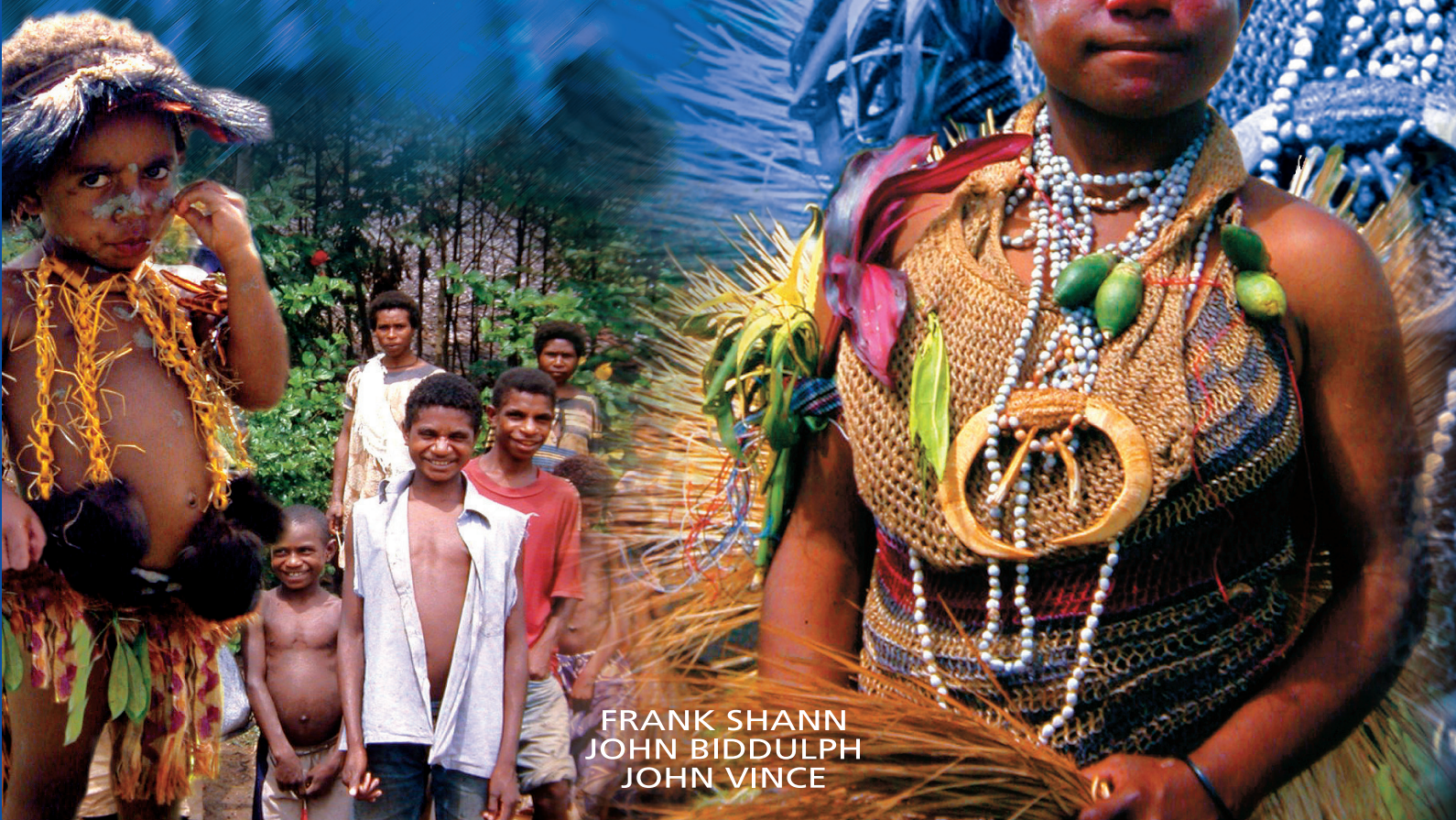


A GUIDE FOR DOCTORS PROVIDING HEALTH SERVICES FOR CHILDREN

PAEDIATRICS FOR DOCTORS IN PAPUA NEW GUINEA



FRANK SHANN
JOHN BIDDULPH
JOHN VINCE

Second Edition 2003

PAEDIATRICS FOR DOCTORS IN PAPUA NEW GUINEA

**A GUIDE FOR DOCTORS PROVIDING HEALTH SERVICES
FOR CHILDREN**

FRANK SHANN

Professor and Director of Intensive Care
Royal Children's Hospital, Melbourne, Australia
Formerly Specialist Medical Officer (Paediatrics)
Goroka Base Hospital, Papua New Guinea

JOHN BIDDULPH

Formerly Senior Specialist Medical Officer (Paediatrics)
Health Department of Papua New Guinea
and
Professor of Child Health
University of Papua New Guinea

JOHN VINCE

Professor of Child Health
University of Papua New Guinea
Formerly Specialist Medical Officer (Paediatrics)
Health Department of Papua New Guinea

SECOND EDITION 2003

Copyright © 2003 by Frank Shann and John Vince

ISBN 9980-85-411-1

Cover photographs by Trevor Duke

Illustrations by Peter Kempthorne

Published by the PNG Department of Health with the funding assistance of AusAID

JOHN BIDDULPH

Sadly, John Biddulph died at the age of 62 in January 1998, well before this second edition of Paediatrics for Doctors in Papua New Guinea was published. The first edition of the book was a result of collaboration between John and Frank Shann, who was working as a Paediatrician at Goroka Base Hospital, and incorporated much of John's original book for Resident Medical Officers. John was involved in the early stages of the preparation of the Second Edition.

John Biddulph spent almost the whole of his professional life working towards improving the health of Papua New Guineans - particularly Papua New Guinean children and their mothers. He was a man of extraordinary vision, determination and dedication, qualities coupled with those of compassion and self discipline. Many of the ideas he believed in were years ahead of their time - but without exception these ideas and his stance on important issues of health care and medical and nursing education have been proven correct. Whilst the reasons for the substantial improvements in the health of children between the 1960s and the early 1980s were many and complex, there is no doubt that John Biddulph and his ideas and drive played a substantial role.

John saw paediatrics and child health not just as a hospital specialty but as a subject covering all areas of health care, education, and community and political involvement. He saw that doctors working in Papua New Guinea must not only be excellent clinicians, but also supporters and educators of health staff, and advocates for children at community and government levels. He wrote a number of practically based textbooks, including Paediatrics for Health Extension Officers and Nurses, Paediatrics for Resident Medical Officers and the first edition of the current book. As the first Professor in Child Health at the University of Papua New Guinea, he set the foundations of the undergraduate and postgraduate courses in child health. In addition, he was instrumental in the establishment of the Post-Basic Paediatric Nursing course, and was personally involved in Health Extension Officer teaching. He was very much involved in the development of the Standard Treatment Book for Common Illnesses of Children. At the government and political level he was a prime mover in the introduction of the Baby Food Supplies (Control) Act of 1976 - the landmark legislation protecting breastfeeding in Papua New Guinea. He was also a member of many Health Department committees, such as the Immunisation Committee and Pharmaceutical Committee, contributing his wisdom and experience to the development of policy making in these areas of Public Health.

John Biddulph led by example. It has been a privilege to have worked with him as a colleague and a role model.

We hope that doctors working with children in Papua New Guinea will find this edition of Paediatrics for Doctors in Papua New Guinea useful. We hope, too, that they will appreciate and practice the broad approach to child health that John Biddulph exemplified.

John Vince
Frank Shann

ACKNOWLEDGEMENTS

Several people have helped in the production of this second edition of Paediatrics for Doctors in Papua New Guinea. Drs Noel Yaubihi and Gertrude Didei helped with the revision of the section on paediatric anaesthesia, and Dr Bage Yominao revised the section on eye diseases. Dr Paddy McMaster revised the neonatal section and contributed to a number of other sections. Dr Graham Ogle revised previous sections on paediatric endocrinology, and contributed a new section on diabetes (with Dr McMaster) and hypoglycaemia. Dr Hanny Friesen contributed the section on HIV infection. We thank all these colleagues. Whilst this second edition of Paediatrics for Doctors in Papua New Guinea was being prepared, the seventh edition of Child Health for Health Extension Officers and Nurses in Papua New Guinea by Biddulph, Stace and Danaya was also in preparation. A great deal of the practical advice contained in both books is based on the experience of paediatricians in Papua New Guinea, and we wish to acknowledge, with thanks, the indirect input of all our paediatric colleagues through the Paediatric Society of Papua New Guinea.

The use of word processing has greatly facilitated the task of preparing a second edition. It has, however, also created problems as a result of the use of different programmes at various stages and by different people. We are indebted to Kim Ang for undertaking the job of creating uniformity in presentation, if not in style.

Finally, we thank the Australian Agency for International Development (AusAID) for funding the book's production, and Dr John Christie of AusAID's PNG Health Services Support Program (HSSP) for his invaluable support.

CONTENTS

JOHN BIDDULPH	i
ACKNOWLEDGEMENTS	ii
CONTENTS	iii
PREFACE	vii
INTRODUCTION: CHILD HEALTH IN PAPUA NEW GUINEA	1
PAPUA NEW GUINEA - HEALTH INDICES.....	3
THE PROBLEMS OF CHILD HEALTH IN PAPUA NEW GUINEA.....	5
THE 10-STEP CHECKLIST - ALL CHILDREN.....	11
RECOMMENDED BOOKS ON COMMUNITY CHILD HEALTH.....	12
ABBREVIATIONS.....	13
ABDOMINAL DISTENSION	15
AMOEBIASIS	16
ANAEMIA	17
ANAEMIA - PERSISTENT OR RECURRENT	20
ANAESTHETICS	22
ANAESTHETICS - DRUG DOSES	27
ANAPHYLAXIS	29
ANTIBIOTICS.....	31
ANTISEPTICS.....	32
ARTERIAL PUNCTURE	34
ARTHRITIS (ACUTE) - ONE JOINT.....	35
ARTHRITIS OR ARTHRALGIA (ACUTE) - MORE THAN ONE JOINT	36
ARTHRITIS - CHRONIC.....	38
ASCITES - ASPIRATION - ABDOMINAL PARACENTESIS	39
ASCITES WITHOUT OTHER OEDEMA.....	40
ASTHMA.....	41
ATAXIA - ACUTE OR SUBACUTE.....	45
BIRD RESPIRATOR.....	46
BLEEDING DISORDERS IN CHILDREN.....	50
BLOOD CULTURE.....	53
BLOOD PRESSURE MEASUREMENT IN INFANTS	54
BLOOD TRANSFUSION - INDICATIONS.....	55
BONE MARROW ASPIRATION	57
BOOKS	59
BURNS	60
CANCER ORIS.....	62
CARDIAC ARREST.....	63
CARDIOMEGALY	66
CAROTID ANGIOGRAM	67
CENTRAL VENOUS CATHETERISATION.....	68
CEREBROSPINAL FLUID - EXAMINATION	70
CEREBROSPINAL FLUID - RESULTS	71
CHILD ABUSE.....	72
CHOLERA	75
CHRONIC RAISED INTRACRANIAL PRESSURE	77
CISTERNAL PUNCTURE.....	80
COLOSTOMY AND ILEOSTOMY - STOMA MANAGEMENT.....	81
COMA.....	82
CONGENITAL MALFORMATIONS AND SURGICAL REFERRAL.....	85
CONVULSIONS - ACUTE PRESENTATION.....	88
CONVULSIONS - RECURRENT.....	92
CRETINISM (ENDEMIC).....	94
CROSSMATCH OF BLOOD	95
CUT DOWN	97
DEVELOPMENTAL MILESTONES	99
DIABETES MELLITUS.....	101

DIARRHOEA.....	103
DIARRHOEA - DIAGNOSIS AND MANAGEMENT SUMMARY	107
DROWNING.....	108
ECG INTERPRETATION	109
ENDOTRACHEAL INTUBATION	113
EOSINOPHILIA	115
EQUIPMENT	117
EYES.....	122
FAMILY PLANNING	126
FEMORAL VEIN PUNCTURE.....	129
FEVER	130
FEVER OF UNKNOWN ORIGIN	131
FLUID AND ELECTROLYTE THERAPY	133
FOREIGN BODIES	137
FROG BREATHING	139
GLUCOSE 6 PHOSPHATE DEHYDROGENASE DEFICIENCY (G6PD).....	140
HEAD CIRCUMFERENCE.....	141
HEALTH CENTRE VISITS	143
HEART DISEASE - CONGENITAL.....	147
HEART DISEASE - INNOCENT (FUNCTIONAL) MURMURS.....	150
HIV INFECTION.....	151
HOSPITALISATION.....	156
HYDROCEPHALUS	158
HYPERVENTILATION	160
HYPOGLYCAEMIA	161
HYPOTHYROIDISM	162
IMMUNISATION.....	163
INFANT FEEDING	167
INTRAOSSEOUS INFUSION.....	171
INTRAVENOUS CATHETERS.....	172
INTRAVENOUS PYELOGRAM.....	175
JAUNDICE IN CHILDREN - SUMMARY	176
JAUNDICE IN CHILDREN - HAEMOLYSIS	177
JAUNDICE IN CHILDREN - HEPATITIS.....	178
JOURNALS.....	180
LACTATION - DRUGS CROSSING INTO BREAST MILK.....	181
LACTATION INDUCTION	182
LEPROSY	184
LIMP	189
LIVER BIOPSY	190
LUMBAR PUNCTURE.....	192
LUNG ASPIRATION	193
MALARIA - SUMMARY.....	194
MALARIA - TREATMENT AND COMPLICATIONS	197
MALIGNANT DISEASE.....	204
MALNUTRITION - INTRODUCTION	210
MALNUTRITION - MANAGEMENT - SUMMARY.....	213
MALNUTRITION - DIAGNOSIS.....	214
MALNUTRITION - INPATIENT TREATMENT	215
MALNUTRITION - OUTPATIENT TREATMENT.....	220
MEASLES.....	221
MENINGITIS.....	223
MENINGITIS - CRYPTOCOCCAL.....	227
MICTURATING CYSTOURETHROGRAM.....	229
MILK FEEDS.....	230
MILK MIXTURES.....	231
MYCOBACTERIUM ULCERANS.....	232
MYELOGRAM.....	233
NEONATES - ADMISSION.....	234
NEONATES - APNOEA.....	236

NEONATES - ASPHYXIA	238
NEONATES - BORN BEFORE ARRIVAL (BBA).....	240
NEONATES - CONVULSIONS	241
NEONATES - DRUG DILUTIONS	243
NEONATES - DRUG DOSES.....	244
NEONATES - EXCHANGE TRANSFUSION	247
NEONATES - FLUIDS AND FEEDS.....	250
NEONATES - INFECTION (NEONATAL SEPSIS).....	251
NEONATES - JAUNDICE	254
NEONATES - LOW BIRTH WEIGHT	256
NEONATES - MECONIUM ASPIRATION	261
NEONATES - MENINGITIS	262
NEONATES - MILK FEEDS	264
NEONATES - NECROTISING ENTEROCOLITIS	265
NEONATES - PATENT DUCTUS ARTERIOSUS.....	267
NEONATES - PHOTOTHERAPY	269
NEONATES - RESPIRATORY DISTRESS	271
NEONATES - RESUSCITATION	273
NEONATES - TETANUS	275
NEONATES - UMBILICAL VEIN CATHETERISATION	277
NEPHROTIC SYNDROME	279
NORMAL VALUES.....	280
OEDEMA.....	282
OSTEOMYELITIS, PYOMYOSITIS AND SEPTIC ARTHRITIS	284
OTITIS MEDIA	285
PAEDIATRIC RULES.....	286
PAEDIATRIC SURVEILLANCE UNIT.....	288
PARALYSIS - ACUTE.....	289
PARALYSIS - CHRONIC - FLACCID.....	293
PARALYSIS - SPASTIC (HYPERTONIC).....	295
PERICARDIAL EFFUSION AND PERICARDIOCENTESIS.....	297
PERITONEAL DIALYSIS	300
PERITONEAL TAP - DIAGNOSTIC	301
PERTUSSIS (WHOOPING COUGH).....	302
PIGBEL (ENTERITIS NECROTICANS)	304
PLEURAL EFFUSION	308
PNEUMONIA.....	310
PNEUMONIA - PROLONGED.....	314
PNEUMOTHORAX	315
POISONING	317
POISONING - BITES AND STINGS.....	333
POISONING - PLANTS	335
POISONING - SEAFOOD INGESTION.....	336
PRIMARY HEALTH CARE	337
RABIES.....	339
RENAL FAILURE - ACUTE	340
RENAL FAILURE - CHRONIC.....	343
RENAL TUBULAR ACIDOSIS	345
SEPTIC SHOCK	346
SEXUALLY TRANSMITTED DISEASE	347
SKIN DISEASES	349
SNAKEBITE.....	351
STRIDOR.....	354
STRONGYLOIDIASIS.....	356
SUBACUTE SCLEROSING PANENCEPHALITIS (SSPE).....	358
TETANUS.....	360
THALASSAEMIA.....	362
TRACHEOSTOMY	364
TROPICAL SPLENOMEGALY SYNDROME (TSS).....	366
TUBERCULOSIS	367

TYPHOID.....	373
ULTRASONOGRAPHY.....	375
UPPER RESPIRATORY TRACT INFECTION (URTI).....	376
URETHRAL CATHETER.....	377
URINARY TRACT INFECTION AND URINE COLLECTION	378
URINARY TRACT INFECTION - TREATMENT.....	381
URINE TESTS	382
VAGINAL DISCHARGE (PREPUBERTAL).....	384
WHEEZING	385
WHITE CELL COUNT OVER 40,000 PER CMM.....	386
YAWS	387
DRUG DOSES - HOSPITAL.....	389
IV AND ORAL FLUIDS	397
INDEX.....	398

PREFACE

This book provides practical advice about how to deal with the day to day problems that face doctors responsible for the health of children in Papua New Guinea, and is based on the practical experience of the authors and their colleagues. Most of the information is directly relevant to the care of children in other developing countries. The book is complementary to Paediatric Priorities in the Developing World by David Morley, and no attempt has been made to duplicate Morley's discussion of how to organize the delivery of health care in countries such as Papua New Guinea.

The book is not intended to be comprehensive; the management of some conditions is discussed briefly or not at all because it is intended that the reader refer to a standard work such as Stanfield's Diseases of Children in the Subtropics and Tropics, Nelson's Textbook of Pediatrics or the Oxford Textbook of Medicine. In some cases, the investigations and management suggested in this book will not be possible at a small hospital; referral might then have to be considered, in consultation with a specialist paediatrician. A number of procedures are described that are only infrequently performed at hospitals, but they are included because, when they are indicated, it is often difficult to find information on how to do them, particularly in children.

The book deals mainly with curative hospital paediatrics. As can be seen from the Introduction (p.1) the authors do not regard curative hospital paediatrics as the only function of doctors working with children in Papua New Guinea. You are strongly urged to read:

- Morley D. Paediatric Priorities in the Developing World, Butterworths (at least up to p.75) and Lancet 2:1012-1014,1976.
- King M. Medical Care in Developing Countries, Oxford University Press (at least the first 13 chapters), Lancet 1:679-681,1972, and
- the Introduction to this book (p.1).

This second edition retains the problem-based format of the first. It has been updated and includes several new sections.

References are by no means exhaustive. Some are to original work, some deal with matters of controversy, some are up to date recommendations, and some are reviews. Doctors are encouraged to become familiar with and to use Medline searches and to use the Internet to keep up with new developments.

INTRODUCTION: CHILD HEALTH IN PAPUA NEW GUINEA

In spite of the recent developments in the mining and oil industries and the high profile given to urban centres in the media, it is important to understand that Papua New Guinea is still - in 2002 - a predominantly rural country, with 85% of the population living in rural villages. Furthermore, a substantial proportion of the population in the urban areas is socially and financially disadvantaged. More than 40% of the population is less than 15 years old, and more than 15% less than 5 years. Most deaths in children - both in rural areas and in urban hospitals - are caused by acute infectious diseases, such as pneumonia, malaria or diarrhoea, that can be prevented or cured by simple and inexpensive means. Malnutrition often contributes to death from these diseases. Whilst effective and cheap preventative and curative strategies are known and should be readily available, there are major difficulties in getting services to the people.

Most of a doctor's medical education is carried out in an urban setting. Great attention is paid to conditions and topics which are especially "interesting" and "topical". The majority of teaching materials - textbooks and journals - are produced in countries with very different disease patterns and far greater resources and levels of health expenditure than Papua New Guinea's. It is imperative that doctors training and working in Papua New Guinea have a very firm grasp of the base on which the country's health services has been founded and developed. Important Health Indicators for the country are shown on p.3.

THE ROLE OF DOCTORS

Doctors responsible for children in Papua New Guinea have a dual role. Whilst based in urban centres and supervising hospital services (both preventative and curative), they must also equally strive to improve the delivery of health care to rural villages and to disadvantaged urban communities. To do this effectively, local beliefs and practices must be taken into account (Lancet 2:152-154,1988). Doctors cannot routinely deliver health care to rural villages and urban settlements themselves, but they must undertake a major role in training and supporting the paramedical workers who do this. Hospital staff can and should be trained to look after routine cases so that doctors are free to leave the wards and undertake their training and supportive roles.

Doctors caring for children in Papua New Guinea must:

1. Train hospital ward and outpatient staff to treat patients using Standard Treatments . Standard Treatment regimens are an essential part of the delivery of health care in Papua New Guinea (Tropical Doctor 19:126-130,1989). Doctors must NOT spend all their time treating the lucky few who are able to come to their hospital. These will consist almost entirely of children who live less than 16 km away. Think of all the others!
2. Encourage and supervise Maternal and Child Health (MCH) services in their area. Participate in regular in-service courses for Child Health Nurses.
3. Regularly visit the health centres in their area (see p.143). This is NOT just to see sick patients, but primarily to teach and support health centre staff.
4. Once or twice a year try to get the senior Health Extension Officers (HEOs) and nurses from each of the rural health centres in their area to come and work in the ward as residents for a week. They should be given plenty of responsibility and plenty of teaching.
5. Encourage and support the HEOs and Community Health Nursing staff in their area to visit and teach their primary health workers (Community Health Workers) regularly. HEOs and nurses are essential to the doctor's proper functioning. Like the doctor, they have an important responsibility to teach others. Health centres must not be little hospitals in the bush that just treat people who come to them.
6. Be prepared to support and be involved in other health initiatives affecting their communities, eg:
 - a. Participation in radio and television programmes dealing with issues of health

- b. Advise, teach and support Village Health Aides (VHAs) if a VHA programme is functioning in their area. VHAs are village people chosen by their peers for a brief training in the treatment or prophylaxis of common diseases such as pneumonia, diarrhoea, malaria and malnutrition. This involves village people in their own health care, and makes it much more widely available (see Lancet 2:1012,1976).

Think about the child's past and future health

This book concentrates on the immediate management of a child who is ill. A child's illness, however, is usually only a brief incident in his or her life. It is important that the doctor who cares for a child during an episode of illness does not neglect either the child's past or future.

The past: enquire about past illnesses, the health of the family, the child's birth history and developmental progress. Look at the child's Health Record Book, which most mothers will have with them. This book provides a valuable record of the child's past health, weight (growth) record and immunisation status. It also gives the child's birth date, so that you can work out his or her exact age.

The future: before discharging a child from hospital, write down in their Health Record Book the reason for the admission, the admission number and any specific follow-up treatment required. This will be a valuable record for the future.

Provide comprehensive care

Sick children in Papua New Guinea frequently suffer from several diseases at once. Whilst treating the child's main illness do not neglect other illnesses, eg malnutrition, anaemia or scabies. Check the child's immunisation status while he or she is in hospital, and remedy any deficiencies found. Utilise the child's time in hospital to provide the family with some health education. If the mother is pregnant, ensure that she attends the antenatal clinic. Educate families about family planning and ensure that those wishing to utilise the service attend the family planning clinic.

PAPUA NEW GUINEA - HEALTH INDICES

Demographic data

Population	5.2 million
Population density	62/sq km (inhabited area)
Crude birth rate	34 per 1,000
Population growth rate	3.2% per year
Rural population	85%
Population <15 years	42%
Population <5 years	15%
Population >65 years	1.6%

Mortality rates (per 1,000 live births)

Neonatal	35
Post neonatal	43
Infant	77
1-4 years (child)	23
Under 5 years	100
Maternal (per 1,000 deliveries)	3-4

Leading causes of death 0-10 years

1. Pneumonia
2. Malaria
3. Diarrhoea
4. Meningitis
5. Tuberculosis
6. Typhoid
7. Malnutrition
8. Perinatal asphyxia
9. Low birth weight
10. Congenital abnormalities

Other indicators - children

Low birth weight	23%
Malnutrition	35%
Proportion fully vaccinated	39%

Other indicators - mothers

Fertility (births per woman)	4.8
Professional antenatal care	78%
Professionally supervised births	53%
Tetanus toxoid during pregnancy	69%

Other indicators - water and sanitation

Adequate safe water - Urban	84%
- Rural	17%
Adequate sanitation - Urban	82%
- Rural	11%

Education indicators

Adult literacy rate	64%
Male:female literacy	4:3

Health expenditure

Proportion of government budget spent on health	8-9%
Proportion of health budget spent on rural areas	65%
Proportion of health budget spent on urban areas	35%
Average per capita health expenditure	24-30 kina
Average per capita spent on rural dweller	25 kina
Average per capita spent on urban dweller	75 kina

Theoretical health facilities and manpower

Aidpost	1 per 1,700 population
Health centre or subcentre	1 per 8,500 population
Hospital bed	1 per 1,000 population
Access to health facility	96%

Health manpower

Community health worker	1 per 1,700 population
Nurse	1 per 1,300 population
Health extension officer	1 per 16,500 population
Doctor	1 per 15,500 population

Supervision (or lack of it!)

68% of all health facilities were not visited by a doctor in 1995.
41% of all health facilities were not visited by a provincial health officer in 1995

SOURCES

1996 National Demographic and Health Survey, National Statistical Office
1996 Strengthening Reproductive Health Services, Baseline Survey of 4 Provinces UNFPA
1996 National Health Plan 1996-2000 Vol 1
1998 State of the World's Children UNICEF
2000 National Population Census

THE PROBLEMS OF CHILD HEALTH IN PAPUA NEW GUINEA

1. **Large numbers of children.** Approximately two fifths of the population is under the age of 15 years, one third under the age of 10 years and 15% less than 5 years. Less than 2% are over the age of 60 years.
2. **Rural population.** Most of the population lives in rural areas. Despite the drift of people to the towns, 85% of the population still lived in rural areas in 2000.
3. **Squatter settlements.** Many of those living in the urban areas live in squatter settlements with very poor hygiene facilities and present an increasing problem to the urban health services. This situation is common to many other countries (Br Med J 296:1683,1988).
4. **Poor communications.** Due to terrain and heavy rainfall.
5. **Low literacy rate.** In spite of improvements in the last few years, only 70% of children of primary school age and 15% of children of secondary school age attend school. Less girls than boys attend school.
6. **Poor sanitation.** Most areas do not have a plentiful supply of clean drinking water, nor do they have satisfactory waste disposal.
7. **Limited financial resources.** The annual per capita expenditure on health currently approximates 30 kina (whilst the amount has increased in the last year, health expenditure per capita in real terms of purchasing power has fallen since the early 1980s).
8. **Shortage of professional manpower.** The doctor-population ratio is 1 to 15,500. But most of the doctors live in urban areas; in rural areas the doctor-population ratio is closer to 1 to 50,000.
9. **Cross-cultural concepts of health.** Many villagers do not understand the germ theory of disease, but attribute illness to sorcery.
10. **Widespread malnutrition.** Malnutrition rates vary widely between different regions: malnutrition is often worst in inland lowlands areas (where there is no fishing and malaria is common), intermediate in highlands areas (where there is no fishing, but malaria is uncommon) and least in developed coastal areas (where the diet is supplemented with fish and antimalarials are available).
11. Young children frequently have more than one disease.

THE INFANT MORTALITY RATE AND UNDER 5 MORTALITY RATE

Overall, the infant mortality rate in the early 2000s approximates 80 per 1,000 live births with an under five mortality rate of between 100-120 per 1,000 live births. It is higher in remote areas and considerably lower in urban areas. About half the infant deaths are in babies less than one month old (neonatal deaths), and the other half are in infants 1-11 months old (post-neonatal deaths). These figures are typical for a developing country.

A most dramatic drop in infant mortality was documented in the Wam area of the East Sepik Province of Papua New Guinea, from 350 per 1,000 in 1959 to 75 per 1,000 in 1969. This was brought about by a combination of environmental factors and medical intervention: the ready availability of antimalarials and penicillin through the aidpost, the immunisation of infants, antenatal immunisation of mothers with tetanus toxoid, education of families (about hygiene, sanitation and nutrition) and oral rehydration for diarrhoea provided through the community health clinics. It is important to stress that it was auxiliary health workers in aidposts and clinics, supported by an active doctor, who were responsible for this remarkable fall in infant mortality. Hospital medicine played very little part.

Between 1960 and 1980 the mortality rates in Papua New Guinea dropped substantially. From the mid 1980s however, they increased, as a result of the breakdown of rural health services. In the middle 1990s the mortality rates appeared to fall slightly, possibly partly as a result of the Child Survival Programme

launched in 1992, the aim of which was to improve rural health services for children and their mothers. This fall, however, was not sustained. It remains to be seen how the new Provincial Government reforms of 1997 will affect health care and mortality rates, but early signs are not very promising.

THE COMMON CHILDHOOD ILLNESSES

Ninety per cent of hospital paediatric admissions are because of infections. Half these children are malnourished. The problem is basically one of infection plus malnutrition, each of which adversely interacts on the other.

The six common infections causing hospitalisation of children are pneumonia, diarrhoea, malaria, meningitis, tuberculosis and typhoid. These six infections are responsible for more than half the paediatric admissions, and four-fifths of hospital paediatric deaths. As well as being a large group, young children are also a vulnerable group. Each of these six infections can be readily recognised and treated by nurses or health extension officers.

A list of the common childhood serious illnesses (excluding the newborn) both in hospital and in the community is:

- Pneumonia and other respiratory tract infections
- Malaria
- Diarrhoea (or gastroenteritis)
- Tuberculosis
- Anaemia
- Meningitis
- Malnutrition
- Measles
- Intestinal worms
- Skin infections (sores, scabies, tinea)
- Accidents
- Typhoid
- Conjunctivitis
- Otitis media (acute and chronic)
- Whooping cough

Again, it is stressed that all the above diseases can be readily diagnosed and managed by paramedical workers who have been adequately trained. A few of the more serious cases may need transfer to a hospital staffed by a doctor. The paramedical workers can recognise those cases that need referral, and send them on after providing initial treatment.

Papua New Guinea is unfortunately in the early stages of what will be a very major HIV epidemic. Children with HIV infection are likely to present predominantly with the common illnesses rather than with the more esoteric features associated with AIDS in developed countries.

