations' *NEJM* 337:1142
2) Thomas S 1990 *Wound Management and dressings* London: Pharmaceutical Press
3) Ryan TJ 1997 'Global curriculum for wound management' *Tropical Doctor* 27 Suppl 1: 31
4) Brown RP 1992 'Knotting technique and suture materials' *Br. J. Surg* 79:399
5) Tera H & Aberg C 1975 'Tensile 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 election *followed by a below-knee revision* results in a subsequent above-knee ampulation 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 unfavourable! 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 energy 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 stabilised 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, Metro) within 12-24 hours must be *urgently debrided* because of the often unsuspected extent of the underlying infection - if a foot is left to suppurate 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, Klebsielia*, coliforms), *Streptococci* and anaerobes (mostly bacteroides and clostridia). Intravenous antibiotics should be continued for at least *48-72 hours after defervescence* 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 amputees may 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 stump-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.faecalis*, and anaerobes including *B.fragilis* and *clostridia*. In chronic cases, consider referral for a 'revascularisation/ampulation' at KEH which may acheive a better functional level.

'VROT-FOOT'

This is an *idiopathic neurotrophic foot* invariably found in *males <50 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
1) Robbs JV 1993 'Simple surgical audit - benefits and spin-offs of un-esoteric research' *S.A.J Surgery* 31 2 51
2) Huizinga et al 1983 'Prevention of wound sepsis in amputations' *S A M.J.* 63:71
3) Pollock and Ernst 1993 'Use of doppler pressure measurements in predicting success in amputation of the leg' *Am J. Surg* 139 303
4) Dagogo-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 feet in blacks' *S.A.M.J.* 49: 2157

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.

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. Comminution / 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 flap)
- 3C - associated arterial injury (requiring repair for limb preservation)

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 Gde. 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 *toileted 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 II 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 ooze 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 Ngwelezane within the first 48 hours so that external fixator/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

then be closed by suture or grafting. The wound that remains sloughy for days and cannot be closed in this way has usually not been adequately debrided - secondary infection and necrosis has taken hold.

All Grade III injuries should be referred as an emergency to Ngwelezane 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-kneebackslab for tibial #s
- Perkin's traction for femoral #s
- Narrow, mid-arm sling or light U-slab for humerus #s
- closed methods for forearms (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 1994 'Management of the open fracture - ten commandments' *Trauma and Emergency Medicine* 11.1 pp.963

2) Gustilo RB, Anderson JT 1976 'Prevention of infection in the treatment of 1025 open fractures and long bones: retrospective and prospective analysis' *AM. J. Bone Joint Surgery* 72.229

3)Antrum RM and Solomkin JS 1987 'A review of antibiotic prophylaxis for open fractures' *Ortho. Rev.* 16:248

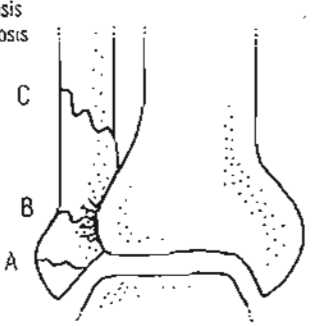
4) De Wind CM 1999 'Management of open tibial fractures' *Trop. Doc.* 29:46

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 1 mm shift implies 40+% loss of joint congruity.Examine the joint carefully for instability. If bony tenderness is absent from the posterior 6 cm of tibia and fibula a fracture is very unlikely and X-ray unnecessary. If a # is present clinically, order 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.

Weber Classification
A. Below level of Syndesmosis
B. At the level of Syndesmosis
C. Above level of Syndesmosis



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. Temporary admission may be required for 48-72 hours for elevation of the leg and mobilisation.

All other #s should be admitted. A backslab or Quigley traction should be applied and the limb elevated for 24-48 hours (this doesn't apply to clinically dislocated ankles which should be reduced IMMEDIATELY).

80-90% can be managed by closed methods. Reduction should be accurate using three point fixation and check X-rays taken post-reduction and weekly for two weeks. If the 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-III 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) Lindsjo U 1985 'Classification of Ankle fractures: the Lauge-Hansen or AO systems?' *Clin Orth.* 199:12

2) Stiell et al 1995 'Multicentre trial to introduce the Ottawa ankle rules for use of radiography in acute ankle injuries' *B.M.J.* 311 594

3) Bewes PC 1995 'The management of ankle fractures' *Trop.Doc.* 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 a *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 Ngwelezane after their primary debridement. Patients with *Gde II- 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 the tibia. 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) Dehne and Nitz 1961 'The treatment of fractures by direct weight-bearing' *J Trauma* 1.514
2) Laushcke FH and Shangula K 1995 'Assessment and management of common tibial injuries in a district hospital' *Trop. 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.

HAEMARTHROSIS

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 unanaesthetised 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 *valgus/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 analgesia/sedation*. *Grade II/III injuries* should be managed in a *'stove – pipe' P.O.P. at 30° flexion for 6-8 weeks*. *A*

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 stove-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 stove - 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 McMurrays 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 *MRJ 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 meniscectomy* (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 *stove-pipe P.O.P. at 90° flexion for 4 weeks*.

Further Reading
1) Mitsou A and Vallianatos P 1988 'Clinical diagnosis of ruptures of the anterior cruciate ligament, a comparison between the Lachman test and the Anterior drawer sign' *Injury* 19:427
2) Wilson-Macdonald J et al. 1990 'Arthroscopy in acute knee injuries - a prospective clinical trial' *Injury* 21:165

FEMUR FRACTURES

NECK FRACTURES

Most helpfully 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 (?~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 usefully 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 iliopsoas 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 fit 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, unflexed supracondylars in young patients, unrotated unicondylar 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 unicondylar #s, the patient should be referred to Ngwelezane for internal fixation with an intramedullary nail inserted via the knee joint.

Further Reading
1) Ingle R and Ingle P 1996 'The functional management of femoral shaft fractures' *S.A. Family Practice* 17.9 406
2) Bewes P 1974 'Fractures of the femur in a tropical context : a revaluation of Perkin's traction' *Trop Doc* 4:64

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 DJJ 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:

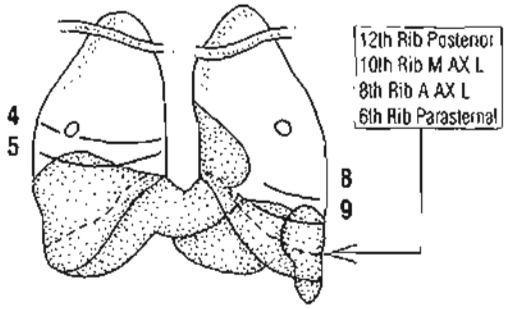
- immediate *needle thoracostomy* for *tension pneumothorax*
- an *occlusive dressing* taped on three sides for *sucking/blowing wounds*
- *splinting of flail segments*/placing the patient with the flail side up
- control any severe external source of *haemorrhage* e.g. intercostal artery

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 and # 1st rib is often associated with major neurovascular /tracheobronchial injuries. # of 7 or more ribs carries a risk of intrathoracic 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.

Surface Marking of the Pleura



emphysema) or the patient deteriorates. *Intercostal tube drainage is indicated for all larger pneumothoraces*, after any needle thoracostomy and in all blunt chest injuries. *Do not suture the chest wound before inserting a drain* unless you are sure there is no pleural penetration clinically or the CXR shows no significant pneumothorax. The tube should remain until there is no leak for 24 hours - persistent leakage unresponsive to gentle suction (-15 cm water) on the tube may signify a tracheo-bronchial injury or pulmonary laceration and the patient should be referred for thoracotomy.

HAEMOTHORAX

Physical findings may be equivocal but the diagnosis can usually be confirmed by *aspiration and erect CXR* (<1000 ml can be missed on a supine film, <200 ml on an erect). Intercostal drainage helps to tamponade the bleeding and enables monitoring of blood loss - use the largest tube that you can (>24F usually). A *massive haemothorax is defined as >300 ml/hr for 3 hours* and is usually associated with injury to a smaller systemic artery, major vessels or heart. Thoracotomy is required. This may also be necessary if the haemothorax clots and cannot be evacuated by tube after 7 days drainage.

FLAIL CHEST

Usually double #s of 3+ ribs anterolaterally resulting in *paradoxical chest movement* and in some cases rapidly deteriorating ventilation due to chest pain, underlying lung damage and possibly 'pendeluft'. In the initial stages, *the flail segment should be stabilised* by splinting with sandbags, strong *analgesia* provided and *respiration monitored* clinically and by pulse oximetry. Intercostal nerve block may be useful. However, many flail segments are associated with an underlying pulmonary contusion and progressive hypoxia - positive pressure *ventilation may be required* in severe cases and *early transfer is wise.*

PULMONARY CONTUSION

Often the result of a deceleration injury or high velocity gunshot wound (spalling[splintering]/inertia/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+% 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 temporising effect of cardiac tamponade. *Pericardiocentesis is a temporary measure and emergency cardiorapphy 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 Durban. *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
1) Demetriades D et al. 1988 'The management of penetrating injuries of the back. A prospective study of 230 patients' *Ann. Surg.* 207.72
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 scaphoid 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, aortography, bronchoscopy) and repair.

Further Reading
1) Jones KW 1980 'Thoracic trauma' *Surgical Clinics of N. America* 60:4 pp. 957
2) Graham JM 1979 'Penetrating Trauma of the Lung' *J of Trauma* 19:665
3) Muckart DJJ 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+%) 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 esp. 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, put 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
1) Huizinga et al. 1987 'Selective management of abdominal and thoracic stab wounds with established peritoneal penetration: the eviscerated omentum' *Am.J.Surg.* 153:564
2) Demetriades D and Rabinowitz B 1984 'Selective conservative management of penetrating abdominal wounds: a prospective study' *Br.J.Surg* 71(2):91
3) Coupland RS 1996 Review: 'Abdominal wounds in war' *B.J.Surg* 83:1505

PELVIC INJURIES

These are generally *high energy injuries* resulting from a tractor or car accident, though isolated pubic ramus #s may occur in the elderly after a fall. Mortality is high and may approach 50 % for 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 tamponaded by the soft tissues. Look for *buttock/perineal/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 / lower limbs. 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 lelt/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 urethrogram* – inject (Omnipaque 5-10 mls, mixed with KY jelly if necessary to increase viscosity) either via syringe and fingers 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 <1 cm displacement)
B. *Rotationally unstable but vertically stable* (Posterior Sacro-iliac ligaments intact)
   - anteroposterior 'open book #'
   - Lateral 'closed book #'
C. *Rotationally and vertically unstable* (Posterior Sacro-iliac ligaments disrupted)
   - unilateral
   - bilateral
   - with associated acetabular #

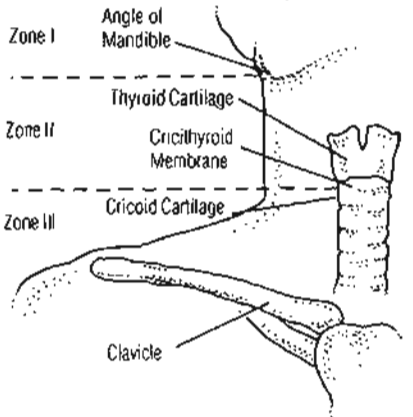
Management

- *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) Tile M 1988 'Pelvic ring fractures - should they be fixed?' *J. Bone & Joint Surgery* 70B:1
2) Trafton PG 1990 'Pelvic ring injuries' *Surgical clinics of N. America* 70:655
3) Cass AS 1984 'Urethral injury in the multiply injured patient' *J. Trauma* 24:901

PENETRATING NECK INJURIES

The neck can be divided anatomically into *three zones*.



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 pelusion 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 haemopneumathorax (try to include the neck right up to the mandible to see the upper airway sillhouette).

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/haematemesis.

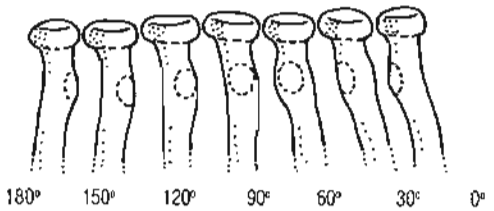
All other patients, including those whose haemorrhage has stopped, those with non-progressive surgical emphysema and those with isolated nerve/brachial plexus injuries should be *admitted*. Arteriography may be indicated for zone I and III injuries esp. if the mediastinum is widened or there is a bruit in ahaemodynamically stable patient. A short period of observation will help to eliminate the possibilty 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) Asensio et al 1991 'Management of penetrating neck injuries' *Surgical clinics of N. America* 71:267
2) Demetriades D 1985 'Penetrating injuries of the neck' *Ann. R.C.S. Eng* 67 71

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/slackening of the cast is a constant problem. However, in children, if a reasonably accurate reduction can be achieved, the # will always unite and remodelling may be able to improve any residual deformity. However, this process is most efficient in the metaphysis/epiphyseal 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.



ROTATION OF THE RADIAL TUBERCLE

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 inferior radio-ulnar joint to exclude Monteggia or Galleazi #/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 screening 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 epiphyseal 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 fending 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

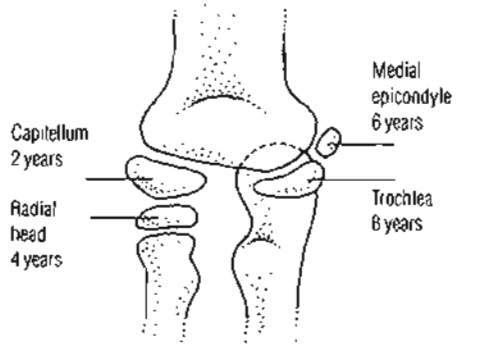
Usually a # of the distal 1/3 with complete off-ending and the proximal fragment pulled into mid-pronation by pronator teres. Check for an associated dislocation of the inferior radio-ulnar joint (ulnar head distal to radial).#s higher up the radial shaft are associated with varying degrees of supination 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 hitches. 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 Ngwelezane - they may continue conservative management in a straighter AE POP with 30° of elbow flexion but internal fixation is often carried out.

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 EPIPHYSES 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 Ngwelezane for the third attempt within the first week. Lateral and apical 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.*

Even if the radial pulse does not return immediately after the reduction, if there are no signs of ischaemia it will usually return within 2-3 days. However, the patient should be closely monitored and if ischaemia develops the backslab should be opened, the elbow extended and if there is no improvement the brachial artery must be explored.Remember that reversed supracondylar fractures should not be reduced in the same way as the ordinary type Supracondylar #s in adults are frequently T-shaped or more severely comminuted. If so, use a collar and cuff but refer within the first week for assessment at Orthopaedic clinic as internal fixation may be required.

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 repulation, 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° flexion* but it is essential to review at one week since further displacement can occur. *>2mm 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 - *>5mm 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° 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 - if it is the interosseous membrane is completely torn and the # very unstable (Essex-Lopresti #/ dislocation). *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 Ngwz.

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
1) Minkowitz B 1994 'Supracondylar humerus fractures. Current trends and controversies' *Orth Clin N. America* 25 581
2) Wilson NIL et al. 1988 'Treatment of fractures of the medial epicondyle of the humerus' *Injury* 19 342
3) Bewes PC 1989 'Supracondylar fractures in children' *Trop. Doc.* 19:172

UPPER ARM FRACTURES

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 (appropriate in 90+%) is a broad arm-sling (impacted) 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 IIIs) and Fracture -dislocations (Neer VIs) - 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
Spak I 1978 'Humeral shaft fractures. Treatment with a simple hand sling' *Acta. Orth. Scand.* 49:234

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 on 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(decerebrate)	2
None	1

Best verbal response

orientated	5
disorientated 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, haemotympanum, 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 hrly 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 concious 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

Resusicate and re-evaluate before consultation.

- Shocked and/or hypoxic patients
- Patients with an initial *GCS of 3-4 from blunt trauma* (probably have irremediable 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 indriven bone fragments
- GCS 15 and localising signs (usually a cerebral contusion, often following a knobkierrie 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 could not be immediately arranged (e.g. during the night).

Further Reading
1) Mendelow et al. 1983 'Risks of intracranial haematoma in head-injured adults' *B.M.J.* 287:1173
2) Wilkinson HA and Rosenfeld SR 1983 'Furosemide and Mannitol in the treatment of acute experimental intracranial hypertension' *Neurosurgery* 12 405
3) Bullock MRR and Du Trevou MD 1988 'Prevention of death from head injury in Natal' *S.A.M.J.* 73:523
4) Jager JY 1990 'Coma scales in paediatric practice' *Am J. Dis Child* 144 (10):1088

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 approachs 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 /or a burst #).
- Isolated *transverse or spinous 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 in 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 sterno-costal 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 para/quadriplegia, neurogenic shock and an atonic bladder. +ve 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 -5 sensory dermatomes and toe 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* = quadriparesis + mild spinothalamic impairment and preserved proprioception. *Posterior cord syndrome* = dysaesthesia in neck/arms/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 I odontoid peg #s) may be managed as an outpatient in a hard collar for ~ 6 weeks. Minimally displaced Jefferson (blow-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/analgesia + 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 acheived 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 of her own pressure areas as soon as possible and this should become a lifelong habit. Quadriplegics 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 hrly 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/labia. 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 intertermittent 'semi-sterile' (!) catheterisation is taught. The patient should self-catheterise 6-8 hrly with a Jacques catheter lubricated with soap and water, and either boil 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). Quadriplegics will require a long-term urethral catheter.
- Stool softener (liquid paraffin +/- glycerine suppositories) with intermittent manual evacuation as required.
- Sucutaneous heparin 5,000 u. 8 hrly.

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

Further Reading

1) Bracken MB et al. 1992 'Methylprednisolone or naloxone treatment after acute spinal cord injury : 1 year follow-up data. Results of the second National Acute Spinal Cord Injury Study' *J. Neurosurg* 76 1-23

2) Bewes PC 1978 'Management of traumatic paraplegia' *Trop.Doc* 8 187

2) Hill VB & Davies WE 1988 'A swing to intermittent clean self-catheterization as a preferred mode of management of the neuropathic bladder for the dextrous spinal cord patient' *Paraplegia* 26:405

3) Fisk N 1985 'Traumatic paraplegia in Northern Tanzania' *Trop. Doc.* 15:23

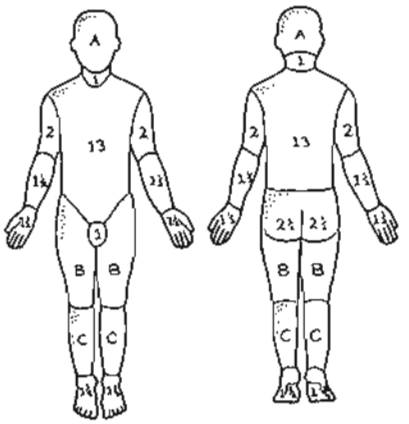
BURNS

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/food. 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 Hlabisa, many of those with a prolonged hospital stay, repealed 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, hyperaemic, 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



Age	<1	1 - 5	5	10
Head	10 %	9%	7%	6%
Thigh	3%	3%	4%	5%
Leg	2%	3%	3%	3%

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 8 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 is <0.5 ml/kg/hr.*

Give *Morphine 0.2 mg /kg i.v. 4-6 hrly* titrated to analgesic effect. *Tilidine drops (1 drop per year of age - max. 10 drops) given sublingually 3-4 x /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 Gentamicin should however be given if the patient becomes systemically unwell during this period (possible early β-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 escharotomy* - 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 in sterile conditions. Subsequent infection should be treated with topical 0.05% Chlorhexidine solution. Silver Sulphadiazine 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 Clairwood 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 Aserbine 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 desloughed and not over-granulated) and the patient is not anaemic.

Burns >50% have a very poor prognosis and palliative care is most appropriate. *Patients with 15-50% burns should be discussed with Ngwelezane/King Edward Hospital.* Ensure that the patient is adequately resuscitated prior to transfer.

Further Reading
1) Chopra M et al. 1997 'Paediatric burns in a rural South African district hospital' *S.A.M.J* 87:600
2) Sowemimo GO 1993 'Burns care in Africa: reducing the misery index' *J. Burns Care and Rehab* 14:589

LIGHTNING INJURIES

These are *super high voltage* (10×10^7V) D.C. discharges of a very brief duration. Fortunately the extremely high current ($\underline{<3} \times 10^5$ A) generated usually 'flashes 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-T changes will be evident on ECG in 10-30% and arrythmias may occur - *admission for 24 hours for observation* is recommended.

Burns are typically superficial with a splashed-on appearance ('Liechtenberg'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 diptested 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
1)Castle WM and Kreft J 1974' A survey of deaths in Rhodesia caused by lightning' *Cent. Afr. Med J.* 20 93
2) Cherington M 1995 'Lightning injuries' *Ann. Emerg. Med* 25:517

UROLOGY

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 operation is considered.
4. *Less clearly specified sexual/cultural reasons*. Though ritual circumcision was abolished in the Shakan 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 hydrocoeles 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 *hydrocoelectomy* (younger), or adhesis can be carried out (instil 250 mg Tetracycline in 15 mls of N/Saline + 5 ml Lignocaine 2% into the hydrocoele).

In infants, *congenital hydrocoele* due to a patent processus vaginalis usually subsides spontaneously and only requires *herniotomy 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 torted 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 10^5/year (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&Es 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 urethroplasty* 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* (PSA <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 *Cyproterone acetate 100mg t.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 Cr). 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 ZB 1997 'A pathological analysis of clinical adenocarcinoma of the prostate in black patients' (letter) *S.A.M.J.* 87:344
3) Partin and Oesterling 1994 ' The clinical usefulness of Prostate specific antigen : update 1994' *J. Urology* 152 1358
4) Urassa M et al 1997 'Male circumcision and susceptibility to H.I.V 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 trangressing 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 (PIPJ free to move, DIPJ 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.

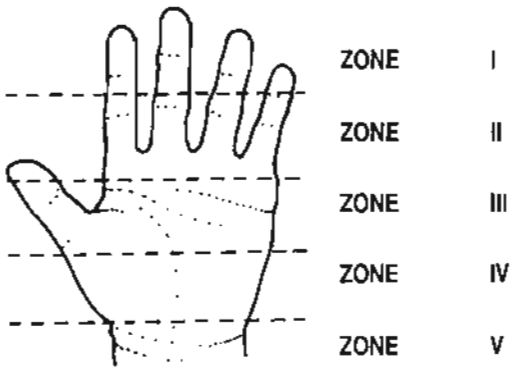
Dislocated fingers usually result from extreme hyperextension and must be urgently reduced. Splint MCP joints flexed and IP joints extended if the reduction is unstable. Neighbour strapping and early active movements can be started if stable. MCP dislocations frequently 'button-hole' through the volar 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 *bushknife 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).

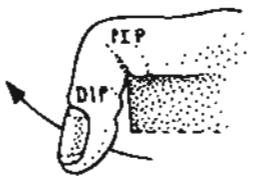
Extensors - *E.digitorum communis* is supplemented by *E.indicis proprius* and *E. digiti minimi* but repair of the last two is not essential so almost all repairs on the dorsum of the hand involve *one layer* of tendons. Since they have a common muscle bulk the tendons tend not to retract too far. Be 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.



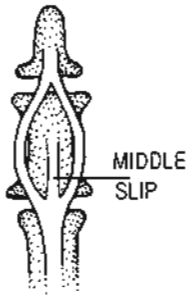
Flexors - Zone I repairs (distal to the proximal IP crease) are distal to the insertion of *F.digitorum superficialis* and only involve the *profundus* tendon. Always check that *superficialis* is intact by doing *Apley's test*-anchor the other fingers flat on the table and ask the patient to flex the 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

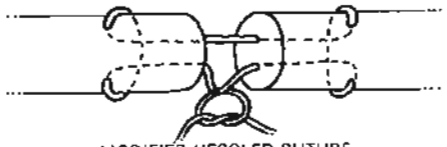
STEADY THE PHALANX



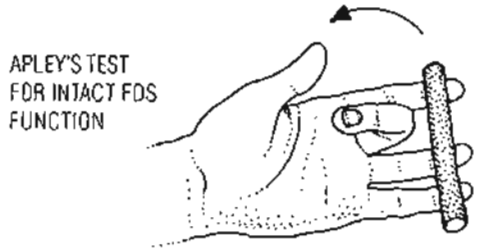
ELSONS MIDDLE SLIP TEST
If the middle slip is ruptured, there is no extension of the P/P joint and the D/P joint stiffens and extends a little.

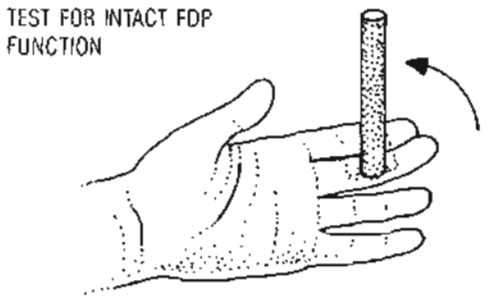


3 SLIPS OF THE EXTENSOR EXPANSION.



MODIFIED KESSLER SUTURE





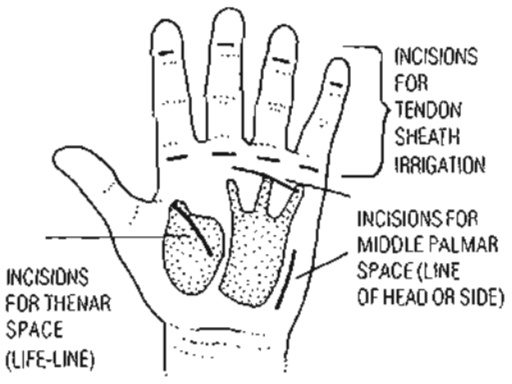
preferably are done as a primary repair since the tendons in this area rapidly retract. Remember however that there are two layers of tendons – if it appears that both are interrupted the patient is better referred to a skilled surgeon.

Flexor repairs in other zones should be referred - the results of delayed primary suture are no worse than primary suture, so toilet the wound and refer to Ngwelezane within 48 hours.

INFECTIONS

Pulpspace infections of the fingers compromise the bloodsupply and quickly lead to osteomyelitis of the distal phalanx - they should be urgently drained through a small diamond-shaped incision of the pad

Paro/eponychiae - can often be managed by simple incision and drainage +/- lifting the cuticle around the nail. However,may require removal of the lateral/proximal nail respectively to ensure adequate drainage in advanced cases.



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 *Kanavel's signs* 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 Cloxacillin 1g 6 hrly i.v., Gentamicin 240 mg o.d. i.v. + Metronidazole 1g b.d. p.r. in 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° dorsi-flexion 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) Mennen U & Howells CJ 1991 'Human fight-bite injuries of the hand. A study of 100 cases within 18 months' *J. Hand Surgery* 16:431

2) Neviaser RJ 1978 'Closed tendon sheath irrigation for pyogenic flexor tenosynovitis' *J. Hand Surgery* 3:462

3) Lindsay JR 1996 '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

SNAKEBITE

Incidence ~80 per 10^5/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* (*iBobosha, iBululu*) - severe cytotoxicity sometimes progressing to haematological effects especially thrombocytopaenia
- *Mozambique spitting cobra* (*uMfezi*) - similar to the above and may also cause snake venom opthalmia.
- *Black and green mambas* (*iMamba*) - 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 (isiBiba) administered. Almost half 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 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,ptosis, 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 - paracelamol +/- 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 delined 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 /yr 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 edrophonium 10 mg (adult) or 0.25 mg/kg (child) in order to establish whether neuromuscular blockade is competitive - if improvement can be demonstrated it may be possible to maintain the patient on Neostigmine 0.05-0.1 mg/kg 4 hourly 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 debridement /exploration is best delayed until 5 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 denuded and subsequently necrose. Fasciotomy 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 splinting 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 anti-venom with a rash, arthralgia and fever. It should respond to a short course of prednisolone.

Further Reading
1) Wilkinson D 1994 'Retrospective analysis of snakebite at a rural hospital in Zululand' *S.A.M.J* 84:844
2) Moran NF et al 1998 'High incidence of early anaphylactoid reaction to SAIMR polyvalent snake antivenom' *T R S T.M.H.* 92:69
3) Newman WJ et al 1997 'Traditional therapy for snake bite in a rural African community' *Ann. Trop. Med. and Parsitol.* 91:967
4)Premawardhena AP 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

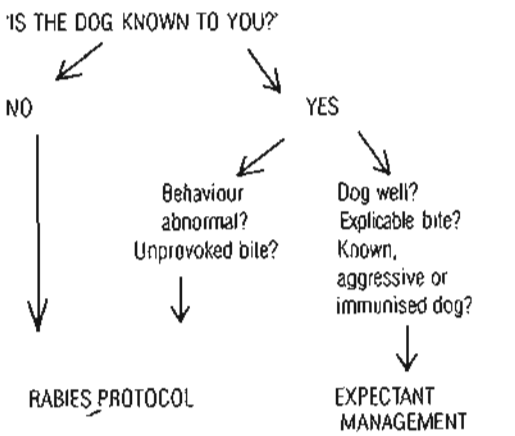
DOG BITE

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 sylvatic 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 proven rabid animal is ~ 50% overall. Most bites occur in children often when passing/entering a neighbour's kraal. Treat a comment 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 have been only twenty 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 managment of dogbites! 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 -



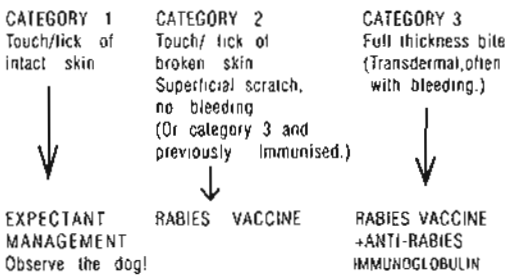
Wound Management (for all patients)

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 *tetanus toxoid* where appropriate. *Antibiotics* - Give prophylactic Ampicillin and Cloxacillin for 3-5 days if the wound is clinically contaminated or the 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 -



- **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 total (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+% 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 humans. *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 decline consent authority may be sought from the magistrate or medical superintendent under the *Human Tissue Act of 1983.*

For further information,
or for help concerning a case, phone:
Pasteur Merieux Rabies Hotline KwaZulu-Natal
☎ 031 3603111

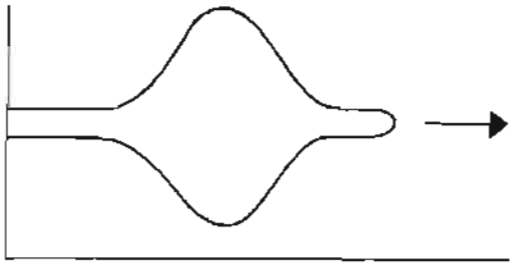
Further Reading
1) Department of Health 1997 *Guidelines for the medical management of Rabies in South Africa*
2) Swanepoel R 1993 'Rabies in South Africa' *Ondersterpoort J.Vet. Res.* 60(4):325
3) Cleveland S 1998 'The growing problem of rabies in Africa' *T.R.S.T.M.H.* 92:131

GUNSHOT WOUNDS

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 C D E

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 i.v.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. 12/89 302-309
2) Swan and Swan 1991 'Principles of ballistics applicable to the treatment of gunshot wounds' *Surgical Clinics of N. America* 71:2 221
3) Coupland R 'War wounds of limbs' *I.C.R.C.*

BONE AND JOINT SEPSIS

Septic arthritis usually presents as an acute monoarthritis of a weight-bearing joint (80% lower limb - knee commonest site in adults, hip>knee>ankle in children). *S. Aureus* 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. H.I.V. +ve patients often have atypical pathogens especially *S. pneumoniae* and Gram negative organisms. Polyarticular involvement may accompany a staph/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 sub periosteal abscess. This interrupts the blood supply from the periosteum and infarcts 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 *gcaba marks* may already have been made by a traditional healer - do not assume that it is these that have caused the inflammation! Important differentials of acute monarthritis in adults are crystal synovitis and reactive arthropathy. In children be on the look-out for Rheumatic fever (classically migratory) and Perthe's 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! Fluctuance 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 G A. 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).

	NORMAL	INFLAMMATORY	SEPTIC
Volume	3.5 ml	>3.5 ml	>3.5 ml
Viscosity	High	Low	Variable
Colour	Clear	Yellow, translucent	Yellow-green, turbid
WBC/mm	<200	2-10 x 10^3	>10 x 10^3
PNMLs	<25%	>50%	>75%
Glucose	= serum	>25 mg/dl	<25 mg/dl
Gram stain	Negative	Negative	Positive

Management

1) *Analgesia, Splintage* (skin traction for hips)
2) Give *Cloxacillin 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. *Substitute Ampicillin for erythromycin in septic arthritis*. *Ceftriaxone* 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 arthrotomy* - needle

*Joint fluid examination

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 *formal 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, book the patient for a 'second look' in theatre. After the acute stages have settled the patient should be discharged on *Co-Trimoxazole 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-Trimoxazole 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 self 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/saucerisation 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 DS et al 1996 'An 18 year clinical review of septic arthritis from tropical Australia' *Epidemiology and Infection* 117(3) 423
2) Learmonth 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) Saraux A et al 1997 'HIV infection as a risk factor for septic arthritis' *Br. J. Rheum* 36(3):333
4) Chiedozi LC 1979 'Pyomyositis : a review of 205 cases in 112 patients' *Am. J. Surg.* 137,255

A community survey of the Ingwavuma 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 (59%) 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 perforating 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 Hlabisa in 1999. Our eye services are currently co-ordinated by a trained ophthalmic nurse practitioner.

EYE INFECTIONS

- Chloramphenicol 1% eye ointment applied 6 hrly 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 stat im.
- 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 hrly

LID PROBLEMS

- Generalised lid cellulitis or infection localised to the lacrimal gland (dacryoadenitis) can usually be treated with oral Penicillin V 500 mg 6 hrly for 5 days. Infection of the lacrimal sac (dacryocystitis) requires iv Penicillin 5 Mu 6 hrly 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 conjuctival 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 hrly 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 hrly 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 evert 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 $\leq$ 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 intraocular 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 hrly. When the symptoms have subsided and in cases of chronic glaucoma, give Pilocarpine 1-4% drops 8hrly.
- 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
1) Cook CD and Stulting AA 1995 'Impact of a Sight Saver clinic on the prevalence of blindness in Northern KwaZulu' *S.A.M.J* 85:28
2) Cook CD et al. 1993 'Prevalence and causes of Low vision and blindness in Northern KwaZulu' *S.A.M.J.* 83.590
3) Cook CD et al. 1998 Is anterior chamber lens implantation after intracapsular lens extraction safe in rural black patients in Africa? A pilot study in KwaZulu-Natal, South Africa' *Eye* 12:821
4) Cook CD et al. 1991 'Perforating eye injuries in KwaZulu' *S A M.J.*

ORAL MANIFESTATIONS OF HIV

CONDITION	CLINICAL APPEARANCE	TREATMENT
FUNGAL INFECTION		
CANDIDOSIS		Topical/systemic
• 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 dorsum of tongue and hard palate (CIT-NIP)	As above
• hyperplastic	White plaques that cannot be wiped off, most commonly occurs on the buccal mucosa	As above
• angular cheilitis	Red fissures radiating from the corners of the mouth	Nystatin/Ampholero B ointment applied 4 x daily Miconazole 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 take metronidazole 200mg 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	
• NUP	Rapid and progressive soft tissue loss, destruction of alveolar bone	
• NS	Ulcero-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, alveolar ridges - ulcers are shallow round/elliptical and coalesce - may be chronic	Acyclovir 400 - 600mg 5 x daily for 10 days
• varicella zoster	More than one dermatome may be affected - multiple shallow, small ulcers, unilaterally distributed along the 5th cranial nerve usually the palate - pain, neuropraxia and tenderness are usually present	Supportive measures Acyclovir 800mg 5 x daily for 10 days
• cytomegalovirus	Single ulcers without initial formation of vesicles - both shallow/deep ulcers may be seen	Acyclovir 800mg 5 x day/ 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
• HPV	Oral warts may be exophytic, granular/cauliflower like, asymptomatic and commonly isolated	Surgical excision, laser therapy/cryosurg

CONDITION	CLINICAL APPEARANCE	TREATMENT
NEOPLASMS		
• kaposi's sarcoma	Blue, black flat or lobulated lesion seen on the hard palate and gingiva, can occur at other sites	Vinblasite intra-lesional 0,2mg/ml or 0,1 ml/cm of tumour Surgical excision Radiotherapy Improve oral hygiene
• lymphoma	Macules/non-healing ulcers anywhere in mouth	Chemo and radio therapy
ORAL ULCERS		
• aphthous ulcers	May be minor/major/herpetiform, may be persistent	Treatment depends on severity Topical steroids .0,1% triamcinolone acetonide tds applied to lesion Beclamethasone spray - 1 puff 3 to 4 times day Beclamethasone 0,5mg tablet dissolved in a tablespoon of water - use mouthrinse 3-4 times day for 2-3 min
SALIVARY GLAND		
• xerostomia	Dry mouth	Sugarless gum/candy Bethanechol 25mg tds po
• parotid swelling	Enlarged parotid glands uni/bilateral	No specific treatment

MEDICINE

MALARIA

Hlabisa lies at the unstable southern edge of the African Malaria belt. Transmission is typically hypoendemic, usually January to April depending on rainfall, but may be becoming holoendemic in adjacent health districts bordering Mozambique, due to widespread cross-border migration of index cases and the supplantation 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 parasitaemias. 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 Mtubatuba 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+°C as a rule but may be apyrexial on admission, don't expect a tertian pattern in the acute phase)
- Headache
- Characteristic "paroxysms"
- Mild diarrhoea 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 (&/or U>20 and Cr >260)
- Jaundice (&/or Bili>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 (Katquick P.f. 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-quantitiatively graded in our lab and treatment decisions are invariably based on the clinical features. All malaria patients that are admitted must have FBC, U+Es 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 artemether and 120 mg lumefantrine per tablet) according to the table below. (Use weight if available rather than age.)