PAEDIATRIC NURSES

Many hospitals have one or more paediatric nurses. These nurses have received post-basic training in paediatrics. They are able to screen, diagnose and treat the common illnesses in children. Many have had years of practical experience and are likely to know more about the practical aspects of diagnosing and caring for sick children than an inexperienced doctor. They provide invaluable assistance to doctors and should be highly respected. Much of the doctor's work relating to sick children can be delegated to the paediatric nurse.

THE IMPORTANCE OF PREVENTION

Most of the common childhood illnesses can be prevented. When they do occur, they can be managed by standard treatment regimens close to people's homes. Prevention requires immunisation, health supervision, improved nutrition, better sanitation, health education, family spacing, maternal education and raised living standards.

COMMUNITY HEALTH CLINICS

About 78% of mothers receive some professional antenatal care, and about 53% of mothers receive some professional help with childbirth - usually in a hospital or health centre. About 90% of children under one year old are enrolled at community health clinics, many of which are run by the churches. In theory, most villages should receive a monthly visit; larger or more accessible villages should be visited more often, and more remote villages are likely to receive fewer visits. In practice, health clinics in many areas function intermittently or not at all. Attendance at community health clinics varies widely from place to place.

At present, about 90% of young children have received BCG vaccine, but less than two-thirds have received a third dose of triple antigen or Sabin vaccine, or routine measles vaccine. Less than 40% have been fully immunised.

The basic health care for children provided by community health clinics should be:

1. Supervision of growth using a weight chart.
2. Health education on nutrition, hygiene and family spacing.
3. Immunisation: BCG, triple antigen, Sabin, measles, hepatitis B (and, hopefully, *Haemophilus influenzae*).
4. Treatment for common diseases.
5. Referral of children with major illnesses to health centre or hospital.
6. Supervision of outpatient treatment for tuberculosis and leprosy.
7. Home visiting, particularly for "at risk" children who have failed to attend the clinic.
8. Records: both vital statistics (births, deaths) and work output (numbers seen, immunisations given).

All Health Workers should be familiar with and use the TEN STEP CHECK LIST in the front of the Standard Treatment Book (p.11).

PRIMARY HEALTH CARE

It is now accepted that everyone has the right to a basic level of health care. It is also accepted that health care should be fully integrated with the activities of other sectors involved in community development, and that the community itself should be actively involved in the formulation and implementation of its health care activities. Primary health care is, therefore, defined as "essential health care made universally accessible to individuals and families in the community by means acceptable to them through their full participation, and at a cost the community and country can afford".

The role of the doctor is to supervise and support the auxiliary staff who provide the primary health care.

The role of the hospital is to back up the community health services by providing secondary and tertiary care for patients referred to it by primary health care workers.

PRIORITIES FOR CHILD HEALTH SERVICES

Taking the above factors into account, it should be clear that the priorities for child health services are:

1. Trained auxiliary staff, effectively supervised, and adequately supported.
2. Simple, inexpensive drugs and equipment.
3. Mobility - a mobile service going out into the community.
4. Cooperation and working with the community.
5. Fitting in with the cultural beliefs and life-styles of the community.

It is vital that doctors working in Papua New Guinea appreciate these priorities. The problems and solutions in providing health care for rural villagers are quite different from traditional medical practice. Yet the unthinking tendency is to transpose primary health care as practised in the city hospital outpatient department or physician's private clinic to the rural health centre. The results are a health care system that is "over-centralised, over-professionalised, over-fragmented, over-expensive and over-mystified".

The following facts must be faced up to:

1. Most sick children are not brought to the hospital or health centre.

2. Rural villagers and doctors have quite different perceptions about health and disease.
3. Doctors are often either ignorant of or ignore the villagers' attitudes, beliefs and customs concerning health and disease.
4. Health is usually low on the list of villagers' perceived needs.
5. Doctors' training has often focused on the illnesses of the urban elite, not the promotion of the health of the rural poor. The doctor has been conditioned to see (and to publish) rare and exotic diseases. Yet the children of the rural areas and of the urban poor have a monotonous plethora of respiratory infections, diarrhoeal diseases and skin infections aggravated by malnutrition, anaemia and polyparasitism.

Clearly, doctors cannot meet the mass health care needs of villagers. Their training has rendered them unsuitable, and their cultural gap with the villagers is too great. Also they are far too expensive. The doctor's role is to organise the health team and to assess and help attempt solutions to the health problems of the whole community. Doctors must be brave enough to leave the sanctuary of their "disease palace" (hospital) with its expensive and sophisticated technology, and travel out to the world of the rural villages and urban settlements, where the action is.

TRADITIONAL CHILD-REARING PRACTICES

It is essential that doctors realise that many of the traditional child-rearing practices in Papua New Guinea are incomparably more biologically based than those of present western society. Two important examples are the care of children in hospital by their families and breast feeding.

Mothers looking after their own children in hospital provides the following advantages:

- Breast feeding can be maintained
- Physical care provided
- Emotional support given
- Mothers provide a 24 hours per day nurse for their child at no cost
- The mother can be taught about nutrition, hygiene, family spacing etc
- Because the baby sleeps with the mother, the mother's body will keep the baby warm at night.

This traditional practice of relatives caring for their children in hospital must be encouraged. Never place young children in "metal cages" (cots). Let them sleep with their mothers on low beds.

BREAST FEEDING

This is much the best way for a baby to be fed (see Jelliffe DB and Jelliffe EFP, Human milk in the modern world, Oxford University Press, 1978). It is impossible ever to produce from cow's milk a breast milk substitute mimicking human milk. Breast milk is safe, simple and inexpensive. It contains antibodies and other substances that protect the baby from infections, and contains growth factors for the developing brain. It contains all the nutrients required by the baby for the first four to six months of life. It also has a major family planning effect due to lactational amenorrhoea. Every effort must be made to promote breast feeding and to discourage artificial feeding. In Papua New Guinea, feeding bottles, teats and infant feeding cups (with perforated spouts) may only be obtained legally on prescription from a registered pharmacist. The Papua New Guinea Health Department Infant Feeding Policy prohibits the use of feeding bottles or feeding cups in hospitals and health centres. If a baby has to be artificially fed, it is far safer to use a cup and spoon.

It is important to note that, in conformity with WHO recommendations, the current Health Department policy in relation to women infected with HIV is that they should be encouraged to breast feed. This is because the risks of the baby dying from infection and malnutrition as a result of not breast feeding are considered greater than the relatively small risks of acquiring HIV infection through breast feeding. Current understanding of the balance of risks is that breast feeding in HIV positive women should probably continue for around 6 months. For the individual HIV infected woman with an appreciation of the requirements for artificial feeding and the means to support it, the doctor may well choose to advise this. Artificial feeding is not, however, synonymous with bottle feeding. Reduction of perinatal transmission by giving nevirapine to mother and baby is likely to be introduced in the foreseeable future.

STANDARD TREATMENT BOOKS

Standard Treatment books have been used in Papua New Guinea for more than 25 years and are an integral part of paediatric practice. The treatments are revised regularly by the Paediatric Society of Papua New Guinea, and they are effective, practicable and safe. Doctors are very strongly encouraged to use these treatments. There are occasions when a different treatment is indicated - but in such instances the doctor must be able to fully justify why he or she has deviated from the standard treatment. The Paediatric Society welcomes suggestions for changes or additions to the book, but it is important that, if changes are made, the new treatments should have proven superiority to what is already there, and that everyone changes at the same time. Doctors should use the books themselves and encourage their health worker colleagues to do likewise.

SELECTIVE INTERVENTION PROGRAMMES

The most well known of these interventions is the **GOBI** campaign, sponsored by UNICEF (GOBI stands for Growth Monitoring, Oral Rehydration Solution, Breast Feeding and Immunisation). There can be little doubt that this campaign has had a major beneficial impact - but there has also been considerable debate as to whether or not selective interventions are the best way to improve child health. Many would argue that the four components of GOBI - all very important - should be fully integrated as parts of overall child health programmes, rather than seen as a specific “package”, and there is currently a move by WHO and UNICEF towards a more broad based and inclusive approach. The GOBI acronym was expanded to GOBI-FFF and the campaign included fertility regulation, or family planning, female literacy and, where appropriate, food supplementation.

The **Baby Friendly Hospital Initiative** is another highly publicised initiative of UNICEF. It has made significant impact in some countries in which breastfeeding was uncommon, but the long term outcome remains to be determined. In Papua New Guinea, where almost all mothers breast feed from birth the campaign has been important in focussing attention on those areas such as antenatal education and the importance of immediate postdelivery contact between mother and baby which have in the past been less than ideal in some hospitals and health centres.

Vitamin A Supplementation during infancy has resulted in significant benefits for children in areas of the world where clinical and sub-clinical vitamin A deficiency is prevalent. Whilst clinical Vitamin A deficiency is uncommon in Papua New Guinea, the prevalence of sub-clinical deficiency is high enough to warrant a vitamin A supplementation programme (which is now approved policy).

INTEGRATED MANAGEMENT OF CHILDHOOD ILLNESS (IMCI)

This is the current WHO and UNICEF programme designed to improve child health worldwide. It has three components, improving individual patient management, improving health care delivery and health system management, and improving community knowledge and health practices (Lancet 350:1226,1997). In the first component, the aim is to provide not only curative but also preventative health care to the child and family. The programme is based on a checklist which includes the child's nutritional and immunisation status. The programme has used some of the ideas including the 10-step checklist, which were delivered during the child survival programme. The current 10-step checklist (p.11) has been modified by the Paediatric Society in conjunction with WHO and the Health Department from the original, first produced by Dr Keith Edwards. Recently, a checklist for use in the assessment and care of neonates and young infants has been introduced.

REFERENCES

- Adekan DA, Adeodu OO, Thurnham DI. Ann Trop Paediatr 17:223-227,1997. Comparative effects of malaria and malnutrition on plasma concentrations of antioxidant micronutrients in children.
- Bang AT, Bang RA, Beitule SB et al. Lancet 354:1955-1961,1999. Effect of home-based neonatal care and management of neonatal sepsis on neonatal mortality: a field trial in rural India.
- Cash R et al. The UNICEF GOBI-FFF Programme. London: Cromm Helm.

Review. *Trans Roy Soc Trop Med Hyg* 83:10-18,1989. Targeted interventions.

Danaya R. *PNG Med J* 38(3) 159-162,1995. Childhood disabilities in Papua New Guinea.

Duke T. *Lancet* 354:1291-1294,1999. Decline in child health in rural Papua New Guinea.

Duke T, Michael A Mgone J et al. *Bull World Health Organisation* 80(1):16-25,2002. Aetiology of child mortality in Goroka, Papua New Guinea.

Garner P, Heywood P, Baea M et al. *PNG Med J* 39(1):6-11,1996. Infant mortality in a deprived area of Papua New Guinea: priorities for antenatal services and health education.

Hosseini S, Biswas R, Kabir I et al. *Br Med J* 316:422-426,1998. Single dose vitamin A in acute shigellosis in Bangladeshi children: randomised double blind controlled trial.

King M, Elliott C. *Br Med J* 315:1441-1443,1997. To the point of farce: a Martian view of the Hardinian taboo - the silence that surrounds population control.

Nacul LC, Kirkwood R, Arthur P et al. *Br Med J* 315:505-510,1997. Randomised, double-blind, placebo controlled trial of efficacy of vitamin A treatment in non-measles childhood pneumonia.

Saskia de Pee, West CW, Mulihal et al. *Lancet* 346:75-81,1995. Lack of improvement in vitamin A status with increased consumption of dark green leafy vegetables.

Semba RD, Munasir Z, Beeler J et al. *Lancet* 27:1330-1332,1995. Reduced seroconversion to measles in infants given vitamin A with measles vaccination. (Editorial commentary by Ross D, p.1317-1318).

Shankar AH, Genton B, Semba RD et al. *Lancet* 354: 203-209,1999. Effect of vitamin A supplementation on morbidity due to *Plasmodium falciparum* in young children in Papua New Guinea: a randomised trial.

Tulloch J. *Lancet* 354 (suppl 11):16-20,1999. Integrated approach to child health in developing countries.

WHO Collaborative Study Team on the Role of Breastfeeding on the Prevention of Infant Mortality. *Lancet* 355:451-455,2000. Effect of breastfeeding on infant and child mortality due to infectious diseases in less developed countries: a pooled analysis.

THE 10-STEP CHECKLIST - ALL CHILDREN

Listen carefully to what the mother says but also always check 10 things about **every child** that you see at the clinic or aidpost:

STEP		*Page
1	ASK ABOUT FEEDING, ASSESS GENERAL APPEARANCE Decide if the child looks TOO SICK	2
2	ASK ABOUT COUGH, LOOK AT THE CHEST Check for fast breathing and chest indrawing	85
3	ASK ABOUT DIARRHOEA, CHECK FOR DEHYDRATION Look at the eyes, mouth and check skin elasticity	30
4	ASK ABOUT FEVER, FEEL FOR NECK STIFFNESS If in doubt, do a lumbar puncture	68
5	ASK ABOUT MEASLES, LOOK FOR SIGNS OF MEASLES Look for rash, runny nose, red eyes, mouth sores	66
6	ASK ABOUT TIREDNESS, CHECK FOR PALLOR Look at palms, nails and conjunctiva	5
7	ASK ABOUT EAR PROBLEMS Look for ear discharge	77
8	CHECK THE WEIGHT Look at the weight graph	57
9	ASK ABOUT FEEDING Check feeding habits	63
10	ASK FOR IMMUNISATIONS Check the immunisation record	44



**DISCUSS WITH THE MOTHER/PARENTS THE PROBLEMS THAT YOU HAVE FOUND,
THE TREATMENT THAT YOU WILL GIVE AND WHAT THEY SHOULD DO.**

*Page numbers of appropriate sections in Standard Treatment Book.

RECOMMENDED BOOKS ON COMMUNITY CHILD HEALTH

UNICEF. The state of the world's children. Oxford: OUP, annual. (Sometimes available from UNICEF office in Port Moresby).

Children, women and families in Papua New Guinea. A situation analysis. Pt Moresby: UNICEF and PNG Dept Health, 1996.

Standard treatments for common illnesses of children in PNG . 7th ed. Pt Moresby: Paediatric Soc PNG, 2000.

Stanfield P. Diseases of children in the subtropics and tropics. 4th ed. London: Edward Arnold, 1991.

Morley D. Paediatric priorities in the developing world. London: Butterworth.

King M. Medical care in developing countries. Oxford: OUP, 1966.

Williams C. Mother and child health. Oxford: OUP, 1994.

Morley D. My name is Today. London: MacMillan, 1986.

Amonoo-Lartson R, Ebrahim GJ, Lovel Hand Rankin JP. District health care. London: MacMillan, 1996.

Werner D. Where there is no doctor. London: MacMillan, 2000.

Abbatt F, McMahon R. Teaching health care workers. London: MacMillan, 1993.

Biddulph J, Stace J, Danaya R. Child health for health extension officers and nurses in Papua New Guinea. 7th edition. Pt Moresby: Dept Health, 1998.

Useful Addresses

TALC. Teaching Aids at Low Cost. PO Box 49, St Albans, Herts AL1 5TX, UK. Fax: 054 41 727 846852. E-mail: talc@talcuk.org. Website: www.talcuk.org. Supplies individual books, library sets, tape slide sets, and other teaching aids at very low cost. Produces a new catalogue each year.

Healthlink Worldwide. Cityside, 40 Adler Street, London E1 1EE, UK. Website: www.healthlink.org.uk. This organisation provides free booklets and information on a number of important issues including child health and AIDS.

ABBREVIATIONS

See also Antibiotics - symbols (p.31).

ADH	antidiuretic hormone	IV	intravenous
AFB	acid fast bacilli	IVP	intravenous pyelogram
AI	aortic incompetence	JVP	jugular venous pressure
ALT	alanine amino transferase	LP	lumbar puncture
Amp	ampoule	LTB	laryngotracheobronchitis
ANF	Antinuclear factor	MCH	maternal and child health
AP	Anteroposterior	MCH	mean corpuscular haemoglobin
APH	Antepartum haemorrhage	MCHC	mean corpuscular haemoglobin concentration
APO	aidpost orderly	MCT	medium chain triglyceride
ASD	atrial septal defect	MCV	mean corpuscular volume
ATS	antitetanus serum	MDI	Metered Dose Inhaler
BD	twice a day	MI	mitral incompetence
BP	blood pressure	MI	morphological index
BS	blood slide	MOF	milk oil formula
BT	bleeding time	MP	malarial parasites
Bx	biopsy	MUAC	mid upper arm circumference
Cap	capsule	Mx	Mantoux
CCF	congestive cardiac failure	NG	nasogastric
CHNS	child health nursing services	No.	number
CNS	central nervous system	OIC	officer in charge
CoAo	coarctation of aorta	OPD	Outpatient Department
CPAP	continuous positive airways pressure	ORS	oral rehydration solution
CSF	cerebrospinal fluid	PAN	polyarteritis nodosa
CT	clotting time	PDA	patent ductus arteriosus
Cx	cervical	PEEP	positive end expiratory pressure
CXR	chest x-ray	PHD	Public Health Department
EBM	expressed breast milk	PHEO	Provincial Health Extension Officer
ECG	electrocardiograph	PHO	Provincial Health Officer
EDTA	edetate disodium	PNG	Papua New Guinea
EMO	Epstein-Macintosh-Oxford	PPD	purified protein derivative
ENL	erythema nodosum leprosum	pr	per rectum
ESR	erythrocyte sedimentation rate	PT	prothrombin time
ETT	endotracheal tube	PTT	partial thromboplastin time
FBE	full blood examination	QID	four times a day
FDP	fibrin degradation products	RBC	red blood cell
FG	French gauge (outer diameter in mm x 3)	RDS	respiratory distress syndrome
FSM	full strength milk	RHC	rural health centre
FSS	full strength Sunshine (milk)	SBE	subacute bacterial endocarditis
G6PD	glucose 6 phosphate dehydrogenase (deficiency)	SBR	serum bilirubin
Hb	haemoglobin	SFD	small for dates
HbF	foetal haemoglobin	SGA	small for gestational age
HEO	health extension officer	SGOT	serum glutamic-oxalacetic transaminase
HIV	human immunodeficiency virus	SLE	systemic lupus erythematosus
HMD	hyaline membrane disease	SMO	specialist medical officer
HPD	high protein diet	SPPS	soluble plasma protein solution
HPF	high power field	SSMO	senior specialist medical officer
ICP	intracranial pressure	STD	sexually transmitted disease
IM	intramuscular	SVM	70% alcohol
INAH	Isoniazid	SXR	skull x-ray
IPPR	intermittent positive pressure respiration	Tab	tablet
		TB	tuberculosis

TDS	to be taken 3 times a day	UPNG	University of PNG
TGV	transposition of great vessels	URTI	upper respiratory tract infection
TID	three times a day	VDRL	Venereal Diseases Research Laboratory
TIG	tetanus immunoglobulin	VHA	village health aide
TOF	tracheo-oesophageal fistula	VMA	vanillyl mandelic acid
TPNG	Territory of Papua and New Guinea	VSD	ventricular septal defect
TSS	tropical splenomegaly syndrome		

ABDOMINAL DISTENSION

ACUTE DISTENSION IN INFANTS

Acute abdominal distension is common in small children with severe sepsis such as pneumonia, meningitis or septicaemia. Hypokalaemia is common in these children; measure serum potassium if this assay is available. If serum potassium cannot be measured but you can do an ECG, the ECG will show flat T waves if there is hypokalaemia.

Acute abdominal distension requiring surgery is uncommon in infants, but is suggested by bilious vomiting (very suggestive of obstruction in a neonate), distension with active bowel sounds, or signs of peritonitis (particularly rebound tenderness). Perforation can be detected by the presence of gas under the diaphragm on an erect chest x-ray (NOT an erect abdominal x-ray). Surgical causes to remember include intussusception and obstructive bands - often in association with malrotation.

1. If intranasal oxygen is being given, make sure the catheter is not pushed in too far (into the oesophagus) and that the flow rate is not more than 1 litre per minute.
2. Pass a size 8 nasogastric tube and aspirate. Give nil by mouth.
3. Give IV fluids. If hypokalaemia is present or suspected, up to 0.4 mEq/kg/hour of KCl (1 gram = 13.3 mEq = 13.3 mmol) can be infused IV in an emergency, but 0.2 mEq/kg/hour is much safer.
4. Give IV ampicillin and gentamicin (unless there is meningitis in which case give chloramphenicol). Abdominal distension can be due to chloramphenicol toxicity (gray syndrome) in babies up to 6 months old.
5. Watch carefully for signs of mechanical obstruction.

ACUTE DISTENSION AFTER 12 MONTHS OF AGE

In older children in the Highlands of Papua New Guinea, acute abdominal distension may be caused by enteritis necroticans, or pigbel (p.304). Pigbel is always accompanied by severe abdominal pain. Appendicitis is becoming more common and is a well recognised cause of peritonitis. In children less than 2 years of age it may present with diarrhoea and vomiting rather than with abdominal pain. Typhoid should also be considered.

CHRONIC ABDOMINAL DISTENSION

Chronic abdominal distension in children in Papua New Guinea is usually due to hepatosplenomegaly, ascites, Hirschsprung's disease or neoplasm.

Hepatosplenomegaly is usually caused by tropical splenomegaly syndrome (p.366), thalassaemia (p.362), leukaemia, or, rarely, storage diseases. Miliary tuberculosis is also associated with hepatomegaly.

Ascites: see pages 39, 40.

Hirschsprung's disease: refer for rectal biopsy.

Neoplasm: the neoplasms that most often cause abdominal distension are Wilm's tumour, Burkitt's lymphoma, neuroblastoma, and non-Hodgkins lymphoma.

AMOEBIASIS

This is disease due to infestation with *Entamoeba histolytica*. An asymptomatic carrier state is common in many countries. Some strains of *E histolytica* are non-pathogenic (Lancet 1:561,1988).

DIARRHOEA OR DYSENTERY

There is diarrhoea with variable amounts of blood and clear mucus. There may be moderate fever and abdominal pain. Repeated examination of fresh stool may be necessary to make the diagnosis: look for trophozoites in the acute stage and cysts during remission. The treatment for amoebiasis is tinidazole 50mg/kg as a single dose daily for 3 days, or metronidazole 15 mg/kg TID for 5 days. High fever and pus in the stools suggests Shigellosis or Campylobacter rather than amoebiasis.

LIVER ABSCESS

This is uncommon in children. It presents with fever and liver tenderness (usually of the right lobe). In many cases the right diaphragm is raised, there is right shoulder tip pain, or there are right basal crepitations or a small effusion. Less than half such patients have *E histolytica* in their stools. The abscess may rupture into the peritoneum, pleura, lungs or pericardium. Haematogenous spread may occur to brain, lung or spleen.

1. Give tinidazole 50 mg/kg daily for 5 days or metronidazole 15 mg/kg TID for 10 days.
2. If the abscess is on the point of rupturing it is best to aspirate, under ultrasound guidance if possible. Otherwise surgical drainage is not now recommended.

REFERENCES

Stanfield JP. Diseases of children in the subtropics and tropics. 4th ed. London: Arnold, 1991, p.739.
Strachan WD. Lancet 1:561-562,1988. A test for pathogenic *E histolytica*.
Price M. Diseases and health services of PNG, PHD, 1973, p.351.

ANAEMIA

Definition: haemoglobin (Hb) below 10 g/dl.

See also Anaemia - Persistent or Recurrent, p.20.

TESTS

If the child is well apart from anaemia, it is reasonable to do only a haemoglobin, then treat with iron, antimalarials, folic acid and albendazole. Repeat the Hb in 4 weeks. If there is no improvement, further investigation is indicated (see p.21).

More comprehensive investigation includes:

- a blood film and red cell indices (MCV, MCH, MCHC)
- a blood slide for malarial parasites

Other investigations may be indicated as follows:

- | | |
|---|-------------------------------------|
| • hepatosplenomegaly or bone changes: | Hb electrophoresis or HbF |
| • lymphadenopathy, and/or purpura: | bone marrow biopsy |
| • jaundice: | see the section on jaundice (p.176) |
| • a family history of TB, weight loss or cough: | Mantoux, CXR, 3 gastric aspirates |
| • severe illness and high fever | blood cultures (SBE, typhoid). |

The great majority of children with anaemia have iron deficiency anaemia as shown by the hypochromic microcytic red blood cells seen on the blood film. A dimorphic blood film (hypochromic microcytic red blood cells and macrocytic red blood cells) is quite common. Polymorphs are often hypersegmented, and this together with the macrocytes suggests a folate deficiency. This dimorphic blood film is most suggestive of iron deficiency in the presence of infection, particularly in a malnourished child. Malaria is a major contributor to anaemia. Hookworm may also contribute, especially in children over 2 years old.

TREATMENT

A child with a Hb 5-10 g/dl is usually treated as an outpatient. A child with a Hb under 5 g/dl is usually admitted.

Antimalarials

1. Treatment

For mild to moderate anaemia (Hb 5-10 g/dl), give the standard 3-day treatment for uncomplicated malaria (infant Camoquin/chloroquine daily for 3 days and single dose Fansidar).

For severe anaemia (Hb <5 g/dl), give the standard treatment for complicated/severe malaria.

2. Prophylaxis

Give prophylactic infant Camoquin or chloroquine weekly for 12 weeks.

Iron

There is some evidence that iron therapy lowers resistance to infection; on the other hand, iron deficiency may impair immunity (for good reviews, see Br Med J 296:660-4,1988 and Ann Trop Paediatr 18(S):S81-S87,1998). In children with anaemia and severe sepsis (eg pneumonia, meningitis), packed cells should be transfused, but it is best to withhold Imferon until the infection has resolved, especially if the child is malnourished. Do not forget to give the Imferon later. Iron deficiency may affect mental development and behaviour (see J Pediatr 102:519,1983 and Indian J Pediatr 51:427-428,1984).

1. Oral iron

Oral iron can be given if compliance can be assured. The dose of elemental iron is 6 mg/kg/day for four weeks (ferrous sulphate 1 mg = 0.3 mg elemental iron). MAKE SURE that the parents understand the need to keep iron tablets in a safe place.

2. Intramuscular

Iron may be given by deep intramuscular injection of iron dextran (Imferon) (but oral iron therapy, if compliance can be assured, is preferable).

Weight (kg)	Doses of Imferon (ml)				Total
	Day 1	Day 2	Day 3	Day 4	
3 - 5.9	3	-	-	-	3
6 - 9.9	3	2	-	-	5
10 - 14.9	4	3	-	-	7
15 - 19.9	4	4	2	-	10
20 - 29.9	4	4	4	3	15
>30	5	5	5	5	20

It is kinder to give half the dose (max 2.5 ml) into each buttock.

3. Total Dose Infusion

- In older children where more than 10 ml of Imferon is required, it can be given intravenously as a Total Dose Infusion (TDI).
- Add the required amount of Imferon to 100 ml of 4.3% dextrose saline and run in slowly through an intravenous drip.
- Anaphylaxis is a recognised though uncommon complication of TDI. Make sure you have the facilities available for management of an anaphylactic reaction. (see p.29).

DO NOT GIVE IMFERON if the child:

- is less than 1 month old
- has severe malnutrition
- is very sick
- has a fever above 38 °C.

Give the Imferon when the child has improved. If a blood transfusion is given to treat anaemia, the doses of Imferon are smaller (see Standard Treatment Book).

Folic acid

Give 1 tablet each week for 3 months (at the same time as prophylactic antimalarials).

Albendazole

Give albendazole (for hookworm).

Diet

Encourage the mother to give protein food (eg meat, fish) and dark green leafy vegetables.

Chronic infection

Treat any chronic infection

Blood transfusion

See also p.55. Transfuse any child with a Hb less than 3 g/dl.

ONLY transfuse children with a Hb of 3-6 g/dl if they have:

- severe infection (severe pneumonia, severe acute malaria, meningitis or TB)
- OR heart failure (hepatomegaly and pulse over 160)
- OR kwashiorkor.

Blood transfusion is necessary in anaemia caused by acute blood loss from haemorrhage or haemolysis. It is NOT usually necessary in chronic iron deficiency anaemia with a Hb of 3 g/dl or more. When blood is transfused for chronic anaemia, packed cells should be used if possible. If whole blood is used, then double the volume will be needed.

Discard any blood left out of the refrigerator for more than 12 hours. Always give antimalarials after any blood transfusion. You must still give iron, antimalarials, albendazole and folic acid, even after giving a transfusion.

Weight (kg)	Volume of packed cells (ml)	Rate		Frusemide
		ml/hr	drop/min	
3-5	100*	20	5	0.5 ml (5 mg)
6-9	150*	25	7	0.75 ml (7.5 mg)
10-14	250*	50	15	1 ml (10 mg)
15-19	400**	75	20	1.5 ml (15 mg)
20-29	500**	100	25	2 ml (20 mg)
*Crossmatch 1 unit of packed cells		**Crossmatch 2 units of packed cells		

REFERENCES

- Bojang KA, Van Hensbroek MB, Palmer A et al. Ann Trop Paediatr 17:355-359, 1997. Predictors of mortality in Gambian children with severe malarial anaemia.
- Bojang KA, Palmer A, Boele van Hensbroek M et al. Trans Roy Soc Trop Med Hyg 91(5):557-561, 1997. Management of severe malarial anaemia in Gambian children.
- Choudhry VP. Indian J Pediatr 51:427-428, 1984. IQ and iron deficiency.
- Crane GG. Am J Clin Nutr 27:1079, 1974. Pathogenesis of anaemia in PNG.
- Oppenheimer SJ. Ann Trop Paediatr 18(S):S81-S87, 1998. Iron and infection in the tropics.

ANAEMIA - PERSISTENT OR RECURRENT

SUMMARY

The crucial test is the reticulocyte count.

Reticulocytes over 2%

Exclude **blood loss**: history, PR, stool occult bloods.

If there is no blood loss, the child has **haemolysis**:

- looks sick (see p.177): suspect malaria, septicaemia or autoimmune disease
- not very sick: suspect malaria, autoimmune disease, G6PD or thalassaemia.

Do a blood slide, Coomb's test, G6PD assay and Hb electrophoresis or HbF.

Reticulocytes 2% or less

Aplasia: do a marrow biopsy.

Stop drugs causing aplasia.

Look for an underlying illness: leukaemia, TB, uraemia, SBE, Still's disease or lead poisoning (all may cause a mild reticulocytosis).

Check that the child actually got iron, antimalarials, folic acid and albendazole.

DIAGNOSIS

Reticulocytes: THIS IS THE CRUCIAL TEST.

Full blood examination and platelets.

Blood slide.

If the reticulocyte count is over 2%, the child has blood loss or haemolysis:

- exclude **blood loss**. Ask about haematemesis, melaena or blood in the stool. Do a PR yourself and inspect the faeces. Check again that iron was given. Test the stools for occult blood 3 times if the blood film is hypochromic and microcytic despite iron
- if there is no blood loss, the child has **haemolysis**:
 - looks sick (see p.177): suspect malaria, septicaemia or autoimmune disease
 - not very sick: suspect malaria, autoimmune disease, G6PD or thalassaemia. Do a blood slide, Coomb's test, G6PD and Hb electrophoresis or HbF.