DOSAGE CHART FOR CO-ARTEM®

WEIGHT	AGE	NO. OF TABS
10 - 14 kg	1 - 5	1
15 - 24 kg	6 - 8	2
25 - 34 kg	9 - 12	3
≥35 kg	≥13	4

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 malaria ward (or ICU when the ward is full). They should be catheterised and routine observations including RR and HGT recorded hourly.
- If no loading dose has been given at clinic, give it IV - 20mg/kg in 500mls 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

- Oliguric 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 500 ml infusion), followed by dopamine 2.5mg/kg/min if necessary. Refer EARLY if dialysis is looking 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.
- Thrombocytopoenia is common in malaria and is not in itself an indication of severity. Platelet transfusion is only likely to be required if Plts<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 febrifugal 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
1) Freese J et al. 1994 'The in vitro sensitivity of South African isolates of P. falciparum to amodiaquine, chloroquine, mefloquine, quinine and sulphadoxine/pyrimethamine' *S.A.J.Sci.* 90:417
2) Moorthy V and Wilkinson D 1997 'Severity of Malaria and level of Pfalciparum transmission' *Lancet* 350:362
3) Sharp and Le Suer 1996 'Malaria in S. Africa - the past present and selected implications for the future' *S.A.M.J.* 86 83
4) Warrell D et al. 2000 Severe and Complicated Malaria (3rd edition) *T.R.S.T.M.H.* 94 Supp 1

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 estimated on tuberculin surveys is ~ 1+% for the black population (suggesting intense transmission). In Hlabisa, we have experienced a rise in case-load of 400% over the last 8 years with an estimated incidence in 1995 of ~400 per 10^5/year. 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 at as 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+% 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 regime 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* (esp. weight loss, night sweats/fever, suspicious CXR changes etc.) may be prescribed *Amoxycillin 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 with *pleural 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 PTBs and any sputum -ves with typical CXR appearances*
- *Other smear or culture positives (Histology, CSF etc.)*
- *Children under 5 years with a confluent or ulcerating tine 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 diagnoses 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 Hlabisa as compared with TB culture results was 77%). Defaulters 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 intercurrent bacterial infections and adverse drug reactions.

T.B. patients in our district are managed *solely* through the *D.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.

PULMONARY T.B.

Comprises *two-thirds of our cases*. *75-85% are sputum positive*. Primary T.B. is essentially a disease of children and Post-primary T.B. of adults. 'Progressive primary/ Transitional T.B. is rarely observed where the whole spectrum of CXR changes may occur shortly after a primary infection in adolescents.

Note that sputum status and clinical response to trial of antibiotics in general take priority over the CXR appearances - so very few suspected PTB cases are admitted direct to T ward. Note that only *~ 10% of culture proven cases demonstrating cavitation on CXR are smear -ve*

Note that *no CXR appearance is in itself pathognomonic of T.B.* - important differentials are silicosis, destructive pneumonias (esp. klebsiella, staphylococci), lung abscess and bronchiectasis. The CXR should always be interpreted in the clinical context. Chest signs are often surprisingly scanty even when the CXR shows marked changes.

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 hemi-thorax* 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 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 left with a drain after decortication/pneumonectomy and require thoracoplasty.

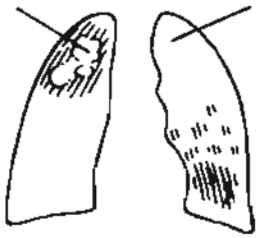
Further Reading

1) Richter et al. 1994 'Diagnosis of T.B. in patients with pleural effusion in an area of H.I.V. infection and limited diagnostic facilities' *Trop. Geo. Med.* 46:5 pp.293

2) Lee et al. 1988 'Corticosteroids in the treatment of tuberculous pleurisy' a double-blind, placebo-controlled, randomized study' *Chest* 94:1256

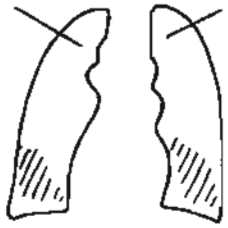
3) Burgess LJ et al. 1994 'Epidemiological study of pleral effusions at Tygerbergh hospital' XIIth. South African Pulmonology Society Congress *S.A.M.J.* 84 (11):773

'Classical Post-1° P. T.B.'



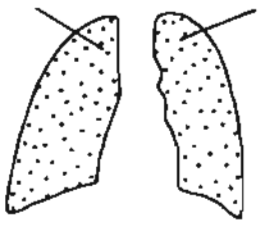
-Commences in 'vulnerable' broncho-pulmonary segments apl/post UL or apl LL
-Cavitation
-Bronchogenic spread to other parts of the same or opposite lung

'Atypical P.T.B.'



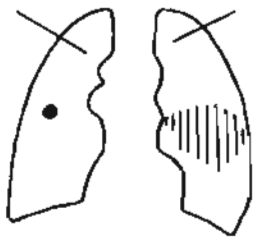
-LZ infiltrates
-Lymphadenopathy
-hilar
-R paratracheal stripe
-aorto-pulmonary window
-sub-carinal
-related to HIV, old age, alcoholism or diabetes

'Miliary T.B.'



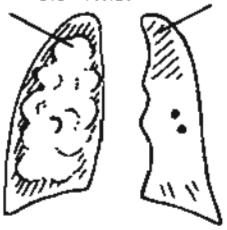
- 1-2 mm generalised homogenous 'millet seed' infiltrates
-results from haematog enous dissemination of 1° or post 1° P.T.B.
- overpenetrated views under bright light may show early lesions in outer rib spaces

'Primary T.B.'



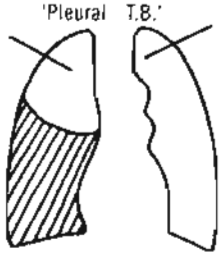
-Small 1° 'Ghon' focus is a 1-2 cm nodule in any area of the lung
-Enlargement of hilar and/or mediastinal nodes follows usually with spontaneous resolution
- Segmental/lobar collapse from bronchial compression/ulceration or bronchopneumonia from rupture of a node may be complications.

'Old P.T.B.'



-Interstitial infiltrates and loss of volume
-Calcification
- Bronchiectatic 'tramlines'
-'Destroyed lung'

'Pleural T.B.'



In children, small and transient following 1° infection.
In adults, >50% of a hemithorax and related to H.I.V.
Characteristic meniscus or shift on decubitus film

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 HIV* - 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. *Macroscopic 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
1) Bem et al 1993 'The value of wide-needle aspiration in the diagnosis of tuberculous lymphadenitis in Africa' *AIDS* 7:1221
2) Perenboom et al 1994 'Diagnosis of tuberculous lymphadenitis in an area of H.I.V infection and limited diagnostic facilities' *Trop Geo. Med* 46:5 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 S_3* just after the second heart sound. CXR will demonstrate *cardiomegaly* after ~ 250 ml have accumulated and *calcification on lateral* in some cases of constriction. US/S diagnosis rests on demonstration of both *anterior and posterior echo-free (dark) areas* between the right and left ventricular free walls and the echo-bright pericardium which persist 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 need not necessitate aspiration in itself if 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 *pericardiectomy* at an interval.

Further Reading
1) Strang JIG et al. 1988 'Controlled clinical trial of complete open surgical drainage and of prednisolone in the treatment of tuberculous pericardial effusion in Transkei' *Lancet* ii 759:63
2) Strang JIG 1990 'Echoes from the third world: two dimensional echocardiography in a developing country' *S.A M.J.* 77:85

ABDOMINAL TUBERCULOSIS

The *peritoneal* form has been the commoner in our practice especially since the advent of HIV but we also see classic cases of *tuberculous enteritis* which usually affect the ileo-caecal junction presenting with a *right iliac fossa mass, pain and weight loss*. In most of our patients abdominal infection is probably due to *M.tuberculosis* but some cases may still be due to *M. bovis* which is always resistant to pyrazinamide though this probably does not affect the adequacy of standard chemotherapy.

Peritoneal T.B. presents with abdominal swelling from *ascites or 'plastic peritonitis'*. CXR may show associated pulmonary T.B. in <40%. *US/S* may confirm the presence of fluid, thickened/stellate/amorphous-looking peritoneum and mesenteric or retroperitoneal *lymph nodes* especially in the pancreatic region. *Culture of ascitic fluid has an 80% yield but smear <5%*. However, *most cases of exudative lymphocytic ascites warrant a trial of T.B. treatment* pending culture results. Probably the quickest way to confirm the diagnosis in the dry form is to do a *mini-laparotomy* though peritoneoscopy is available at King Edward Hospital

Further Reading
1) Gilinsky NH et al. 1983 'Abdominal tuberculosis - a ten year review' *S.A.M.J.* 64:22 pp.849
2) Shukla HS et al. 1982 'Peritoneal biopsy for diagnosis of abdominal tuberculosis' *Postgraduate Medical Journal* 58:226
3) Jain R et al 1995 'Diagnosis of abdominal T.B.- sonographic findings in patients with early disease' *Am. J. Roentgenology* 165:1391

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 basal 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, fluctuations in conscious 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 *IIIn, IVn and Vn palsies* and *peripheral pareses* 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/mm^3 in 90%, note that PNMLs 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

>85% and is highest on the first specimen.

TBM may be staged according to the British M.R.C. system viz. *stage I - undisturbed consciousness, no neurological signs, stage II - disturbed consciousness but not coma and focal 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 C.S.F. penetration and should be continued for at least *12 months* (substitute ethionamide for ethambutol in children). *Cerebral tuberculomata*, 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/dl (Froin's syndrome)* and/or signs of arachnoiditis clinically. Patients who deteriorate with *suspected hydrocephalus* should be transferred for *C.T. scan* and assessment for *V-P shunting*.

Further Reading
1) Donald PR 1996 'Paediatric meningitis in the Western Cape Province of South Africa' *J Trop.Paeds* 42 256
2) Alarcon F et al. 1990 'Tuberculous 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 paraspinal abscess. The radiological appearances include *disc-space narrowing* ('discitis'), *anterior wedging* of two adjacent vertebra (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 Ngwelezane 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 vertebra will *almost always require surgery* to remedy the imminent spinal collapse. All other patients (including Frankel Cs and Ds) 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.

* Frankel classification of spinal injuries
- A - no sensory or motor function
- B - sensory but no motor function
- C - sensory and motor function but not useful
- D - sensory and motor but not normal
- E - normal

Further Reading
1) Boachie-Adjei O and Squillante RG 1996 'Tuberculosis of the spine' in *Orthopaedic clinics of N. America* 27 1 pp 95
2) Ogle JW 1994 'Angular kyphosis as an indicator of the prevalence of Pott's Disease in Transkei' *S.A.M.J.* 84:614

DRUG TREATMENT

In Hospital

	Adults <50 kg	Adults >50 kg	Children
ISONIAZID (H)	250 mg	300 mg	15 mg/kg
RIFAMPICIN (R)	450 mg	600 mg	10 mg/kg
PYRAZINAMIDE (Z)	1500 mg	2000 mg	30 mg/kg
ETHAMBUTOL (E)	800 mg	1200 mg	

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)

	Adults <50 kg	Adults >50 kg	Children <10 kg	Children 10-15 kg
ISONIAZID(H)	600 mg	900 mg	100mg	200mg
RIFAMPICIN (R)	600 mg	600 mg	150 mg	150mg
PYRAZINAMIDE(Z)	3000mg	3500mg	500mg	750mg
ETHAMBUTOL (E)	2000mg	2500mg	-	-

	Children 16-20	Children 21-25	Children 26-30	Children 31-35 kg
ISONIAZID(H)	300 mg	400 mg	400mg	500mg
RIFAMPICIN (R)	150 mg	300 mg	300 mg	300mg
PYRAZINAMIDE(Z)	1000mg	1250mg	1500mg	1500mg
ETHAMBUTOL (E)	-	-	-	-

Further Reading
1) Davies GR et al. 1999 'Twice weekly Directly Observed Treatment for HIV infected and uninfected Tuberculosis patients Cohort study in rural South Africa *AIDS* 13: 811
2) Connolly C et al 1999 Relapse and mortality 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 nocte 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.

	Day 1	Day 2	Day 3
ISONIAZID (H)	50 mg	300mg	full dose
RIFAMPICIN ®	75 mg	300 mg	full dose
PYRAZINAMIDE (Z)	250 mg	1000 mg	full dose
ETHAMBUTOL (E)	100 mg	500 mg	full dose

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

Further Reading
1) Girling DJ 1982 'Adverse effects of anti-tuberculous drugs' *Drugs* 23:56
2) Zent C 1994 'Toxicity of anti-tuberculous medication' *S.A.J.Epi and Infection* 9(1):5

SEXUALLY TRANSMITTED DISEASES

These are very common in Hlabisa 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 these only 65% were judged to have been treated adequately according to current provincial guidelines STDs are widely held to be *ukufa kwabantu par excellence* and the first consultation will often be with a traditional healer - remember that the patient's conception 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 ~8% and this seems to play a large part in our perinatal mortality. Other bacterial STDs especially 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 quinolone 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 im stat + erthythromycin 500 mg q.d.s p.o. for 5 days

In those allergic to penicillin, erthythromycin 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

Metronidazole 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 Clo-trimazole 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 *WR* on all patients and give a date for follow-up if the results will not be available the same day. If +ve, 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 though 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: randomised controlled trial' *Lancet* 346: 530

2) Wilkinson D 1999 'Unrecognised sexually transmitted infections in rural South African women: a hidden epidemic' *B.WHO* 77.22

3) Harrison A et al 1998 '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%. Heapatocellular 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 KingEdward Hospital.
- Alcohol - although *isizulu* (sorghum/maize beer) has relatively low alcohol content (2-3%) other much more potent home-made spirits (30+%^{o}) are widely available e.g *isiqatha*.
- Siderosis (traditionally common feature of liver biopsies in African men, possibly related to iron overload from cooking pots and a polygenic predisposition. Porphyria 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.v.c. webs 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/infestations.
- Biliary obstruction in younger patients may be related to wandering ascarids but in older patients more often to gallstones or pancreatic malignancy.

Our patients often present late with gross ascites but excepting gynaecomastia, leuconychia and subtle skin changes, signs of chronic liver disease will usually be absent.

Investigations

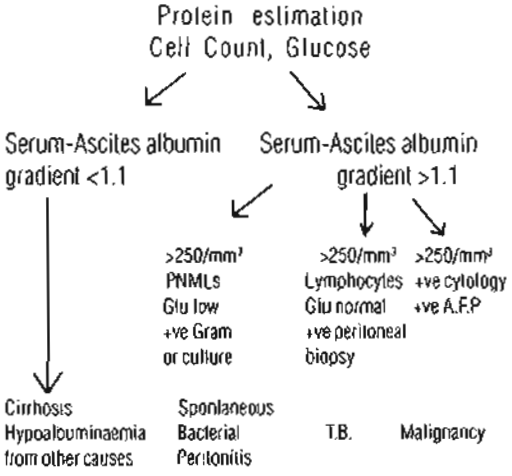
- *Routine work-up* should always include *LFTs, U & Es and INR.* Important differentials of primary liver disease in the jaundiced patient are malaria (ParaSight F) and haemolytic anaemia (Hb, Retic count and diptest urine for urobilinogen/bilirubin). Always exclude cardiac failure/constrictive pericarditis in cases of ascites and diptest 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 *brightly echogenic enlarged liver* and are hard to miss. *Cirrhosis* is characterised by a *small bright liver* with an irregular *'morse-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 3° bile ducts running alongside peripheral portal veins. The upper limit of normal for the common bile duct diameter is *0.6 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 -



- Certain *2nd line investigations* can be carried out in Durban. *α-foetoprotein* 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 -

Points scored for increasing abnormalities

	1	2	3
Encephalopathy (grade)	None	1/2	3/4
Ascites	None	Mild	Mod/Severe
Bilirubin (μmol/l)	<25	25 - 40	>40
Albumin (g/l)	>35	28 - 35	<28
Prothrombin time (s prolonged)	1-4	4-6	>6

Grade A = 5-6, Grade B = 7 -9, Grade C = 10 - 15

Patients admitted with *decompensated/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. Ceftriaxone should be started if S.B.P. is suspected clinically/on tap.

Further Reading

1) Gines P 1987 'Comparison of paracentesis and diuretics in the treatment of cirrhotics with tense ascites - results of a randomised study' *Gastroenterology* 93:234

2) Menzies et al. 1986 'Tuberculous peritonitis in Lesotho' *Tubercle* 67:47

3) Abdool Karim et al. 1993 'Hepatitis C infection in urban and rural KwaZulu/Natal' *S A M.J.* 83 191

4) Parekh D et al. 1987 'Gall stone disease among black South Africans' *S.A M.J.* 72 23

5) Maharaj B et al. 1986 'Causes of hepatomegaly at KEH VIII hospital, Durban A prospective study of 240 black patients' *S.A.M.J.* 69:183

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 (DCMO). It was notable that in women pericardial 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+% of autopsies for cardiovascular disease in RSA. A proportion of such cases present clinically in a normotensive 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. 'Shoshun' 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 *Frusemide 40-80 mg slowly i.v.*
- Add *Morphine 5-10 mg slowly i.v.* if severe left-sided symptoms.
- *Glyceryl Trinitrate* by continuous i.v. infusion (at an initial rate of *100µ g/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 *Hydrallazine 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 vein.
- If a precipitating arrythmia 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 two 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 beri-beri* 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 S_1 may be loud and an S_4 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. S_1 is typically soft and an S_3 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 rheumatic activity 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 prosthesis (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-receptors 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 (+/- Dypridamole) - 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
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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, chorea or erythema marginatum, which should be remembered when applying the Jones Criteria (see below).

REVISED JONES CRITERIA FOR RH. F

Major Manifestations

Carditis
Polyarthritis
Chorea
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, β-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 Cardiac clinic (WWH).

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.*

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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 over half presenting in heart failure and a *mortality of 40+%*.

The Duke Criteria may be quite useful in making a logical clinical diagnosis. Recovery rates from blood cultures at Hlabisa 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%). There

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, Glomerulonephritis
- Echo: consistent with I.E. but not meeting major criteria
- +ve blood culture but not meeting major criteria

Definite endocarditis = 2 major/3 minor+1major / 5minor/ I E rejected = firm alternative diagnosis
Clinical resolution in <4 days

Subacute cases with no evidence of embolisation (probably streptococcal) should receive Penicillin G 2-4 Mu 4 hrly + Gentamicin 3mg/kg once daily for 4 weeks. In more *severe cases* (possibly staphylococcal) give Cloxacillin 2g 4 hrly + 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 1 week of treatment.*

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CHEST SEPSIS

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 lit 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 *Amoxycillin 500 mg p.o. 8 hrly.*
- If *severe* give *Benzylpenicillin 2-4 Mu (1.2-2.4 g) i.v. 6 hrly.*

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).