If the reticulocyte count is 2% or less, the child has aplastic anaemia:

- do a bone marrow biopsy
- stop any drugs known to cause aplasia, eg cytotoxic drugs, indomethacin, gold, aspirin, chloramphenicol, sulphonamides, streptomycin, isoniazid, tetracycline, chlorpromazine, chlorothiazide or carbamazepine
- look for an underlying chronic illness: eg leukaemia, TB, uraemia, SBE, SLE, Still's, lead poisoning (all may occasionally cause a mild reticulocytosis).

IMPORTANT CAUSES OF PERSISTENT OR RECURRENT ANAEMIA

1. Resistant malaria

This is defined as falciparum trophozoites (not gametocytes) present within 14 days (28 days in low endemicity areas, for example in a large city) of an **adequate and supervised** course of antimalarials.

Treat for Treatment failure malaria (see p.199).

2. **Recurrent malaria**

This suggests that maintenance weekly antimalarials were not taken, or that the child has resistant malaria.

3. **Haemolysis**

Suggested by a stable or falling haemoglobin with reticulocytes over 2% (3% in a child less than 12 months old) in the absence of blood loss:

- looks sick (see p.177): malaria, septicaemia or autoimmune disease
- not very sick: malaria, autoimmune disease (p.177), G6PD (p.140), or thalassaemia (p.362).

4. **Leukaemia**

Do a marrow biopsy if leukaemia is suggested by the blood film, thrombocytopaenia, purpura, lymphadenopathy or hepatosplenomegaly.

5. **Gastrointestinal haemorrhage**

This is suggested by occult blood in the stool or frank melaena. It is usually due to oesophageal varices, duodenal ulcer, hookworm, colonic polyps, anal fissure or Meckel's diverticulum.

6. **Anaemia secondary to chronic illness**

Eg TB, chronic urinary infection, uraemia or malignancy.

If malaria, haemolysis, leukaemia and GI bleeding have been excluded, do:

urea, chest x-ray, Mantoux, 3 gastric aspirates for AFB, and keep a 4-hourly temperature chart (to detect fever from TB).

OTHER INVESTIGATIONS MAY BE INDICATED

1. Fever or cardiac murmur: blood (stool cultures (SBE, typhoid)).
2. Frontal bossing, maxillary hyperplasia, hepatosplenomegaly: haemoglobin electrophoresis or HbF for thalassaemia.
3. Fever, arthritis, butterfly rash: ANF and SLE cells, rheumatoid factor, ESR (for SLE and Still's disease).
4. Anorexia, vomiting, constipation, irritability, drowsiness, ataxia, fitting: blood lead, blood stipple cells, urine glucose, x-ray long bones (for lead poisoning).

ANAESTHETICS

PAEDIATRIC ANAESTHETICS AT SMALL HOSPITALS

A doctor confronted with the prospect of operating on a young child in a small hospital is often more worried about the anaesthetic than the surgery. It is better to avoid operating on infants under 12 months old in these circumstances if you can; delay the procedure until the child is older, or refer him to a hospital that has an anaesthetist and a surgeon.

Operative mortality is greatly increased if the Hb is less than 8 g/dl (Lancet 1:727-9,1988).

Useful references are Tropical Doctor 10:66-71,1980, chapter 22 of Medical Care in Developing Countries by M King and A Manual of Anaesthesia for the Small Hospital by FN Prior. Both of these books are available from TALC, PO Box 49, St Albans, Herts AL1 5TX, UK. Fax: 054 41 727 846852. E-mail: talc@talcuk.org. Website: www.talcuk.org.

KETAMINE

Ketamine is an extremely useful agent for the inexperienced anaesthetist. It produces adequate anaesthesia and marked analgesia without depressing respiration or blood pressure. The patient remains in reflex control of his airway, although care must be taken to ensure airway patency by keeping the chin up - especially in very small children. Vomiting is rare. Surgical anaesthesia under ketamine is difficult to describe, but fairly easy to detect. The child's eyes are open, but he has a vacant stare. Eyelash, corneal and pharyngeal reflexes remain, but he does not respond to pain.

The patient should be fasted for 4-6 hours before ketamine anaesthesia, and suction should be available. Avoid using ketamine with an inhalational anaesthetic such as halothane, because the combination may cause a low pulse rate and blood pressure.

A dose of ketamine of 5-10 mg/kg IM will cause loss of consciousness in 3-4 minutes, and an IV cannula can then be inserted if further anaesthesia is required. With IV ketamine, 1-2 mg/kg will usually induce anaesthesia for about 5 minutes; this may need to be repeated once or twice in babies if ketamine is used as the sole anaesthetic agent.

The main disadvantages of ketamine are:

- a prolonged recovery time with procedures lasting longer than about 30 minutes, particularly if the IM route is used. Always give ketamine IV if you can, and use IV infusion for longer procedures
- postoperative restlessness and hallucinations. This is rare in children under 8 years of age. Give IM diazepam if it occurs. In older patients, give diazepam 0.3 mg/kg oral preoperatively, or 0.05-0.1 mg/kg IV at induction
- hypotension and bradycardia may occur postoperatively
- profuse salivation is common, so it is usual to premedicate with atropine 0.2-0.3 mg IM
- convulsions occur occasionally
- ketamine is moderately expensive.

MINOR PROCEDURES

1. Fast the child for 4-6 hours.
2. Premedication of atropine IM 15-30 minutes before the procedure. Lower doses of ketamine will be required if pethidine is given as well as atropine, but the child will be slower to wake up and a longer period of observation will be needed.
3. IV or IM ketamine.

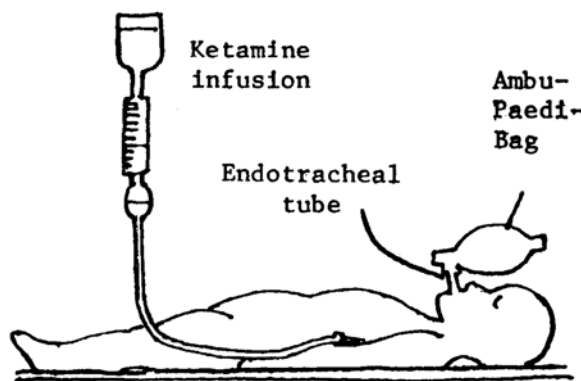
OPERATIONS LONGER THAN 30 MINUTES NOT NEEDING MUSCLE RELAXATION

1. Fast the child for 4-6 hours.
2. Give a premedication of atropine and pethidine IM 30 minutes before the operation.
3. Add 1-2 mg/kg of ketamine to 100 ml of 4.3% dextrose in 1/5 normal saline. Run this intravenously at 2-5 drops/minute until surgical anaesthesia is achieved (vacant stare, no response to pain). Then slow the drip to 1-2 drops/minute to maintain anaesthesia. Stop the ketamine infusion 5-7 minutes before the end of the operation. See PNG Med J 37:209-213,1994.

OPERATIONS NEEDING MUSCLE RELAXATION

DO NOT ATTEMPT THIS UNLESS YOU CAN INTUBATE WELL.

1. Fast the child for 4-6 hours.
2. Give a premedication of atropine and pethidine IM 30 minutes before the operation, and paracetamol 40 mg/kg rectally.
3. Induce anaesthesia with ketamine 1-2 mg/kg IV or 5 mg/kg IM.
4. Add 1-2 mg/kg of ketamine to 100 ml of 4.3% dextrose in 1/5 normal saline. Run this intravenously at 2-5 drops/minute.
5. Give oxygen via bag and mask, and paralyse the patient with suxamethonium 1 mg/kg IV (use a dilute solution of 10 mg/ml and give 0.1 ml/kg).
6. Intubate the patient, and then maintain paralysis with pancuronium 0.08 mg/kg or alcuronium 0.25-0.5 mg/kg as required.



**Ketamine
infusion
anaesthesia
with
muscle relaxation.
See Tropical Doctor
8:68-72, 1978 and
10:66-71, 1980.**

7. Ventilate the patient with an Ambu-Paedi bag. Oxygen at 1 litre/min can be fed into the bag in patients with shock, severe anaemia or lung disease. Squeeze the bag just hard enough to produce full expansion of the chest (do not squeeze too hard in small children).
8. Stop the ketamine infusion 10-15 minutes before the end of the operation.
9. At the end of the operation, reverse the muscle relaxant with IV atropine and neostigmine. NEVER give neostigmine until the child has made some spontaneous movements, showing the relaxant is starting to wear off. Extubate (with positive pressure on the bag) only when the child is fully awake and breathing normally.

PAEDIATRIC ANAESTHESIA USING A BOYLE'S MACHINE

An appropriate anaesthetic machine, paediatric breathing circuits, endotracheal tubes, masks, laryngoscopes and oximeter must be available. The medical officer giving the anaesthetic must be able to intubate, and it is highly desirable to have someone available to assist the anaesthetist.

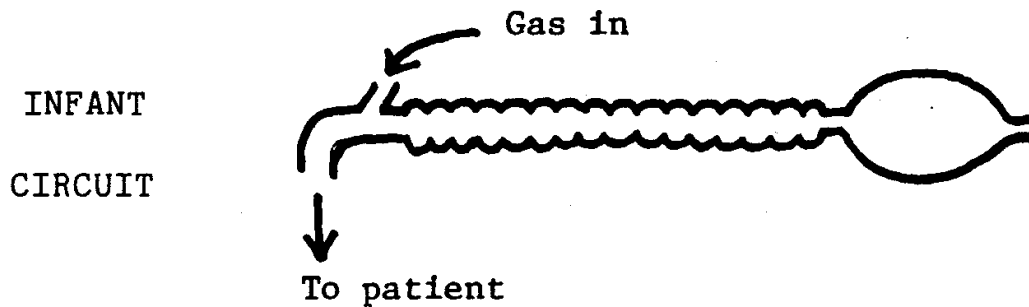
Ketamine or EMO-ether anaesthesia will often be the method of choice even at larger hospitals. However, the use of a Boyle's machine is preferable in babies under 3 months, in very ill patients and in children with upper airways obstruction due to croup or epiglottitis (see p.354).

Anaesthetic circuits on a Boyle's machine

Always use a gas flow of 3-4 litres/minute.

Up to 20 kg: infant circuit

Over 20 kg: adult circuit with inspiratory and expiratory tubes



NEONATES AND INFANTS UP TO 3 MONTHS OF AGE

DO NOT ATTEMPT THIS UNLESS YOU CAN INTUBATE WELL.

KEEP THE BABY WARM - wrap him in cotton wool and Alfoil or Glad-wrap.

REPLACE BLOOD OR FLUID LOSS.

1. Insert an IV drip 4 hours before the operation, then give nil orally.
2. If the baby has a bowel obstruction or abdominal distension, pass an NG tube, aspirate, then remove the tube just before the anaesthetic.
3. Give IV atropine. Anaesthesia is usually induced with an inhalational agent such as halothane - lower concentrations are needed in young infants.
4. Awake intubation is now rarely attempted, even in newborn babies. Babies tend to need a larger dose of suxamethonium, such as 1.5-2.0 mg/kg IV (or 3 mg/kg IM). Then intubate with an appropriate sized tube (see p.113).
5. Ventilate with an infant circuit using oxygen 1 litre/min and halothane 0.5-1.0%. If extra analgesia is needed, a small dose of 0.05 mg/kg of morphine is preferable to nitrous oxide in young infants.
6. Paralyse the baby with IV pancuronium. The response to this drug is very variable in small babies, and the dose has to be titrated against the response. Most babies need about 0.1 mg/kg. Larger doses may be needed, but the action is then often greatly prolonged.
7. At the end of the operation, reverse the pancuronium with IV atropine and neostigmine. NEVER give neostigmine until the baby has made some spontaneous movements, showing that the pancuronium is starting to wear off. Extubate (with positive pressure on the bag) only when the baby is fully awake and breathing normally.

RELAXANT ANAESTHESIA IN A CHILD OVER 3 MONTHS OF AGE

DO NOT ATTEMPT THIS UNLESS YOU CAN INTUBATE WELL.

1. Give no solids for 6 hours preoperatively, and no clear fluids for 4 hours preoperatively. Insert an IV drip 4 hours preoperatively in infants under 12 months of age.

2. Give IM atropine and pethidine 30 minutes before the operation.
3. If there is a bowel obstruction or abdominal distension, pass an NG tube, aspirate, then remove the tube just before the anaesthetic.
4. Give IV thiopentone SLOWLY - just enough to put the child to sleep (usually 5 mg/kg in an infant, and 3-4 mg/kg in an adolescent).
5. Paralyse with IV suxamethonium, then intubate.
6. Ventilate with oxygen 1 litre/min and nitrous oxide 2 litre/min, using an infant circuit for children weighing less than 20 kg and an adult circuit for those over 20 kg.
7. When the suxamethonium wears off and spontaneous respiration recommences, give IV pancuronium or alcuronium.
8. At the end of the operation, reverse the pancuronium with IV atropine and neostigmine. NEVER give neostigmine until the child has already made some spontaneous movements, showing that the pancuronium is starting to wear off.

If the child is shocked or has severe lung disease:

- do not give a pethidine premedication
- ventilate with 100% oxygen at 3 litre/min (no nitrous oxide)
- supplement the anaesthetic with halothane 0.3% OR small doses of IV pethidine (0.5 mg/kg, which is 0.1 ml/kg of pethidine 50 mg diluted to 10 ml).

NON-RELAXANT ANAESTHESIA IN A CHILD WEIGHING MORE THAN 20 KG

The use of a mask in a small child makes it difficult to maintain the airway and increases the dead space. It is better to use ketamine or a relaxant anaesthetic in children weighing less than 20 kg.

1. Give pethidine and atropine IM half an hour before operation.
2. If the child has a bowel obstruction or abdominal distension, pass an NG tube, aspirate, then remove the tube just before the anaesthetic.
3. Have the child breathe oxygen 1 litre/min and nitrous oxide 2 litre/min through the mask.
4. Gradually introduce halothane up to 2%. When the child is asleep, increase the halothane to 4% until automatic respiration has been established and no other spontaneous movement is occurring, then reduce to 2% following the surgical incision. Gradually reduce to 1% over the next hour.
5. Ensure that the airway remains patent at all times.

If halothane is not available, anaesthesia can be induced with thiopentone, then maintained with oxygen, nitrous oxide and small doses of IV pethidine (0.5 mg/kg, which is 0.1 ml/kg of pethidine 50 mg diluted to 10 ml). Halothane is an excellent anaesthetic agent. But it is rather expensive, causes bradycardia unless atropine is given, is a poor analgesic, often causes hypothermia then shivering during recovery, is incompatible with adrenaline and ketamine, and may cause spasm on extubation.

EMO-ETHER ANAESTHESIA WITHOUT RELAXATION IN CHILDREN OVER 20 KG

Ether is one of the safest anaesthetic agents for the inexperienced anaesthetist, and it is cheap. But it is highly inflammable, has a prolonged induction and recovery time and often causes postoperative vomiting.

1. Fast the child for 4 hours.
2. Give a premedication of atropine and pethidine IM 30 minutes before the operation.
3. The prolonged unpleasant ether induction can be avoided by using IV thiopentone for induction. Give only just enough to produce sleep - overdose depresses respiration, reduces the inhalation of ether and is dangerous.

4. The EMO inhaler with Oxford Inflating Bellows, a tightly-fitting face mask and an oral airway can then be used to administer an air-ether anaesthetic with the patient breathing spontaneously without the need for bottled nitrous oxide or oxygen. The EMO was specially designed for use at rural hospitals. A detailed description of its use can be found in *Medical Care in Developing Countries* by Maurice King, p22:12 to 22:18 and in *A Manual of Anaesthesia for the Small Hospital* by FN Prior.
5. After thiopentone induction, an ether-oxygen anaesthetic (usually with nitrous oxide too) can be administered with a Boyle's machine. However, the concentration of ether cannot be measured accurately on a Boyle's machine, the mixture of ether and oxygen is highly explosive and bottled oxygen is required.

SPINAL ANAESTHESIA

This can only be used when patient co-operation can be guaranteed, so that it is unsuitable in most children under 12 years of age. The technique is described by Maurice King on p22:3 to 22:12 of *Medical Care in Developing Countries*.

NERVE BLOCKS

Specific nerve blocks (eg intercostal, caudal) can be given for postoperative pain relief, but these should only be administered by an experienced person. Never use lignocaine with adrenaline for nerve blocks in the fingers, penis or any other end-artery organ.

REFERENCES

- King M. Primary anaesthesia. OUP, 1986.
Brown TCK, Fisk GC. Anaesthesia for children. Blackwell, 1992.
Yanbihi N, Oberoi GS. PNG Med J 37:209-213, 1994. Ketamine and epidural block.

ANAESTHETICS - DRUG DOSES

Anaesthetics - Drug Doses		Weight (kg)	Under 3	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
ATRACURIUM (TRACURIUM) 25 mg/2.5 ml			NB DILUTED								
➤ ADD 7.5 ml STERILE WATER			0.5	1	1.5	2.5	3.5	5	USE UNDILUTED		
• initial bolus dose (0.5 mg/kg) IV	ml		0.25	0.5	1	1.5	2	3	USE UNDILUTED		
• further doses (0.3 mg/kg) IV	ml								1.75	2.25	2.5
• UNDILUTED. Initial bolus IV	ml								1	1.5	1.5
• further doses IV	ml										
ATROPINE. Amp 0.6 mg/ml, IM or IV (0.015 mg/kg)			NB DILUTED								
• ADD 3ml STERILE WATER	ml		----- (0.1 ml/kg) -----			-	-	-	-	-	-
• Undiluted	ml		-	-	0.25	0.25	0.25	0.5	0.75	1	1
KETAMINE (KETALAR). Amp 500 mg/10 ml. Give atropine first.			NB DILUTED								
➤ SLOW IV			----- (0.2 ml/kg) -----			-	-	-	-	-	-
• take 1 ml, ADD 4 ml STERILE WATER (2 mg/kg)	ml		-	-	-	0.5	0.75	1	1.5	1.75	2-3
• undiluted (2 mg/kg)	ml										
• further injections: give half dose (1 mg/kg)											
➤ IM											
• first dose (10 mg/kg)	ml		0.2 ml/kg	0.75	1.5	2.5	3.5	5	7	9	10
• further doses (5 mg/kg)	ml		0.1 ml/kg	0.5	0.75	1.25	1.75	2.5	3.5	4.5	5
MIDAZOLAM (HYPNOVEL). 10 mg/2 ml, 15 mg/3 ml			NB DILUTED								
➤ Take 1.0 ml and ADD 4 ml STERILE WATER								1.25	1.75	2.25	2.5
• for sedation (0.01-0.1 mg/kg) IM	ml		0.15	0.2	0.35	0.5	0.75				
• for anaesthetic induction (0.15-0.3 mg/kg) IV	ml		0.5	0.75	1.5	2.5	3.5	----- USE UNDILUTED -----			
➤ UNDILUTED	ml		-	-	-	-	-	1	1.75	2	2
NEOSTIGMINE (PROSTIGMIN), IV. Give atropine first.			NB DILUTED								
• Amp 0.5 mg/ml, ADD 4 ml STERILE WATER (0.05 mg/kg)	ml		(0.5 ml/kg)	-	-	-	-	-	-	-	-
• Amp 0.5 mg/ml, undiluted (0.05 mg/kg)	ml		-	0.5	0.75	1.25	1.75	-	-	-	-
• Amp 2.5 mg/ml, undiluted (0.05 mg/kg)	ml		-	-	-	-	-	0.5	0.75	1	1

Anaesthetics - Drug Doses		Weight (kg)	Under 3	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
PANCURONIUM 4mg mg/2 ml			NB DILUTED								
➤ ADD 2 ml STERILE WATER.											
• initial bolus dose (0.06 mg/kg) IV	ml		0.1	0.25	0.5	0.75	1	1.5	2	2.75	3
• further doses (0.01 mg/kg) IV	ml		-	-	-	-	-	0.25	0.25	0.5	0.5
➤ UNDILUTED											
• initial bolus IV	ml		-	0.125	0.25	0.375	0.5	0.75	1	1.25	1.5
• further doses IV	ml		-	-	-	-	-	0.125	0.25	0.25	0.25
PETHIDINE. Amp 50 mg/ml or 100 mg/2 ml			NB DILUTED			NB DILUTED			NB DILUTED		
• IV during anaesthetic, DILUTE 1 ml with 9 ml STERILE WATER (0.5 mg/kg)	ml		----- (0.1 ml/kg) -----			1	1.5	2	3	4	5
• IM as premedication, undiluted (1-1.5 mg/kg)	ml		-	-	-	0.75	0.5	0.75	1	1.25	1.5
SUXAMETHONIUM (SCOLINE). Amp 100 mg/2 ml, IV (mg/kg)			NB DILUTED								
• ADD 8 ml STERILE WATER	ml		----- (0.2 ml/kg) -----			-	-	-	-	-	-
• Undiluted	ml		-	-	-	0.25	0.5	0.5	0.75	1	1-2
THIOPENTONE (PENTOTHAL). Amp 0.5 gram/20 ml											
• APPROXIMATE dose is 5 mg/kg IV	ml		----- 0.2 ml/kg -----			3	4	5	7	9	10
VECURONIUM 4mg/2 ml											
➤ ADD 2 ml STERILE WATER											
• initial bolus dose (0.1 mg/kg) IV	ml		0.25	0.5	0.75	1	1.75	2.5	3.54	UNDILUTED	
• further doses (0.05 mg/kg) IV	ml		-	0.25	0.25	0.5	1	1.25	1.75	2.25	2.5
➤ UNDILUTED											
• initial bolus	ml		-	0.25	0.25	0.5	1	1.25	1.75	2.25	2.5
• further doses	ml		-	-	-	0.25	0.5	0.5	1	1	1.25

ANAPHYLAXIS

This causes obstruction of upper (stridor) and lower (wheeze) airways, vasodilation and loss of plasma volume with hypotension, vomiting and inhalation, and cardiac arrhythmias.

1. **Urgently give adrenaline:** dilute 1 ml of adrenaline 1/1,000 with sterile water to make 10 ml (1/10,000).

Give 0.1 ml/kg (approximately 0.5 ml per year of life) up to a maximum of 5 ml. Give SLOWLY IV if shocked. Give intramuscularly if not shocked. Repeat IV or IM adrenaline every 15 minutes if necessary (or give as an infusion, see below).

With a sting or subcutaneous injection: inject an equal volume of adrenaline subcutaneously around the site to reduce absorption of antigen.

With an intramuscular injection of antigen: do NOT inject adrenaline into the site (it will increase absorption).

2. **Oxygen.**
3. **Pressure bandage:** if the injection or sting is into a limb, apply a firm pressure bandage and splint the limb to stop the poison spreading.
4. **Airway:** an oropharyngeal airway or tracheostomy may be required.
5. **Nasogastric tube:** insert a 10 FG tube, aspirate, then leave the tube on free drainage.
6. **Hydrocortisone:** give hydrocortisone 10 mg/kg IV stat. This does not act for some hours, but should be given for its late effect. Adrenaline is more important.
7. **Promethazine** (phenegan): 1.25 mg/kg IV slowly (0.05 ml/kg of 50 mg/2ml).
8. **IV fluid:** 20 ml/kg of saline (or Haemacel) repeated as necessary to maintain systolic blood pressure at 90 or more.
9. **Inotropic support:** If hypotension persists despite IV fluid.

Adrenaline: dose 0.05-1.0 microgram/kg/min. Add 0.6 ml/kg of 1 in 1,000 into 100 ml N saline. Run at 0.5-10 ml/hr. Adrenaline is the preferred inotrope in anaphylaxis.

Dopamine: in low doses (1-5 microgram/kg/min) produces vasodilatation and increased blood flow through the kidneys. In these doses it is often used with dobutamine. In higher doses (10-20 microgram/kg/min) it increases heart rate and increases blood pressure from peripheral constriction.

Run at 5 microgram/kg/min initially and increase to a maximum of 20 microgram/kg/min (Add 3 mg/kg to 50 ml 5% dextrose or N saline and run at 5 ml/hr, increasing to 20 ml/hr).

Dobutamine: less effective in infants than in older children. Dose 1-20 microgram/kg/min.

Isoprenaline: use 1/5,000 amps (0.2 mg/ml). Dose 0.05-0.5 microgram/kg/min.

- **Weight under 10 kg:** put isoprenaline 0.3 ml/kg in 100 ml saline in the burette. Run at 25 ml/hour (drop rate will depend on whether you are using a standard 15 drops/ml or a paediatric 60 drops/ml burette) which is 0.5 microgram/kg/min. Adjust the rate according to the BP (normal 90/60).
- **Weight 10 kg or more:** put isoprenaline 0.15 ml/kg in 100 ml saline in the burette. Run at 50 ml/hour which is 0.5 microgram/kg/min. Adjust the rate according to the BP (normal 95/70).

(NB: Accurate monitoring of inotrope infusions is essential. Use a constant flow pump if at all possible.)

10. **Inhaled salbutamol:** if the patient has a wheeze: .

- **Weight under 10kg:** 2.5 mg salbutamol to make up to 2 ml with N saline.
 - **Weight 10kg or more:** 5 mg salbutamol to make up to 2 ml with N saline.
- Give this every 15 mins or continuously if necessary.

REFERENCES

Fisher MM. Med J Aust 149:34-8,1988. Acute anaphylactic reactions.
Advanced paediatric life support. The practical approach. 3rd ed. London: BMJ Books, 2001.
Helpful guidelines for using inotropes with syringe drivers - and for many other aspects of paediatric intensive care procedures - are given in Shann FA. Drug doses. Intensive Care Unit, Royal Children's Hospital Parkville, Victoria 3052, Australia. Available from Secretary, Paediatric Society of Papua New Guinea.

ANTIBIOTICS

SYMBOLS

A	Chlortetracycline	NS	Mitroxolinum
AL	Furaltadone	NV	Novobiocin
B	Bacitracin	OB	Cloxacillin
BX	Phenethicillin	OL	Oleandomycin
C	Chloramphenicol	OX	Oxacillin
CAZ	Ceftazidine	P	Penicillin G
CB	Methicillin	PB	Polymyxin B
CEC	Cefaclor	PM	Paromomycin
CL	Cephalexin	PN	Ampicillin
CN	Gentamicin	PY	Carbenicillin
CR	Cephaloridine	RD	Rifampicin
CRO	Ceftriaxone	RL	Sulfamethoxazole
CT	Colistin Sulphate	RM	Rifamide
CTX	Cefotaxime	RV	Rifamycin
D	Demethylchlortetracycline	S	Streptomycin
DA	Clindamycin	S3	Compound Sulphonamides
DO	Doxycycline	SD	Sulphadiazine
E	Erythromycin	SH	Spectinomycin
F	Nitrofurantoin	SM	Sulphamerazine
FC	Nitrofurazone	SP	Spiramycin
FD	Fusidic Acid (Sodium Salt)	ST	Sulphathiazole
FR	Furazolidone	SXT	Sulfamethoxazole/Trimethoprim (cotrimoxazole)
FY	Framycetin	SZ	Sulphadimidine
G	Sulphafurazole	T	Oxytetracycline
GS	Methacycline	TE	Tetracycline
K	Kanamycin	TH	Sulphamethizole
KF	Cephalothin	TL	Tylosin
KY	Sulphamethoxypyridazine	TS	Thiosporin
MY	Lincomycin	VA	Vancomycin
N	Neomycin	W	Trimethoprim
NA	Nalidixic Acid		
NI	Nitroxolinum		

Antibiotics which are used systemically (or drugs like neomycin that are related to a systemic drug) should not be used topically, except in the eyes. Sores and ulcers should not be treated with topical antibiotic creams or powders.

ANTISEPTICS

ACRIFLAVINE

This is an acridine derivative. It is a slow-acting antiseptic, bacteriostatic against many gram positive bacteria but less effective against gram negative bacteria and fungi. Acriflavine emulsion is a useful treatment for infected sores and ulcers, but it should only be applied until the sore is clean because it slows healing.

EUSOL

Eusol is no longer recommended for routine use since it can damage healthy tissue. It has been replaced by chlorhexidine or normal saline (itself a good antiseptic).

CHLORHEXIDINE (SAVLON)

Chlorhexidine is the active constituent of Savlon; the cetrimide is a detergent which increases the penetration of chlorhexidine. A 1 in 100 solution of Savlon is made by adding a 20 ml sachet of Savlon Hospital Concentrate to 2 litres of water. This rather dilute solution can be used to clean sores and most wounds, but it is liable to contamination with gram negative bacteria and should be used within one week. Savlon 1 in 30 (a 20 ml sachet in 600 ml water) should be used for very dirty wounds or for cleaning equipment before sterilization.

1% chlorhexidine in 70% alcohol (Hexol) is effective in cleaning hands before surgery or at the bedside. It should be rubbed on for 2 minutes, then the hands allowed to dry by evaporation. Hexol can also be used to clean skin preoperatively. Alcoholic solutions should not be used on wounds or sores.

CRYSTAL VIOLET (GENTIAN VIOLET)

A 1% aqueous solution is effective against Candida and gram positive bacteria, but is much less active against gram negative bacteria. It is therefore a useful antiseptic for use on skin sores. Crystal violet is cheap.

HEXACHLOROPHANE 3% EMULSION (PHISOHEX)

This is used only for skin antiseptics. It is highly active against gram positive organisms, but less active against gram negative ones. Its action is slow, but repeated applications result in a progressive reduction in skin flora. Phisohehex should not be used on a neonate on more than one occasion, because hexachlorophane may be absorbed through a baby's skin and cause brain damage.

IODINE SOLUTION

This is often supplied as 1% iodine in 70% alcohol. It is very effective for skin disinfection. Occasionally, topical iodine causes a severe constitutional reaction with fever and rash. Therefore, iodine solution should be washed off the skin after use. Iodine in alcohol should not be used to treat wounds and sores: it is painful, is rapidly inactivated by tissue substances and it delays healing.

70% ALCOHOL (SVM)

This is a good antiseptic for cleaning the skin before an injection is given. Cleaning should be vigorous enough to remove any dirt present on the skin. Over 90% of the bacteria are killed if the skin is kept moist with SVM for 2 minutes. The alcohol should be allowed to evaporate before the injection is given, or it will cause pain.

RECOMMENDED ANTISEPTICS

ALLOW AT LEAST TWO MINUTES FOR THE ANTISEPTIC TO WORK.

Cleaning hands: wash well with soap and water, then apply 70% alcohol or Hexol.

Cleaning skin before injections: 70% alcohol for 2 minutes.

Cleaning skin preoperatively: 1% iodine in 70% alcohol for 2 minutes (wash off after use).

Small sores: wash with aqueous Savlon or normal saline and apply crystal violet.

Large, infected sores: wash with aqueous Savlon or normal saline, and apply acriflavine emulsion daily.

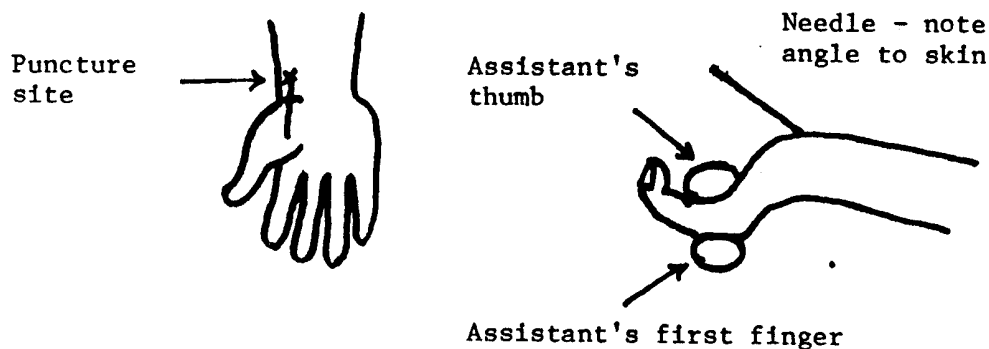
REFERENCES

Goodman and Gilman. The pharmacological basis of therapeutics, 9th ed. New York: McGraw Hill, 1996.

ARTERIAL PUNCTURE

Uses:

- to take a sample for blood gases
 - to take blood for routine tests (eg Hb, cross match, blood culture, electrolytes) from small fat infants, or a child with collapsed or poor veins.
1. Use a 23 or 25 gauge scalp vein needle (or a 23 or 25 gauge needle on a 5 ml syringe). Do not use local anaesthetic. Do NOT use a tourniquet.
 2. Have an assistant hold the hand palm uppermost with the wrist SLIGHTLY extended. Clean the skin with 70% alcohol.
 3. Palpate the radial artery at the wrist (on the side nearest the thumb).
 4. Puncture the skin over the pulse just proximal to the skin crease at the wrist. While still gently palpating the pulse with the index finger of your left hand, hold the needle in your right hand and slide it slowly up along the line of the artery angled at about 30 degrees to the skin.



5. When the artery is punctured, blood will run back along the scalp vein tubing. You may have to advance or withdraw the needle slightly to get a good flow of blood. Attach a syringe to the tubing and gently suck out the blood you need. You may need to have several attempts before successful puncture of the artery. Check that the needle is not blocked before each attempt.
6. Withdraw the needle, and get your assistant to press FIRMLY over the puncture site with a dry swab for AT LEAST 2 minutes.

ARTHRITIS (ACUTE) - ONE JOINT

See also Limp (p.189).

BEWARE OF BACK PAIN in children: it is always pathological. The most usual cause in Papua New Guinea is tuberculosis of the spine but other causes such as malignancy occur.

BEWARE OF NECK PAIN: this is always pathological. It may be due to cervical tuberculosis. A syndrome of atlanto-axial dislocation (?due to ligamentous laxity) may occur in children. It may result in cord transection and death. The child often has a history of upper respiratory tract infection, then presents with pain and marked limitation of movement of the neck. A cervical collar and surgical referral are indicated, and cervical traction may be needed.

FEBRILE PATIENT

Pyogenic arthritis

If a patient has arthritis of one joint and a fever, the joint must be aspirated to exclude pyogenic arthritis. Send the fluid for microscopy and culture. *Staphylococcus aureus* is the usual organism, but *Salmonella* species, *Haemophilus influenzae type B* and other bacteria may also cause arthritis. Do not be put off by a history of trauma (many children with pyogenic arthritis have a history of recent trauma). In hospital, it is best to start treatment with chloramphenicol and cloxacillin until sensitivities are known. Treatment with either cloxacillin or chloramphenicol should continue for a minimum of four weeks.

AFEBRILE PATIENT

Traumatic arthritis

Even if there is no history of trauma, a period of symptomatic treatment and observation is indicated in an afebrile child with mild monoarthritis (but always be wary of septic and tuberculous arthritis). If the child lives in a filarious area, take midnight blood films.

ARTHRITIS OR ARTHRALGIA (ACUTE) - MORE THAN ONE JOINT

RHEUMATIC FEVER

Polyarthritis or polyarthralgia is usually migratory, shifting from one larger joint to another. Knees, ankles, elbows, wrists and hips are frequently involved. Occasionally only one joint is affected.

The diagnosis is based on the Modified Jones Criteria:

Major	Minor
Carditis	Fever
Polyarthritis	Arthralgia
Chorea	High ESR
Erythema marginatum	Prolonged PR interval
Subcutaneous nodules	
PLUS	

Evidence of a preceding Group A streptococcal infection

Two major, or one major and two minor criteria plus evidence of a preceding streptococcal infection indicate a high probability of rheumatic fever.

Notes

1. A prolonged PR interval cannot be used as a minor criterion if carditis is a major criterion. Similarly, arthralgia cannot be used together with polyarthritis.
2. Interpretation of ESR in Papua New Guinean children is difficult. In some children with chronic or recurrent non-specific infections it may be high, whilst in others with ovalocytosis it may be very low.
3. If there is polyarthritis and a fever, always do an ECG to check for a prolonged PR interval:
 - Greater than 0.16 secs (4 small squares) in children less than 10 years old
 - Greater than 0.18 secs in children 10-12 years old.

Reference

American Heart Association guidelines for the diagnosis of rheumatic fever. Jones Criteria 1992 Update. JAMA 268:2069,1992.

Treatment

Acute illness

1. Give a single dose of benzathine penicillin or a 10-day course of amoxycillin.
2. Give high dose aspirin (80-100 mg/kg/day in four doses) until signs of inflammation settle, and then continue with lower doses (30-50 mg/kg /day) for 2-3 weeks.
3. If there is evidence of moderate or severe carditis, a course of steroids - starting with prednisolone 2 mg/kg/day - is usually given instead of aspirin, with the latter being given during and for two to three weeks after the steroids are tapered off to prevent rebound.

Prophylaxis

1. All patients with rheumatic fever should be placed on long term prophylaxis - either with monthly benzathine penicillin injections or with daily penicillin V (WHO recommendations 1988, are for

twice daily but this is probably less practical than once a day). Duration of prophylaxis is controversial, but should probably be at least until the age of 21 years in Papua New Guinea.

2. If surgery or a dental procedure is performed in a patient with rheumatic heart disease, appropriate prophylaxis should be given (ampicillin or amoxycillin for dental procedures, amoxycillin and gentamicin for abdominal or genitourinary procedures).

PYOGENIC ARTHRITIS

This occasionally causes polyarthritis (suspect gonococcal infection). It is nonmigratory: once involved, a joint does not spontaneously recover. Take a blood culture and joint aspirate from any child who looks very ill, or does not respond promptly to aspirin, then give cloxacillin and chloramphenicol until the organism is identified and continue with chloramphenicol or cloxacillin for a minimum of 4 weeks.

NON-SPECIFIC ARTHRITIS

Common. When rheumatic fever and pyogenic arthritis have been excluded:

- if in a filarious area, give diethylcarbamazine (Hetrazan)
- give aspirin 25 mg/kg QID until asymptomatic, then stop aspirin and observe. If after 4 weeks the arthritis still returns each time aspirin is stopped, suspect juvenile rheumatoid arthritis (Still's Disease) - more correctly called juvenile chronic arthritis
- in older children and adults, Reiter's syndrome and gonococcal infection are common causes of tropical polyarthritis (Lancet 1:1103,1989). Reiter's disease does not respond to aspirin; try indomethacin (dose is 0.25-1 mg/kg/dose 6-8 hourly after meals).

REFERENCES

Salfield S. Med J Aust 1:264-267,1975. Filariar arthritis in PNG.
Stein CM et al. Lancet 1:1103-1104,1988 . Aetiology of tropical polyarthropathy.

ARTHRITIS - CHRONIC

Chronic arthritis is not very common in children in Papua New Guinea, but when it does occur it can be incapacitating. However, with regular attention, and access to analgesic and anti-inflammatory agents, life for these children can be dramatically improved.

When a child with chronic arthritis is first seen, an attempt should be made to exclude infective causes of tropical arthritis such as filariasis. It should also be remembered that tuberculosis can present with a chronic monoarthritis or polyarthritis. A diagnosis of recurrent rheumatic fever should also be excluded. Yaws may also present with a chronic osteomyelitis, that may mimic chronic arthritis.

If these conditions have been excluded, the diagnosis is likely to be either reactive arthritis or juvenile chronic arthritis (JCA) - which used to be called juvenile rheumatoid arthritis, or Still's Disease.

REACTIVE ARTHRITIS

In this condition, the history is usually one of recurrent attacks of acute rather than persistent or chronic arthritis. Attacks are often precipitated by febrile illnesses such as respiratory or gastrointestinal infection.

Reactive arthritis usually responds well to short courses (1-2 weeks) of an anti-inflammatory agent:

- aspirin in doses of 25 mg/kg per dose 4 times a day for 3 days, then 7-12 mg/kg/dose 4 times a day
- indomethacin 0.5-1.0 mg/kg/dose 2-3 times a day.

JUVENILE CHRONIC ARTHRITIS

This is a complex disorder with several subtypes:

- Polyarticular, rheumatoid factor negative
- Polyarticular, rheumatoid factor positive
- Pauciarticular, Type 1 (ANA +, HLA-B27 -)
- Pauciarticular, Type 2 (ANA -, HLA-B27 +)
- Systemic.

In general, girls are more frequently affected than boys. In the pauciarticular forms, the large joints are most commonly affected and irido-cyclitis may be present. The cervical spine may be involved with limitation of movement. The natural history of the disease is of exacerbations alternating with relative quiescence.

TREATMENT

The mainstay of treatment is aspirin (25 mg/kg/dose 4 times a day initially, reducing to 7-12 mg/kg/dose 4 times a day), or other non steroidal anti-inflammatory drugs (NSAIDs). Of the latter, indomethacin (0.5 mg /kg /dose 2 or 3 times a day) is the only one likely to be available through the Health Services.

In a Western setting, patients not adequately controlled with NSAIDs, may be satisfactorily treated with low dose methotrexate (10-15 mg/m² weekly, oral or IM), but there is concern that it may not be safe to use this drug in a tropical environment. In very severe cases, it may well be worth trying methotrexate if the FBC can be monitored regularly and INAH given as well.

Acute exacerbations can be treated with prednisolone 1-2 mg/kg daily for 2-4 weeks, with the dose gradually reduced over a further 2-4 weeks. Long term treatment with steroids is contraindicated, and steroids should only be used when adequate doses of NSAIDs are not controlling the symptoms. Courses of steroids should always be accompanied by INAH.

Iridocyclitis is a medical emergency and requires application of topical steroids and dilating agents, and the attention of an ophthalmologist if available. Physiotherapy may help to maintain joint mobility.

ASCITES - ASPIRATION - ABDOMINAL PARACENTESIS

See also Peritoneal Tap (p.301).

This should only be done as a diagnostic procedure and NOT to remove ascites, except with very severe distension or in terminal malignancy.

1. Make sure the child's bladder is empty.
2. The child should be supine and held firmly.
3. Scrub your hands.
4. Clean the skin with iodine.
5. Check that a grossly enlarged liver or spleen does not extend to the puncture site.
6. Aspirate the ascites through a 21 gauge disposable needle on a 10 ml syringe inserted midway between the pubic symphysis and the umbilicus in the right lower quadrant or in the midline. Ascitic fluid should flow freely into the syringe.
7. Wash off the iodine with 70% alcohol (SVM).

ASCITES WITHOUT OTHER OEDEMA

Exclude nephrotic syndrome, cardiac failure, hookworm oedema, malnutrition, acute nephritis, cirrhosis and strongyloides (see Oedema, p.282).

The major causes of ascites without generalised oedema are TB, malignancy, pyogenic ascites and chronic liver disease.

TESTS

Urine: protein, blood and casts

Blood: ALT, alkaline phosphatase, bilirubin, albumin, globulin
alpha fetoprotein if hepatoma suspected

Chest x-ray

Gastric aspirate for AFB x 3

Mantoux

4-hourly temperature chart (for evidence of TB)

Aspiration of ascites: protein, cell count, gram stain, culture, AFB stain, and AFB culture. Cytology is UNRELIABLE.

Ascites without other oedema		
	Protein gram/l	WCC/cmm
Malignant	over 25 (75%)	over 1,000 (50%) variable types
TB	over 25 (50%)	over 1,000 (70%) usually lymphocytes
Pyogenic	over 25	polymorphs
Cirrhosis	under 25 (95%)	under 250 (90%)
Portal vein thrombosis (splenomegaly)	under 25	few or none
Constrictive pericarditis	under 25	few or none

MANAGEMENT

If you are unsure whether the patient has TB or malignancy, always consider a trial of TB therapy. For example, consider giving TB therapy if the patient refuses laparotomy, or is too sick to have a laparotomy, or while you are waiting for a biopsy report (even if the lesion is macroscopically malignant).

High protein diet.

Albendazole

Antimalarials.

Hydrochlorothiazide and Span K. If these do not reduce the ascites, consider adding frusemide and spironolactone. In addition, weigh the child and measure the abdominal girth twice a week.

If the ascites has been controlled and the diagnosis is still unclear, consider laparoscopy or exploratory laparotomy, and liver biopsy. Liver biopsy may enable the diagnosis of active chronic hepatitis, which may respond to prednisolone 1 mg/kg TID gradually reducing to 5-10 mg/day (keep the ALT normal). Give steroids only if the Hepatitis B antigen is negative.

ASTHMA

See also Wheezing (p.385).

A number of changes have occurred in the management of asthma over the last few years. These have resulted from:

1. The demonstration of the fact that young children can be treated using metered dose inhalers (MDI) with a spacing device and that, properly used, these may be as effective as a nebuliser.
2. The realisation that inflammation is a very important component of the aetiology of asthma.
3. The demonstration of the fact that, in patients with frequent or chronic asthma, attacks may be prevented by prophylactic anti-inflammatory treatment with low dose inhaled steroids or cromoglycate.
4. The realisation that theophylline is not usually required if beta sympathomimetics are given in adequate dose - and theophylline may cause side effects.

CLASSIFICATION OF ASTHMA

The traditional classification has been into Mild, Moderate and Severe but this is best used for presentation of an acute attack or exacerbation.

In determining the long term management of children with asthma it is convenient to classify them into one of the following three groups:

1. **Mild infrequent episodic.** Seventy five percent of children fall into this category. Mild to moderate attacks occur less than once a month, and the children are completely symptom free in between.
2. **Frequent episodic.** Moderate or severe attacks occur more frequently than once a month but the children are normal or near normal in between. Twenty percent of children fall into this category.
3. **Persistent (chronic).** There are frequent acute exacerbations in a child who is symptomatic on most days or nights and who usually has abnormal lung function tests. Fortunately this only applies to about 5% of children.

Patients with infrequent episodic asthma are treated for the individual attacks.

In those with frequent episodic or chronic asthma efforts should be made to prevent attacks and exacerbations.

MANAGEMENT OF PATIENTS WITH AN ACUTE ATTACK OR EXACERBATION

Mild

Inhaled salbutamol via nebuliser or MDI and spacer. Repeat every 2-3 hours as needed.

Dose for nebuliser:

- 5 mg diluted up to 2 ml with normal saline for children >10 kg
- 2.5 mg for those <1 year or <10 kg.

Severe

Asthma is severe if the children are:

- too breathless to talk
- too breathless to feed
- breathing at >50 breaths/min
- tachycardic (pulse >140/min).

Asthma is life threatening if the children are:

- cyanosed
- making poor respiratory effort (silent chest)
- very tired or exhausted
- agitated or with reduced consciousness.

Immediate management

1. Give oxygen 10 litre/min via a face mask if available, or 2 litre/min by nasal cannula if not (it is possible that putting in a nasal cannula may cause agitation and deterioration).
2. Give nebulised salbutamol as soon as possible (5 mg to older children, 2.5 mg to those less than 1 year or less than 10 kg). If no nebuliser is available, use a MDI and a spacer. Give one puff every few seconds until improvement occurs - up to a maximum of 20 puffs.
3. Give either prednisolone 2 mg/kg orally, or intravenous hydrocortisone 100 mg 6 hrly.

If there is improvement:

1. Give nebulised salbutamol 0.15 mg/kg (or MDI and spacer if no nebuliser) from 1 to 4 hourly as required.
2. Continue prednisolone for 4 to 5 days and then stop (in areas where tuberculosis is common, it is advisable to “cover” this steroid treatment with daily INAH at standard doses).

If there is no improvement or if there are life threatening features:

1. Give nebulised salbutamol 0.5% (or MDI and spacer if no nebuliser) continuously, if necessary.
2. Give intravenous aminophylline 10 mg/kg over 60 minutes, followed either by an infusion of 1 mg/kg per hour or 6 mg/kg 6 hourly infused over one hour. If the child has been taking oral aminophylline, do not give the 10 mg/kg loading dose of aminophylline. Use 4.3% dextrose in 0.18% normal saline as the maintenance fluid.

These suggested guidelines for the management of children with asthma are summarised in the table on page 44.

Notes

1. Insertion of an intravenous line, whilst desirable in severely ill children, may be difficult and may cause deterioration in the child's condition. Administration of beta agonists is the most important aspect of management, and steroids can be given orally. An intravenous line can be established, if necessary for intravenous medication and for maintenance of hydration, after the inhaled beta agonist has been given.
2. If inhaled beta agonists are not available, give subcutaneous adrenaline in a dose of 0.01 ml/kg of 1 in 1,000.

PROPHYLACTIC TREATMENT FOR CHILDREN WITH FREQUENT EPISODIC OR CHRONIC ASTHMA

There are two types of prophylactic medication which may be available:

1. **Sodium cromoglycate** (Intal). It is available both as a MDI and as a Spinhaler. It may take some 2 to 3 weeks before beneficial effects are evident, and does not help all patients. Furthermore, it is expensive.
2. **Inhaled steroids**. Both beclomethasone (Becotide) and budesonide (Pulmacort) are available in MDI. The Health Department currently purchases Becotide but it is important to realise that there are three strengths: Junior - 50, Regular - 100 and Forte 200 micrograms per puff. A new steroid preparation, Fluticasone, is more expensive and does not offer any great advantage over the older drugs.

Inhaled steroids are taken regularly - usually twice daily in the lowest dose that gives reasonable control. Doses can be increased to cover periods of exacerbation.

The ideal is for patients with frequent episodic or chronic asthma to be maintained on regular inhaled cromoglycate or steroids, and to require infrequent recourse to inhaled salbutamol (or similar beta agonist).

The regular use of inhaled steroids can make a very major improvement in the lives of patients with frequent or chronic asthma.

Notes

1. Spacing devices. These are available commercially, but they are expensive. Home made spacers can be made with cordial bottles. A plastic coffee cup with a hole cut in the base can serve as a mask. Spacers should be washed regularly, rinsed thoroughly in clean water and left to dry, to reduce static charges and deposition of the inhaled drug on the walls of the device.
2. Technique. It is imperative you demonstrate the technique of using a MDI with or without a spacer, and that you make sure the patients or parents can perform it satisfactorily. For spacing devices, it is best to give the required number of puffs - usually two - one at a time, with the patient taking 6 breaths from the spacer in between. Check the patient's (parents') technique each time you see them.
3. Nebulisers. Nebulisers should be available at all hospitals and health centres. They are usually driven by an air pump. It is important not to remove nasopharyngeal oxygen in a child with severe asthma during nebulising. Nebulisers can be driven by oxygen with a flow rate of 8 litre/min in children with very severe asthma - but this may be difficult to achieve with leaking equipment.
4. Salbutamol tablets are effective and are widely available. They can be used if a nebuliser or MDI is not available. Dose is:

Weight	Dose
6-14.9 kg	¼ tab six hourly
15-29.9 kg	½ tab
30 kg or more	1 tab

5. Some patients have only one asthmatic episode. For those who have repeated episodes it is important that they be seen if at all possible on a regular basis, so that the need for prophylaxis can be determined and the dose controlled. This will prevent acute exacerbations and the need for hospital admissions.

Summary of management of children with asthma

Infrequent episodic asthma (75%)

Beta sympathomimetics by MDI with spacer, or nebuliser.

If severe attack:

- Oxygen if available
- Frequent beta sympathomimetic by nebuliser or MDI/spacer (1-4 hourly as necessary, continuous in extreme cases)
- Short course steroids (with INAH cover if indicated)
- Intravenous aminophylline in those not responding.

Frequent episodic asthma (20%) and chronic asthma (5%)

Regular inhaled steroids by MDI with spacer - lowest dose to give good control.

Beta sympathomimetics by MDI with spacer or nebuliser as required.

If deterioration in control:

- Increase dose of inhaled steroids using a MDI with spacer.

If severe acute attack or exacerbation:

- Oxygen if available
 - Frequent beta sympathomimetic by nebuliser or MDI/spacer (1-4 hourly as necessary, continuous in extreme cases)
 - Short course steroids (with INAH cover if indicated)
 - Intravenous aminophylline in those not responding.
-

REFERENCES

- Guidelines on the management of asthma. Thorax 52 (Suppl): S1,1997.
- Consensus view: management of acute asthma. J Paediatr Child Health 29:101-103,1993.
- Editorial. Br Med J 314:1061-1062,1997. Spacer devices in the treatment of asthma.
- Vince JD. PNG Med J 39(4):329-337,1996. Management of asthma in children in Papua New Guinea.
- Zar HJ, Brown G, Donson H et al. Lancet 354:979-982,1999. Home-made spacers for bronchodilator therapy in children with acute asthma: a randomised trial.

ATAXIA - ACUTE OR SUBACUTE

Do not confuse ataxia with weakness of the legs (p.289).

SUBACUTE SCLEROSING PANENCEPHALITIS (SSPE)

Ataxia may be an early sign of the progressive neurological deterioration of SSPE. Other prominent features are myoclonic jerks. Papua New Guinea has one of the highest incidences of this condition ever recorded (see p.358).

POSTERIOR FOSSA LESION

(Tuberculoma, tumour, subdural or epidural haemorrhage): causes ataxia, headache, vomiting, papilloedema, neck stiffness, and sometimes head tilt. See Chronic Raised Intracranial Pressure (p.77).

ACUTE CEREBELLAR ATAXIA OF CHILDHOOD

This usually comes on over a few hours after a febrile illness in a child aged 1 to 5 years. The ataxia is mainly of gait, and there is often generalised hypotonia and dysarthria. The CSF may have mild lymphocytosis. Most cases recover fully.

BENIGN PAROXYSMAL VERTIGO OF CHILDHOOD

There are intermittent attacks of ataxia and vertigo, usually lasting several minutes.

OCCULT NEUROBLASTOMA

Ataxia of gait occurs with opsoclonus (irregular, hyperkinetic, multidirectional, spontaneous eye movements) and myoclonic jerks of the face and body. Do a urine VMA, chest x-ray (look for a mediastinal mass), an x-ray of the abdomen (look for suprarenal calcification) and an IVP.

ACUTE INTERMITTENT FAMILIAL CEREBELLAR ATAXIA

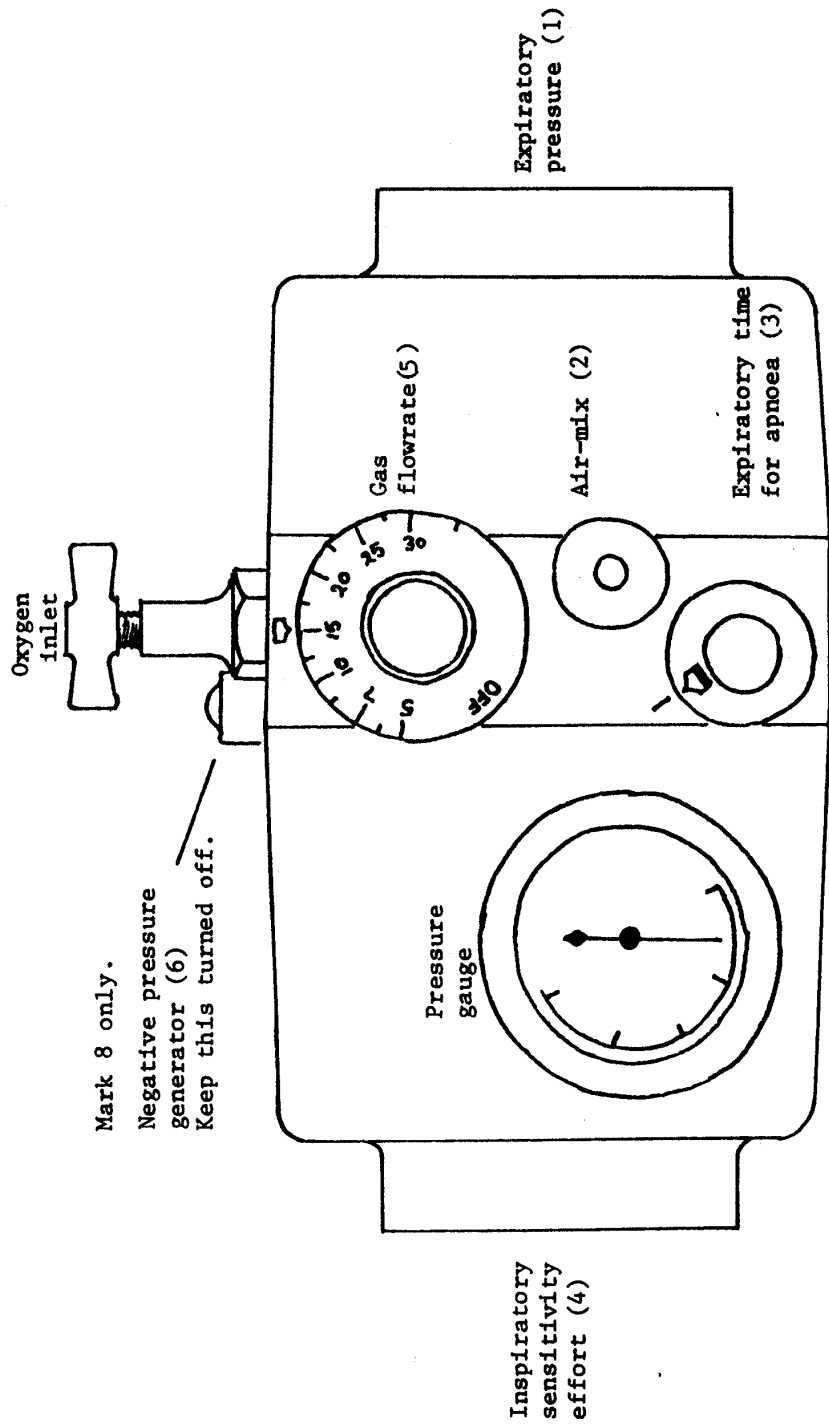
There is familial ataxia of gait, with intention tremor and dysarthria.

MINOR MOTOR EPILEPSY, PETIT MAL EPILEPSY, CHOREA

DRUG INDUCED

Ataxia may be caused by phenytoin, primidone or diazepam.

BIRD RESPIRATOR



BIRD RESPIRATOR

Mark 7, 8, 9 or 10

Assisted ventilation is rarely indicated in children in developing countries, where the risk of a complication from ventilation is very high. Ventilation is occasionally appropriate in children with snakebite or acute paralysis. It should NOT be used for children with apnoea due to meningitis.

Intermittent positive pressure ventilation (IPPV) can be delivered by a Bird respirator. This machine is pressure cycled. A small inspiratory effort by the patient triggers the respirator, and gas flows at the rate determined by the flowrate control (5). Gas continues to flow into the patient's lungs until the pressure in the system rises to the level set on the expiratory pressure control (1), then the flow stops until the patient triggers the respirator again. If the patient is too small or too weak to make even the small effort required to trigger the gas flow, automatic start of the gas flow can be achieved by turning on the expiratory time control (3). The air-mix control (2) adjusts the concentration of expired oxygen to 40% (knob out) or 100% (knob in).

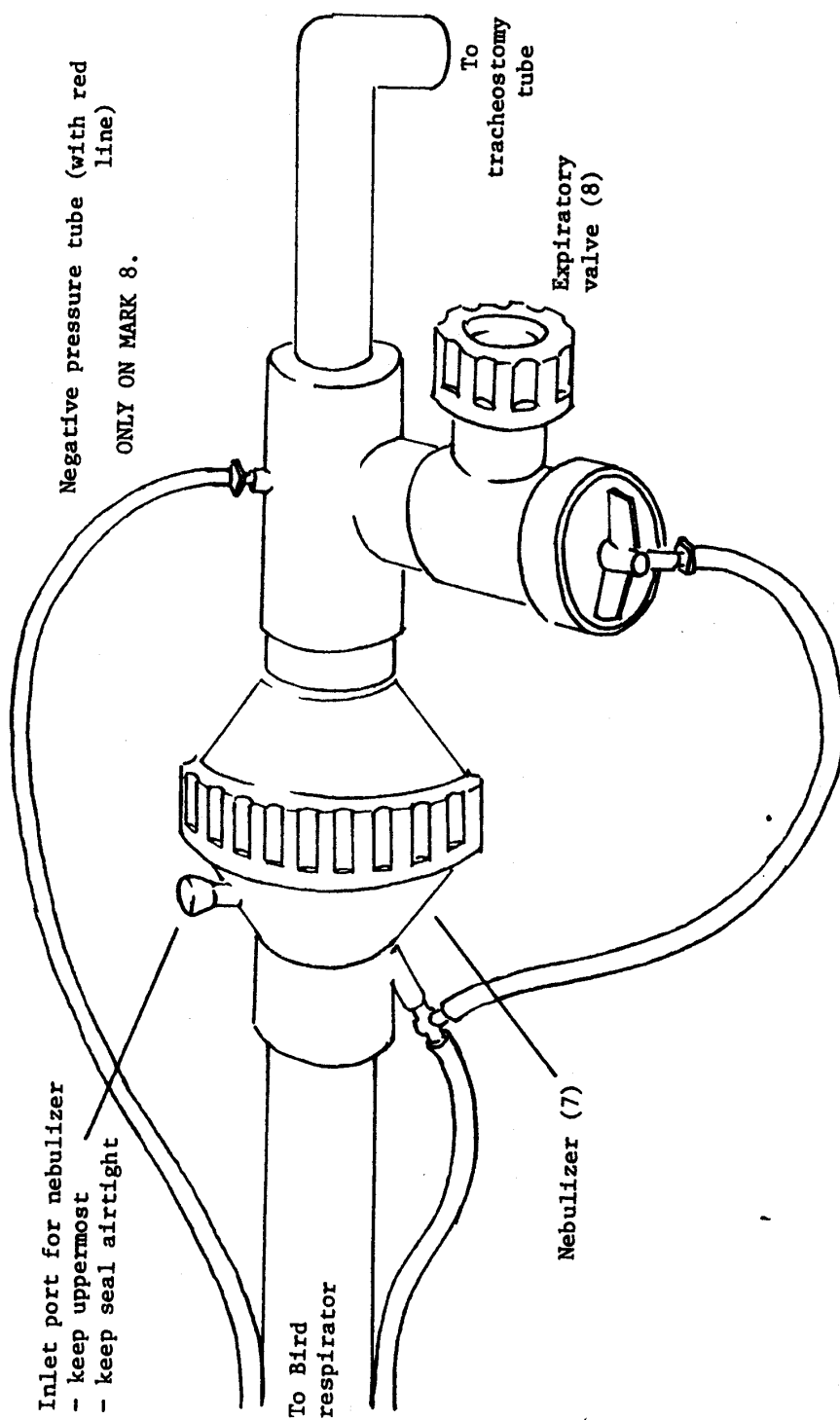
1. Intubate the patient. If the patient is likely to require assisted ventilation for more than 4 or 5 days a tracheostomy should be considered.
2. Set the controls on the Bird as follows:
 - Expiratory pressure (1): 15 (this sets the depth of inspiration)
 - Air-mix (2): pull out and lock (this gives 40% oxygen)
 - Expiratory time for apnoea (3): off
 - Inspiratory sensitivity effort (4): 10 (determines the inspiratory effort from the patient that is required to start gas flow)
 - Gas flowrate (5): off (sets length of inspiration)
 - Negative pressure generator (6): off.
3. Check that the breathing circuit is correctly connected (see diagram) and that there are no leaks.
4. Put 2 ml sterile water in the nebuliser (7). Do NOT inject through the red cap on the nebuliser inlet: remove the cap, inject, replace the cap. Inject 2 ml of sterile water each hour. Check that a fine mist appears in the nebuliser with each inspiration.
5. Connect the Bird to an oxygen supply.
6. Turn the gas flowrate control (5) to 15, then immediately connect the breathing circuit to the endotracheal or tracheostomy tube.
7. If the patient breathes in but gas does not flow, put the inspiratory sensitivity effort control (4) on a lower setting, eg 5 to 10.
8. Watch the excursion of the chest in inspiration. Alter the expiratory pressure control (1) until it is on the lowest setting that gives full excursion, eg 10 to 15.
9. Adjust the gas flowrate control (5) to alter the length of inspiration. A lower setting (eg 5 to 10) gives a longer inspiration than a higher setting (eg 10 to 15).