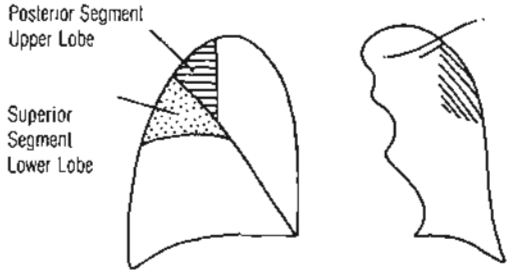
CHRONIC COUGH

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 10^5/ 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 manifested 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 Penicillins owing to *gram negative especially Pseudomonas colonisation* - our only option in this situation is oral *Ciprofloxacin 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.



ASPIRATION - VULNERABLE BRONCHOPULMONARY SEGMENTS

EMPYEMA

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 *28F* 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 Cardiothoracics 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 Mu 6 hrly i.v.* until definite clinical improvement. If progress is slow, add iv chloramphenicol 1 gm 6 hourly. Continue *oral amoxycillin* for up to *3 months*.

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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, menorrhagia, 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 bd is better tolerated than tds. 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 >95)

Always ask about alcohol intake. Other causes should be considered than B12 and folate deficiency- reticulocytosis (haemorrhage/haemolysis)

- liver disease aside from alcohol
- phenytoin (interferes with folate)
- aplastic anaemia and myelodysplastic syndromes and myeloma

Do not tranfuse 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 Bco. strong tablets. Be aware that in pernicious anaemia, and small bowel disease (blind loop syndrome, crohns etc), there will be no response. High dose folic acid -5mg od for four months and hydroxycobalamin 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 urobilinogen and then Coombs test in suspected AIHA. Discuss with haematologist.

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 2/3 of patients previously admitted were on inhaled steroids and in most the dose was only 200 µg /day. Many patients struggle to master inhaler technique but almost 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.
- *Loeffler'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 β_2 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 β_2 agonist
(salbutamol 100µg MDI) 2 puffs p.r.n. 6hrly

↓

Inhaled steroids
(Budesonide 100µg MDI) 2 puffs 12 hrly*

↓

Increase the dose of inhaled steroid up to 2000µg daily

↓

Add slow release theophylline tablets b.d.

↓

Prednisolone (lowest dose possible)

*Beclomethasone 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 β_2 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 phenytoin 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 alt 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 severily -

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
- O_2 40-100% by mask (be wary about giving any >28% in older smokers!) Use the pulse oximeter to gauge the minimum F_iO_2 that maintains saturations >90%.
- Salbutamol 5 mg O_2-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

- Salbutamol 5mg iv. If no improvement, give into 1 litre of saline or 5% dextrose and run at 2ml/min increasing up to 6ml/min. if necessary
- Aminophylline has not been shown to be of benefit in acute asthma
- Adrenaline nebuliser 0.5 mg may be used as a last ditch measure in extremis
- If ventilation is clearly going to be required, the best method is to paralyse, intubate and sedate with a ketamine infusion which has bronchodilating properties.

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HYPERTENSION

The prevalence of hypertension defined as 160/95 in community surveys of rural Zulus has been estimated at 8.3 % 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 o.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 Korotkov 1 (first appearance of sounds) and diastolic as Korotkov 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 HGT and diptest urine on all patients. If abnormal, take random blood glucose and / or U&Es /Crealinine.

- In all patients, *try non-pharmacological measures first*
  - *lose weight*
  - *reduce alcohol intake*
  - *regular exercise*
  - *reduced salt diet* (No Knorrox 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') i 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 *Hydrallazine 20 mg b.d.*

↓

Review in one month
Ensure compliance

↓

Hydrallazine 50 mg b.d.

If still uncontrolled consider changing to / adding 2nd line agents e.g Prazosin 1mg b.d. increasing to 5mg 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, hydrallazine 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 Hydrallazine 25-50 mg t.d.s. or nifedipine 10-20 mg t.d.s. In all cases investigate for a 2° 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 hydrallazine 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.

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DIABETES MELLITUS

Prevalence generally <1% in rural Africa but approaching 4% in urban Zulus. 'IDDM' 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 (reactive 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 or 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 diptest 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 unconcious 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/failure to rise of the plasma Na^+). Hypotonic solutions e.g Darrows 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 -

serum K^+	replacement rate
<3.0	40 mmol/l
3.0-4.0	30 mmol/l
4.0-5.0	20 mmol/l
5.0-6.0	10 mmol/l
>6.0	none

- *Low dose i.v. insulin* inhibits the uncontrolled lipolysis and gluconeogenesis of the ketotic state - give *5 u. Actrapid stat 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 mmol/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 hypertensive 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.
- Hypernatraemia 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 'fast 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
1) Huddle and Gill 1989 'Reducing acute hyperglycaemic mortality in African diabetic patients' *Diabetic Medicine* 6:64
2) Huddle and Gill 1993 'Hypoglycaemic admissions amongst diabetic patients in Soweto, South Africa' *Diabetic Medicine* 10:181
3) Adrogue et al 1989 'Salutary effects of modest fluid replacement in the treatment of adults with diabetic ketoacidosis' *J.A.M.A.* 262:2108
4) Alberti KGGM 1977 'Low dose insulin in the treatment of diabetic ketoacidosis' *Arch. Int. Med.* 137:1367
5) McLarty DG et al. 1990 'Diabetes in tropical Africa: a prospective study 1981-1987 II Course and prognosis' *B.M.J.* 300:1107

NEUROLOGY

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 Phenytoin 300 mg/day who had neither side effects nor seizures demonstrated 'potentially toxic' levels in 38%. Epileptics in our community suffer considerable morbidity, especially from burns, and most are dependent on some sort of disability grant.

Diagnosis

It 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.
- *Febrile 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 diptest the urine for protein. Check HGT 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 focal/ poorly controlled fits or neurological signs.

Treatment

- Start after *two seizures* (or one, if focal, as the likelyhood 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 folate 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 hyponatraemia, 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 1g 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 Phenobarbitone to another drug unless they are experiencing troublesome side-effects
- *Stopping treatment.* In practice, acheiving 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 EPILEPTICUS

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 100 mg 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 electolytes +/- Ca^{2+} and consider a cause e.g. infection/ SOL 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.* 66:21
2) Dansey et al. 1992 'Seizures and neurocysticercosis in black men' *S.A.M.J.* 81:424
3) McFadyen 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* 78 587
4)The First Seizure Trial Group 1993 'Randomised clinical trial on the efficacy of anti-epileptic drugs in reducing the risk of relapse after a first unprovoked tonic-clonic seizure' *Neurology* 43 478
5) MRC Antiepileptic drug withdrawal study group 1993 'Prognostic index for recurrence of seizures after remission of epilepsy' *B.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 commonly 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 esp. 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, T.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 at 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. >200/120 after 48 hours bed 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./W.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 TIAs 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.

Further Reading
1) Joubert J 1991 'The MEDUNSA Stroke Data Bank - an analysis of 304 patients seen between 1986 and 1987' *S.A.M.J.* 80:567
2) Subramanyan R et al. 1989 'Natural History of Aorto-arteritis (Takayasu's disease)' *Circulation* 80:429
3) Celani et al 1994 'Comparability and validity of two clinical scores in the early differential diagnosis of acute stroke' *B.M.J.* 308:1674
4) Kassell N et al 1990 'International co-operative study on timing of aneurysm surgery' *J.Neurosurg.* 73:18
5) Malenga J 1997 'Stroke incidence rates among black residents of Harare - a prospective community-based study' *S.A.M.J.* 87:606

MYELOPATHY

'Idiopathic myelopathy' is a common problem for African physicians - half of cases go unexplained. 'Konzo' is a non-progressive spastic paraparesis resulting from thiocyanate 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.

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/transection)
- Intrinsic lesions/'Transverse Myelitis' (Tumours, HIV, Schistosomiasis, Vascular - classically bilateral dissociated sensory loss)
- Posterior column syndromes (Tabes Dorsalis and Sub-acute Combined Degeneration - charaterised by +ve Romberg's sign and absent ankle jerks)
- HTLV-1 myeloneuropathy (usually spastic weakness and absent ankle jerks with little sensory loss)

Management

- Check Hb, RPR, urine/stool for schistosome 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 +/- macrocytosis, take blood for serum B_{12} and give 1000 µg B_{12} stat i.m. daily for a week + Folate 5 mg daily. Though established spinal cord damage from megaloblastosis 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 B_{12}/Folate
  - HIV, HTLV-1, RPR all negative, no evidence of TB/bacterial spondylitis or schistosomiasis.

Further Reading
1) Bhigjee et al. 1993 'Prevalence and transmission of HTLV-1 infection in Kwazulu/Natal' *S.A.M.J.* 83:665
2) Van der Ryst E and Smith MS 1994 'A high prevalence of HTLV-1 antibodies in HIV-1 seropositive patients with neurological disease' *S.A.J Epidemiology and Infection* 9:1 p.20
3) Cliff J & Nicala D 1997 'Long term follow-up of konzo patients' *T.R.S.T.M.H.* 91:447

PERIPHERAL NEUROPATHY

Many in-patients complain of burning dysaesthesia/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.B 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 Amitryptyline (25-50 mg nocte) may be helpful.

Further Reading
1) Cosnett J.E. 'Neurological disease in Natal' in *Tropical Neurology* Spillane (ed.) Oxford University Press
2) Max MB et al. 1992 'Effects of desipramine, amitryptyline and fluoxetine on pain in diabetic neuropathy' *N.E.J.M.* 326:1250

HIV/AIDS

KwaZulu-Natal is undergoing an explosive epidemic of HIV infection, outstripping the worst case projections of Doyle, especially in urban areas. This has undoubtedly been fuelled by the fragmented social structure and high prevalence of STDs. At Hlabisa,unlinked anonymous serosurveys of antenatal attenders have documented a rise in seroprevalence from 7.9% in 1993 to 32% by 1999 and this has already been reflected in clinical practice - most notably by a 400% increase in TB caseload and the occurrence of several high-profile AIDS-defining illnesses (Kaposi's Sarcoma, Cryptococcal meningitis and Oesophageal Candidiasis). The number of in-patients fulfilling the criteria for AIDS is still relatively small but is doubling every year and in 1997 constituted 5% and 2% of female and male medical patients respectively. However, the epidemic is affecting all aspects of our clinical work and undoubtedly altering the prevalence and presentation of many more common illnesses.

In our district the sex ratio of infection is F:M 1.4:1 and the peak incidence is in the third decade. A series of micro-epidemics seems to be occurring, with the worst affected areas being KwaMsane and Nkundusi. The attitude of the community seems still to be generally one of 'denial' and though major efforts are underway to encourage community awareness and disclosure, home-based care for the majority of our patients is still a distant prospect.

Further Reading
1) Wilkinson D and Hapgood LC 1994 'The evolving HIV epidemic in a rural hospital in Zululand from 1989 to 1993' *Epi. Comments* 21(1)
2) Floyd K et al 1999 'Admission trends in a rural South African hospital during the early years of the HIV epidemic' *J.A.M.A.* 282:1087

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 -

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 diarrhoea 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 diarrhoea 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

- HIV Encephalopathy
- &/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 a 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+% 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 indedpendently of CD4 count - it may appear as *plaques, flat erythematous patches or angular cheilitis*. First line treatment is *Nystatin* oral suspension *100-500,00 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)*.

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

- *Scabies* (v prevalent infestation locally, typical distribution - give Benzyl Benzoate 25%)
- *Folliculitis* (staphylococcal infection usually on the chest/armpits in males - give oral cloxacillin)
- *Seborrheic dermatitis* (caused by blastospheric form of Pityosporum 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* (escp hypersensitivity to T.B. drugs)
- *'Prurigo'* (idiopathic mononuclear/eosinophilic infiltration of subcutaneous tissues) - systemic antihistamines (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 Hlabisa, though KS has only become a significant clinical problem in the last few years. *'Benign'* lesions (small lesions confined to skin wilhout 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

1) Wilkinson, D et al 1999 'Prevalence of infection with HHV8 / KSHV in rural South Africa' *S.A.M.J.* 89.554

2) Stein et al. 1994 'Epidemic AIDS related Kaposi's Sarcoma in Southern Africa. experience at the Johannesburg General Hospital 1980-1990' *T R S T.M.H* 88 434

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 Mycobacteria-other-than-tuberculosis (M.O.T.T.s) though prevalent in South African miners, are exceedingly uncommon in our patients.

BACTERIAL INFECTIONS

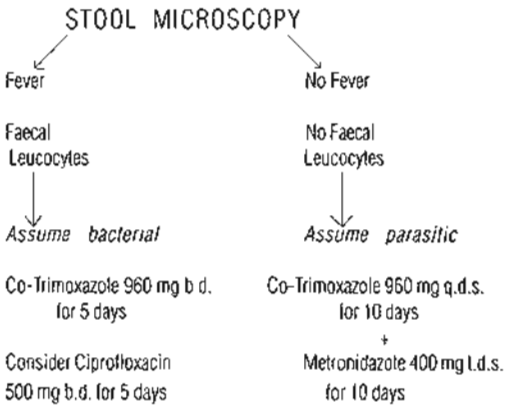
Invasive pneumococcal disease (lobar pneumonia, bacteraemia,meningitis, sinusitis) is a common cause of mortality in our patients. *Non-typhoidal salmonella and E. coli* may also cause fatal septicaemia.Prompt recognition and treatment with Ampicillin 500 mg 6 hrly and Gentamicin 240 mg daily -unless 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 *Amoxycillin* or *Co-trimoxazole prophylaxis* should be considered in these patients.

Further Reading
1) Gilks C et al 1990 'Life-threatening bacteraemia in HIV-1 seropositive adults admited to hospital in Nairobi, Kenya' *Lancet* 335: 545
2) Anglaret 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,shigella or campylobacter) or a treatable infestation (isosporiasis, amoebiasis, giardiasis). Management of patients with chronic diarrhoea should proceed as follows -



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

Give Albendazole 2 tabs stat to all patients as 40+% will be infested 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 hrly* should be started.

Further Reading
1) Batchelor BI et al. 1996 'Microbiology of HIV associated bacteraemia and diarrhoea in adults from Nairobi' *Epidemiol. Infect.* 117:139
2) Kelly P et al. 1996 'Albendazole chemotherapy for treatment of diarrhoea in patients with AIDS in Zambia: a randomised double blind controlled trial' *B.M.J* 312 1187

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.R. 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 *Sulphadoxine /Pyrimethamine 500mg/12.5 mg ii stat and then ii b.d.* The differentials are toxoplasmoma, 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 (+ve C.S.F. W.R.) 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 bronchoscopy 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 /desaturation 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./i.v. for 14 days + Prednisolone 40 mg daily if severely hypoxic.* Secondary prophylaxis with Co-trimoxazole 960mg daily or 3x/week is indicated.

Further Reading
1) Zar HJ et al. 2000 'Pneumocystis carinii pneumonia in HIV infected patients in Africa - an important pathogen?' *S.A.M.J.* 90:684

STDS

Always ask about 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.

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 cold clinical situation the permission of the

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
1) Wilkinson D et al. 1997 'On-site HIV testing in resource-poor settings: is one rapid test enough?' *AIDS* 11:377
2) The Voluntary HIV-1 Counselling and Testing Efficacy Study Group 2000 'Efficacy of Voluntary Counselling and Testing in individuals and couples in Kenya, Tanzania and Trinidad: a randomised trial' *Lancet* 356:103
3) Seidel G 1996 '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 Ubombo 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 ungulate 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*
- *'bilharziomas'* 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 *peritoneal/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 diptest of the same urine.
- *S. mansoni* - 3 wet stool preparations +/- rectal snips/ squashs. 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 stat 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

1) Cooppan RM et al. 1986 'Morbidity from urinary schistosomiasis in relation to the intensity of infection in the Natal province of South Africa' *Am.J.Trop.Med.Hyg.* 35:765

2) Cooppan RM et al. 1987 'Urinary reagent strips in the screening of children for schistosomiasis in RSA' *S.A.M.J.* 72:459

3) Schutte CH et al 1983 'Effectiveness of Praziquantel against South African strains of S. haematobium and S. mansoni' *S.A.M.J.* 58:66

PARASITOLOGY

Infestation with soil transmitted helminths is common in our patients - in surveys of under-25s in the Ubombo/Ingwavuma 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 fortunately *uncommon (<1%).*

The nutritional significance of these infestations in children is the subject of some debate, but it 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* (wheezy children with transient pulmonary infiltrates and eosinophilia, caused by migrating ascarid larvae - do not de-worm at presentation as 'verminous pneumonia' may result when the larvae die)
- *Small bowel obstruction* due to ascarid '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.*
- *'Sandworm'* is 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 Calliphorid flies is occasionally encountered in small children or neglected wounds in adults. *Cordylobia* species may be easily expressed from their holes after application of liquid paraffin but more aggressive species not responding to this treatment may require debridement +/- irrigation with chloroform or ether.

Treatment

Treat according to the infestation identified on stool microscopy - though in clinics/O.P.D. it is acceptable to treat blind 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)
- NICLOSAMIDE 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 infestations)
- TOPICAL THIABENDAZOLE solution 4x/day for 1 week - for *'sandworm'*

Further Reading

1) Schutte CHJ et al. 1981 'Intestinal parasitic infections in Black scholars in Northern Kwazulu' *S A M.J.* 25 p.137

2) Moodley D et al. 1991 'Cryptosporidium infections in children in Durban' *S.A M.J.* 79:295

3) Bradley and Buch 1994 'The prevalence of Ascaris and other helminth infestations in children attending a rural Natal hospital and its clinics' *S.A.J. of Epidemiology and Infection* 9:2 p.42

4) Taylor M et al. 1995 'Targeted chemotherapy for parasite infestations in rural black pre-school children' *S.A.M.J.* 85:870

5) Appleton CC 1996 'The distribution of common intestinal nematodes along an altitudinal transect in Kwazulu/Natal, South Africa' *Ann Trop Med. & Parasitology* 90:181

NEPHROTIC 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 antigenaemia. In adults and children, the commonest cause is membranous or membrano-proliferative 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 >3.5g/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/S* to assess the renal size (a gross discrepancy may indicate renal vein thrombosis).

Management

- *Restrict salt* to approx 1500mg/day.
- Give *Frusemide* to mobilise the oedema (dose up to 500 mg b.d 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 look-out for *complications* -especially venous thrombosis and infection (pneumococcal peritonitis 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. immunosupressants).