If the patient is too weak to breathe at all, you will have to turn on the expiratory time control (3) to set the desired time of expiration. In an older child or adult, inspiratory and expiratory time should be equal and respiratory rate should be about 12 breaths per minute. Ideally, controls (1), (3) and (5) should be adjusted so that the pCO₂ is 40mmHg, but blood gas analysis is rarely available in Papua New Guinea. Without blood gas analysis it is difficult to correctly ventilate a patient who is too weak to trigger the respirator. The advent of non-invasive pCO₂ monitors makes this much easier - though the CO₂ monitors are very expensive.

In coastal areas of mainland Papua New Guinea, snakebite envenomation is common and paediatricians must be able to use the Bird ventilator comfortably and confidently. The most important monitoring of a ventilated patient is done by routine observations of colour, pulse rate, blood pressure and, in particular, by watching timing, character and magnitude of the chest movements.

If the patient is cyanosed on the ventilator:

1. push the air-mix knob in (this delivers 100% oxygen to the patient)
2. check that the endotracheal or tracheostomy tube is not blocked
3. check that the patient does not have a pneumothorax.



BREATHING CIRCUIT

for Bird Respirator Mark 7, 8, 9, 10 or 14

THE MOST COMMON LIFE THREATENING PROBLEM ENCOUNTERED IN LOOKING AFTER A VENTILATED PATIENT IS A BLOCKED ENDOTRACHEAL TUBE. IF IN DOUBT REMOVE THE TUBE, VENTILATE WITH BAG AND MASK AND REINTUBATE AS NECESSARY.

1. If the gas flows continuously and does not turn off:
 - a. Check the circuit for leaks
 - b. Check that the expiratory valve (8) is not stuck
 - c. Check that the respirator valve (a thin metal rod that runs between the centres of controls (1) and (4)) is not stuck by pushing it to the left towards the inspiratory sensitivity effort control (4)
 - d. Increase the gas flowrate (5) and/or reduce the expiratory pressure setting (1).
2. If the gas flow turns off and on very rapidly:
 - a. Check that the endotracheal or tracheostomy tube is not blocked
 - b. Check that the tubing is not kinked.

NOTES

1. If you want to ventilate a child under 12 months of age, you should use an infant breathing circuit (number B9993-646 for Mark 7 Bird, number 993-427 for Mark 8 Bird).
2. With the air-mix control out (40% oxygen) and the flowrate control on 10 litre/min, a Bird respirator uses about 1.7 litre/min of oxygen.
3. Illustrated instructions for Bird respirators and a parts list can be obtained from Bird Products Corporation, 1100 Bird Center Drive, Palm Springs, CA92262, USA (phone 619-778-7200, fax 619-778-7274). Be sure to specify the model of your respirator (eg Mark 7, Mark 8).
4. If the child is likely to be ventilated for more than 1-2 days, cimetidine should be given to reduce the risk of stress ulcer.

BLEEDING DISORDERS IN CHILDREN

COAGULATION, PLATELET AND CAPILLARY DISORDERS OF HAEMOSTASIS

History

	Coagulation defects	Platelet and capillary defects
Sex	Usually male	Tend to be female
Transmission	Often sex-linked	Seldom hereditary
Age onset	Early in hereditary cases	Any age
Prior trauma	Usual (but often slight)	Often none
Site of bleeding	Deep (eg joints, muscles, gastrointestinal)	Superficial (eg petechiae on skin, mucous membranes)

Screening tests

	Hess test	Bleeding time	Clotting time	Clot retraction	Platelet count
Coagulation	N	N	Abn	N	N
Capillaries	Abn	N	N	N	N
Platelets	Abn	Abn	N	Abn	(Abn)

Hess test: place a cuff at 50 mmHg on the arm for 5-7 minutes. Over 10 petechiae in an area 2.5 cm in diameter is abnormal.

Bleeding time: make two small nicks in the skin, wipe off the blood with filter paper every 15 seconds. Bleeding normally stops within 7 min.

Clotting time: put 5 ml of blood in a PLASTIC tube, and tilt it every 30 seconds. Compare the clotting time with blood from a normal control.

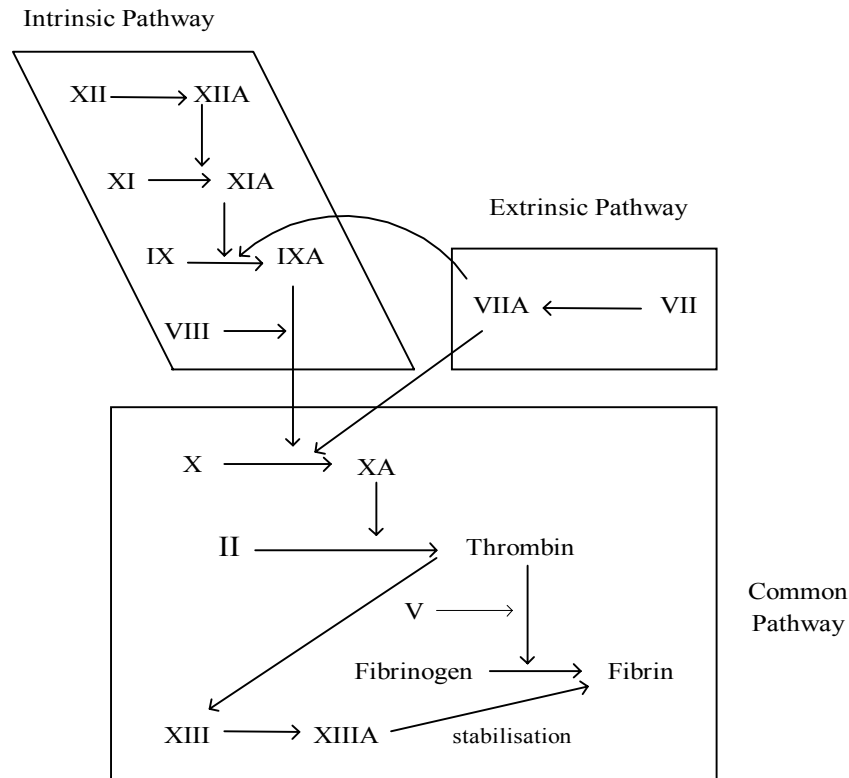
Clot retraction: the clot should shrink to half the original volume in 1 hour. This gives a rough guide to platelet and fibrin function.

Platelet count: bleeding is rare with more than 30,000/cmm. The count may be inaccurate. In thrombasthenia, there is a normal count but poor function.

Coagulation tests

Prothrombin Time (PT) tests the Extrinsic and Common Pathways.

Partial Thromboplastin Time (PTTK, APTT) tests the Intrinsic and Common Pathways.



COMMON INHERITED BLEEDING DISORDERS IN PAPUA NEW GUINEAN CHILDREN

1. **Factor VIII deficiency, Haemophilia A.** A sex-linked condition. Often a family history of affected males. Varies in severity.

Diagnosis: Normal bleeding time, normal PT, prolonged PTTK. Prolonged PTTK corrects with cryoprecipitate

Treatment: Cryoprecipitate (made in PMGH Blood Bank) or Factor VIII Concentrate (if available)

2. **Factor IX deficiency, Haemophilia B, Christmas disease.** Also sex-linked. Not usually as severe as Haemophilia A.

Diagnosis: Normal bleeding time, normal PT, prolonged PTTK. Prolonged PTTK corrects after Fresh Frozen Plasma (FFP)

Treatment: FFP.

3. **Factor XIII deficiency.** Autosomal recessive. Factor XIII is clot stabilising factor. Relatively common in PNG, which has one of the highest prevalences of this condition in the world. Presents with chronic, rather than acute bleeding and can present with umbilical cord bleeding.

Diagnosis: Bleeding time, PT and PTTK normal. Specific factor XIII assay available at PMGH

Treatment: FFP

4. **Von Willebrand's disease.** Autosomal dominant. Presents with epistaxis, excessive bruising or unexpected postoperative haemorrhage.

Diagnosis: Platelet count normal, bleeding time prolonged, PT normal, PTTK normal or may be prolonged

Treatment: Cryoprecipitate.

COMMON ACQUIRED BLEEDING DISORDERS IN PAPUA NEW GUINEAN CHILDREN

1. **Disseminated intravascular coagulation.** This is seen in very ill septicaemic children, or occasionally in those with severe malaria.

Diagnosis: Platelet count is reduced, bleeding time prolonged (oozing from venepunctures), PT and PTTK prolonged. Fibrin degradation products (FDP) positive.

Treatment: Treat the underlying condition and replace coagulation factors with FFP.

2. **Idiopathic thrombocytopenic purpura (ITP).** Children not usually very ill

Diagnosis: Platelet count reduced, bleeding time prolonged, PT and PTTK normal. Bone marrow shows increased megakaryocytes

Treatment: Only indicated if platelets fall below 30,000/dl. Treat with prednisolone starting at 1-2 mg/kg/day and tail off after 3-4 weeks. Platelet concentrates if available and platelet count is extremely low

3. **Leukaemia or other cause of bone marrow replacement.** Children usually very ill.

Diagnosis: Platelets reduced, usually very high WCC, abnormal blood film, abnormal bone marrow.

Treatment: For the underlying condition

BLOOD CULTURE

This must be done CAREFULLY, or the result may be very misleading.

1. Good veins to use are in the cubital fossa or on the back of the hand. You can also use arterial puncture (p.34).
2. Put a firm tourniquet on the limb. Do not make it too tight: make sure you can still feel the radial pulse at the wrist.
3. Clean the skin VERY WELL with swabs of iodine. Scrub all the dirt off the skin. Wait at least 2 minutes until all the iodine is dry.
4. WITHOUT TOUCHING THE PUNCTURE SITE WITH YOUR FINGERS, puncture the vein and withdraw 3-6 ml of blood.
5. Swab the lid of the culture bottle with iodine before you inject the blood.
6. Change the needle on the syringe and inject the blood into the culture bottles (The most effective ratio is 1 ml of blood to 5 ml of culture medium).
7. Put the labelled bottles into the incubator immediately. Do NOT refrigerate them.
8. Clean the iodine off the skin with 70% alcohol (SVM).

BLOOD PRESSURE MEASUREMENT IN INFANTS

In infants and young children, you should use the widest blood pressure cuff that will fit on the upper arm. A cuff that covers the whole upper arm is more accurate in children than one that covers only two thirds of the upper arm (J Pediatr 92:934-938,1978). If you do not have a cuff of the correct size you cannot take the blood pressure (make sure you get a cuff for next time).

Pump up the cuff to 120 mmHg. This is usually enough, and higher pressures just disturb the child. Measure the pressure as the mercury is allowed to fall VERY slowly.

1. Listen over the brachial artery with a small stethoscope. This is the most accurate method, but if you cannot hear anything



2. Palpate the radial or brachial artery. This gives only systolic pressure. If you cannot feel the pulse well



3. Use the flush method. Firmly compress the hand and forearm in one hand to squeeze all the blood out, THEN pump up the cuff to 120 mmHg while still squeezing. Stop squeezing, then allow the mercury to fall 5 mm at a time, waiting for 5 seconds at each step. The end-point is when the limb suddenly goes pink, and is about 5 mmHg less than the true systolic blood pressure. Repeat the measurement two or three times.

Upper limits of normal blood pressure (mmHg)	
0-3 years	90/60
4-7 years	95/70
8-10 years	100/70
11-12 years	105/75

BLOOD TRANSFUSION - INDICATIONS

See also Crossmatch of Blood (p.95).

PACKED RED CELLS

The quantity of packed cells required (in ml) is equal to the rise in haemoglobin required x the child's weight in kg x 4:

eg: a 10 kg child with a Hb of 4 g/dl, whom you want to transfuse to a Hb of 12 g/dl needs $(12 - 4) \times 10 \times 4 = 320$ ml of packed cells.

Neonates

In the first 2 weeks of life, transfuse if the Hb is less than 10 g/dl. From 3-4 weeks of age, transfuse if the Hb is less than 10 g/dl if the baby is sick, or transfuse if the Hb is less than 8 g/dl if the baby is well.

Acute haemolysis

Transfuse if the Hb is less than 6 g/dl.

Severe infection

(eg severe pneumonia, meningitis, osteomyelitis, severe acute malaria or TB). Transfuse if the Hb is less than 6 g/dl.

Cardiac failure

Transfuse if the Hb is less than 6 g/dl.

Severe anaemia

Transfuse any child with a Hb less than 3 g/dl.

WHOLE BLOOD

The quantity of whole blood required (ml) is equal to the rise in haemoglobin required x the child's weight in kg x 8:

eg: a 10 kg child with a Hb of 4 g/dl whom you want to transfuse to a Hb of 12 g/dl needs:
 $(12 - 4) \times 10 \times 8 = 640$ ml whole blood.

Acute blood loss

The need for transfusion is assessed by estimating amount of blood lost and the child's pulse rate and blood pressure, rather than on the Hb (which may take many hours to fall).

Pigbel

Transfuse whole blood if the Hb is less than 6 g/dl.

WHEN GIVING A BLOOD TRANSFUSION

1. Give frusemide (Lasix) IM or IV at the beginning of the transfusion UNLESS YOU ARE TRANSFUSING TO REPLACE ACUTE BLOOD LOSS.
2. Only use blood which has been properly grouped and cross-matched.

3. Make certain that the CORRECT bag of blood is given to the patient.
4. When giving blood at 20 ml or 25 ml/hour, try to use a paediatric measuring burette to measure the rate of the transfusion.
5. Only remove a bag of blood from the Blood Bank refrigerator when you are ready to start the transfusion.
6. Never transfuse blood that has been out of the refrigerator for more than 24 hours.
7. All patients receiving a blood transfusion should receive antimalarials.
8. If the patient develops fever or skin rash, or becomes ill, then:
 - a. stop the transfusion
 - b. give promethazine IM.

(Note that in many instances it is necessary to transfuse children who have a fever - eg those with severe infection and profound anaemia. In such instances, the fever is not a contraindication to transfusion.)

REFERENCE

Talonu T. PNG Med J 20:23-5,1977. Potentially dangerous group O blood.

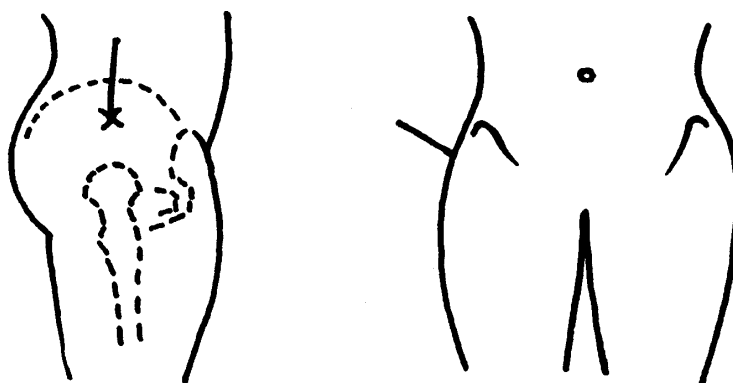
BONE MARROW ASPIRATION

OBTAINING THE SPECIMEN

1. Several sites are available:

Site	Age
Anterior or posterior iliac crest	All ages
Tibial or femoral shaft	Birth to 2 years
Medial malleolus of tibia	Birth to 5 years
Sternum	Adults ONLY

2. Several types of needle can be used:
 - a. **Simple needle with stylet:** the needle with stylet in place is pushed through the cortex into the marrow, the stylet is removed and marrow is aspirated.
 - b. **Turkel trephine needle:** the needle and stylet are pushed down to bone, the stylet is removed and replaced with the trephine which is used to bore through the bone cortex into the marrow. The needle is then pushed into the marrow, the trephine and biopsy specimen are removed, and aspiration is performed as well if desired.
3. The bone marrow can be aspirated or biopsied. Only aspiration of marrow from the anterior iliac crest using a simple needle with stylet will be described here.
4. Sedate the child with pethidine 30 minutes before the procedure.
5. Have the child lying on one side and firmly held by two assistants.
6. The site for puncture is 2-3 cm below the top of the iliac crest, at a point one quarter of the distance from the anterior superior iliac spine to the posterior superior iliac spine. Clean the skin with SVM (alcohol skin preparation solution) over a 10 cm radius from this point.
7. Infiltrate skin, subcutaneous tissue and periostium with 1% lignocaine.
8. Push the needle with stylet in place through the skin in the direction of the child's legs at about 45 degrees to the long axis of the body until bone is reached.
9. Then push harder, twisting the needle clockwise and anticlockwise, until a definite 'give' with decreased resistance shows that the needle has gone through the bone cortex and entered the marrow cavity.



10. Remove the stylet and attach a 5 ml syringe. Aspirate ONLY 0.1 to 0.2 ml of marrow into the syringe. Stop aspirating, and then remove the syringe from the needle. If a large specimen is aspirated it is likely to be diluted with peripheral blood from small vessels in the marrow. If a sample is required for culture, use a second syringe.
11. Remove the needle from the patient.

12. Make 4 to 8 marrow films on glass slides that have been **SCRUPULOUSLY** cleaned with 95% alcohol and lens paper. The technique is the same as that for peripheral blood films for malaria. A **SMALL** drop is placed near the frosted end and allowed to spread along the end of a slightly narrower spreader slide. The spreader slide is then pushed smoothly but firmly towards the opposite end of the specimen slide. The size of the drop should be such that the film which results is approximately two thirds the length of the transparent part of the specimen slide. If the film goes off the end of the slide, it is likely to be unsatisfactory. As a general rule, the more anaemic the patient, the smaller should be the size of the drop to be spread. For medical officers who are not good at spreading films, it is probably wise to have a technician on hand for this purpose if possible. It is acceptable to mix the specimen with EDTA to prevent clotting, but if the procedure is done smoothly this is usually not necessary.
13. If a satisfactory specimen has been obtained, you should see small marrow fragments (from 0.25 mm to 1 mm diameter) in the tail of the film. They are easy to see if you hold the slide up to the light. If no fragments are present, it may be difficult for the haematologist to tell whether the marrow cavity has been actually entered. Also, marrow fragments are essential for assessing total cellularity and iron stores.

FIXING THE SLIDES

1. The slides should be thoroughly dry before fixing.
2. They should be fixed **WITHIN 1 TO 2 HOURS** of spreading.
3. They should be fixed in **ABSOLUTE METHANOL** (preferably **ANALAR**, analytical grade) for 15-20 minutes. Care must be taken to prevent the methanol from being contaminated with water.
4. They should be left in air to dry after fixing.
5. One or two peripheral blood films, preferably obtained at the same time as the marrow, should also be made. These can be fixed with the marrow slides.

SENDING THE SPECIMENS TO A HAEMATOLOGIST

1. Make sure all the relevant clinical information is included in a letter.
2. Include any relevant blood counts that have been done (Hb, WCC, platelets, reticulocytes). When a peripheral film is sent, it is not necessary to report WBC differential or RBC morphology.
3. Include 1 or 2 well spread peripheral films.
4. Ensure that the slides are carefully packed to prevent breakage.

REFERENCE

Amato D. PNG Med J 20:15-22,1977. 700 bone marrow aspirations in PNG.

BOOKS

See also Journals (p.180).

Medical books can be bought from Heffer's Bookshop, 20 Trinity Street, Cambridge CB2 3NG, England, and Waterstones Booksellers, 82 Gower St, London WC 1E, England. Tel 020 7636 1577. Email: enquiries@gowerst.waterstones.co.uk. American books can be bought from Union Bookshop, 500 Milberry Union, San Francisco, CA 94122, USA. Useful material is available from TALC (Teaching Aids at Low Cost), PO Box 49, St Albans, Herts AL1 5TX, UK. Fax: 054 41 727 846852. E-mail: talc@talcuk.org. Website: www.talcuk.org. It takes 3 to 6 months for books to get from England or America to Papua New Guinea by surface mail.

The following books should be available at every hospital:

Crofton J, Horne N and Miller F. Clinical Tuberculosis. Macmillan^{1,2}
Dreisbach RH. Handbook of Poisoning. Lange
ABC of Ophthalmology. Br Med J Publications
Illingworth RS. Common Symptoms of Disease in Children. Churchill
King M. Primary Child Care Vol 1 and 2. OUP
Jones PG. Clinical Paediatric Surgery. Blackwell
Nelson WE. Textbook of Pediatrics. Saunders.
Roberton NRC. Neonatal Intensive Care. Edward Arnold.
Roberton NRC. Manual of Normal Neonatal Care. Edward Arnold.
Robinson and Roberton. Practical Paediatrics. Churchill Livingstone.
Stanfield, Brueton, Chan, Parkin and Waterston. Diseases of Children in the Subtropics and Tropics. Edward Arnold.

The following are additional books that may be useful at hospitals with specialist paediatric units:

Advanced Paediatric Life Support. The Practical Approach. 3rd ed. London: BMJ Books, 2001
Avery GB. Neonatology. Lippincott
Illingworth RS. The Normal Child. Churchill Livingstone
Isaacs D and Moxon ER. A Practical Approach to Paediatric Infections. Churchill Livingstone.
Roberton NRC. Neonatology. Churchill Livingstone.
Smith DW. Recognisable Patterns of Human Malformation. Saunders.
Swinscow TDW. Statistics at Square One. Br Med J Publications.
Waterlow J. Protein Energy Malnutrition¹.

¹ These can be obtained from TALC (see p.12).

² This is included in TALCs list of 17 books for a district hospital available at a very reasonable price (approx 250 kina). The list includes "Care of the Critically Ill Patient" and "Gastroenterology in the Tropics and Subtropics". Both written and edited by people who have worked in Papua New Guinea.

BURNS

Management of a child with extensive burns or scalds must take the following into account:

1. Death in the first few days after a burn is usually caused by surgical shock secondary to a diminished circulating blood volume. Fluid very similar to plasma is lost at the damaged site. This fluid loss occurs very soon after the burn occurs, is maximal in the first eight hours and progressively diminishes for 48 hours after the burn.
2. Death more than 48 hours after the burn is usually caused by infection. The wound initially is sterile, but contamination often occurs after the injury.
3. Anaemia may develop rapidly once the initial phase of haemoconcentration has passed.
4. The initial stage of surgical shock impairs renal function, so that oliguria must be watched for and managed if it occurs.
5. Tissue breakdown that results from the burn is associated with release of potassium into the extracellular fluid, and a raised level of potassium in the serum.
6. Protein depletion occurs due to loss of protein in the fluid that exudes from the burn site. Adequate nutrition is important in the convalescent stage.
7. Late effects of burns are contractures, which should have been prevented by adequate attention earlier.

TREATMENT

1. Sedation: give morphine or pethidine in correct dose. Chloral hydrate is often helpful.
2. Replacement of losses of water, electrolytes and protein: any child with more than 10% of his surface area burnt requires IV fluid:

Surface area of body	0-3 years	Over 3 years
Head and neck	18%	12%
Trunk and groin	32%	38%
Both arms	20%	20%
Both legs	30%	30%

- a. **IV fluid required during the first 8 hours after a burn:**
 - Volume: $2 \times \text{weight in kg} \times \text{surface area of burn} = \text{ml required}$.
 - Type of fluid: Use Hartmann's solution, if available, or normal saline, if not.
 - Blood: One quarter of the quantity of the above fluid should be given as whole blood if the burns are both deep and extensive.
 - b. The volume of **IV fluid required during the next 16 hours** is half the amount given during the first 8 hours. Use 4.3% dextrose in 0.18% normal saline.
 - c. The volume of **IV fluid required during the second 24 hours** is also half the amount given during the first 8 hours. Use 4.3% dextrose in 0.18% normal saline.
3. Oral fluids: the child should drink his estimated daily fluid requirements. This is in addition to the fluid given intravenously. Potassium containing fluids, such as fruit juices or coconut juice, should be given by mouth.
 4. Renal failure: urine output must be monitored carefully. Any child with more than 20% body burns should have the amount of urine produced each hour measured. Urine output should be AT LEAST 1 ml/kg/hour. If the urine output falls below this, extra IV replacement fluid is needed. If urine output does not improve over 1 hour, IV mannitol should be given. The dose of mannitol is 0.25 g (1.25 ml/kg) of 20% mannitol given IV over 5 minutes. If this produces good urine flow, a repeat dose can be given after 3 hours.

5. Assessment of the patient: keep an hourly chart of the following, to tell you whether the patient is receiving sufficient IV fluid (you must be prepared to give more fluid if necessary):
 - a. the presence or absence of restlessness and thirst
 - b. pulse rate
 - c. blood pressure
 - d. temperature
 - e. urine output
 - f. Hb level (done 4 hourly until it is normal, and then daily).
6. Prevention of infection:
 - a. give tetanus prophylaxis: give tetanus toxoid (and tetanus immunoglobulin if the child has not been immunised)
 - b. give benzyl (crystalline) penicillin IV 6 hourly
 - c. keep the burn open, and apply silver sulphadiazine cream 6 hourly
 - d. nurse the child under a mosquito net
 - e. nurses should wash their hands and put on a gown and mask before dressing the burn
 - f. the child should be nursed on clean sheets.
7. Adequate nutrition and Hb must be maintained after the early stage of surgical shock has passed. Blood transfusions may be required.
8. Prevention of contractures: adequate splinting and physiotherapy is required to prevent the development of contractures.
9. Skin grafting is often necessary once oedema fluid has subsided, the burn area is clean and anaemia has been corrected.
10. Occupational therapy. Children with burns may have to stay in hospital for many weeks. Make sure that they are kept happy and occupied. This is best achieved by allowing their mother to stay with them.

CANCNUM ORIS

Cancnum oris is severe gangrene of the cheek or upper lip caused by anaerobic spirochaetes. A large hole may appear in the child's face, and there may be necrosis of the maxilla and hard palate. Systemic illness may occur. Cancnum oris usually affects children 2-5 years old. Malnutrition, measles and malaria are predisposing factors.

TREATMENT

Metronidazole is extremely effective against the anaerobic spirochaetes. It may have to be given by NG tube. Tinidazole is an effective alternative.

Give benzyl (crystalline) penicillin IM 6 hourly.

Give antimalarials.

Treat malnutrition, by NG feeding if necessary.

Reconstructive surgery will usually be necessary when the child's general condition has improved after several weeks or months.

REFERENCES

- Enwonwu CO, Falkler WF, Idighe EO et al. Am J Trop Med Hyg 60(2):223-232, 1999. Pathogenesis of cancrum oris (noma): confounding interactions of malnutrition with infection.
- Lazaruo D, Hudson DA. S Afr Med J 87(10):1379-1382, 1997. Cancnum oris - a 35-year retrospective review.
- Tempest MN. Trop Doctor 1:164-168,1971. Cancnum oris.

CARDIAC ARREST

AIRWAY

In an unconscious child the most likely thing to obstruct the airway is the tongue. The child may start breathing again if the head is positioned correctly. This is in the neutral position in infants with a chin lift putting the end of your finger under the tip of the chin on the bone (take care not to obstruct the airway more by pushing below the chin under the tongue). In older children the cervical vertebrae should be flexed forwards but the head tilted back on the atlanto-axial joint: sniffing the morning air. Use a hand on the forehead to tilt the head back and a finger under the angle of the mandible to lift the jaw up.

LOOK for chest movements

LISTEN for breath sounds

FEEL for breath with your cheek over the child's mouth and nose, looking down onto the chest.

Inspect for a foreign body. If visible remove with a pair of Magill's forceps. You are more likely to push an object further down if you try using your fingers.

Intubate (ONLY if you are expert):

Weight	Size of ETT
Under 3 kg	2.5 mm
3 - 5 kg	3.0 mm
6 - 8 kg	3.5 mm
9 - 11 kg	4.0 mm
12 - 15 kg	4.5 mm
16 - 19 kg	5.0 mm
20 - 23 kg	5.5 mm
24 - 29 kg	6.0 mm
If weight unknown: $\text{Size of ETT} = \frac{\text{Age}}{4} + 4$	
In croup, use an ETT 0.5 mm smaller	

BREATHING

Ventilate with 100% oxygen using a self-inflating bag (eg Ambu or Laerdahl) through the ET tube or with a mask over the nose and mouth keeping the airway open. Look for chest expansion, check the seal around the mask and the face, adjust the position of the head in case the tongue is still obstructing the airway. If you are using an ET tube listen over the left upper zone to check the ET tube is not down the right main bronchus.

Give 5 rescue breaths.

Then reassess whether breathing has started spontaneously. If not, go onto "C".

CIRCULATION

Assess the cardiac output feeling for major pulses (femoral or brachial and in older children carotid). Feel for 5 seconds. Give external cardiac massage if the pulse is absent or less than 40 or very weak. If there is a good pulse continue ventilation if still apnoeic.

Lay the child on his/her back on a hard surface. For infants this may not be necessary if your hands are put round the baby's chest with the thumbs on the sternum. Use two fingers for infants, and the heel of your hand for older children. Press at the junction of the middle and lower thirds of the sternum. Depress the sternum 1.5-2.5 cm for infants, 2.5-3.5 cm for small children and 3-4.5 cm for larger children. Give 5 cardiac compressions then 1 breath: watch each breath to ensure good chest expansion. Continue with a rate of about 20 cycles per minute.

This must continue and any further manoeuvres should only be contemplated if there are a third pair of hands.

DRUGS

Adrenaline

ONLY if there is a third person and no response to the above.

If there is an IV cannula already sited:

- give 1/10,000 adrenaline 0.1 ml/kg (1 ml of 1/1,000 added to 9 ml IV fluid) as a fast bolus followed by a bolus of 20 ml of IV fluids

If there is no IV cannula and the child is intubated:

- give the adrenaline through the ET tube.