Further reading
1) Seedat YK 1978 'The Nephrotic syndrome in the Africans and Indians of South Africa-a ten year study' *T.R.S.T.M.H.* 72:5
2) Schiepatti A et al. 1993 'Prognosis in untreated patients with Idiopathic Membranous Glomerulonephritis' *N.E.J.M* 329:85
3) Imperiale TF 1995 'Are cytotoxic agents beneficial in Idiopathic Membranous Nephropathy? A meta-analysis of the controlled trials' *J.Am.Soc.Neph* 8:1553

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 prevelent 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 abruption/eclampsia/puerperal sepsis). Mortality is still high (~50% at one year) but usually results from complications (esp. 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-catheterisation 'tubular paralysis' in chronic retention). *Physical signs are non-specific* so you may miss the diagnosis if you don't check the U&Es! 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 Frusemide* (the prognosis is better if you can avoid prolonged oliguria) - followed 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 of saline* titrated with the B.P. if persistent hypotension seems to be responsible.
- Take an *E.C.G.* if the *potassium is >6.0 mmol/l* - any flattening of the P wave or widening of the QRS complex must be promptly treated. Give *Calcium Gluconate 10 ml 10% slowly i.v.* over ~ 1 minute (repeated once if no effect) followed by *i.v. insulin / dextrose (10 u Actrapid + 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 sulphonate 15g oral or 30g rectal 6 hrly.*
- 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* as tolerated. Give cimetidine to prevent stress ulceration.
- Be alert to the possibility of *secondary sepsis*.
- Hypertension should respond to *Nifedipine*.
- Patients without an obvious precipitant and a persistently active urinary sediment, hypertension etc should be referred for *renal biopsy* (≤15% 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
1) Chugh et al. 1989 'Changing trends in acute renal failure in third world countries - Chandigarh study.' *O.J.Med* 73:1117
2) Rasmussen and Ibels 1982 'Acute Renal Failure.' *Am J.Med* 73:211
3) Graziani et al. 1984 'Dopamine and Frusemide in oliguric renal failure' *Nephron* 37:39
4) Seedat et al. 1993 'Acute renal failure in Blacks and Indians in South Africa - Comparison after 10 years' *Nephron* 64:198

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, emulsifying 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 is *localised to the flexures* where constant scratching leads to lichenification 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.
- *Seborrheic Dermatitis.* Probably due to the fungus *Pityosporum 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 off 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 *Whitfields 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, vesiculation 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. haemopoietic 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 systemically 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 (hairs 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 taped 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 *Whitfields 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.

- *Tinea Pedis (Ringworm of the feet):* Usually found in young men (especially if working in wellington boots). Can be severe and is a common entry site for cellulitis. Treatment is with potassium permanganate (1 in 10 000) foot soaks. Prevention is careful drying between the toes, cotton socks and well-aerated shoes.
- *Tinea Capitis (Ringworm of the scalp):* Commonly children, and presents as single or multiple patches of broken or distorted hairs/alopecia often with scaling of the skin. Most resolve with puberty but may result in permanent alopecia especially in the more severe 'Favus' or 'Kerion' forms characterised by suppuration or thick crusts. Treatment is with *griseofulvin 10 mg/kg up to 500 mg daily for 4-6 weeks.*
- *Tinea Unguium (Ringworm of the nails):* Yellow discoloration and distortion of the nails, which are often brittle. Treatment is with griseofulvin or ketoconazole and has to be prolonged: 6 months for fingernails, 18 months for toenails, and is therefore only indicated in exceptional circumstances.
- *Pityriasis versicolor:* Common in hot humid weather, pregnancy, steroid therapy and the immunocompromised. Family outbreaks often occur. Small irregular pale patches gradually coalesce to form the *'continent with surrounding islands' pattern*. The *trunk is most commonly affected*. It can mimic vitiligo. A single dose of ketoconazole (400mg) is probably the most simple and effective treatment.
- *Scabies :* Caused by the mite, *Sarcoptes scabiei*, it is *extremely common*. Classically it causes an *itchy rash* with *pruritis very much worse at night and the presence of tortuous thread-like burrows is pathognomic*. The distribution is typically *between the fingers*, around the *wrists*, the *waist, buttocks, inner thighs, ankles and the nipples in women and the penis in men*. The lesions can be obscured by *excoriation* from scratching and can become *infected*. Atypical severe infestations can occur in the immunocompromised with a widespread dermatitis. Treatment is with *25% benzyl benzoate emulsion* painted into every nook and cranny from the neck down after a good scrub and left on for 24-48 hours then washed off thoroughly. All family members should be treated at the same time and bed linen changed immediately after treatment. Treatment can exacerbate any underlying dermatitis and the *itch and rash may persist for many days after treatment*. Aqueous cream and occasionally weak steroids are indicated.

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 50mg nocte 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 contract 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 viracidal therapies, all treatments are aimed at debulking the wart and stimulating an inflammatory reaction in the hopes 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 *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: subungal* 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 excision 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 - 100 mg/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 dorsum 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 referrable 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. *Delirium* 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 >8 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 apyrexial 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
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4) Abdool Gaffar MS et al 1992 'The white cell count in Typhoid Fever' *Trop.Geo.Med* 44:23
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AMOEBIASIS

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 shigella pandemic. Stool surveys identify E. 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 ABSCESS

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 +/- atelectasis. The diagnosis is *usually confirmed by US/S* - a typical cystic structure will usually be present with through 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 'last 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 colitis 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 tense abscess wall, but the other concern here is to exclude a bacterial abscess)
- *raised hemi-diaphragm* (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 abdominally* (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 +/- Eosin 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 8 hrly 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 Durban)

2) Jackson TFGH 1995 'Amoebiasis - resolution of a centuries old mystery and its impact on our understanding of the disease' *C.M.E.* 13:8 pp 903

3) De le Rey Nel et al. 1989 'Indications for aspiration of amoebic liver abscess' *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 Sulphamethoxazole, 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 Shigellosis 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. Amoebic 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, PITs, U&Es and ask about urine output*. Be alert to 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

- Ceftriaxione 50 mg/kg/day slow i.v./i.m. o.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 urea and cannot be restored with Frusemide 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

1) Chopra M et al. 1997 'Epidemic Shigella dysentery in children in Northern KwaZulu/Natal' *S.A.M.J.* 87:48

2) Pillay DG 1997 'An outbreak of shiga bacillus in KwaZulu/Natal, South Africa' *J. Infection* 34(2):107

3) Bennish ML 1991 'Potentially lethal complications of Shigellosis' *Rev Inf.Dis.* 13(S4):S319-24

PEPTIC ULCER DISEASE

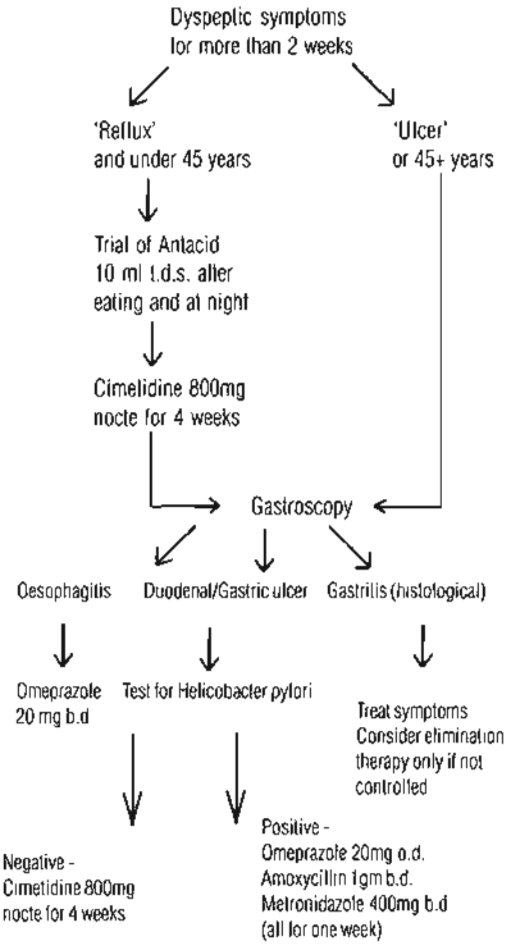
Dyspepsia may affect up to 30% of rural Africans but the majority are not related to peptic ulceration. However, peptic ulcer disease can no longer be considered uncommon in black South Africans - the incidence of admission for this illness increased 12x at KEH VIII between 1950 and 1976. We have operated for four perforated D.U.s at our hospital since1995 - 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 -

- *Reflux type* - complain of heartburn, acid brash and burning retrosternal discomfort made worse by lying down and large meals.
- *Ulcer type* - complain of epigastric pain often worse at night and releived 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).



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 ~3 per 10^5 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 reinfection and duodenal ulcer recurrence' *GUT* 36:554
2) Segal I et al. 1983 'Duodenal and gastric ulcer in Soweto' *S.A.M.J* 64(20):???
3) Mayor-Davies JA et al. 1985 'Surgery for complications of peptic ulceration in urban black patients' *S.A.M.J.* 67(4):545
4) Holcombe C et al 1991 'The prevalence of symptoms of dyspepsia in north eastern Nigeria: a random community-based study' *Tr. Geo. Med.* 43.209
5) 1999 'Diagnosis and Management of Dyspepsia - clinical guidelines' *S.A.M.J* 89.897

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 and 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, pneumatocoeles 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 unrousable 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, peanutty taste. They contain curcin, a ricin-like poison that causes q severe gastroenteritis. The plant is commonly used by local traditional healers as a purgative. The clinical presentation is of abdominal pain, diarrhoea,vomiting and sometimes dehydration. The condition is fortunately self-limiting and supportive treatment only is required.

JIMSON WEED '*ILOYI*'

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 - ibotenic 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 cyclopeptides) is characterised by a delay in initial symptoms for 6-24 hours followed by profuse vomiting and choleraic diarrhoea. A 48-72 hour remission then supervenes but is often followed by acute hepatic and renal necrosis which is fatal in 50+% of cases. It is only in this phase that neurological symptoms may become prominent as a result of metabolic encephalopathy. Megadose penicillin therapy is thought to oppose the effects of amanitins and doses of 1 Mu/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 dipping domestic animals and stock and is also used as a spray around farm compounds to control cockroachs. 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 neuro-muscular junctions e g. generalised 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 gastro-intestinal 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 radio-opaque. The clinical features are biphasic with initial abdominal pain, diarrhoea and vomiting of black fluid which subsides as the iron enters the reticulo-endothelial 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 / 1gm 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 acidotic 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 oesophagus but cause severe gastric lesions. Pass an n.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/laparotomy 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

1) 1995 'Pesticidal poisoning in South Africa 1980-1994' *Epi. Comments* 22:112

2) Reed RP and Conradie 1997 'Epidemiology and clinical features of paraffin (kerosene) poisoning in rural African children' *Ann. Trop. Paeds* 17:49

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PALLIATIVE CARE

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 1gQDS
NSAIDS:	Ibuprofen 200-800mg TDS
Weak opioids:	Codeine, Codeine/Paracetamol mix, Valloran (children)
Strong opioids:	Morphine, Pethidine

On each step, build up to the maximum dose 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. carbamazopine.

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.

BREATHLESSNESS

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 negative 5) 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 nystagmus can 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 hypovolaemia; 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 drowsiness 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, foetal 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 make 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 50-90 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

1) Bennish ML 1994 'Cholera: Pathophysiology, Clinical Features and Treatment', in Wachsmuth, Blake and Olsvik (eds) *Vibrio Cholera and Cholera: Molecular to Global Perspectives*

2) Cairncross and Feachem 1983 *Environmental Health Engineering in the Tropics*

3) Christie AB 1987 'Cholera' in *Infectious Diseases* Vol 1, 4th ed.

4) WHO 1993 *Guidelines for Cholera Control* (Good tables for fluid replacement)

PSYCHIATRY

PSYCHIATRY

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 better 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/Nystagmus/VIn palsies (Alcohol withdrawal)
  - Asterixis/Jaundice/Asciles (hepatic encephalopathy)
  - Hypertension (hypertensive encephalopathy)
  - HGT (hypoglycaemia, often alcohol-related).

Routinely check the WR and any other tests (Blood glucose, Katquick, 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, Utshwala, Isiqatha 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 100mg b.d (tds 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 tds Anticholinergic medication does not need to be given routinely. If the patient does develop troublesome extrapyramidal side-effects give Orphenadrine 50 mg tds.

Discharge

Usually the MO covering psychiatry will discharge the patients 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 morning). 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 tds 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 G2/2 (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 50-75mg 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 iiii 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 t.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

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3) Ensink K et al. 1998 'Expression of schizophrenia in black Xhosa-speaking and white English-speaking South Africans' 88:883

4) Solomons K et al. 'Toxic cannabis psychosis is a valid entity' *S.A.M.J.* 78:476

5) Bhagwanjee et al 1998 'Prevalence of minor psychiatric disorders in an adult African rural community in South Africa' *Psychol. Med.* 28:1137

6) Thompson C 1994 'The use of high dose anti-psychotic medication' *Br J. Psych.* 164:448

7) Sellers EM et al 1983 'Diazepam loading : simplified treatment of alcohol withdrawal' *Clin. Pharm. Ther.* 34:822

PAEDIATRICS

GASTROENTERITIS

Acute infective GE is a common cause of morbidity and mortality in South African children, with data from 1968 - 1985 suggesting that 43.5% of deaths in black children in the postneonatal period were attributed to GE. In the Hlabisa Hospital paedriatric audit (1995 - 1996) acute GE was the primary diagnosis in 21% of in-patient deaths, with dysentry 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, intussusseption 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 hyponatraemia)
  - consistency
  - colour - blood/mucus (? dysentery - see separate protocol)
- FEEDING: type and volumes of food and fluids tolerated vomiting
- 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 4hours

ADMIT

- anyone requiring a drip
- any child
  - <1mth old
  - <3rd centile or Kwash
  - recurrent GE ie 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*
- Intraosseous line if can't get IV within 5 minutes.
- Give O_2
- 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 cupfuls 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
- Malnourished / immunocompromised -Ampicillin and Gentamicin
- Shocked (likely septicaemia) - Ampicillin and Gentamicin
- Dysentry - Nalidixic acid or Ceftriaxone (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 Myelinolysis has not been described in acute hyponatraemia in children.

- If *Na^+ < 125 mmol/l* give *Ringer's at 10ml/kg/hr* - repeat U+E after 8hr Change to 1/2D+D when >125mmol/l.

B. *Hypernatraemia*:

Usually fat, well-nourished children on formula feeds. Implies significant intracellular hypertonicity and dehydration and giving hypotonic solution risks rapid osmosis into cells and cerebral oedema. Suspect if rubbery skin or jittery/irritable with less dehydration than expected.

- If *Na^+ >150mmol/l* give *1/2 D+D 1L + 40ml 8.4% NaHCO3* at MAX *10ml/kg/hr.*

Change to 1/2D+D after 24 hrs.

Repeat U&Es after 4 hours, Na^+ should fall at ≤1 mmol / l / hr, bicarbonate may drop the K^+

C. *Hypokalaemia*:

Suspect with acidosis, severe hypotonia, abdominal distension/ileus.

- If *K^+ ≥4. 0mmol/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. 5mmol/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 mmol/l* correct IV i.e. 40mmol 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 food as 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.

- VITAMIN A: give to all - 100,000U if < 1yr, 200,000U if > 1yr. 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 diarhoea.
- MAGNESIUM SULPHATE: 0.5ml IM once weekly if diarrhoea over 1 week.
- FOLATE: 5mg OD 5/7 if diarrhoea over 1 week.

Further Reading

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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-supression (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.
- Aim for normal diet in 3 days - encourage starchy feeds 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:

- *Folate* 5mg OD 7/7
- Mg_2SO_4 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 sticktest for reducing substances: If *>0.5%* pos change to Infasoy. If *-ve* or not better on Infasoy then give 'bowel cocktail'.
  - *Breast fed infant*: Test for reducing substances - If result *positive 2% or more* then change to Infasoy 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*:
    Gentamycin 80mg BD orally 3days. Cholestyramine 1g qds 5 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 Infasoy 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

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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 Hlabisa 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.

Severity*	Criteria	Management
'Mild'	Cough, Fever Respiratory rate <50/min	Paracetamol
'Moderate'	Cough, Fever Respiratory rate >50/min	Paracetamol Amoxycillin
'Severe'	Cough, Fever Respiratory rate >50/min Intercostal recession Cyanosis Unable to drink	See Below

*Young infants and 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, may 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. poor feeding, convulsions, abnormal sleeping, etc. It may be difficult to distinguish pneumonia from septicaemia, UTI or meningitis. Have 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 O_2 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 1litre/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 Amoxycillin (non severe)

- <1 year 10-15mg / kg tds
- 1-5 years 125-250mg qds
- >5 years 250-500mg tds (total 8 days)

IV Ampicillin (severe)

- <1 yr 15-20 mg/kg qds
- 1-5 yrs 125-250 mg qds
- >5yrs 250-500 mg qds

Switch to amoxycillin 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 GroupB strep and Gram negatives

Benzyl penicillin 20-25 mg/kg qds iv/im AND *Gentamycin* <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 Gentamycin or Ceftriaxone. (risk gram negatives)

Bronchodilator if wheezy

<1yearAtrovent nebs 125mcg in 2 ml saline tds

>1year 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, <5years 120mg, >5 years 500mg 4-6 hourly, max qds
- **Further Investigations**
  CXR is not usually indicated unless there is doubt about the diagnosis, poor response to initial therapy or complications are otherwise suspected. Consider a KatQuick 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 chloramphenicol 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 owing to succesful 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, a trial of steroids is usually indicated if HIV status is known.

Further Reading
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CROUP

H. influenzae B epiglottitis has been an extremely rare condition in South African series and this might reflect a tissue tropism 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 barking cough
- starting 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 (diptheria)
- dysphagia or if the patient prefers a sitting position (epiglottitis, retropharngeal absecess)
- systemic toxicity e.g. with rash (Staph tracheitis)

Check the CXR for an inhaled radio-opaque FB/segmental hyperinflation and look at the upper airway sillhouette.

ASSESSMENT OF SEVERITY

Grade	Criteria	Treatment
I	INSPIRATORY Obstruction only	Observe in OPD/at home if favourable conditions
II	INSPIRATORY + PASSIVE EXPIRATORY Obstruction	Admit Nebulised Adrenaline* half hourly
III	INSPIRATORY + ACTIVE EXPIRATORY Obstruction (i.e. visible /palpable abdominal contraction or palpable pulsus paradoxus)	Admit Continuous nebulised Adrenaline If no improvement in 1 hour, consider intubation and transfer
IV	RESPIRATORY FAILURE Cyanosis, apathy, marked intercostal recession	Continuous nebulised Adrenaline Intubate and transfer for ventilation

**Adrenaline* nebulisers - 2 ml 1:1000 in 2 ml 0.9% Saline

Note: Stridor may become softer as the obstruction becomes more severe.

- Give humidified 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 tachyphylaxis 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% O_2 throughout. Spray the pharynx and cords with Lignocaine and pass a flexible ETT (2.5 mm if <6/12, 3.0 mm if 6-18/12 and 3.5 mm if 18/12 - 3 years). If the tube won't pass easily with gentle pressure don't force it, use the next smaller size. Magills 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
1) Hussey G 1994 'Epidemiology of invasive H influenzae infections in Cape Town, South Africa' *Ann.Trop.Paeds* 14 97
2) Kairys SW et al. 1989 'Steroid treatment of Laryngotracheitis: A meta-analysis of the evidence from randomised trials' *Paediatrics* 83:683
3) Jacobs S 1994 ' Validation of a croup score and its use in triaging children with croup' *Anaesthesia* 49.903

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 *per 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 faltering 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 T.B. 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 marasmics and 10% of kwashs are HIV +ve and highly susceptible to gram negative septicaemia, often from co-existing and undeclared UTIs.

Weight for age (% of expected)	Oedema present	Oedema absent
80-60	KWASHIORKOR	U.W.F.A.
<60	MARASMIC-KWASH	MARASMUS

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 hypoalbuminaemia. 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 kwash on the ward.

ON ADMISSION

- O.R.S. +/- breast milk only p.o. 2 hrly for 24 hours.
- Keep child warm, check temperature and HGTs 6hrly.
- Check Hb, U&Es and diptest 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 D+D 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 1/2D+D 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 B 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 B Co. 1 tab. daily p.o.
- Vitamin C I 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 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, hyponatraemia, 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 1litre.

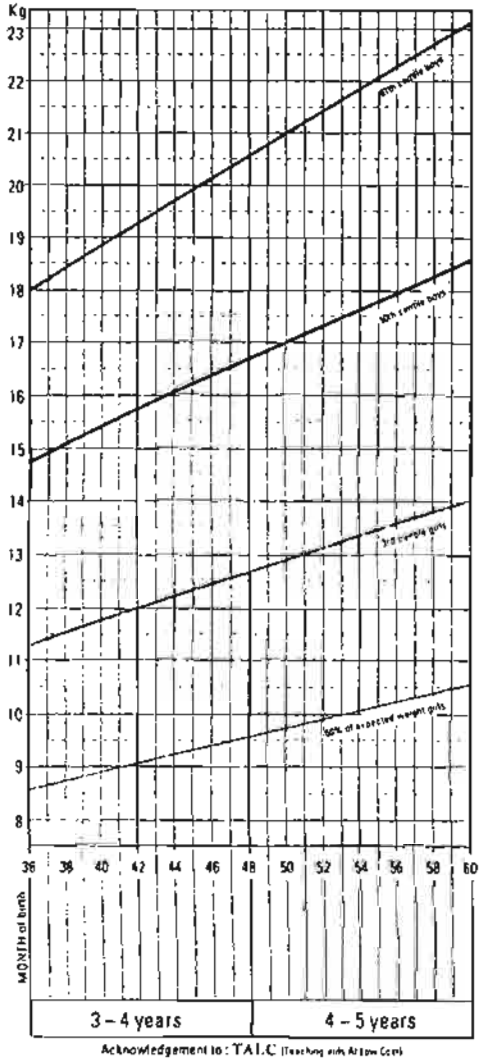
Further Reading

1) Wilkinson D et al. 1997 'Reduction of in-hospital mortality of children with malnutrition' *J. Trop.Paeds* 42:114

2) Macintyre UE 1994 'Protein-Energy malnutrition - results of rehabilitation' Supplement to *S.A.M.J.* July

2) Cameron N and Kgamphe JS 1993 'The growth of South African rural black children' *S.A.M.J.* 83:184

4) Zollner E and Cartier ND 1993 'Breast feeding and weaning practices in Venda 1990' *S.A.M.J.* 83:580



Acknowledgement to: T.A.I.C (Teaching with All Low Cost)

Road to Health Chart

Department of Health

IMPORTANT: always take this card with when you visit any health clinic, doctor or hospital, and present the card on school entry.

Child's name ☐ 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. / 10 min. | Gestational age (wks) | Serology

Mother's file numbers – Antenatal / Delivery

IMMUNISATIONS		PRIMARY Date given	PRIMARY Signature	BOOSTERS Date given	BOOSTERS Signature
B.C.G.	1			2	
Polio	0			4	
	1			5	
	2				
	3				
DTP	1			4	
	2			DT	
	3				
Hep.B	1				
	2				
	3				
Meas	1			2	
Other	1			3	
	2			4	
Vit A	1			4	
	2			5	
	3				

Remember to discuss child spacing

GW 6/123

Clinic 1 | Address

Clinic 2 | Address

Mother's name

Father's name

Caretaker if 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 — yes
- Is this baby a twin — yes
- Is this baby bottle fed — yes
- Does the mother need more family support — yes
- Are any brothers or sisters underweight — yes
- Are there any other reasons for taking extra care for example – tuberculosis, single parent etc. — yes

Vision screening ($4\frac{1}{2}$ – 6 yrs) | date | Result L: R:

Hearing screen (7 to 9 months) | date | Manchester Rattle used ☐ | Result L: R:

CARD GIVEN AND MOTHER TAUGHT BY

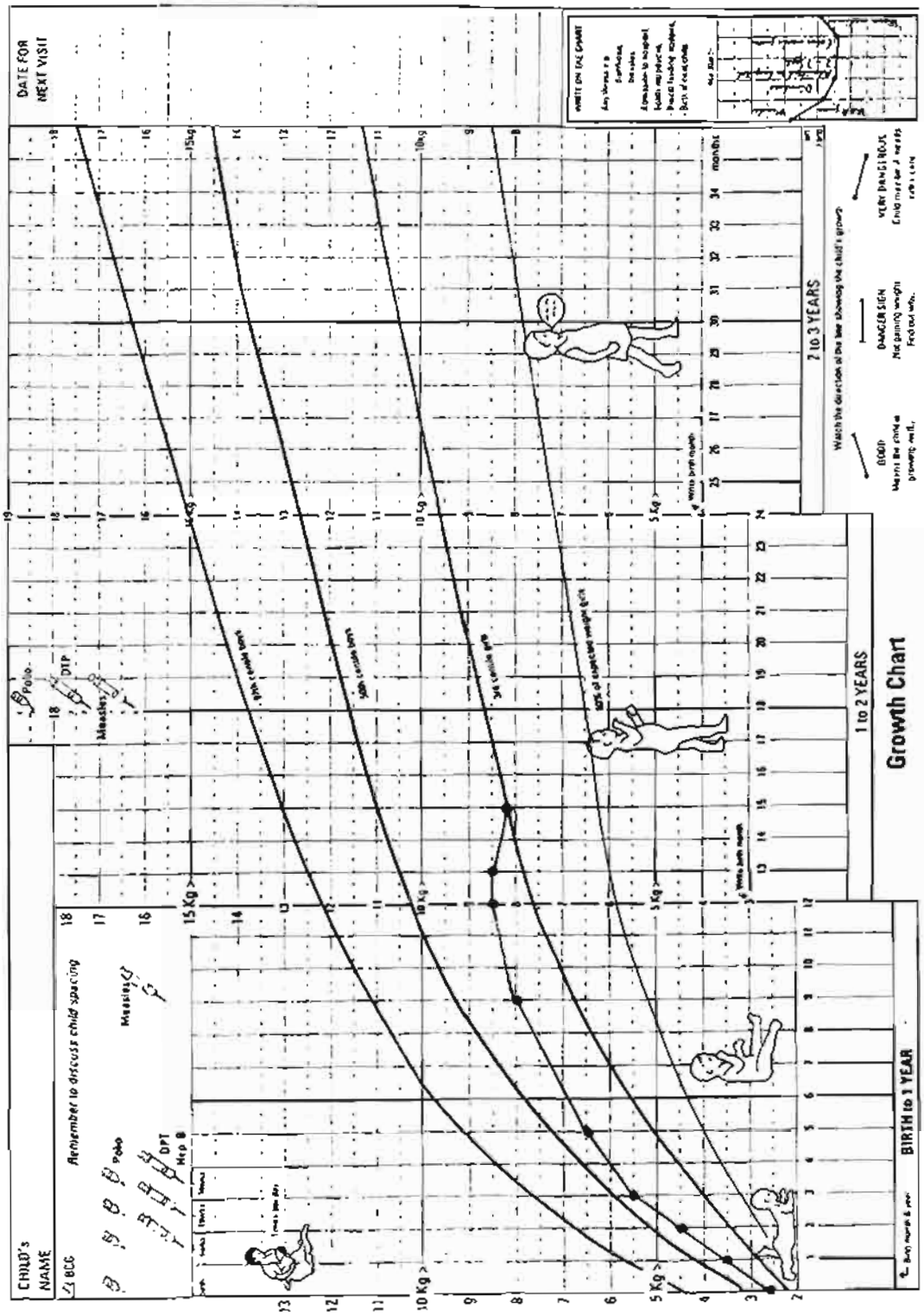
ORAL REHYD. RATION DATES: Taught / Used

TUBERCULOSIS SCREENING

Heaf / Mantoux / Tine	Date	Grade

TB contact

TB notified


CHILD's NAME
DATE FOR NEXT VISIT
BCG
Remember to discuss child spacing
Polio
DPT
Hep B
Measles
BIRTH to 1 YEAR
1 to 2 YEARS
2 to 3 YEARS
Growth Chart
50% of expected weight gain
3rd centile mark
Watch the direction of the line showing the child's growth
GOOD
DANGER SIGN
VERY DANGEROUS

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, fading 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

- *Amoxycillin 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 + Cloxacillin 125-250 mg 6hrly i.v.*
- Diarrhoea - usual supportive care + *Metronidazole 200 mg o.d. for 5 days* at/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) Whittle HC et al. 1979 'Severe ulcerative herpes of mouth and eye following measles' *T.R.S.T.M.H.* 73:66
3) Naidoo S & Meyers K 1994 'The measles epidemic' *S.A.M.J.* 84:125
4) Shann F et al. 1997 'Meta-analysis of trials of prophylactic antibiotics for children with measles , inadequate evidence' *B.M.J.* 314:334
5) Hussey GD 1996 'Clinical problems in measles case management' *Ann Trop.Paeds* 16 307

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 Ubombo 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 or 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, defer lumbar puncture pending resuscitation in shocked/dehydrated patients.

Csf	Normal	Bacterial	Tuberculous	Viral
Cells	0-5/mm^3 All lymphocytes	>1000/mm^3	<500/mm^3	<500/mm^3
Protein	≤0.45 g/l	>1.5 g/l	1-5 g/l	<1 g/l
Glucose	50+% of blood	<50% of blood	<50% of blood>	50% of blood

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 0.1g/l Protein to the normal values for each 1000 red cells present.

Management

- Give O_2 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 1-2x/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 cocco-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 6hrly* i.v. while waiting for LP results. If the gram stain shows *Gram +ve cocci/Gram -ve diplococci* change to *Penicillin 1-2 Mu 6 hrly*. If the LP shows Gram -ve cocco-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&Es - 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 20mg/kg slow i.v. if not well controlled. If convulsions continue give 5mg/kg maintenance doses daily.
- If there is *no significant change* after 48-72 hours -
  - *Repeat the Na'* to exclude significant hyponatremia.
  - *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 + Ethionamide 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 empyema/brain abscess is high in this group.

Further Reading

1) Hussey G et al. 1997 'Epidemiology of post-neonatal bacterial meningitis in Cape Town children' *S.A.M.J.* 87 51

2) Ciana G et al. 1995 'Effectiveness of adjunctive treatment with steroids in reducing short-term mortality in a high-risk population of children with bacterial meningitis' *J.Trop.Paeds.* 41 164

3) Raju N et al. 1995 'Cranial sonography in pyogenic meningitis in neonates and infants' *J.Trop.Paeds.* 41.68

4) Barson WJ et al. 1985 'Prospective comparative trial of ceftriaxone versus conventional therapy for treatment of bacterial meningitis in children' *Paed Inf.Dis* 4:362

5) Rennick G et al. 1993 'Cerebral hygeniation during bacterial meningitis in children' *BMJ* 306.953

OTITIS

OTITIS EXTERNA

Inflamed ear canal with normal drum? ~50% fungal. Post-auricular LNs 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 otoscopy or auricular cellulitis

- Clo-trimazole 1% cream can be used for refractory fungal infections.
- Primary dermatological disease of the ear canal should be considered if there is no improvement.

OTITIS MEDIA

Most of our cases present late, usually with long-established chronic, suppurative 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 Amoxycillin p.o. Myringotomy in the acute phase is almost never required but may be indicated at Ngwelezane if pain does not resolve in 24-48 hours on antibiotics.
- *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 in! 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/serosanguinous discharge suggesting tuberculosis/cholesteatoma. The last two especially are associated with cranial nerve (mostly VIIn) 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 audiology is relatively favourable.
- *Middle ear effusion* (chronic serous 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

1) Froom J et al. 1997 'Antimicrobials for acute otitis media? A review from the International Primary Care Network' *B.M.J.* 314.98

2) Van Rooy CH et al. 1995 'Diagnosis and treatment of ear disease among children in the Ellisras district. An outreach programme' *S.A.M.J* 85:675

3) Claassen AJ & Hough AP 1987 'A microbiological study of acute otitis media in Bloemfontein' *S.A.M.J.* 71:15

4) Liebowitz LB et al. 1987 'In vitro 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/ecthyma 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 cell casts* on urine microscopy and a *+ve A.S.O. titre (1 : 160 or greater in 90+%)*. Throat and skin swabs have a low sensitivity (~30%).
- Order 4 hrly obs inc B.P., CXR, U&Es/Cr, Albumin/Total Protein. Make sure accurate fluid charts are kept, weigh and diptest urine daily.
- *Restrict fluids* on admission to 15 ml/kg/24 hrs and thereafter to the previous days output.
- *Low-salt diet* (NO Cheesy puffs!). 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 %ile 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, O_2 5-8 l/min by mask.
  - *Morphine 0.1 mg/kg i.v..*

- *Frusemide 2 mg /kg i.v.* over 5-10 minutes.
- Repeal 4 mg/kg if no prompt reponse
- 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
1) Hallett AF 1977 ' Post-streptococcal glomerulonephritis in African children' *T.R.S.T.M.H.* 71(3):241

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 *3-4+ proteinuria on diptest* 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/ Cr, 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 peritonitis.
- *+ve 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 +/- enalapril* for persistent heavy proteinuria. *HBsAg -ve* patients should be given a *trial of steroids* (2 mg/kg/day max. 80 mg for 6 weeks then 0.5 mg/kg /day for a further 6 weeks) If there is no response, refer for assessment and biopsy. Treat any other specific precipitating infection (esp. Syphilis and schistosomiasis).

Further Reading
1) Coovadia HM et al. 1993 'Hepatitis B 's' and 'e' antigen carriage in childhood nephrotic syndrome predicts membranous glomerulonephritis' *Ann.Trop.Paed.* 13:79
2) Adhikari M et al.1983 'Absence of 'true' minimal change nephrotic syndrome in African children in South Africa' *J.Trop.Med.Hyg.* 86:223
3) Bhimma R et al. 1997 'Nephrotic syndrome in South African children: changing perspective over 20 years' *Paed.Neph.* 11:429

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	B. Probable Tuberculosis	C. Confirmed 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 repiratory disease 2.3 With painless swelling in a group of superficial LNs	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*	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 ^

* 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.

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 same room, breastfeeds) with a tuberculous adult carries a 5 year 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 atelectases, alveolar/interstital infiltrates and pleural effusion.
- 2° 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 Manloux but can be semiquantitatively graded according to the following method. It is not designed to draw blood!

GRADING OF THE TINE TEST

Grade	Description	Result	
0	No palpable reaction/papules <2mm	Negative	
I	One or more papules >2mm	Doubtful	
II	Coalescence of two or more papules but no ring	Positive	
II+	Ring of induration	Positive	
III	Smooth dome shaped swelling	Positive	
IV	Ulcerated/Vesiculated	Treatment mandatory	

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 sputum 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 in 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 meningeal 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
1) Cundall et al. 1986 'Diagnosis of PTB in malnourished Kenyan children' *Ann. Trop. Paeds* 6:249
2) Parisi et al. 1994 'Pictorial review of the usual and unusual roentgen manifestations of childhood tuberculosis' *Clin Imaging* 18 149
3) Schaaf HS et al. 1995 'Respiratory tuberculosis in childhood - the diagnostive value of clinical features and special investigations' *Paed Inf Dis J.* 14 . 189
4) Colditz GA et al. 1994 'Efficacy of B.C.G. vaccine in the prevention of tuberculosis . meta-analysis of the published literature' *J.A.M.A.* 271 (9) . 698
5) Jeena PM et al 1996 'Effects of the human immunodeficiency virus on tuberculosis in children' *Tub. Lung Dis.* 77.437

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	MINOR SIGNS
Weight loss/FTT	Generalised lymphadenopathy
	Oral candidiasis
Chronic Fever > 1 month	Repeated common infections
	Persistent cough > 1 month
Chronic Diarrhoea >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
1) Yeung S et al. 2000 'Paediatric HIV infection in a rural South African district hospital' *J. Trop.Paeds.* 46:107
2) Madurai S et al. 1996 'Use of HIV1 specific immunoglobulin G3 as a serological marker of vertical transmission' *J Trop.Paeds.* 2.359
3) LePage P et al. 1998 'Care of human immunodeficiency virus-infected children in developing countries' *Paed.Inf.Dis.J* 17:581
4) Fawzi W et al. 1998 'Vitamin A supplements and mortality among HIV positive and negative children in Tanzania' Abstract 42331 *12th World AIDS Conference*
5) Bobat R et al. 1999 'Mortality in a cohort of children born to HIV infected women from Durban South Africa' *S.A.M.J.* 89:646

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 unbooked 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-intradication 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

Age	*Vaccine*
Birth	TOPV, BCG*
6 weeks	TOPV, DTP, Hep B Hlb
10 weeks	TOPV, DTP, Hep B Hlb
14 weeks	TOPV^, DTP, Hep B Hlb
9 months	Measles
18 months	TOPV, DTP, Measles
5 years	TOPV, DT

* 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-Guerin)* - 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 of 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 and swelling at the injection site may occur for up to 24 hours.
- Hlb (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 *<0°c*).
- -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* at all times and *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
1) Wilkinson D et al. 1997 'Maternal and child health indicators in a rural South African health district.' *S.A.M.J.* 87:456
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.* 86:1128
3) *Ministry of Health 1995* 'Immunisation that works! The vaccinators manual of the expanded programme on immunisation in South Africa'

BREASTFEEDING

Klabisa 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.

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 breastmilk, 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.