If there is no IV cannula and the child is not intubated:

- spend no more than one minute attempting to site an IV cannula. If unsuccessful after one minute, give it intraosseously (see Intraosseous Infusion, p.171). Again, give a 20 ml flush to follow.

Continue CPR; if no response after 3 minutes give 1/1000 adrenaline 0.1 ml/kg (or 1/10,000 give 1 ml/kg). Continue this every 3 minutes.

IV fluids

After the first bolus of adrenaline give 20 ml/kg of 0.9% normal saline, Haemacel or, if not available, whatever IV is closest at hand. Draw up with a syringe and push in as fast as the cannula will allow.

Bicarbonate

Has not been shown to improve survival. Consider only if profound acidosis likely. Give 1 ml/kg 8.4% solution. Flush well as it inactivates adrenaline.

WHEN THE HEART and BREATHING STARTS

Oxygen: continue via a nasal cannula or via ET tube if consciousness not regained

IV fluids: consider further 20 ml/kg bolus, then maintenance.

FOLLOWING INITIAL RESUSCITATION

1. IV glucose: give 5 ml/kg of 10% glucose or 2 ml/kg of 25% dextrose in older children.
2. Inotropes: if BP low and 60 ml/kg IV colloid or normal saline given, consider:
 - a. **dopamine** starting at 5 microgram/kg/min increasing to a maximum of 20 microgram/kg/min (add 3 mg/kg to 50 ml IV fluid, run at 5 ml/hr to maximum 20 ml/hr).
 - b. **adrenaline** at 0.05-1 microgram/kg/min (add 0.6 ml/kg of 1:1000 in 100 ml at 0.5 to 10 ml/hr).
OR
 - c. **isoprenaline** 1/5,000 1 ml amps (0.2 mg/ml):
 - *Weight under 10 kg:* put isoprenaline 0.2 ml/kg in 100 ml of 5% dextrose in the burette. Run the drip at 25 ml/hour. Adjust the rate according to the BP (90/60 is normal).
 - *Weight 10 kg or more:* put isoprenaline 0.1 ml/kg in 100 ml of 5% dextrose in the burette. Run the drip at 50 ml/hour. Adjust the rate according to the BP (95/70 is normal).
3. Frusemide (once BP in normal range) 1 mg/kg to maintain renal function.
4. Monitor heart rate, BP, temp, urine output, ECG if available, blood glucose, haemoglobin (cross match), urea and electrolytes, airway and pneumothorax.

NO RESPONSE TO RESUSCITATION

If there is still no heart beat after a second dose of adrenaline the evidence is that the outcome is very poor. Stop resuscitation after 30 minutes. Resuscitation should not normally be attempted in children dying from pneumonia or meningitis; it is rarely successful and the child usually arrests again anyway. Resuscitation is often effective in anaesthetic arrests. If a child arrests in the ward after a relaxant anaesthetic, check whether he or she is still curarized (give IV atropine and neostigmine). If the heart beat returns but the child is apnoeic consider mechanical ventilation if the outlook is good. If there is likely to be severe brain hypoxia or an irreversible pathology stop resuscitation after 1 hour.

REFERENCE

Advanced paediatric life support. The practical approach. 3rd ed. London: BMJ Books, 2001.

CARDIOMEGALY

Children with gross cardiomegaly have a very large heart shadow on chest x-ray. They are often clinically misdiagnosed as having pneumonia with CCF.

Valvular or congenital heart disease rarely produces gross cardiomegaly.

TESTS

- Cardiac ultrasound or echocardiography, if available
- ECG
- Thiamine 50 mg IV (diuresis within 24 hours confirms beri-beri)
- Mantoux.

CARDIOMYOPATHY

The commonest cause is viral myocarditis. Give digoxin and diuretics. Always give a trial of aneurine (thiamine) 50 mg IV, if available, followed by a 5 mg tab BD; there will be a diuresis within 24 hours if the CCF is caused by beri-beri.

PERICARDIAL EFFUSION

(See p.297).

The best way to diagnose pericardial effusion is by simple cardiac ultrasound or echocardiography. All hospitals should have an ultrasound machine available, and all doctors working in PNG should have a basic idea of what a pericardial effusion looks like on ultrasound. If ultrasound or echo is not available, do an ECG (look for low voltages with widespread T wave inversion) and screen the chest if you can (look for reduced movement of the heart, and you may see a separate heart shadow within the effusion).

Tamponade is suggested by an anxious, ill child with Beck's triad of a quiet heart with rising JVP and falling blood pressure. There may also be weak pulses (weaker still on inspiration), hepatomegaly and an impalpable apex beat. Pulsus paradoxus of >10 mm is highly suggestive of tamponade. The presence of a third heart sound suggests heart failure rather than tamponade.

Aspiration of the effusion is URGENT if tamponade is present (see p.298). Culture the fluid for pyogenic organisms and stain it for AFB. Take 3 gastric aspirates for AFB.

Digitalis and diuretics are contraindicated in pericardial effusion.

REFERENCE

Jatyesimi F. Arch Dis Child 84:384-390,1979. Infective pericarditis in Nigerian children. Pericarditis was common, and the diagnosis was often missed (72% were detected only at autopsy), and only 23% had muffled heart sounds.

CAROTID ANGIOGRAM

This dangerous procedure should only be performed in Base Hospital by a Specialist Medical Officer. CT scanning, though very expensive, may be possible for some children and is certainly preferable to a carotid angiogram.

1. Small children will need a general anaesthetic. Angiography is very difficult in children less than 4 years old.
2. Older children should be sedated with pethidine 30 minutes before the procedure, unless this is contraindicated. Infiltrate the skin and subcutaneous tissue of the neck with 1% plain lignocaine (Xylocaine).
3. Cannulate the carotid artery
 - a. either use a disposable 18 gauge angiogram needle with extension set attached
 - b. or use a 20 gauge Medicut and attach an extension set.BE VERY CAREFUL NOT TO INJECT ANY AIR AT ANY TIME.
4. Flush the system with heparinised saline (put 1000u heparin in one litre of saline) and clamp the extension set with artery forceps
5. Use 60% diatrizoate methylglucamine (Urografin). If only 76% is available, dilute 20 ml to 60% by adding 5 ml of sterile water.
 - Under 10 years: Inject 5 ml for each picture
 - 10 years or more: Inject 7 ml for each picture
6. Inject the Urografin RAPIDLY over about 2 seconds. Call 'shoot' as the 75% mark is being passed: the radiographer immediately takes an AP film. This film will show the arterial phase. Flush with heparinized saline, and then clamp the extension set.
7. When the radiographer is ready, repeat the injection and take a lateral film.
8. On occasions, extra information may be obtained by taking extra films 2 and 4 seconds after the 75% mark is passed. These films will show the capillary and venous phases respectively. Do not inject more than 8 times in one day.
9. After you have checked that the pictures are adequate, remove the cannula and apply firm pressure to the site YOURSELF for 5 minutes. Observe the patient closely over the next 2 hours.

CENTRAL VENOUS CATHETERISATION

CENTRAL VENOUS LINES ARE DANGEROUS - ONLY USE THEM WHEN ABSOLUTELY NECESSARY.

They are useful for:

1. measurement of the central venous pressure (CVP)
2. rapid infusion of fluid in a shocked patient
3. infusion of hypertonic or irritant solutions.

The dangers are:

1. arterial puncture
2. pneumothorax: rare with internal jugular catheterization
3. embolus of catheter: NEVER withdraw the catheter through the needle once it has been inserted
4. air embolus (because of negative intrathoracic pressure on inspiration): keep the patient head down during insertion of the catheter, and ensure that there are no leaks in the tubing and that the catheter is never disconnected without being occluded
5. infection: there must be strict asepsis during insertion of the catheter, and meticulous care of the site. The catheter should be removed as soon as possible (or immediately if the child develops a fever).

INSERTION OF CENTRAL LINES

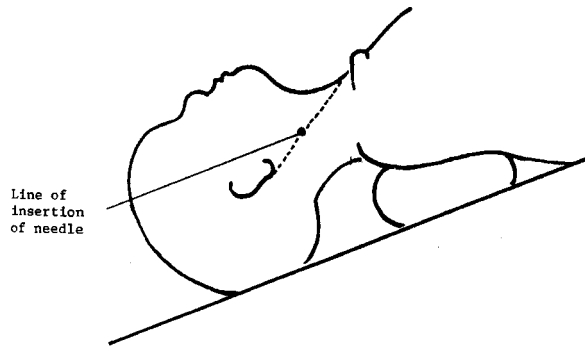
External jugular vein

It is often possible to insert a central venous line through the right external jugular vein, but the catheter may stick at the junction between the external jugular and subclavian veins. It may help to infuse fluid through the catheter while trying to negotiate the junction. The child is placed in the same position as for cannulation of the internal jugular vein: supine, with a small pillow under the shoulders, head down and turned slightly to the opposite side, held by two assistants. The vein is often easily visible at the lateral border of the neck, particularly when the child cries; despite this the vein is often difficult to cannulate.

Internal jugular vein

This is a fairly safe, reliable technique.

1. Scrub your hands and put on sterile gloves.
2. Lie the child supine with a small pillow under the shoulders. Put the bed sloping head down. Have the head extended and turned slightly away from the side on which the catheter is to be inserted (the right side is usually easier). Have one assistant hold the arms, and another assistant hold the head.
3. Clean the skin on the neck thoroughly with 70% alcohol, and drape.
4. Place a pair of sterile artery forceps, an Intracath, and a sterile 5 ml or 10 ml syringe close by, so that you can easily reach them with your right hand.
5. Pinpoint the exact position of the internal jugular vein by inserting a 25 gauge needle on a 2 ml syringe pointing caudally (towards the feet) and slightly laterally at an angle of 30° to the skin, through a point just (1 cm in an adult) lateral to the pulsation of the common carotid artery and midway (or just below midway) along a line joining the tip of the mastoid process and the insertion of the sternal head of sternomastoid. Exert gentle suction as the needle is inserted, until venous blood runs freely into the syringe. Note the exact position and depth of the needle, then remove it.



6. Swab the skin again with 70% alcohol. Put the 17 gauge needle from a green Intracath (or 14 gauge cream Intracath for adults) loosely on a 5 ml or 10 ml syringe; using a NO TOUCH, STERILE technique, insert it into the internal jugular vein - blood should run back very freely into the syringe. Grasp the hub of the needle firmly with your left hand, and carefully remove the syringe. Pick up the catheter with a pair of sterile forceps and GENTLY thread it into the vein. Do NOT force the catheter in. NEVER pull the catheter out through the needle if it sticks (remove the needle and catheter together). NEVER leave the end of the catheter open to the air (because of the risk of air embolus). Connect the IV line immediately.
7. Holding the catheter in the vein, pull the needle back along the catheter and snap on the needle cover. Put the bed level.
8. It is difficult to secure the catheter firmly in a small child. The best method is that described on pages 173 for securing an Intracath in a scalp vein.

MEASUREMENT OF CVP

Connect a CVP-manometer and giving-set. Do not allow the paper filter in the manometer to become wet. Measure the CVP with the patient supine and the bed level. The normal CVP is 0-3 cm above the sternal angle. The tip of the catheter must be in a large intrathoracic vein, and must be unobstructed (the fluid level in the manometer must swing with respiration).

INTERPRETATION OF CVP

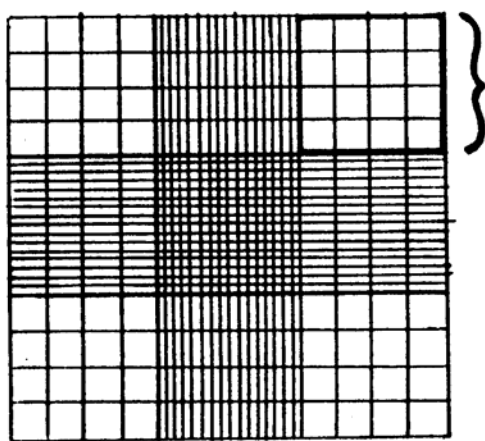
1. Hypotension + low CVP + peripheral vasoconstriction suggests hypovolaemia.
2. Hypotension + high CVP + peripheral vasoconstriction suggests heart failure.
3. Hypotension + low CVP + peripheral vasodilation suggests septic shock or vasodilation due to drugs (eg chlorpromazine).
4. Hypovolaemia can be confirmed by infusing 2 ml/kg of fluid over 2 minutes. In hypovolaemia the CVP will not rise more than 3 cm for every 1 ml/kg infused, and there will be improved peripheral circulation. If overloading occurs, the CVP will rise to more than 10 cm of water.
5. Serial measurements of CVP are of more value than a single reading.

REFERENCE

Rosen M, Latto IP, Ng WS. Handbook of percutaneous central venous catheterization. Saunders, London, 1981.

CEREBROSPINAL FLUID - EXAMINATION

1. Keep one of the two bottles of CSF sealed for later checking and culture by the laboratory staff. Record the appearance of the CSF.
2. Fill a CLEAN counting chamber with unstained CSF (to count the red blood cells).
3. Put ten drops of CSF into a small test tube. Add one drop of acidified crystal violet solution (CSF diluting fluid), mix well and stand for 3 minutes.
4. Fill a CLEAN counting chamber with stained CSF.
5. Carefully lower a CLEAN cover slip onto the counting chamber (avoid trapping air bubbles).



Count the cells in five of these large squares and multiply by two

NEUBAUER
COUNTING
CHAMBER

6. Using the 40x objective lens on the microscope, count the red cells in the unstained CSF. Count five large squares AND MULTIPLY BY TWO. If there are more than 100 red cells per large square, count one large square AND MULTIPLY BY TEN. This gives cells/cmm.
7. Still using the 40x objective, count the lymphocytes and polymorphs in the stained CSF. Count five large squares AND MULTIPLY BY TWO.
8. An approximate CSF protein can be obtained by testing with Albustix. An even more approximate estimation of CSF sugar can be obtained by testing with Clinistix: + is low, ++ or +++ is normal.

CEREBROSPINAL FLUID - RESULTS

NORMAL CSF

	Protein g/l	WBC/cmm	Glucose mmol/l*
Baby under 2.2 kg:			
• Under 1 week	0.5-4.0	0-18	1.1-2.2
• 1-3 weeks	0.5-2.0		
• 3-6 weeks	0.5-1.0		
• 8 weeks	0.4-1.0		
Baby over 2.2 kg:			
• Under 1 week	0.4-1.0	0-15	1.1-2.2
• 1-2 weeks	0.5-1.0		
• 2-4 weeks	0.3-0.7		
Child 0.5-11 years	0.05-0.3	0-5	1.1-5.0
Adult	0.15-0.45	0-5	2.7-4.5

*The CSF glucose is usually half to two-thirds the serum level.

TYPICAL CSF FINDINGS*

	Protein	Cells	Glucose
Meningitis:			
• bacterial	High	Polys	Low
• viral	Normal	Lymphoc	Normal
• tuberculous	High	Lymphoc	Low (50-100)
Encephalitis	High	A few	Normal

*But there is wide variation in CSF findings

TRAUMATIC (UNEVENLY BLOOD-STAINED) LP

Formulae designed to correct for blood contamination of the CSF are inaccurate (Am J Clin 82:95-97, 1984). However, a high proportion of children with bacterial meningitis will have a CSF WBC count observed/predicted ratio greater than 10 (J Infect Dis 162:251-254,1990), where the predicted WBC count is:

$$\text{blood WBC} \times (\text{CSF RBC/blood RBC}).$$

As a rough guide, the number of WBC in the CSF from the blood is 1-2 for every 1,000 RBC.

Allow 1 mg% rise in protein for each 700 RBC.

FROIN'S SYNDROME (CSF PROTEIN OVER 5 G/L)

The CSF is xanthochromic (clear yellow), rapidly clots on standing and contains more than 5 g/l of protein with few cells.

The usual cause is spinal block due to tumour, localised spinal meningitis or epidural abscess.

Froin's syndrome may be caused by acute postinfective polyneuritis (p.291), and rarely by TB meningitis (there will usually be a high lymphocyte count).

CHILD ABUSE

INTRODUCTION

Child abuse is unfortunately a feature of human society.

It was previously thought to be a problem of the affluent countries of the West, but it is now abundantly clear that it occurs in every country and through all strata of society.

It is an extremely difficult problem to deal with - but the first step is recognising that it is a problem in Papua New Guinea and that it is almost certainly common. The second step is to make the diagnosis on affected children - which requires a knowledge of the clinical findings and behaviour patterns associated with child abuse.

DEFINITION

An act - or an omission of normal caretaking duties - which endangers the physical or mental health or development of the child.

CLASSIFICATION

There are four major categories of child abuse. Affected children may be abused in more than one way.

Physical abuse (non-accidental injury)

Suspect this on the basis of:

1. History
 - a. delay in taking child to health facility
 - b. inadequate, discrepant, or excessively plausible explanation for the injury
 - c. previous injury to child or sibling
 - d. failure to thrive.
2. Examination (from top to toe in good light)
 - a. evidence of repeated injury
 - b. evidence of different forms of injury (eg burns and fractures)
 - c. bruising of face and non exposed areas
 - d. lashing marks
 - e. bites
 - f. skeletal injuries (fractures)
 - g. injuries to the mouth
 - h. periorbital haematomas
 - i. head injuries
 - j. abdominal visceral injury
 - k. thermal injury.

Parents may show an unusual reaction to the child's injuries or show abnormal behaviour. The question often arises as to what is non-accidental injury in the context of physical punishment. In some countries, physical punishment is illegal. In countries such as Papua New Guinea where physical punishment is culturally and socially acceptable it is generally understood that physical punishment which results in tissue damage is excessive - and indicative of child abuse.

It is very likely that physical abuse in Papua New Guinea is largely missed. Some affected children may be treated as "routine" cases in surgical wards and clinics.

Sexual abuse

Suspect this on the basis of:

1. History. Often but not always suggestive. Psychosomatic complaints and behaviour disturbances may be the presenting complaints
2. Examination (a careful examination in a good light is essential)
 - a. evidence of STD
 - b. hymenal disruption (old or new)
 - c. abrasions and damage to the the posterior edge of the vagina
 - d. excess relaxation of the anal sphincter
 - e. alterations in vasculature of the hymen and surrounding structures.

Note: It is extremely important that you have another (preferably female) staff member with you when you are examining the child.

Sexual abuse appears to be distressingly common in Port Moresby and probably in other urban centres.

Neglect

This is difficult to distinguish from the effects of severe poverty but should be suspected in any child who is:

1. unusually dirty and smelly
2. covered in severe skin sores (including nappy rash)
3. wearing dirty, smelly clothes.

Emotional abuse

This is difficult to quantify - but has profound effects. It may occur without any other form of abuse - but any child abused in any other way will of course suffer emotional abuse in addition to physical damage.

WHAT TO DO IF YOU SUSPECT CHILD ABUSE

Remember that the interests of the child are paramount.

ADMIT THE CHILD (This can be arranged against the parents' wishes if necessary by contacting the coroner or the coroner's officer - usually a senior police officer).

1. Take a careful history.
2. Do a careful examination in a good light.
3. Make good notes of your findings.
4. Arrange for photographs of injuries if possible - if not, make sure that you have some sketches of the injuries.
5. Do appropriate investigations
 - a. skeletal survey
 - b. coagulation screen
 - c. genital smears
 - d. serology for VDRL.

Further management

The aims are:

1. treat the physical injuries
2. prevent further injuries.

The prevention of further injuries means ensuring that it is safe to send the child home. This will involve trying to identify the perpetrator, and discussing the problem with senior family members, and may require the involvement of the Welfare Department and possibly the Police.

REFERENCES

- Vince J. PNG Med J 33(1):1-4,1990. Child abuse. Editorial.
- McClelland A, Polome H. PNG Med J 33(3):203-206,1990. Sexual abuse of children presenting to the Children's Outpatients Department of Port Moresby General Hospital.
- Riley I, Wohlfahrt D, Carrad E, eds. The management of rape and other sexual offences in Port Moresby. Department of Community Medicine UPNG 1985.
- ABC of child abuse. Meadows R, ed. BMJ Publishing Group, London, 3rd edition,1997.

CHOLERA

Cholera has not been diagnosed in Papua New Guinea. However, conditions are suitable for an outbreak at any time.

Most cholera is caused by the Cholera 01 El Tor strain, although a new strain, type 0139, has appeared in recent years. Asymptomatic carriers who spread the disease are common. *Vibrio* grow in the small bowel and produce a toxin that causes secretion of very large amounts of water and electrolytes into the bowel lumen. Patients pass large amounts of watery stools containing flakes of mucus (rice-water stools) and have copious clear vomitus. Isotonic dehydration develops rapidly with metabolic acidosis, hypokalaemia, haemoconcentration and shock. Patients with severe cholera are apathetic, cold and pulseless with sunken eyes, a husky voice, marked loss of skin turgor, cramps and scanty urine. Children are often drowsy, and they may convulse.

TREATMENT

The general principles of treatment are:

1. Replace water and salt rapidly.
2. Correct acidosis and potassium depletion.
3. Maintain normal hydration until the diarrhoea stops.
4. Reduce the amount and duration of diarrhoea with oral tetracycline.

Untreated severe cholera has a mortality of over 50%. With proper treatment, the mortality is less than 1%.

1. Water and electrolytes

	Severe: Weak or absent pulse	Moderate: Dehydrated, but good pulse	Mild: Minimal or no dehydration
Fast	Hartmann's 40 ml/kg IV		
First 4 hours	2.5% dextrose in half strength Darrow's 60 ml/kg IV over 4 hours	ORS 25 ml/kg/hr oral or NG	ORS 15 ml/kg/hr oral or NG
After 4 hours	ORS 5-15 ml/kg/hr (1.5 x output in previous 4 hours)	ORS 5-15 ml/kg/hr (1.5 x output in previous 4 hours)	ORS 5-15 ml/kg/hr (1.5 x output in previous 4 hours)
ORS: oral rehydration solution should contain Na 90mEq/l in cholera			

In older children and adults, stool output is measured every 4 hours in a graduated bucket placed under a hole cut into a special cholera bed. The bed has a plastic sheet on it, with a sleeve that goes through the hole and funnels stools into the bucket. Monitor stool output, pulse rate and volume, skin turgor, weight, urine output and thirst. Cramps and vomiting should disappear, and the neck veins should fill.

Children treated with Hartmann's solution alone often develop hypernatraemia, hypokalaemia and hypoglycaemia. As soon as they can drink, children should be given as much Oral Rehydration Solution (ORS) as they can take by mouth.

	Concentration (mmol/l)				
	Na	K	Cl	HCO₃	Glucose
Cholera stool (child)	105	25	90	30	-
Hartmann's solution	131	4	111	29	-
½ strength Darrow's solution	61	18	52	27	139
Oral Rehydration Solution	90	20	80	30	110

2. Oral tetracycline. This reduces the duration and volume of diarrhoea by 50% and the duration of *Vibrio* excretion by 50%. As soon as vomiting has stopped (usually after rapid rehydration), give tetracycline 12.5 mg/kg orally 6 hourly for 8 doses. This short course will not damage the teeth, even in small children.
3. Disinfection
 - Hands: wash with soap and water then antiseptic.
 - Thermometers: keep in disinfectant. Wipe with moist cotton wool before use.
 - Linen, bedpans: soak in disinfectant for 2 hours, then wash.
 - Utensils: boil
 - Dead bodies: plug mouth and anus with cotton wool. Cover with a sheet soaked in disinfectant.
4. Vaccination against cholera is not very effective, and lasts a very short time. Strict personal hygiene is much more important in an epidemic.
5. Do not give antiemetics (such as chlorpromazine or Stemetil), diuretics for anuria, analgesics for cramps, stimulants (adrenaline), plasma expanders (SPPS, plasma), or oxygen for cyanosis. Rapid IV rehydration makes all these treatments unnecessary.

REFERENCES

WHO. A manual for the treatment of diarrhoea. WHO/CDD/Ser 80.2/Rev 2 1990.
Candy D. Infectious diarrhoea and vomiting. In: Isaacs D, Moxon ER. A practical approach to paediatric infection, 1996: chap 3.1.

CHRONIC RAISED INTRACRANIAL PRESSURE

There are symptoms over a period of weeks or months. Do NOT do a lumbar puncture in a child with papilloedema. See also Head Circumference (p.141).

SIGNS OF RAISED INTRACRANIAL PRESSURE

1. Headache (worse in the morning)
2. Vomiting (often without nausea)
3. Widened skull sutures, bulging fontanelle, hydrocephalus
4. Papilloedema (rare under 6 months of age)
5. Impaired conscious state.

CAUSES OF CHRONIC RAISED INTRACRANIAL PRESSURE

Space occupying lesions

- tuberculoma, toruloma (cryptococcoma)
- subdural effusion
- neoplasm
- brain abscess.

Block to CSF flow (hydrocephalus)

(See p.158)

- congenital: spina bifida with Arnold-Chiari malformation
 aqueduct stenosis
 congenital cysts
- acquired: infectious (tuberculosis, cryptococcosis)
 post-infection
 post-haemorrhagic
 post-traumatic
 neoplasm.

Pseudotumour cerebri

- lead poisoning
- tetracycline
- hypoparathyroidism
- steroids
- excess or deficiency of vitamin A
- cyanotic congenital heart disease
- Addison's disease
- idiopathic

The diagnosis of acute raised intracranial pressure is dealt with on p.82. The usual causes are meningitis, cerebral malaria, abscess, encephalitis, Reye's syndrome, intracranial haematoma, neoplasm and acute cerebral oedema (eg after trauma or hypoxia).

THE SITE OF AN INTRACRANIAL LESION

Look particularly for:

- signs of raised intracranial pressure
- ataxia
- head tilt or neck stiffness
- cranial nerve palsies
- visual field defects
- paralysis of upward gaze
- long (pyramidal) tract signs (hyperreflexia and upgoing plantars).

RAISED INTRACRANIAL PRESSURE (OF GRADUAL ONSET)

With no focal signs

Is usually due to a midline tumour (eg neoplasm, tuberculoma, toruloma). It is usually in the posterior fossa (look for neck stiffness), but may also be pineal (look for paralysis of upward gaze) or in the region of the third ventricle (look for visual field defects).

Rarer causes are lateral ventricle choroid plexus papilloma, subdural effusion, and pseudotumour cerebri.

With cerebellar ataxia and tremor

Suggests posterior fossa tumour. There may be ocular palsies (III) and V, VI, VII, IX, X or XI cranial nerve palsies, and contralateral corticospinal tract signs. The presence of ataxia and head tilt or neck stiffness strongly suggests a posterior fossa tumour.

With visual field defect

Suggests optic glioma, craniopharyngioma or hypothalamic glioma.

Note: **Focal signs without raised intracranial pressure** suggest cerebral hemisphere (anterior fossa) tumour OR brain stem glioma (suggested by multiple cranial nerve palsies, long tract signs of hyperreflexia and upgoing plantars, without raised intracranial pressure).

INVESTIGATION

1. Do NOT do a lumbar puncture in a child with papilloedema.
2. X-ray the skull and chest, and do a Mantoux and sputum or 3 gastric aspirates for AFB.
3. Where the fontanelle is patent or the sutures splayed, ultrasonography of the ventricular system should be done.
4. Ventricular tap may be indicated to remove CSF (for micro and culture, indian ink for torula, examination for AFB, and measurement of protein and glucose).
5. Cerebral angiography may be possible in some hospitals (see p.67).
6. CT Scan or MRI if available.

DO NOT DO A LUMBAR PUNCTURE IN A CHILD WITH CHRONIC RAISED INTRACRANIAL PRESSURE.

TREATMENT

1. A trial of TB therapy should always be considered.
2. With very high pressure and progressive deterioration of conscious state:
 - a. give mannitol 20% 5 ml/kg rapidly IV. The effect begins in 20 minutes and lasts for 4 hours
 - b. give dexamethasone 2-4 mg IV or IM 6 hourly. The effect commences in 6 hours
 - c. put the head of the bed on blocks
 - d. arrange for surgical decompression as soon as possible if this is indicated
 - e. in an acute emergency, intubate and hyperventilate. This reduces intracranial pressure immediately.
3. If it is felt that surgery is a viable option, discuss the patient with your surgical colleagues.

INTRACRANIAL TUMOURS IN CHILDREN

Posterior fossa neoplasm in children (65%)

	Age (yrs)	Raised ICP	Ataxia	Pyramidal tract*	Cranial nerves	Cure
Cerebellum 40%						
• astrocytoma (20%)	3-8	Early	Unilateral	No	No	
• medulloblastoma (20%)	3-5	Early	Unilateral	No	No	
Ependymoma 10%	0-5	Early	No	Sometimes	Sometimes	Rare
Glioma brain stem 15%	6-8	Rare	No	Bilateral	Bilateral	Rare

*Pyramidal tract signs: hyperreflexia and upgoing plantars

Anterior fossa neoplasm in children (35%)

	Age (yrs)	Raised ICP	Visual loss	Growth failure	Cure
Third ventricle 15%					
• craniopharyngioma (5%)	0-20	Often	Often	Often	60%
• optic glioma (5%)	0-10	Late	Yes	Late	Often
• pineal tumour (3%)	0-15	Yes	Loss up gaze	No	Often
• hypothalamic glioma (2%)	0-1	Yes	Yes	Marked	Rare

Cerebral hemispheres 20%

Astrocytoma, oligodendroglioma, ependymoma, glioblastoma and sarcoma give early focal signs with late rise in intracranial pressure. Lateral ventricle choroid plexus papilloma 5% (0-3 years) causes raised intracranial pressure with hydrocephalus, focal signs rare.

Even if a neoplasm is strongly suspected, a trial of TB treatment and, if a toruloma or cryptococcal meningitis is unlikely or has been excluded, steroids should be given.

REFERENCE

Packer RJ. Current Opinion in Pediatrics 8(6):549-557,1996. Brain tumours in children

CISTERNAL PUNCTURE

Cisternal puncture can be used to obtain CSF (if this is important), or to inject dye for a myelogram if lumbar puncture has failed. It is a DANGEROUS procedure because the needle is very close to the medulla and you can kill the child if you push the needle in too far. **The death of a child following an investigation such as cisternal puncture may be (correctly or incorrectly) attributed by the family to the procedure and the practitioner. Only perform this procedure if:**

1. it is essential
2. the parents clearly understand that it is dangerous, and have signed an informed consent form
3. you have done it before under supervision.

Cisternal puncture must NOT be performed if there is raised intracranial pressure, myelomeningocele (when Arnold-Chiari malformation is likely) or suspected cerebellar malformation.

1. Have an assistant hold the child very firmly lying on his or her side (as for a lumbar puncture) with the neck slightly flexed. Put padding under the head so that the spine is straight. Sedate if necessary.
2. Shave the hair from the occiput and the back of the neck.
3. Scrub and put on sterile gloves.
4. Clean the skin over the back of the neck with iodine.
5. Feel for the small depression in the midline at the junction of the head and neck. One edge is the bottom of the occipital bone of the skull, the other edge is the spine of the axis (C2).
6. With your right hand insert a 4 or 5 cm long lumbar puncture needle with a stylet into the middle of this depression. Keep the needle EXACTLY horizontal but aim it slightly towards the head through the middle of a line joining the left and right external auditory meatus, and towards the glabella. Steady the needle by holding it between the thumb and index finger of your left hand at the point at which it pierces the skin.
7. Push the needle in about half a centimetre. Remove the stylet. If no CSF comes after waiting at least 15 seconds, replace the stylet and push the needle in another 2 mm and again remove the stylet. Continue until CSF is obtained, or until the needle hits the occipital bone or reaches the maximum depth. If the needle hits bone or no CSF is obtained remove the needle and reinsert it, aiming just below the external auditory meatus. Never move the needle up or down or from side to side.