Contact Numbers for more information of BFHI training and implementation.
Nutrition Sub-Directorate, KwaZulu-Natal, Department of Health, Natalia, Pietermaritzburg
☎ 033 395 2726 or 395 2642
Nutrition Directorate, Dept of Health, Pretoria
☎ 012 312 0071
Nutrition Project Officer, UNICEF, Pretoria
☎ 012 338 5000
IBFAN Africa (International Baby Food Action Network), Mbabane, Swaziland
☎ 09268 404 5006


NURSERY

NEONATAL RESUSCITATION

ASSESSMENT:

Check: colour, heart rate and respiratory effort

COLOUR HEART RATE RESPIRATORY	PINK >100/min good	BLUE >80/min gasping	WHITE <80/min apnoeic	WHITE 0/min apnoeic
ACTION	No suction! dry keep warm monitor	gentle suction O_2 by mask ↓ *no response* ↓ hand ventilate ↓ *no response* ↓ chest compressions	gentle suction O_2 by mask ↓ hand ventilate ↓ *no response* ↓ intubate hand ventilate chest compressions i.v. access/drugs	intubate hand ventilate chest compressions i.v. access/drugs

Make sure the resuscitaire 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 H_2O 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 : 1 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 O_2/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 0.1ml/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 foetal haemorrhage and shock give haemaccel/O -ve blood 10-20ml/kg over 10mins. Once perfusion restored Neolyte 5 ml /kg/hr maintenance
3. CHECK HGT - give 3-5 ml/kg/hr Neolyte i.v. if low (Neolyte 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 > 3.5kg. ONLY if persistent bradycardia in the presence of an otherwise satisfactory response
6. SODIUM BICARBONATE - 4.2% (5mls 8.5% Soda bic + 5mls sterile water/saline). Give 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 fault -?O_2 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
  - O_2 (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, regularity, 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

SIGN	SCORE 0	SCORE 1	SCORE 2
Heart rate	Absent	Below 100/mins	Above 100/mins
Respiratory effort	Absent	Weak	Good, cries
Muscle tone	Flaccid	Some flexion	Well flexed
Reflex irritability	No response	Grimace	Cough / sneeze
Colour	Pale or Blue	Pink body, blue limbs	Completely pink

CRITERIA FOR ADMISSION TO NURSERY

Criteria for Admission to Nursery:

- Pre-term <37 w
- Post-term >42 w if intrapartum complications
- Low birth weight <2 kg (if >2 kg & no other problems observe on perinatal ward and home if breastfeeding well)
- Over-weight-for-age
- Low Apgar (<6 at 5 mins) / required resuscitation
- Meconium aspiration and other intrapartum complications (CPD, Foetal distress etc.)
- All otherwise unwell babies (Respiratory distress, Seizures, Cord sepsis, Diabetic mother etc.)

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 squirted 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. neolyte.
- *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 stool 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 D et al. 1999 'Impact of prematurity on admissions to the neonatal nursery of a rural South African rural hospital' *J Trop.Paeds* 45:76
2) Simkiss D 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 - ivi for the first 24 - 48 hours until

- meconium passed
- abdomen soft and not distended

Then give EBM (or PRENAN) via the NGT as below:

DAY	IVI (ML/KG/DAY)	MILK
1	75	0
2	50	25
3	50	50
4	50	75
5	50	100
6	0	150

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 ivi 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.

DAY 1 - 60
2 - 90
3 - 120
4 - 150 ml/kg/day

- Add 25-30 ml/day if under phototherapy or diarrhoea.
- Weigh daily - loss of >10% birth weight =>increase needed
- Use Neolyte 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 - 0.90% 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 then 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 NAN 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 <36.5 °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 tempreture 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 O_2, add 10 ml 4% Sodium Bicarbonate to the drip and check HGT.
- Consider i.v. antibiotics if there is a suspicion of infection.

ROUTINE INCUBATOR TEMPERATURES (°C)

Birth weight	Days						
(kg)	0	5	10	15	20	25	30
1	35.5	35	35	34.5	34	33.5	33
1.5	35	34	33.5	33.5	33	32.5	32.5
2	34	33	32.5	32	32	32	32
2.5	33.5	32.5	32	31	31	31	31
3	33	32	31	30	30	30	30

NORMAL RANGES FOR NEONATES

FBC		U&E
Hb : 14 - 21.5	Na 132-142	BR(T) 68-120umol/l
HCT 53.6-68 4%	K 5 - 7 5	BR(C) 0-3 "
MCV 100 - 140	Cl 96-106	ALP 42 - 200U/L
PLT 150 - 400	Ur 1 6 - 4.9	ALT 10 -60 "
WCC 9 - 35	Cr 20 - 60	AST 10 -42 "
	Ca 1.7 - 2.6	Tot Prot 41 - 74 g/l
	Mg 0.7 - 1 0	ALB 32 - 50 "
	CSF	
LEUCS	PROTEIN.	GLUC>50 mg/dl
<5 : 75% lymph	20 -45 mg/dl	or75% serum

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 syndromes and Hyaline Membrane disease is virtually unknown >33w gestation. Infection probably accounts for 40+% of clinically identified cases of respiratory distress, and meconium aspiration for a further 10%. Always consider *extra-pulmonary causes*: Hypoglycaemia, Anaemia, Hypothermia, Metabolic Acidosis, Pneumothorax, Diaphragmatic hernia and Congenital heart disease. 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 for all includes:

- Warmth, minimal handling, energy (i.v. Neolyte)
- Observations : RR, recession, grunting, cyanosis, HR, temp
- O_2 until tongue pink -5-6 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 O_2 requirements in 1st 48 hrs - mortality approachs 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 is available at referral hospitals
- Look for and treat other associated complications of asphyxia/prematurity.

TRANSIENT TACHYPNOEA / WET LUNG SYNDROME

Pulmonary maladaptation associated with CS, prenatal/intrapartum asphyxia, maternal sedation, polyhydramnios. May be term or pre-term. Amnio/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: O_2 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% Nsaline + 50% Sod bic. with ETT 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/hypothermia, Gastric aspirate : pus cells, bacteria (only of value before first feed)

CXR: collapse/ consolidation

Management: Ampicillin 50 mg/kg 6-8 hrly + Gentamicin 5 mg/kg o.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 O_2 needs, decreased air entry/ breath sounds, palpable liver (if right) decreased heart sounds transillumination of affected side brighter/CXR

Management:

- mild RD/no cyanosis — >headbox O_2 + 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, anaemia.

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 S_2 - ASD*
  - *Pan-systolic, L. sternal edge => VSD*
  - *Short systolic, heard at the back => Coarctation*
- Bounding/absent pulses
- O_2 - resistant cyanosis due to large R—>L shunt (probable tetralogy)

Management:

- *Fluid restrict* to 60 ml/kg/day.
- *Frusemide 1mg/kg* i.v. stat.
- *Digoxin 0.05 mg/kg/day* divided into 4 doses over 24 hours then 1/4 of this dose daily.
- Transfuse with packed cells if PCV<30 /Hb<10, correct hypoglycaemia and acidosis.
- If *O_2-resistant cyanosis*, stop O_2 and give *Pg E_2 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

APNOEIC ATTACKS

= stopping breathing long enough to cause cyanosis/ bradycardia/ pallor

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, fits, drugs (pethidine), PVH
- GI - reflux, perforation, large feeds, vomiting
- CVS - hypo/hypertension, anaemia+/- PDA, CCF, hypovolaemia (APH)
  - Metabolic - hypoxia/ hypothermia
  - hypo- glycaemia/ -calcaemia/ -natraemia
  - hypernatraemia
  - increased ambient temperature.

Treatment:

- Oxygen - headbox
- Transfusion- 10ml/kg if Hb<10
- If immature (<36wks) - aminophylline : loading dose 5mg/kg then 2mg/kg tds (1mg/kg tds if < 1.5 kgs
- septic screen + ivABx
- Treat other identified causes.

AIRWAY OBSTRUCTION

Due to choanal atresia ->stridor + cyanosis when asleep but pink when awake and mouth open.

Dx: polyhydramnios, failure to pass n.g. tube per nares post-delivery (blocked nose - saline nose drops).

Further Reading

1) Van Rijswijk P & Ingle RF 1996 'Causes of early neonatal respiratory distress in the former Venda - a community based study' *S.A.M.J.* 86:1413

2) Adhikari M & Gouws E 1995 'Meconium aspiration in South Africa' *S.A.M.J.* 85:891

3) Wax JR 1995 'A radiological update on medical diseases of the newborn chest' *Paed.Radiol.* 25:631

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. gonorrhaea* 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
hypoglycaemia	hypotonia oedema scleraema

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.

Age	Total Neutrophil count	% Band forms
Birth	1.8-6.0	<16
24 hours	7.2-12.6	<16
48 hours	3.6-8.1	<13
5 days	1.8-5.4	<12

- *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 licquor. 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/mm^3
  - Lymphocytes 0-10/mm^3
- *Urine microscopy.*

Management

Supportive: ie 0_2, iv Neolyte/ NG feeds) as required. Monitor HGTs and temperature closely.

Antibiotics:

Ampicillin: 62,5 mg qds po 1week 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/per n.g.t. 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 +ve amniotic fluid cultures at the time of rupture. The infection is invariably polymicrobial but addition of anaerobic cover seems to confer 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 screen.

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 septicaemic infant. There is a high incidence of ventriculitis 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. (intrathecal 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 as prophylaxis vs. seizures - then 5mg/kg po od.

NECROTISING ENTEROCOLITIS

Necrosis usually of 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, septicaemia, 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
- Reintroduce 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 panopthalmitis in 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 pussy, 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 for 7 days.
- Treat 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 periumbilical flare. Portal phlebitis, peritonitis and septicaemia may follow. The usual causes are *Staphylococci,Streptococci* and Coliforms but proliferation of *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 hrly till dry. Do not cover!
- Established infection - Surgical spirit 3 hrly + Penicillin and Gentamicin i.v.

NEONATAL TETANUS:

- clear airway, O_2
- no stimulation
- NGT - 30 mins after sedation
- fluids: EBM - small frequent feeds (decrease risk aspiration/regurgitation)
- suctioning & turning after each dose sedation
- Monitor O_2 saturations and pulse and record during spasms
- Diazepam 1mg/kg iv/pr till spasms stop and then 0.25-1mg/kg 4-8hrly
- Chlorpromazine 12.5 mg i.m. 4-6 hrly if spasms not controlled
- Penicillin 25,000 u /kg 6 hrly
- 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

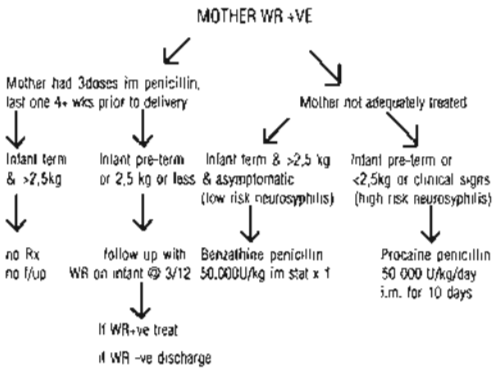
Risk of congenital infection ~50% but only ~10% of babies of untreated WR +ve mothers have clinical signs of Syphilis at birth. These include -

LBW	Syphilitic pemphigus (hands & feet)
Heavy greasy placenta >1/5 of infant's birthweight	Anaemia
	Thrombocytopoenia/ petechiae
Hepatosplenomegaly	Nasal discharge (?blood-stained)
Lymphadenopathy	Metaphysitis on long 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 FTA-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 (aspirate gram stain diagnostic)
- *Treatment*:
  wash - Chlorhexidine bd for 5 days, uncover cord - no nappy!
  Treat cord infection - prevent spread!
  If unwell —>iv Abx
- 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 pustules)
- Monilial rash -
- raised red velvely rash especially aroumd 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 1ml after feeds for 7 days &on mum's nipples/vagina (cream)

Further Reading
1) Manroe et al. 1979 'The neonatal blood count in health and disease I: reference values for neutrophil cells' *J.Paeds.* 95:89
2) Maberry MC et al. 1991 'Anaerobic coverage for intra-amniotic infection: maternal and perinatal impact' *Am J.Perinat.* 8:338
3) Haffejee IE 1984 'A therapeutic trial of cefotaxime vs. Penicillin and Gentamicin for severe infections in children' *J. Anti Chemo* 14 Suppl B 147
4) Laga M et al. 1986 'Single dose therapy of gonococcal opthalmia neonatorum with ceftriaxone' *N.E.J.M.* 315(22):1382
5) Jeena PM & Coovadia HM 1997 'Risk factors for neonatal tetanus in Kwazulu/Natal' *S.A.M.J.* 87:46
6) Sanders RKM 1996 'The management of tetanus 1996' *Trop.Doctor* 26:10

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, polycythaemic.

Symptoms: none or:

decreased brain function: lethargy, hypotonic, weak cry, apnoea, absent moro, cyanosis increased brain function: jiltery, staring, making fists, abnormal eye movements, convulsions sweating.

Sick infants should have - IVI - 10% glucose (neonatalyte) 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 10ml of 50% glucose to 90ml neolyte

**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

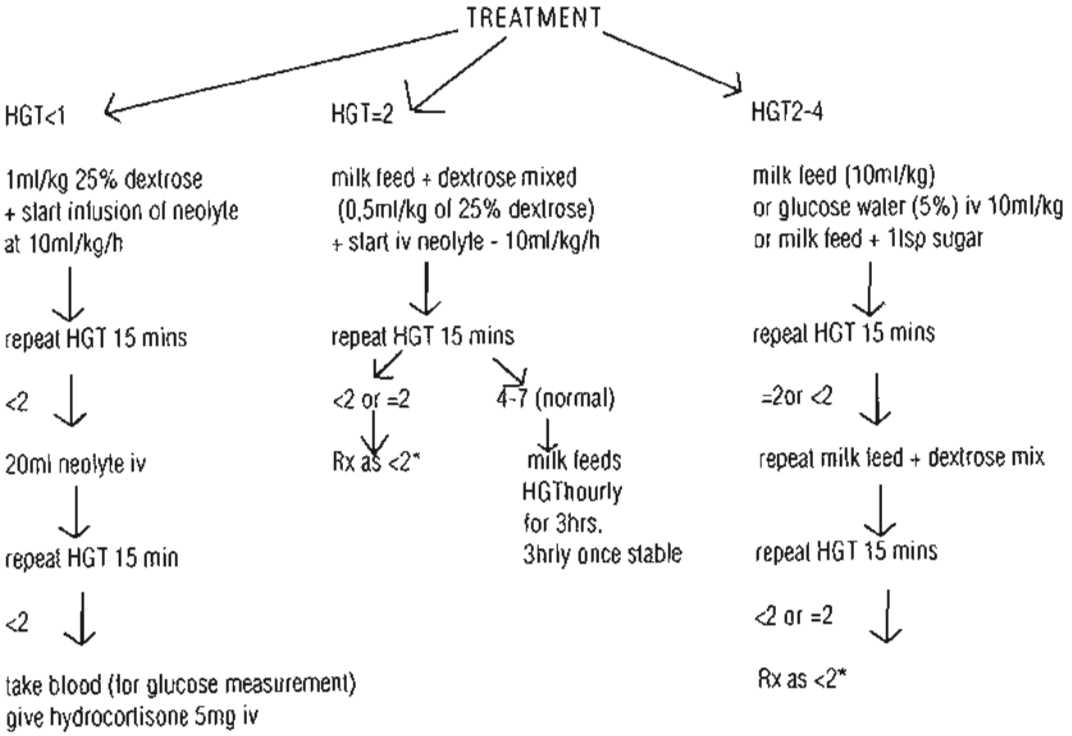
Repeated hypoglycaemia —>?metabolic cause—>refer

HGT monitoring: should be as above until on 100ml/kg/day milk feeds (teach mum to do it if possible).

HYPERGLYCAEMIA = >9,7MMOL/L

Due to: neonatalyte over - infusion, PVH, stress reaction, often to infection.

Causes: polyuria, dehydration, PVH Rx 5% dextrose, milk feeds, if >20mmol/l despite this then infuse insulin at 0,05 - 0,1 Units/kg/hr & strictly monitor the HGT to detect hypoglycaemia 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

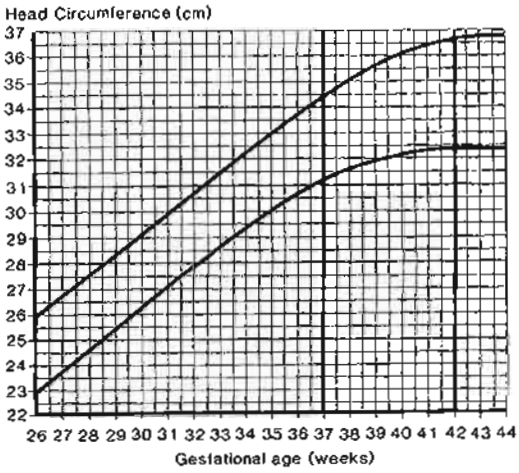
All babies <2,5kg are low birth weight (LBW). They may be *Pre-term* or *Under-weight-for-(gestational)-age*. There are important clinical differences between the two groups -

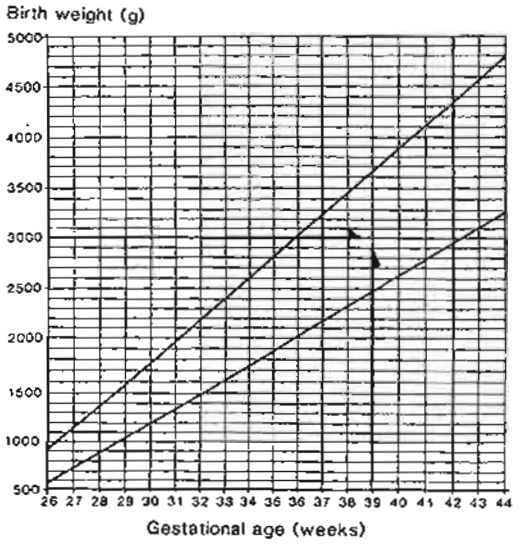
	PRE-TERM	UWFA
Nutrition	adequate	poor
Mental	dull	alert/hungry
Suck	no	yes
Skin	thin	crinkly
Oedema	yes	no

and each has different problems -

	PRE-TERM	UWFA
Hypothermia	yes++	yes
Hypoglycaemia	yes	yes++
Asphyxia	yes	yes++
RDS	HMD/PDA	Mec/Asp
Apnoea	yes	no
Infection	yes++	yes
Hb	high	low

HEAD CIRCUMFERENCE AND WEIGHT--FOR- GESTATIONAL-AGE CHARTS





Source and Further Reading

Woods DL (ed.) 1998 *Perinatal Education Programme: Manual II - Newborn Care'*, 'Gestational Age and Weight, Skills Workshop 17, pp. 6,7

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.

SPLEN SCORE

	0	1	2	3
SKIN	smooth, pink, thin, few veins seen	few creases, light brown,few veins	deep creases, brown thick	wrinkles brown peeling
PLANTAR CREASES	anterior of sole	2/3 of sole	whole	sole
LABIA / TESTES	minora<majora scrotum empty	minora = majora inguinal testes	minora<majora testes high in scrotum	minora covered testes deep
EAR	slow recoil	ready recoil	instant recoil	quick recoil
NIPPLE	flat	bud <5mm	bud >	5mm

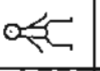
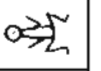
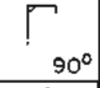
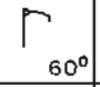
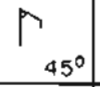
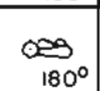
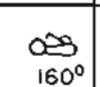
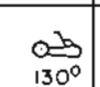
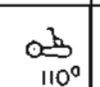
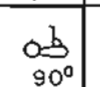
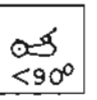
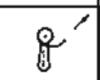
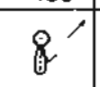
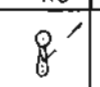
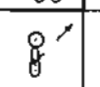
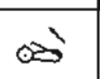
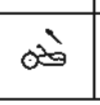
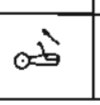
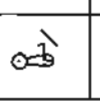
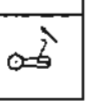
Add the scores of the 5 factors together and add the result to 30 to obtain the gestational age (if >30w)

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.

	0	1	2	3	4	5
Posture						
Square Window (wrist)	 90°	 60°	 45°	 30°	 0°	
Arm Recoil	 180°		 100°-180°	 90°-100°	 <90°	
Popliteal Angle	 180°	 160°	 130°	 110°	 90°	 <90°
Scarf Sign						
Heel to Ear						

Skin	gelatinous red, transparent	smooth pink, visible veins	superficial peeling, &/or rash few veins	cracking pale area rare veins	parchment deep cracking no vessels	leathery cracked wrinkled
Lanugo	none	abundant	thinning	bald areas	mostly bald	
Plantar Creases	no crease	faint red marks	anterior transverse crease only	creases ant. 2/3	creases cover entire sole	
Breast	barely percept.	flat areola no bud	stippled areola 1-2mm bud	raised areola 3-4mm bud	full areola 5-10mm bud	
Ear	pinna flat, stays folded	sl. curved pinna; soft & slow recoil	well-curv pinna; soft but ready recoil	formed & firm & instant recoil	thick cartilage ear stiff	
Genitals ♂	scrotum empty no rugae		testes descending few rugae	testes down good rugae	testes pendulous deep rugae	
Genitals ♀	prominent clitoris & labia minora		majora & minora equally prominent	majora large minora small	clitoris & minora completely covered	

BALLARD SCORE CHARTS

Score	Wks.
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44

BALLARD TABLE

Source and Further Reading

Woods DL (ed.) 1998 *Perinatal Education Programme: Manual II - Newborn Care* , 'Gestational Age and Weight, Skills Workshop 17', pp. 4,5.

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 [illegible] These may indicate low glucose or calcium or have no identifiable cause.

	JITTERS	FITS
Eye deviation or other eyesigns	No	Yes
Responds to stimulation	Yes	No
Dominant movement	Tremor*	Rhythmical jerking**
Movements stop when limb gently flexed	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 (<2.2mmol/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 O_2 - bag and mask if not breathing or still cyanosed in oxygen
- Check HGT immediately: if <or =2 treat as below
- Empty stomach with NGT, nurse prone
- Keep warm
- Give an anti-convulsant:
  a) Phenobarbitone 20mg/kg iv over 5mins
  b) If still fitting after 10 mins give further phenobarb 10mg/kg iv
  c) [illegible] Diazepam 0.2mg/kg iv
  d) [illegible] Phenytoin 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 <or = 2 and infant is fitting: - 3ml/kg 10% dextrose/ neonatalyte IMMEDIATELY and continue with infusion of this @ 6ml/kg/hr for one hour or until HGT >or = 4
- Then reduce the rate to 4ml/kg until HGT stable
- Check HGTs hourly until >or = 4 and before any change in rate of transfusion.

HYPOCALCAEMIA

- If Ca <1.7 mmol 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 FDPover 30 mins.

SUSPECTED OR CONFIRMED MENINGITIS

Benzylpenicillin (200 000u/kg/d) and gentamycin for 14 days or ceftriaxone (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
Phenobarbitone 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 hyperbilirubinaemia.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 Coomb'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 Addinglon. Anaemia due to continuing haemolysis may be a problem as late as 6 months so Hb should be monitored monthly and tranfusion 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.

CLUB FOOT

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/meningomyelocoele and arthrogryphosis (signified by other joint contractures).

Management

Very mild deformities may be treated with strapping (use traction kit strapping, not elastoplast, 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 tenotomy at this time. If deformity remains severe, then further surgery will be required.

If conservative methods fail then a postero-medial release of soft tissues is carried out at six months and the foot maintained in POP for a futher 8 weeks.

Late-diagnosed deformities not responding well to the above methods can undergo a triple (Dunn's) arthrodesis in adolescence.

FORENSIC MEDICINE

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 fortnightly 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 patients' 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:

- Ampulees, 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 Pietermaritzburg.

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 provided 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 GW 24/56 and annex. Lung function data are not essential.

The forms should be forwarded to George Mbonde P.O. Box 4584, Johannesburg 2000
For further information phone ☎ 011 4036322

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 36% 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 *'supression 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 pressurise 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 /or penetrate the peritoneal cavity.
- Lacerations of the hymen. The hymeneal 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 odour 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 for 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 incident/s . 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 Mtubatuba. 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 Comissioner of child welfare, even if charges are not contemplated.

Further Reading
1) Winship WS, Key JA and Jacob WAS 1987 'Examination of sexually abused children ' *S A M.J.* 71:437
2) Larsen JV,Chapman JA and Armstrong A 1996 'Child sex abuse in a rural population' *S.A.M.J.* 86:1432

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.

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 unfounded 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 *douched, 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* for *bacteriology* in the hospital lab and a second *plain damp swab* for 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
- *Ovral* 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
1) Craven SA 1996 'Assessment of alleged rape victims - an unrewarding exercise' *S.A.M.J.* 86:237
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 Mtubaluba 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.

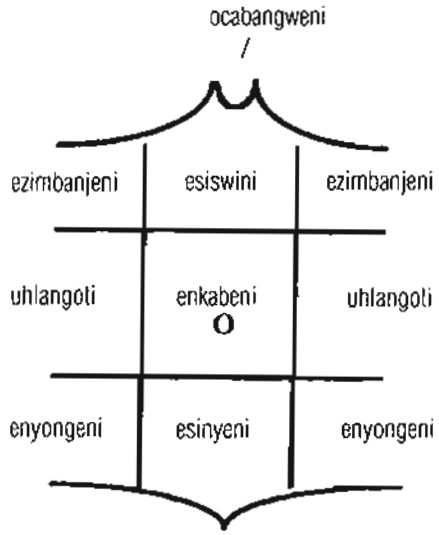
Take note of any suggestive signs of recent trauma or injury. Urine and blood may be taken for toxicology examination but poisoning has historically been a virtually impossible diagnosis to prove in our magistrature.

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 BI-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-mortems 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.

MEDICAL ZULU



COMPARTMENTS OF THE ABDOMEN IN ZULU

BASIC MEDICAL ZULU

The following is a short phrasebook designed for use in consultations. The quicker you are able to communicate at a basic level in Zulu the sooner you will be able to pick up on many aspects of the consultation you would otherwise miss, and the more you will enjoy your work. Attempts to speak with the patients in their own language are generally greatly appreciated and something of a surprise to many!

Sawubona / Sanibonani - Hello (s/pl)
Ngena / Ngenani - Come in (s/pl)
Hlala phansi - Take a seat
Unani? / Uphethwe yini? / Yini ngawe? - What are you suffering from?
Ngiyagula / Angiphilile kahle / Angizizwa kahle - I am sick / not feeling well
Kwaqala nini? - When did this start?
Isikhathi esingakanani? - How long?
Sekunesikhathi / Kade ngagula - I have been sick for a long time
Kuqale ngesonto / ngenyanga edlule - It started last week/ month
Kwaqala kanjani? - How did this start?
Kwenzeke nini lokhu? - When did this occur?
Kwenzeke kuphi? - Where did it happen?

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
Ngiyopha - I am bleeding
Ngiyashisa - I have a fever/feel hot
Ngiyajuluka - I am sweating
Ngiyakhwehlela - I am coughing
Zinjani izikhwehlela? - What is the sputum like?
zimhlophe - white
ziluhlaza - green
zinegazi - blood-stained
Nginciphile emzimbeni - I am losing weight
Ngiphelelwa amandla - I am losing strength
Ngiyaqhaqhazela/Ngiyagodola - I am shivering/having chills
Nginenzululwane - I am feeling dizzy
Nginenxeba - I have a wound

The negative construction for simple statements like those listed is formed by adding the prefix *a-* or *ka-*, deleting the *-ya-* particle and adding the suffix *-i* in place of *-a (e.g. angihudi, angiqubuki)*

Ngephukile - I have a fracture
Nginethumba - I have an abscess
Ngiyaqubuka - I have a rash
Ngihuzukile - I have a bruise
Ngiyahuda - I have diarrhoea (adults)
Uyakapalala - She has diarrhoea (children)
Ukaka kangaki ngelanga? - How many times a day do you pass stools?
Uyakangaki ngaphandle? - How many times a day do you pass stools?
Anjani amakaka? - What are your stools like?
Ngiyaphalaza/hlanza - I am vomiting
isisu siqumbile - my abdomen is distended / I am constipated
angisuzi - I am not passing any flatus
Ngedlulwe yinyanga - I have missed a period
Ngiphuma isisu - I had a miscarriage
Ngicela ukukuhlola - I would like to examine you
Ngeke ngikulimaze - I won't hurt you
Khumula - Take off your clothes
Gqoka - Put on your clothes
Lala ngomhlane/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 uku* - (I request you to) or by adding the suffix *ke* to the listed commands.

Yehla - Lie flat
Phenduka - Roll over
Phefumula - Breathe in and out
Bamba Umoya - Hold your breath
Khipha Umoya - Breathe out
Donza Umoya - Breathe in
Khamisa - Open wide
Yithi 'aahh' - Say 'aahh'
Qoshama - Open your legs wide
Yenza kanje/ngapha - Do it like this (When all else fails!)
Khululeka/Thambisa - Relax
Unganyakazi - Stay still
Ungakhathazeki - Don't worry

Ngikhombise lapho kubuhlungu khona - Show me where it hurts
Ungitshele uma uzwa ubuhlungu - Tell me when you feel pain
Uzosinda -You're going to be well
sizohlola igazi - we will take a blood test
Kufanele uzolinda imiphumela - you must wait for the results
Sizokuhlola umchamo wakho - we'll take a urine test

'You must' is usually expressed by the construction *Kufanele* - or *Kumele*- followed by a verb or infinitive. 'I need' is expressed in most circumstances by *ngidinga* + an object or infinitive.

Uzolashwa - You will be treated
Nanku umuthi wakho - here is your medicine
Phuza amaphilisi zonke izinsuku - Take the tablets every day
Ungaweqisi lo muthi - don't take more than is prescribed
Ekuseni - in the morning
Emini - at lunchtime
Ntambama - in the afternoon
Ebusuku - at night
Phuza lo muthi... - take this medicine....
kabili ngosuku - twice a day
kathathu ngosuku - three times a day
kane ngosuku - four times a day
Phuza umuthi/gwinya amaphilisi ungakadli - take the medicine/tablets before meals
Phuza umuthi/gwinya amaphilisi usudlile - take the medicine/tablets after meals
Phuza umuthi/gwinya amaphilisi uma uzwa ubuhlungu - take the medicine/tablets when you feel pain
Consisela lo muthi endlebeni - put these drops in your ears
Kufanele unciphe - You must lose weight
Gqoka/sebenzisa iKhondom/iJazi - Wear/use a condom
ngicela ubuye mhlaka - 14 February - I would like you to come back on 14 February
Khumbula usuku lokubuyela esibhedlela - Remember your next appointment
Ungalukhohlwa - Don't forget (the date)!

Uzohlinzwa - You need an operation
Sizokuthunga inxeba - we will stitch your wound
Ngizochasisa - I will explain
Ngidinga imvume yakho - I need your consent
Sizokhipha izitishi ngomhlaka - 2 May/ezinsukwini ezinhlanu - we will take the stitches out on 2 May/in 5 days
Kusobala? - Is it clear?
Unemibuzo? - Do you have any questions?

PARTS OF THE BODY

umzimba - body
ikhanda - head
isimongo/ibunzi - forehead
ubuso - face
ishiya - eyebrow
ikhala - nose
isihlathi - cheek
umlomo - mouth
udebe - lip
ulimi - tongue
umhlathi - jaw
isilevu - chin
i/amaZinyo - tooth/teeth
intamo - neck
i/amaHlombe - shoulder/s
in/izingalo - arm/s
i/izindololwane - elbow/s
um/imikhono - forearm/s
isi/izihlakala - wrist/s
is/izandla - hand/s
umu/iminwe - finger /s
isibindi - liver
ubuchopho - brain
ucikicane - little finger
umphimbo - throat
uqhoqhoqho - windpipe
isifuba - chest
amaphaphu - lungs
inhliziyo - heart
izimbambo - ribs
isisu - stomach
ithumbu/isibilini - intestine
isinye - bladder
inyongo - gall bladder
umhlane/iqolo - back
isi/izinqe - buttock/s
i/amathanga - thigh
um/imilenze - leg/s
i/amadolo - knee/s
i/amaqakala - ankle/s
u/izinyawo - foot/feet
u/izinzwane - toe/s
isi/izithende - heel/s
isithupha - thumb
isi /izinso - kidneys
isibeletho - uterus

SELECTED DISEASES

Wake waphathwa yini? - Have you ever suffered from?
Uphuzo - alcoholism
Umfutho wegazi/i'high-high' - hypertension
Isifo socansi - sexually transmitted disease
Udropu - gonorrhoea
Isimungumungwane - measles
Isifuba somoya - asthma
Inqubulunjwana - chicken pox
Ingculaza - HIV/AIDS
Isankonkonku - whooping cough
Uqhuqho or Umalaveva - malaria
Isilepero - leprosy
Isichenene - schistosomiasis
Isisu segazi - dysentery
iTB - TB
Ukufa kwaBantu, e.g. Idliso - Zulu diseases often with no exact European equivalent

HOSPITAL FORMULARY

HOSPITAL FORMULARY

GASTROINTESTINAL SYSTEM

Drug:

Magnesium Hydroxide Mixture
AluminiumHydroxide 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

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)
Ipratropium nebuliser 0.25 /2 ml & 0.5/2 ml
Saline nose drops
Compound Benzoin Tincture
Simple Linctus
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
Frusemide 40 mg tablets
Frusemide 20 mg injection
Reserpine 0.25 mg tablets
Atenolol 50 mg tablets
Hydrallazine 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
Verapamil 5 mg vials for injection
Dopamine 200 mg/5 ml vial for injection
Heparin 5000 u/5 ml for injection
Warfarin 5 mg tablets

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

ANTI-INFECTIVES

Drug:

Phenoxymethylpenicillin 250 mg tablets
Benzylpenicillin 1 Mu and 5 Mu vials
Procaine penicillin 3 g/10 ml vials
Benzathine penicillin 2.4 Mu vial
Ampicillin injection 500 mg vial
Amoxycillin 250 mg capsules and 125 mg/5 ml syrup
Cloxacillin 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 1g suppositories
Doxycycline 100 mg capsules
Ciprofloxacin 250 mg tablets
Nalidixic acid 500 mg tablets and 250 mg/5 ml syrup
Nitrofurantoin 50 mg capsules
Gentamicin 40 mg/ml ampoules for injection
Ceftriaxone 1g vials
Cefazolin 500mg vials for injection
Ketoconazole 200 mg tablets
Fluconazole 50mg capsule
Clo-trimazole 500 mg vaginal tablets
Fansidar (Sulphadoxine 500 mg/Pyrimethamine 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
Albendazole 200 mg tablets
Nystatin suspension 100,000 u/ml

TUBERCULOSIS DRUGS

Drug:
Rifampicin 150 mg, 450 mg and 600 mg tablets
Isoniazid 200mg and 300 mg tablets
Pyrazinamide 500 mg tablets
Ethambutol 400 mg tablets
Ethionamide 250 mg tablets
Streptomycin 1g vial for injection

ANAESTHETIC AND RESUSCITATION DRUGS

Drug:
Ketamine 50 mg/ml injection
Diazepam 10 mg/2 ml injection
Thiopentone 500 mg vials
Suxamethonium 100mg/2 ml vials
Alcuronium 10 mg /2 ml
Neostigmine 2.5 mg/ml ampoules
Atropine 0.5 mg/ml ampoules
Halothane
Adrenaline 1mg/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/ FAMILY PLANNING

Drug:
Oxytocin 1u/ml and 10u/ml vials
Oxytocin 5u + 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
Dinoprost 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
Microval (Levornogestrel 30 µg) 28s
Medroxyprogesterone acetate 5 mg tablets
Norethisterone 5mg tablets
Prempak-N 0.625/5 mg and 1.25/5 mg 28s
Cyproterone acetate 100 mg tablets
Magnesium Sulphate 50% injection 2 ml ampoules
Mefenamic 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
Carbemazepine 200 mg Sustained release tablets and syrup 100 mg/5 ml
Phenobarbitone 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
Clothiapine 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

Zuclopenthixol decanoate 200 mg/1 ml injection
Orphenadrine 50 mg tablets
Amitriptyline 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 elixir
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 /1 ml, 50 mg/1 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
Benzoyl peroxide 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
Benzyl benzoate 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
Clobetasol 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
Acyclovir 3% ointment
Timolol 0.25% - 0.5% drops
Levubunolol 0.5% drops
Pilocarpine1%, 2% & 4 % drops
Acetazolamide 250 mg tablets
Cyclopentolate 1% drops
Atropine 1% drops
Phenylephrine 10% drops
Oxybuprocaine 0.4% drops

EAR, NOSE AND THROAT

Drug:

Acetic acid 1% ear drops
0.9% Saline nose drops
Glycerine ear drops
Beclomethasone 50 µg 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
Thiamine 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 Gluconate elixir (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


INDEX

INDEX

Note: The index excludes names of drugs found in the text and in the hospital formulary.

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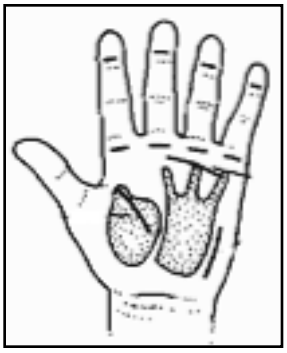
NOTES

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 may 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.

Web-Site Addresses

www.global-help.org
www.orthobooks.org

Send comments, questions, or photo-graphs of a HELP publication in use to:
questions@global-help.org

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