	Under 2kg	Neonate	10kg	20kg	30kg	40kg	50kg	70kg
Usual depth to insert needle (cm)	1	1.5	2	2.5	3	3.5	4	4.5

COLOSTOMY AND ILEOSTOMY - STOMA MANAGEMENT

WHILE THERE IS EXCORIATION

1. Paint the ulcerated area once a day with 10% mercurochrome or aqueous gentian violet and let it dry.
2. Put Orabase Protective Paste on the ulcerated area. If this is not available, use plenty of vaseline.
3. Cover the ulcerated area and surrounding normal skin with plenty of Zinc Cream Compound (camphor 1%, aluminium acetate solution 10%, wool fat 33%, and zinc cream to 100%).
4. Put a gauze dressing over the stoma.
5. When the child has a stoma motion, wash off any faeces or ileostomy fluid gently with warm water. If the Orabase layer has not been broken, just put on more Zinc Cream Compound. The Orabase will have to be replaced 4 or more times a day.

WHEN THERE IS NO EXCORIATION IN A BABY

1. Cover the skin around the stoma with plenty of Zinc Cream Compound.
2. Put a gauze dressing over the stoma. When the child has a stoma motion, wash off any faeces or ileostomy fluid gently with warm water. Put on more Zinc Cream Compound.

WHEN THE CHILD STARTS TO SIT UP

1. Cut a circle of Stomahesive to extend onto normal skin for 2 cm all round the stoma. Cut a hole in the centre just smaller than the stoma.
2. Fit a plastic flange and stoma bag to the Stomahesive.

NOTE

In babies, colostomies can be managed surprisingly well by the use of copious amounts of Zinc Cream or vaseline and regular changing of nappies

COMA

Patients in coma may also have prolonged convulsions. This section should be read in conjunction with that on convulsions.

CAUSES

The causes of coma which should always be considered are:

SHORT (<3DAYS) HISTORY	LONGER HISTORY
cerebral malaria	tuberculous meningitis
bacterial meningitis	space occupying lesion
encephalitis	tuberculoma
hypoglycaemia	toruloma
trauma	brain tumour
hypertension	trauma
Reye's syndrome	chronic poisoning

ALWAYS check the blood sugar (dextrostix should be available)
check the blood pressure
check the blood slide

and

If safe to do so, lumbar puncture - check for papilloedema

Notes

1. Children with a widely open fontanelle do not get papilloedema, and it is safe to do an LP unless they have hydrocephalus.
2. In older children papilloedema is rare with coma of acute onset.
3. If you are in doubt about the safety of doing a lumbar puncture, you should not do it - BUT you must always treat the child for meningitis and cerebral malaria.

MANAGEMENT

1. Nurse the child in the coma position and ensure clear airway.
2. Put in an IV line and check dextrostix.
3. If hypoglycaemic, give either 5 ml/kg of 10% dextrose slowly IV or 1 ml/kg of 50% dextrose over 5 min. If you have no dextrostix it is reasonable to give dextrose as above.
4. Prime investigations (LP and MPS).
5. Commence treatment for bacterial meningitis and for cerebral malaria pending results.
6. Other investigations to be considered:
 - a. Urea and electrolytes (renal failure, hyper or hyponatraemia)
 - b. Liver function tests (Reye's syndrome - elevated enzymes)
 - c. Serum/CSF cryptococcal antigen (available in PMGH CPHL)
 - d. Serum calcium
 - e. CXR (evidence of TB)
 - f. Skull xray (intracranial calcification, skull fracture, splayed sutures, eroded clinoid process)
 - g. Mantoux test
7. Management of underlying cause if found.
8. Management of raised intracranial pressure if appropriate (see p.77).

NOTES ON SOME CAUSES OF COMA

Hypoglycaemia

Hypoglycaemia is common in children with severe infections. It is particularly common in malnourished children. Always check a comatose child for hypoglycaemia with a dextrostix if you can. If you cannot, it is always reasonable to treat for hypoglycaemia with intravenous 10% dextrose (5 ml/kg) or 50% dextrose (1 ml/kg). Hypoglycaemia is a recognised complication of parenteral quinine treatment. Dextrostix should always be monitored if possible when children receive parenteral quinine.

Hyperglycaemic (diabetic) coma

Hyperglycaemic diabetic coma is very uncommon in Papua New Guinean children but will probably become more common. Consider this particularly if the child is acidotic or ketotic.

Poisoning

eg cassava, angel's trumpet flower, arsenic (from preserved wood), lead, pesticides, antihistamines, antidepressants, anticholinergics, aminophylline, phenothiazine and aspirin (see p.317).

Head injury

A history of trauma may be absent if the child is maltreated. Look for cuts, bruises, fundal haemorrhages, papilloedema and focal CNS signs (which may be caused by a recent convulsion).

Space occupying lesion

Haemorrhage, neoplasm, tuberculoma, toruloma or abscess. Ask about recent headaches, vomiting and drowsiness. Look for widened sutures, a tense fontanelle, papilloedema, retinal haemorrhages and focal CNS signs (which may be caused by a recent convulsion). See also Convulsions - Recurrent (p.92) and Chronic Raised Intracranial Pressure (p.77).

Hypertensive encephalopathy

Diastolic pressure over 110 and fundal changes. Suspect renal disease, eg acute nephritis (see p.283).

Reye's syndrome

Reye's syndrome often starts with a prodromal viral URTI followed by severe vomiting and lethargy, then coma. There is often a history of aspirin ingestion. Serum SGOT is usually over 60 units/ml, the blood glucose is usually low and the prothrombin time is usually prolonged. The CSF findings and the serum bilirubin are usually normal. There is fatty infiltration of liver and brain. The immediate cause of death is usually cerebral oedema. Treatment is 20% mannitol 2.5 ml/kg IV 4 hourly for raised intracranial pressure, IV fluid restricted to 10% maintenance and vitamin K 4 mg IM. Peritoneal dialysis and exchange transfusion are not effective.

Encephalitis

Encephalitis is a common cause of coma, but it is important to exclude the other causes before making this diagnosis. The CSF may be normal, or have elevated protein (over 3 g/l) with a few lymphocytes.

GLASGOW COMA SCALE AND CHILDREN'S COMA SCALE

GLASGOW COMA SCALE (4-15 YEARS)		CHILDREN'S COMA SCALE (<4 YEARS)	
Response	Score	Response	Score
EYES:			
Open spontaneously	4	Open spontaneously	4
Verbal command	3	React to speech	3
Pain	2	React to pain	2
No response	1	No response	1
BEST MOTOR RESPONSE:			
Verbal command: obeys	6	Spontaneous or obeys verbal command	6
Painful stimulus:		Painful stimulus:	
Localises pain	5	Localises pain	5
Flexion with pain	4	Withdraws in response to pain	4
Flexion abnormal	3	Abnormal flexion to pain (decorticate posture)	3
Extension	2	Abnormal extension to pain (decerebrate posture)	2
No response	1	No response	1
BEST VERBAL RESPONSE:			
Orientated and converses	5	Smiles, orientated to sounds, follows objects, interacts	5
Disorientated and converses	4		
Inappropriate words	3	<i>Crying:</i>	
Incomprehensible sounds	2	Consolable	<i>Interacts:</i>
No response	1	Inconsistently consolable	Inappropriate
		Inconsolable	Moaning
		No response	Irritable
			No response

REFERENCES

The child with a decreased conscious level. Chap 12, p127-137. In: Advanced paediatric life support. The practical approach, 3rd ed. London: BMJ Books, 2001.

Newton CRJC, Chokwe T, et al. Trans Royal Soc Med Hyg 91(2):161-165,1997. Coma scales for children with severe falciparum malaria.

CONGENITAL MALFORMATIONS AND SURGICAL REFERRAL

ABDOMEN

Exomphalos (omphalocele): Abdominal contents herniate through the umbilical opening. Covered by thick membrane - amniotic membrane fused with peritoneum. **Gastroschisis:** Abdominal contents herniate through an opening in the abdominal wall lateral to the umbilicus.

Cover with sterile gauze soaked in saline, then clear plastic. Discuss urgently with your surgical colleagues. In all but the smallest omphaloceles, commence IV fluids and nil by mouth and prepare for surgery.

Umbilical granuloma: Caution - make sure you are not dealing with a **persistent urachus or vitellointestinal duct** in the base of the cord. Granulomas can either be snipped off and dressed with vaseline gauze and a pad, or they can be treated with copper sulphate or silver nitrate application.

Umbilical hernia: Central. Most close by the end of the first year. Surgery only if not closed by about 5 years.

Paraumbilical: non urgent - closure at 12-24 months.

Vomiting of bile, or vomiting with distension: Suggests bowel obstruction. Commence IV fluids, nil by mouth, antibiotics (ampicillin and gentamicin) and consult your surgical colleagues URGENTLY.

ANUS AND RECTUM

Anorectal malformations: These can be classified into three groups.

1. High: anorectal agenesis with or without urinary or vaginal fistula, and rectal atresia.
2. Intermediate: rectobulbar urethral, or rectovesicular or rectovaginal fistula and anal agenesis without fistula.
3. Low anocutaneous fistula, anal stenosis or microscopic anus and ectopic anus (or anovestibular fistula or cloaca - a single opening for vagina and anus in females).

These abnormalities should be discussed **URGENTLY** with your surgical colleagues. If there is no obvious fistula, or if there is any suspicion of abdominal distension put up an IV, nil by mouth and prepare for surgery.

CHEST WALL

Pigeon chest and funnel chest: No surgery.

EARS

Deafness: Congenital, cretinism, postmeningitis. A hearing aid may help, but these are expensive.

Mastoiditis: A febrile child with a tender mastoid. There is often a swelling, pushing the pinna forwards, a discharging ear, or large cervical nodes. Urgent referral for surgery is indicated; give IV or IM chloramphenicol and oral or NG metronidazole or tinidazole.

EYES

Blocked tear ducts: Common in neonates. Treat infection with oxytetracycline (Terramycin) eye ointment and massage of the nasolacrimal duct. Refer for probing if the eye still waters at 4 months of age.

Congenital cataracts: First available eye specialist referral.

Squints (strabismus): Refer at about 18 months of age.

HEAD AND NECK

Cleft lip and palate: Refer cleft lip at 3 months of age, refer cleft palate at 18 months of age.

Nasal meningocoele and encephalocoele: These seem to be relatively more common than sacral myelo-meningocoele in many developing countries. Early surgery is more difficult, but minimises the subsequent distortion of the face (hypertelorism). Discuss referral with the regional surgeon by letter or telephone. See Arch Dis Child 64:201-4,1989 for a discussion of the variation in neural tube defects from region to region.

Subgaleal inclusion cyst (Odeka's cyst): A soft egg-shaped swelling over the anterior fontanelle which is quite common in many developing countries. Repair is simple, and non-urgent.

HEART

Presenting in the neonatal period with severe respiratory distress, cyanosis or heart failure:

Unfortunately facilities do not exist for surgical management of such children. Transfer overseas for surgery is very difficult and extremely expensive, and is not usually an option.

Less severe presentation: There is currently a programme in PNG in which children with some forms of congenital heart disease and some forms of rheumatic heart disease receive surgery. The children are very carefully selected. The stages of selection are:

1. Diagnosis of heart disease by medical officer.
2. Entering child in the hospital Cardiac Register. This is very important. If there isn't one - start one!
3. Review of child by paediatrician and selection for:
4. Review and selection by paediatric cardiologist.
5. Transfer to Operating Centre.

INGUINAL AND SCROTAL REGIONS

Hydrocoele: Most disappear, refer for surgery if still present at 12 months.

Inguinal hernia: Refer at 2 years if easily reducible, but if strangulation occurs emergency transfer is required. If the hernia is not easily reducible, refer at the time of diagnosis.

Undescended testis: Exclude retractile testis (one that can be brought to the bottom of the scrotum). Surgery is needed if there is no descent by 1 year of age.

LIMBS

Burns contractures: Minimise by early grafting of granulating areas and control of infection.

Contractures of large joints: discuss with a surgeon after 6 months. Contractures of fingers: leave until the child is more co-operative (7 years), then discuss with a surgeon.

Fingers and toes - extra digits, webbing etc: Leave until 7 years old.

Semimembranous bursa: Surgery for pain at any age.

Talipes: It is important to determine if the talipes is “positional” (where it can be easily manipulated into the correct position) or “pathological” (there is often a deep crease in the medial aspect of the foot in such cases). For positional talipes, simple regular gentle manipulation is often sufficient. For the obviously “pathological” type of talipes, surgical referral is indicated. Seek orthopaedic surgical advice if this is locally available - or consult your surgical colleagues for advice as to the correct time for correction. Talipes which is “intermediate” in type often responds to strapping and manipulation.

REFERENCES

Vince JD and Jacob OJ. Congenital abnormalities of the gastrointestinal tract. In: Watters D, Kiire C, eds. Gastroenterology in the tropics and subtropics. Macmillan, 1995: chap 24 (available through TALC).

Practical procedures. In: Biddulph J, Stace J, Danaya R. Child health for health extension officers and nurses in Papua New Guinea. 7th edition. Dept of Health PNG, 1998: chap 38.

Tefuarani N. PNG Med J 43(1-2):65-68,2000. A history of congenital heart disease surgery in PNG.

CONVULSIONS - ACUTE PRESENTATION

Read this in conjunction with section on Coma (p.82).

CONVULSIONS WITH FEVER

Convulsion indicative of underlying brain pathology

- acute bacterial meningitis
- cerebral malaria
- encephalitis
- acute viral meningitis

Convulsions caused by fever in the absence of acute brain pathology (febrile seizures)

Characteristic features are:

- child aged 4 months to 5 years
- no history of afebrile fits
- fever of $>38^{\circ}\text{C}$
- not associated with underlying CNS infection.

Notes

1. Febrile fits occur in about 3% of children. About 1% of children have more than one febrile convulsion. They are most commonly associated with URTI.
2. Febrile convulsion is a diagnosis of exclusion. You cannot make a firm diagnosis without examining the CSF and a blood slide for malaria.
3. In a situation where bacterial meningitis and cerebral malaria are common, ALWAYS TREAT THE CHILD FOR MENINGITIS AND CEREBRAL MALARIA IF THERE IS ANY DOUBT AT ALL ABOUT THE DIAGNOSIS.

CONVULSIONS WITHOUT FEVER

1. Metabolic disorders:
 - a. hypoglycaemia
 - b. hypocalcaemia
 - c. hypomagnesaemia
 - d. hypernatraemia
 - e. hyponatraemia
 - f. pyridoxine deficiency
2. "Idiopathic" cause not ascertained.

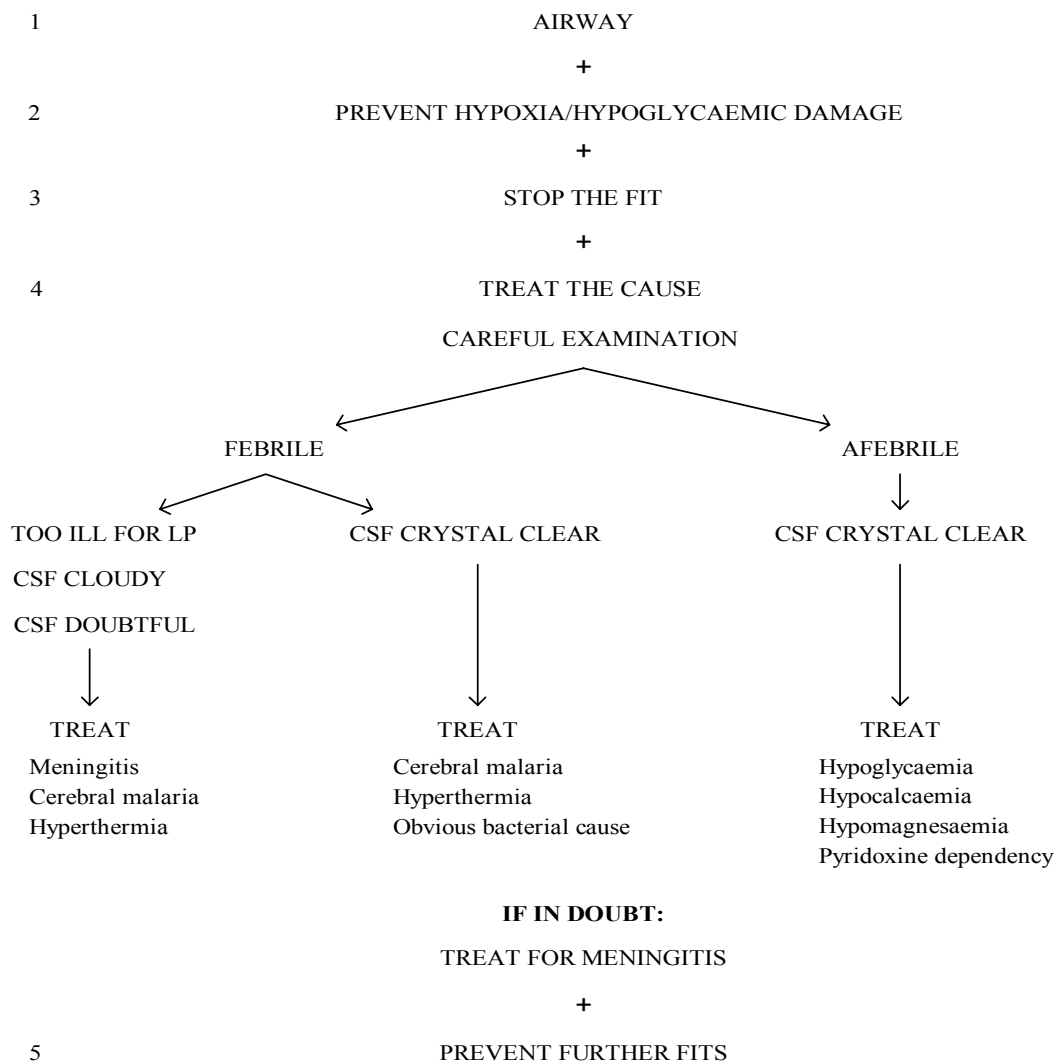
Note

Differential diagnosis includes:

- Tetanus spasms - caused by movement or noise.
- Drug induced extrapyramidal movements/oculogyric crisis (phenothiazine dystonic reaction) - most commonly seen after Stemetil (which should never be used in children). Sometimes occurs after Maxolon (metoclopramide). If you suspect this, give benzotropine (Cogentin 0.02 mg/kg IM or IV). Movements will cease within one to two minutes.

MANAGEMENT OF THE FITTING CHILD

Figure. Management of a fitting child. LP=lumbar puncture, CSF=cerebrospinal fluid.



Management is much easier for two persons than for one - call for help.

1. CLEAR AND MAINTAIN THE AIRWAY

- a. Nurse the child on his/her side in coma position
- b. Gently suck out secretions or vomitus
- c. Use a plastic airway if available
- d. Empty the stomach with a nasogastric tube.

2. PREVENT CNS DAMAGE BY CORRECTING HYPOXAEMIA AND HYPOGLYCAEMIA

- a. Give oxygen if available by nasopharyngeal catheter, prongs or mask
- b. Give IPPR if child is not breathing
 - i. Bag and mask
 - ii. Nasopharyngeal oxygen (2-4 litre/min) and “frog breathing”, mouth to mouth, or mouth to nose
 - iii. Endotracheal tube if prolonged apnoea
- c. Check for hypoglycaemia with dextrostix
 - i. If sugar <3.3 mmol/l (60 mg/dl)
 - Give IV 50% dextrose 1 ml/kg slowly over 1-2 mins
 - or IV 10% dextrose 5 ml /kg over 2-5 mins
 - then, run IV 10% dextrose at “Coma rate” (see p.397)
 - ii. Check the blood sugar regularly with dextrostix.

Note: If you have no dextrostix, give a bolus of dextrose as above - unless there are signs of ketosis or acidosis and a possibility of juvenile onset diabetes mellitus (very rare in PNG).

3. STOP THE FIT

- a. Use either:
 - i. Diazepam 0.25 mg/kg IV or 0.5 mg/kg rectally, or
 - ii. Paraldehyde 0.2 ml/kg deep IM injection or 0.3 ml/kg diluted 1:10 rectally
- b. Repeat if no control after 10 mins
- c. Change to the alternative if no control after 10 mins
- d. If still no control give either:
 - i. Phenobarbitone 15-20 mg/kg slowly IV over 5 min or IM injection, or
 - ii. Phenytoin 15-20 mg/kg slowly IV over 30 min (dilute in normal saline).

Note:

- The combination of diazepam and phenobarbitone may cause respiratory depression.
- Never give IM diazepam or phenytoin. It is ineffective and a waste of drugs.

4. TREAT THE CAUSE OF THE FIT

- a. Do a careful physical examination (including tympanic membranes and throat)
- b. If the child is febrile:
 - i. Investigate
 - do a lumbar puncture unless contraindicated (papilloedema or the child extremely ill)
 - do a blood slide for malaria parasites
 - do a blood culture if available
 - do other cultures urine/pus as appropriate
 - do a white cell count and differential if available
 - ii. Treat
 - If the CSF is turbid or blood stained, or if the LP is unsuccessful or contraindicated
 - o treat for meningitis with chloramphenicol (see p.223), and
 - o treat for cerebral malaria with Artemether or quinine and Fansidar
 - o treat for hyperthermia - cool sponge (this may not be very effective, but does no harm and involves the parents in the child's care).
 - If the CSF is crystal clear (compare with water)
 - o treat for cerebral malaria
 - o treat for hyperthermia
 - o treat other infection if present.

- c. If the child is afebrile:
 - i. Investigate
 - lumbar puncture unless contraindicated (beware partially treated meningitis)
 - blood slide for malaria
 - others, if appropriate and available, eg serum urea, creatinine and electrolytes; serum calcium and magnesium and protein; liver function tests (particularly enzymes).
 - ii. Treat
 - specific cause if it is confirmed or strongly suspected (see below)
 - empirically, if cause not obvious and fits uncontrolled
 - o give 10% calcium gluconate 1.0 ml/kg slowly over 5 minutes
 - o give 50% magnesium sulphate 0.2 ml/kg IM
 - o give pyridoxine 50 mg IV if available or 50-100 mg oral (or by NG tube).

Note: If the fit is completely unexplained, remember the possibility of:

- head injury - search for bruises/localising signs
- poisoning - search for clues in the history and physical examination.

5. PREVENT FURTHER FITS

Unless the child has had a simple febrile convulsion, and if the child has not already received one or the other:

- a. Give a loading dose of either phenobarbitone 15 mg/kg IM, or phenytoin 15-20 mg/kg IV slowly over 30 mins, and
- b. Continue maintenance doses - for the duration of the illness, or for several months if the fits have been very difficult to control.

THE LONGER THE FIT LASTS THE MORE LIKELY THERE IS TO BE PERMANENT BRAIN DAMAGE. NEVER LEAVE A FITTING CHILD UNTIL THE CONVULSION HAS BEEN CONTROLLED. DON'T LEAVE IT TO SOMEONE ELSE.

NOTES ON SPECIFIC METABOLIC CAUSES OF SEIZURES

Hypoglycaemia

Always check for this in any fitting child and treat it if present - or treat it anyway if you can't test for it (IV 10% dextrose 5 ml/kg or 50% dextrose 1 ml/kg)

Hypocalcaemia

Seen in neonatal tetany (very rare in breastfed babies), severe malnutrition, malabsorption, chronic diarrhoea, chronic renal failure, Vitamin D resistance (specific or secondary to renal tubular dysfunction), post-acidosis, hypoparathyroidism. It is often accompanied by hypomagnesaemia

Give 10% calcium gluconate 1 ml/kg IV slowly AND 50% magnesium sulphate 0.2 ml/kg IM.

Hyponatraemia (Na <125 mmol/l)

With normal or high urea and low K, suggests hyponatraemic dehydration. Correct dehydration with half strength Darrows or with normal saline and potassium.

With low urea and low K, suggests increased secretion of ADH (meningitis, severe pneumonia). May need fluid restriction.

With normal or high urea and high potassium, suggests hypoadrenalism. Probably requires treatment with hydrocortisone.

Hypernatraemia (Na >150 mmol/l)

Hypernatraemic dehydration. Rehydrate slowly with half strength Darrows solution at 4 ml/kg/hr.

CONVULSIONS - RECURRENT

See also Coma (p.82), Chronic Raised Intracranial Pressure (p.77) and Neonates - Convulsions (p.241).

In a child with one or more convulsions who is otherwise well:

1. Search for headache, vomiting, widened sutures, tense fontanelle, impaired conscious state or papilloedema (ie signs of raised intracranial pressure).
2. Search for focal neurological signs (cranial nerve, cerebellar or long tract signs).
3. Check the blood glucose level.
4. Test the child's urine for blood and protein, and take the BP.
5. If there is no papilloedema, do an LP.

Before diagnosing idiopathic epilepsy, it is important to exclude space occupying lesions (neoplasm, tuberculoma, toruloma), poisoning (arsenic from preserved wood, lead), uraemia and hypoglycaemia.

Many people in developing countries regard the treatment of epilepsy as the domain of traditional healers, and there is a large morbidity from untreated epilepsy. Watts (Brit Med J 298:807-9,1989) has described a successful control programme in rural Africa that depended on publication of the fact that effective treatment is available, a simple regimen using phenobarbitone, adequate supplies of drugs, free treatment, monthly review clinics, mobile clinics, and an effort to ensure that a patient always saw the same health worker.

TREATMENT

If no specific treatable cause is found, a decision will have to be made about whether to attempt long term anticonvulsant treatment. This depends on:

- the frequency and severity of convulsions
- whether the child is otherwise well
- the wishes of the parents and the child
- the educational level of the parents and child
- the family's access to medical services.

If it is decided to treat the child with anticonvulsants:

Principles of anticonvulsant therapy

1. Aim to use a single drug if possible.
2. Use a drug that is readily available to the patient (this will depend on access to hospital or health centre).
3. Give the correct dose of the drug.
4. Change to another drug only after a fair trial of the one initially selected.
5. Use more than one drug only if satisfactory control with one drug is not achieved.
6. Try and review the patient frequently when starting treatment to ensure adequate compliance and control.

Treatment choices

There are four "broad spectrum" anticonvulsants available in Papua New Guinea.

Phenobarbitone: maintenance dose 5 mg/kg/day

- widely available in health centres
- cheap
- once a day dose
- behavioural side effects seem to be uncommon in Papua New Guinean children

- widely used and effective in low health budget countries.

Phenytoin: maintenance dose 3-8 mg/kg/day

- widely available in health centres
- cheap
- once a day dose
- side effects of nystagmus, ataxia, drowsiness, gum hypertrophy
- comes in 30 mg and 100 mg tablets or capsules, which can easily get mixed up.

Carbamazepine: maintenance dose 10-30 mg/kg/day given BD or TID

- available only from hospitals
- relatively cheap
- few side effects in lower doses
- drug of choice in temporal lobe seizures.

Sodium valproate: maintenance dose 10-50 mg/kg/day given BD or TID

- available (inconsistently) only from hospitals
- expensive
- few side effects
- drug of choice in petit mal epilepsy.

For most patients it is most practical to start with phenobarbitone. The maintenance dose of 5 mg/kg/day can be increased up to 10 mg/kg/day if necessary, and may be divided BD to provide smoother blood levels.

Some children with drop attacks or infantile spasms respond well to carbamazepine. Others may require treatment with a benzodiazepine-nitrazepam (1-5 mg BD) alone or with carbamazepine. Myoclonic seizures are best controlled with benzodiazepine-nitrazepam.

Some children in whom control is difficult to achieve with phenobarbitone, phenytoin or carbamazepine, will respond to sodium valproate.

NOTES

1. Start with low doses and build up gradually to avoid toxic effects.
2. Symptoms such as irritability, drowsiness, nystagmus, ataxia and skin rash suggest side effects.
3. Antimalarials may increase the incidence of convulsions.
4. Avoid the sudden cessation or withdrawal of a drug. Sudden withdrawal is very likely to exacerbate the convulsions.
5. Aim for at least a one year and preferably a 2-year period free of convulsions before gradual withdrawal of anticonvulsant drugs.

REFERENCES

- Danaya RT, Attah Johnson F, Ambihapahar U. PNG Med J 37(1): 3-6,1994. Childhood epilepsy in Papua New Guinea.
- Danaya RT. PNG Med J 38(3) 159-162,1995. Childhood disabilities in Papua New Guinea.
- Vince JD. PNG Med J 35(2) 144-151,1992. Clinical practice. Convulsions in children.

CRETINISM (ENDEMIC)

NEUROLOGIC ENDEMIC CRETINISM

This was once common in parts of Papua New Guinea (including the Jimi Valley and uplands Madang Province). It is caused by maternal iodine deficiency in the first trimester of pregnancy and is the extreme end of the spectrum of iodine deficiency disease which includes subclinical intellectual handicap and endemic goitre. Iodine deficiency is thought to be the commonest cause of preventable intellectual handicap in the world. Neurologic endemic cretinism has been virtually eliminated from Papua New Guinea by mass injection of iodised oil and the addition of iodine to salt, but may still occur in isolated parts of the country. Children with cretinism are intellectually handicapped. They are hypotonic babies, but later develop spasticity (at the knees but not the ankles) with an unusual gait (broad-based, bent and knock-knees, flat everted feet, hips adducted, arms swing poorly with slow shuffling movements, shoulder swaying). There is often nerve deafness or strabismus. Goitre may occur, but thyroid function tests are usually normal. Myxoedematous features are rare (this and other features should allow a clear differentiation from congenital hypothyroidism). It should be suspected with the above clinical features, especially in areas where the visible goitre rate is >5%, deaf and dumb children are common, or where small goitres are frequently present in school children. Medical personnel should report all cases and also check and report if non-iodised salt is being sold in the area. Iodised oil administration to females between 10 and 45 years of age should then be considered.

SPORADIC (MYXOEDEMATOUS) CRETINISM

See Hypothyroidism (p.162).

REFERENCE

Hetzel BS, Pharoah POD. Endemic Cretinism, Institute of Human Biology Monograph Series, No.2, 1971. Goroka, PNG: Institute of Medical Research.

CROSSMATCH OF BLOOD

For crossmatch for exchange transfusion, see p.247. See also Blood Transfusion - Indications (p.55).

1. Collect blood from the patient into a plain bottle (3 ml from a baby, 5 ml from a child, 10 ml from an adult).
2. Let the blood stand for about 15 minutes, until it clots.
3. Centrifuge, and separate the serum into another tube.
4. Group the patient's cells by mixing one drop of cells with one drop of anti-A antiserum, one drop with anti-B and one drop with anti-AB:

Patient's group	Antiserum		
	Anti-A	Anti-B	Anti-AB
A	+	-	+
B	-	+	+
AB	+	+	+
O	-	-	-

+ agglutination, - no agglutination

5. Remove group compatible bag(s) of blood from the Blood Bank fridge (use donor blood of the same group as the patient if you can):

Patient's group	Use donor blood (group)
A	A, O
B	B, O
AB	AB, A, B, O
O	O

6. Tie a knot in the long plastic side tube of the bag of donor blood. Empty the donor red cells from the side tube into a labelled glass tube (one tube for each bag of donor blood).
7. You should now have:
 - one labelled tube of patient's cells
 - one labelled tube of patient's serum
 - one labelled tube of donor cells for EACH bag of donor blood.
8. Mix one drop of each reagent on a crossmatch tile as follows (the example is for two bags being crossmatched):

	Papain	Albumin	Saline	Positive control
	A	B	C	D
Bag 1	Donor cells Pt serum Gluc citrate Papain	Donor cells Pt serum Albumin	Donor cells Pt serum Saline	Donor cells Papain anti-D Gluc citrate
Bag 2	E Donor cells Pt serum Gluc citrate Papain	F Donor cells Pt serum Albumin	G Donor cells Pt serum Saline	H Donor cells Papain anti-D Gluc citrate
Auto control	I Pt cells Pt serum Gluc citrate Papain	J Pt cells Pt serum Albumin	K Pt cells Pt serum Saline	L Pt cells Papain anti-D Gluc citrate

9. Incubate the tile at 37 °C for 15 minutes, then read for agglutination:
- squares D, H and L should all agglutinate (positive control). If they do not, the crossmatch is invalid
 - if squares A, B, C, E, F, G, I, J and K do not agglutinate, the blood is compatible
 - if squares A, B, C, E, F and G agglutinate but I, J and K do not, the donor blood is incompatible with the patient's blood. Check that you have grouped the patient correctly
 - if squares I, J and K agglutinate but A, B, C, E, F and G do not, the blood is compatible but the patient has auto-antibodies (is there haemolytic anaemia?). See p.20
 - if squares A, B, C, E, F and G agglutinate, but not as heavily as I, J and K, the blood may still be compatible - but agglutination is occurring because of auto-antibodies (is there haemolytic anaemia?). Get expert advice if you can before transfusing.

CUT DOWN

Cut downs should only be used as a last resort, or in an emergency. They make the vein useless for future cannulation and have a high incidence of infection. Scalp vein needles or Intracaths are preferable (p.172).

Intra-osseous fluid can be given in an emergency (p.171).

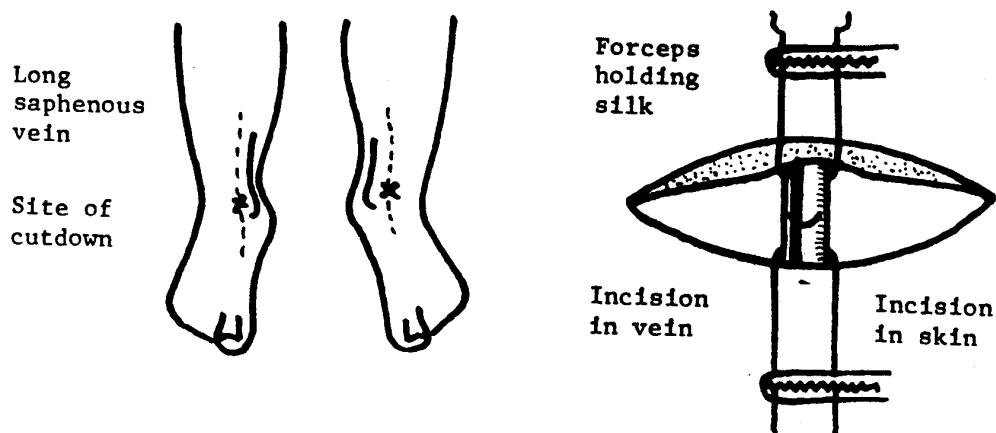
SITES

- The long saphenous vein just lateral to the medial malleolus at the ankle.
- The basilic vein on the medial side of the cubital fossa at the elbow. The cephalic vein at the lateral side of the cubital fossa is less satisfactory.
- The cephalic vein in the groove between biceps and deltoid at the lateral side of the upper arm.

INSERTION

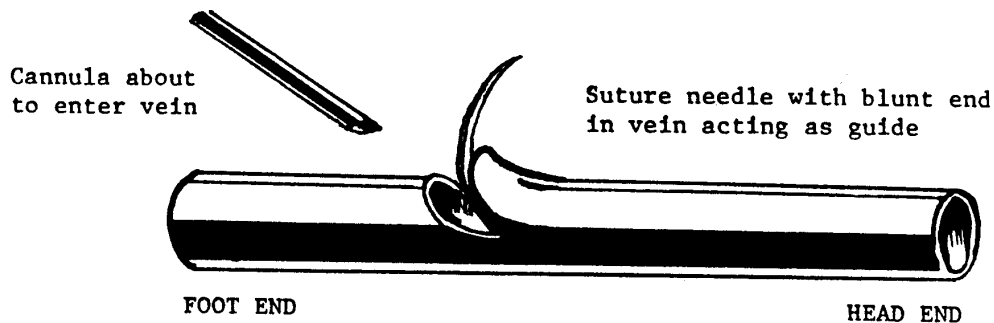
Cannulation of the long saphenous vein will be described.

1. Have an assistant hold the child's leg firmly.
2. Clean the ankle thoroughly with iodine and drape.
3. Inject 1% or 2% plain lignocaine (Xylocaine) into the skin.
4. Apply a tourniquet to the thigh tightly enough to occlude the veins, but NOT tightly enough to occlude the arteries (check that the tibial pulse is still present).
5. Make an incision in the skin from just above the medial malleolus extending 2 cm laterally. Do NOT cut too deeply with the scalpel - ONLY use it to cut the skin.
6. Use careful blunt dissection of the subcutaneous tissue with a pair of artery forceps to expose the long saphenous vein. In a fat child, the vein may be quite deep.



7. Place two loops of silk thread under the vein, one at the top of the wound, the other at the bottom. Place forceps across the two ends of each loop. These can be used to exert gentle tension on the vein and to position it.
8. With a small, sharp pair of scissors make a small V shaped nick in the wall of the vein. Do NOT cut right through the vein. Have the assistant exert gentle traction on the lower loop of thread.
9. Insert the BLUNT end of a curved suture needle into the incision in the vein. This acts as a guide for the cannula.

10. Thread the cannula into the vein. This is often easier to do if you cut a bevel in the end by cutting across the catheter obliquely. The shorter you cut the cannula, the more rapidly fluid will run through it - about 15 cm (6 inches) is often all that is needed. Do NOT leave a thin cannula too long.
11. After the cannula is in the vein a short distance, it is a good idea to infuse fluid through it while you thread it up the vein: this helps it to thread more easily, and makes it less likely to tear the wall of the vein.



Greatly magnified lateral view of venous cutdown on saphenous vein.

12. Remove the silk threads from around the vein. Do NOT use them to tie off the vein.
13. Suture the catheter in with a stitch through the skin just below the incision.
14. Apply antibiotic powder to the wound, and suture the skin. Apply a firm dressing to stop bleeding. Splint the leg securely.

DO NOT LEAVE A CUT DOWN IN ANY LONGER THAN NECESSARY. Replace it with a percutaneous drip as soon as possible.

DEVELOPMENTAL MILESTONES

You should remember four important milestones:

	50% do it by:	97% do it by:
1. Sits unsupported	6 months	9 months
2. Walks 10 steps unsupported	12 months	18 months
3. Speaks 3-4 single words	14 months	20 months
4. Phrase ("Daddy go work")	24 months	36 months

The following more detailed milestones were suggested by Penelope Hubley for Indian village children, and are quoted by Dr David Morley in "See How They Grow", Macmillan, London, 1979.

T = tested by putting the child in a situation where the desired activity is likely to be undertaken.		
A = ask the mother (but, because she is concerned for her child, she is sometimes unreliable).		
Age	Behaviour	
3 months	T	Visually very alert, particularly interested in human faces.
	T	Moves head deliberately to look around him.
	T	Definite response to mother's voice either by becoming quiet or smiling.
6 months	A, T	Can roll over from belly to back.
	T	Placed downwards on face, will lift head and chest well up, supporting himself on extended arms.
	T	If held standing with feet touching a hard surface, the baby will bear his weight on his feet and bounce up and down actively.
	A, T	The baby will show competent reaching and grasping of small objects.
9 months	A, T	The baby will sit alone for ten to fifteen minutes on a firm surface.
	A, T	The baby can move on the floor by rolling or squirming.
	A, T	The baby tries to crawl on hands and legs.
	T	If the baby is held standing, he will step on one foot then the other.
	A, T	The baby can distinguish strangers from known persons. May become distressed with strangers.
12 months	A, T	Can rise to sitting position from lying down.
	A, T	Pulls himself to standing and lets himself down again, holding on to furniture.
	A, T	If the baby's hands are held he will walk. He may even walk without help.
	T	Gives objects to an adult on request (before this age he will not do this).
15 months	T	Will walk unevenly with feet wide apart and arms raised to balance.
	A, T	Speaks 2-6 recognisable words. Understands more.
	T	Points to familiar persons, animals, toys when requested.
18 months	T	Picks up a toy from the floor without falling.
	T	Shows his own hair, nose and eyes on request.
	A, T	Demands desired objects by pointing and making a noise.
	A, T	Explores his surroundings energetically.
2 years	T	Runs safely on whole feet, stopping and starting easily to avoid obstacles.
	A, T	Puts two or more words together to form a simple sentence.
	A, T	Refers to himself by name.
	A, T	Shows correctly when asked, his hair, hands, feet, nose, eyes and mouth and repeats their correct names.

T = tested by putting the child in a situation where the desired activity is likely to be undertaken.

A = ask the mother (but, because she is concerned for her child, she is sometimes unreliable).

Age	Behaviour	
2.5 years	T	Jumps with two feet together.
	T	Can stand on tip-toe if shown.
	A, T	Uses the pronouns, I, me and you.
3 years	T	Stands for a short time on one foot when shown.
	T	Carries out simple conversations and talks about past experiences.
	A	Likes to help with adult activities.
	A	Joins in play with other children.
4 years	T	Hops on one foot.
	A	Climbs ladders and trees.
	T	Gives a connected account of recent experiences.
	A	Needs other children to play with and is alternatively co-operative and aggressive.
5 years	T	Runs lightly on his toes.
	T	Can stand on one foot for 8-10 seconds.
	T	Can hop 2-3 metres forward on each foot.
	A	Undresses and dresses alone.

DIABETES MELLITUS

Diabetes mellitus is rare in children in Papua New Guinea, but may increase in frequency as lifestyles progressively westernise. It is usually due to Type 1, or insulin-dependent diabetes mellitus (of autoimmune aetiology). Fibrocalculous disease of the pancreas (which results in diabetes and is associated with malnutrition) is seen occasionally. Type 2 (non-insulin-dependent) diabetes may also be seen, particularly in overweight adolescents from coastal regions.

Diabetes is a complex disorder. Life-threatening crises (hypoglycaemia or ketoacidotic coma) are frequent unless the child is well managed. Optimal management is by a multi-disciplinary team (physician, diabetic nurse educator, dietitian, social worker), but good results may be obtained by careful management in the provinces.

PRESENTATION

The commonest symptoms of diabetes are polyuria, polydipsia and weight loss. Also frequent are nocturia/enuresis, visual disturbance, abdominal pain, and vaginal itch or thrush. Symptoms will steadily progress. Ketoacidosis presents as decreased consciousness or coma, vomiting, dehydration, abdominal pain, Kussmaul-type hyperventilation and the smell of ketones.

Differential diagnosis includes UTI, diabetes insipidus, renal disease and stress hyperglycaemia. It is important to think of diabetes in atypical cases of severe abdominal pain, “pneumonia” and “gastroenteritis”.

DIAGNOSIS

Made by demonstrating a persistently elevated blood sugar level (BSL). Glucose tolerance tests are almost never necessary. Therapy should commence immediately after diagnosis - don't send the child home to come back to clinic! An abdominal X-ray should be done to exclude fibrocalculous disease of the pancreas. Urea and electrolytes, urinalysis, (and blood gases if unwell) should be performed if possible, and sometimes a CXR or urine MCS is indicated.

TREATMENT

Two types of insulin are used - short acting (Actrapid, Humulin R, or similar), and long acting (Isophane, Humulin NPH, or similar). NB: Insulin is usually in the strength 100 Units/ml, so 10 Units = only 0.1 ml.

1. Ketoacidosis

If shock is present, resuscitation should be carried out with 10-20 ml/kg of normal saline or Haemaccel given quickly.

Insulin must be commenced as soon as possible. Short-acting insulin 50 Units is loaded into 50 ml of normal saline and given at 0.1 ml/kg/hr (equating to 0.1 U/kg/hr of insulin) in a side drip. If an infusion cannot be loaded quickly, commence therapy with 0.1 ml/kg given SC or IM every hour.

Fluid need should be calculated from maintenance and deficit, and then administered initially as normal saline, half of the 24 hour requirement given in the first 8 hours and the remainder in the next 16. Monitor BSL half to one hourly. Once the BSL has fallen to 12-15 mmol/l, fluids should be changed to 4% dextrose and 0.18% saline (or half normal saline made up to 5% dextrose if available). Do not stop insulin infusion. It is often necessary to add extra dextrose to the saline/dextrose infusion to maintain BSL above 4 mmol/l. Electrolytes, pH and urine ketones should be monitored every 2-4 hours. Substantial potassium replacement is necessary, and should be commenced as 30 mmol/l of fluid as soon as urine output is confirmed.

Intravenous bicarbonate is only very rarely indicated and may be harmful in some cases. Excessive fluid administration should be avoided, and hypernatraemia presents added dangers. Cerebral

oedema is a rare but catastrophic complication of management, and, if suspected, mannitol should be administered immediately.

This is a life-threatening condition - advice should be obtained from experts if you are inexperienced.

2. Once ketoacidosis has resolved, or initial therapy if not ketoacidotic.

Commence regular insulin as below, starting at 0.25 U/kg/day in two divided doses. Two-thirds of the daily dose should be given 30 min before breakfast and one-third 30 min before the evening meal. On each occasion, two-thirds is given as “long” acting and one-third as “short” acting. Titrate dose to response, aiming to avoid hypoglycaemia and keep BSL 4-8 mmol/l. Generally around a total of 0.5 U/kg/day is needed for prepubertal children, and 0.5-1 U/kg/day for pubertal children. Insulin requirements may fall temporarily in the first few months (the honeymoon period).

3. Long-term management

Details are beyond the scope of this entry. A full protocol tailored to PNG conditions may be obtained on request from the UPNG Professor of Child Health. Caloric intake should be spread through the day, with three meals and six snacks, and regular exercise. As much as possible, the choice of foods should conform to the normal family diet. Simple carbohydrate (“sugar”) foods, drinks and sweets should be avoided. BSL should be monitored with glucometers at home if possible, otherwise test urine. Parents and child need education on injection technique, diet, exercise, recognition and management of hypoglycaemia, “sick days” and recognition of ketosis. Much encouragement and support is needed. Complications should be monitored for and managed appropriately.

DIARRHOEA

Diarrhoea can be defined as the passage of frequent stools of a consistency more fluid than usual.

Diarrhoea is one of the top four causes of death and morbidity in Papua New Guinean children.

WHO now classifies diarrhoea into three types - and this classification is very useful in guiding treatment.

1. Acute watery diarrhoea. The majority of cases. Most deaths in this group are due to dehydration and are preventable by oral rehydration therapy (ORT) or if necessary intravenous rehydration therapy (IVT). Antibiotics are usually not indicated.
2. Dysentery. Diarrhoea with blood mixed through the stool. This accounts for relatively few cases but has a significant mortality. Antibiotic therapy is usually indicated in addition to rehydration.
3. Persistent diarrhoea. Diarrhoea which persists for more than 14 days. (In PNG 7 days is taken as the defining period). This accounts for the smallest number of cases but has the highest mortality (up to 20-30%). It is almost invariably associated with severe malnutrition and affected children require intensive medical treatment.

DEHYDRATION - AND REHYDRATION USING ORT OR IVT

The main cause of death in diarrhoeal disease of children is dehydration. Emphasis must therefore always be placed on prevention of dehydration and correction of dehydration if it is present.

Prevention - more fluids more often

This is the most important part of the prevention of dehydration. However, it is not something which is naturally done - fluids are often withheld if a child has diarrhoea. In discussing the management of her child with a mother it can be useful to use the example of a child being like a plastic bag containing water. If the bag gets a leak (if the child gets diarrhoea) it goes slack (like dehydration) and more water must be put in to keep it full. In the case of the child, the leak usually stops after 2 to 3 days.

Most "home" fluids are suitable to give in the early stages of diarrhoea, before dehydration occurs. Coconut water, tea, mueli water (lemon water), soups from cooking vegetables, "Guava water" (water boiled with guava leaves - a traditional treatment in some parts of the country) and plain water or water with a heaped teaspoon of sugar in a large mug (sugar-water) are all reasonable.

Treatment

Once dehydration occurs it is necessary to use Oral Rehydration Solution - either the Unicef product - ORS - (1 sachet dissolved in 1 litre of water) or a home made solution made of a heaped teaspoon of sugar and a finger pinch of salt in a large mug of water. ORS works very well, and depends on the active co-transport of glucose and sodium across the intestinal cell membrane - a process which is much more robust than the other sodium transporting mechanisms. Water is carried with the sugar and salt. Even severely dehydrated children can be satisfactorily rehydrated with ORS. "Cereal based" Oral Rehydration Solutions containing starch from cereals and salt are as effective, if not more so, than glucose based ORS. They are not widely used in Papua New Guinea but water from cooking rice or vegetables and containing a little salt (the amount used for normal cooking) is an efficient form of ORS.

Some countries have a policy of "a packet of ORS readily available for every household". In Papua New Guinea the policy is in general to reserve ORS for treatment of dehydrated children in health centre or hospital - and to rely on home fluids for the prevention of dehydration.

The step by step management of children with diarrhoea is detailed in the Paediatric Standard Treatment Book and is summarised in the chart (p.107).

IMPORTANT POINTS TO REMEMBER

1. Always encourage mother to continue breastfeeding (unless there is evidence of severe lactose intolerance).
2. Give food to children with diarrhoea - they require more calories, not less.
3. Always check for other illnesses.

MILD DIARRHOEA

No signs of dehydration.

Treat the child as an outpatient. Discuss with the mother the importance of giving extra fluids every time the child has a watery stool and give some fluids to the child to drink under supervision.

Examine and treat any other infection present.

Encourage mother to continue breastfeeding and to give extra food.

Encourage the mother to bring the child back for reassessment if the diarrhoea becomes worse, becomes bloodstained or continues for more than two days, if there is persistent vomiting, if the child becomes very thirsty or loses interest in feeding and drinking or if there is persistent fever.

DIARRHOEA WITH DEHYDRATION

Signs of dehydration start to appear when the child is 4-5% dehydrated (4-5% of body weight). Signs of severe dehydration and shock appear when the child is 10% dehydrated or more.

ORT

If the child has signs of dehydration but is not shocked, is not vomiting frequently and does not have a distended abdomen, ORT using the WHO/UNICEF ORS is likely to be successful in rehydrating the child. The child should be given as much as he or she can drink - at least a big cup every hour.

Note: It is better to give small sips very frequently than to try and force the child to drink a large amount all at once if the child is unwilling to do so, or if he vomits after a large amount. One teaspoon (5 ml) every minute is 300 ml in an hour - a substantial amount!

IVT

If the child has signs of severe dehydration, shock, refuses to drink, is vomiting repeatedly or has a distended abdomen (the likelihood of a hypokalaemic paralytic ileus) IVT will be required. The standard fluid is half strength Darrow's solution. This fluid contains Na 61, K 18, Cl 52, and HCO₃ 27 mmol/l in 2.5% dextrose. It is specifically designed for treatment of diarrhoea and is suitable for the initial management of all forms of severe dehydration, hypo-, iso- and hypertonic. It is given in steps of 20 ml/kg as fast as possible with regular reassessment until signs of dehydration improve, and then is maintained at a rate of about 150 ml/kg per 24 hours.

IVT can be discontinued when the child is drinking satisfactorily.

If you are unable to put in an intravenous line, give fluids by an alternative route.

Other routes of giving fluids:

1. **Nasogastric.** This can be used if the abdomen is not distended, the child is not vomiting repeatedly and is not shocked. Make sure the tube is in the stomach, and splint the child's arms so that he cannot dislodge it. ORS or half strength Darrow's are ideal. 20 mls/kg can be given every hour.
2. **Intraosseous** (as in treatment of shock, see p.171).
3. **Intraperitoneal.** This cannot be used if there is abdominal distension, or if the child is profoundly shocked. The fluid to be given must be warmed to approximately body temperature.

A scalp vein needle is inserted horizontally just under the skin in the midline about 1 cm below the umbilicus. The drip is fully opened and the scalp vein lifted from the horizontal to the vertical. It is gently pushed into the peritoneal cavity. On entering the cavity, fluid will start to run freely.

Once a child reaches a health care facility he should not die of dehydration.

DRUG TREATMENT OF CHILDREN WITH DIARRHOEA

Most children with diarrhoea do not require antibiotics.

Drugs are given as follows:

1. Antimalarials
 - a. If the child is not dehydrated but febrile, treat as for uncomplicated malaria (see p.197).
 - b. If the child is severely dehydrated and febrile, treat as for complicated malaria (see p.197).
2. Antibiotics
 - a. If the child is shocked and possibly septicaemic, give chloramphenicol.
 - b. If the child is severely malnourished, give ampicillin and gentamicin.
 - c. If the child has dysentery, give cotrimoxazole.
3. Antiprotozoal agent (tinidazole or metronidazole)
 - a. If the child is severely malnourished.
 - b. If the child has severe diarrhoea for more than 3 days despite treatment.
 - c. If the child has dysentery (give for 3 days).
 - d. If the child has persistent diarrhoea.
4. Anthelmintics (Albendazole)
 - a. If the child has severe diarrhoea.
 - b. If the child has persistent diarrhoea.
 - c. If the child has oedema (give for 3 days).
 - d. If the child has dysentery.

DIARRHOEA WITH BLOOD - DYSENTERY

If there is only a small amount of blood and the child is not sick, no special treatment is required. Keep the child under review.

If the child has blood and mucus mixed through the stool, looks sick, has malnutrition, has a small but persisting amount of blood or has a large amount of blood, treat with:

- antimalarials (as for complicated malaria)
- cotrimoxazole
- tinidazole
- albendazole.

BEWARE THE POSSIBILITY OF INTUSSUSCEPTION. Children often present late without the characteristic history of colicky screaming attacks.

PERSISTENT DIARRHOEA

(More than 7 days - see above).

This is a serious condition associated with a very high mortality. Children with persistent diarrhoea are almost always severely malnourished and require intensive treatment. They require:

- intensive nutrition support (including trace elements zinc and magnesium and vitamins, see p.216). Parenteral nutrition may occasionally be required (see p.306)
- tinidazole
- albendazole

- antimalarials, if febrile (as for Complicated Malaria, p.197).
- antibiotics, if severely malnourished, febrile or showing any signs of septicaemia (ampicillin and gentamicin)
- nystatin, if signs of mucocutaneous candidiasis.

Lactose intolerance

Children with persistent diarrhoea may have temporary lactose intolerance. This diagnosis is likely if they have a red “burnt” perineum. Test the stool for lactose using Clinitest tablets (5 drops of liquid stool, 10 drops of water and 1 Clinitest tablet. A yellow, orange or red colour after 1 minute denotes the presence of a reducing substance). Management of children with lactose intolerance includes the withdrawal of breastmilk (or other lactose containing milk) for 2 to 3 days, and its substitution by a low lactose milk if available (such as Pregestemil, Ensure, Digestelact, Nutramigen or Infasoy) or by other forms of nutrition if not available. It is important that mother expresses her breasts to prevent soreness and discomfort, and understands that breastfeeding should continue when the diarrhoea settles.

REMEMBER THE POSSIBILITY OF TYPHOID AND HIV INFECTION.

REFERENCES

- Conroy RM, Elmore-Meegan M, Joyce T et al. Lancet 348:1695-1697,1996. Solar disinfection of drinking water and diarrhoea in Maasai children: a controlled field trial.
- Dale CB. J Diarr Dis Res 5(4),1987. Symposium on Super ORS.
- Gomae A. Lancet 2:145-148,1988. Impact of an ORS campaign in Egypt.
- Gore S, Fontaine O, Pierce N. Br Med J 304:287-291,1992. Impact of a rice based oral rehydration solution on stool output and duration of diarrhoea:meta-analysis of 13 clinical trials.
- Han AM, Sleight A, Vince J et al. PNG Med J 38(4):272-277,1995. Persistent diarrhoea in children admitted to Port Moresby General Hospital.
- Howard P, Alexander ND, Atkinson A et al. J Trop Paediatr 46:10-13,2000. Bacterial, viral and parasitic aetiology of paediatric diarrhoea in the highlands of Papua New Guinea.
- PNG Med J 38(4):257-338,1995. Focus issue on enteric infections.
- Pratt D, Smith D, Barker J et al. PNG Med J 35(1):22-25,1992. Rotavirus diarrhoea in children in the highlands of Papua New Guinea.
- Shann FA. PNG Med J 22:98-100,1979. Metronidazole for chronic diarrhoea.
- Sodemmann M, Jacobsen MS, Molba KK et al. Trans Roy Soc Trop Med 93:65-68,1999. Episode specific risks factors for progression of acute diarrhoea to persistent diarrhoea in West African children.
- Vince JD. PNG Med J 38(4):262-271,1995. Diarrhoea in children in Papua New Guinea.
- World Health Organisation. The rational use of drugs in the management of acute diarrhoea in children. Geneva: WHO, 1990.

DIARRHOEA - DIAGNOSIS AND MANAGEMENT SUMMARY



	START HERE		
	A	B	C
1. Look at: • Condition • Eyes • Tears • Mouth and tongue • Thirst	Well, alert Normal Present Moist Drinks normally	Restless, irritable Sunken Absent Dry Thirsty, drinks eagerly	Lethargic or unconscious; floppy Very sunken and dry Absent Very dry Drinks poorly or not able to drink
2. Feel • Skin pinch • Pulse rate • Limb temperature	Goes back quickly Less than 120/min Warm limbs	Goes back slowly 120-160/min Warm limbs	Goes back very slowly More than 160/min Cold limbs
3. Check C, then B, then A	←	←	←
4. Decide	The child has NO SIGNS OF DEHYDRATION	If the child has two or more signs in Col B, there is SOME DEHYDRATION	If the child has two or more signs in Col C, there is SEVERE DEHYDRATION
5. Diagnosis	MILD DIARRHOEA	MODERATE DIARRHOEA	SEVERE DIARRHOEA
6. Treat diarrhoea	More fluids more often at home	Oral Rehydration Solution (ORS) under supervision	IV half-strength Darrow's (HSD) as an inpatient
7. Other treatment	Treat fever if present	For uncomplicated malaria	For severe malaria plus IM chloramphenicol
8. Review	Daily	Every 3-4 hours	Every hour

NOTES

1. Always continue breast feeding and give food to older children.
2. Bloody diarrhoea or persistent diarrhoea, see p.105.
3. Examine all children carefully for other illnesses.

DROWNING

The immediate threat to life comes from hypoxaemia caused by lung damage (stiff lungs, pulmonary oedema and A-V shunting are present), shock, hypothermia, renal failure and metabolic acidosis. The long-term prognosis in survivors depends on the degree of cerebral damage from hypoxaemia at the time of drowning.

FIRST AID RESUSCITATION ON SITE IS OF VITAL IMPORTANCE.

BEWARE THE DANGER OF CERVICAL INJURY IN A DIVING ACCIDENT.

Previous emphasis on differences between drowning in salt and fresh water was unwarranted. Steroids have no place in the management of drowning.

TREATMENT

On admission to hospital:

1. Give oxygen and gradually rewarm the child.
2. Intubate, and arrange for tracheostomy if necessary.
3. Pass a nasogastric tube and aspirate stomach contents (IMPORTANT - abdominal distention often causes respiratory embarrassment in drowned children).
4. Positive pressure ventilation with PEEP may be required to correct hypoxaemia (but it will often not be available).
5. Insert an IV - into a central vein if possible. If the child is shocked give a rapid infusion of saline or (preferably) SPPS 20 ml/kg.
6. Give 2 ml/kg of 8.4% sodium bicarbonate slowly IV. Monitor blood gases frequently if possible.
7. Take blood for grouping, Hb and electrolytes.
8. Pass a urinary catheter.
9. Take a chest x-ray.
10. Aminophylline IV may help if there is a wheeze.
11. Commence broad spectrum antibiotics.

Subsequent management involves:

- adjustment of the inspired oxygen concentration and ventilation to maintain arterial oxygen tension (and avoid cyanosis)
- administration of 8.4% sodium bicarbonate IV to correct metabolic acidosis (if blood gas estimation is available)
- adjustment of the blood volume with SPPS or plasma (of the same blood group as the patient) using central venous pressure and urine output as guides.

REFERENCE

The child with an electrical injury or near drowning. Chap 21, p.205-210. In: Advanced paediatric life support. The practical approach. 3rd ed. London: BMJ Books, 2001.

ECG INTERPRETATION

PAPER SPEED

The ECG is normally recorded at 25 cm/sec in which case 1 mm = 0.04 sec.

P-WAVE

Reflects atrial activity.

Duration shorter than in adults: • infants: 0.04 - 0.07s • adolescents: 0.06 - 0.1s

Height 2.5 mm

RIGHT ATRIAL HYPERTROPHY (RAH)

Increased p-wave amplitude in II; V1; V4R (Rt. sided)

Occurs in pulmonary stenosis
pulmonary atresia
tricuspid atresia

LEFT ATRIAL HYPERTROPHY (LAH)

Biphasic p-wave (later depolarisation of LA).

Occurs in: mitral valve disease
LV obstruction and disease

Varying p-wave morphology may indicate wandering atrial pacemaker.

PR INTERVAL

Atrial depolarisation varies with age and rate:

Normal range of PR interval (time in secs)				
Heart rate	0-1 month	0-12 month	3-8 years	12-16 years
<60	-	-	-	0.1-0.19
60-100	-	-	0.1-0.16	0.1-0.17
100-140	0.08-0.11	0.08-0.12	0.1-0.14	
140-180	0.08-0.11	0.08-0.12	0.1-0.14	
>180	0.08-0.09	0.08-0.11		

Prolonged: slight prolongation may be normal
myocarditis (eg acute rheumatic fever)
ischaemia
drugs
hyperkalaemia

Short: Wolff Parkinson-White
Lown-Ganog-Levine
glycogen storage disease

Variable: wandering atrial pacemaker
Wenkebach phenomenon

QRS COMPLEX

Ventricular activity

Duration: 0.06 - 0.08 secs

Prolonged: ventricular hypertrophy
 bundle branch block
 electrolyte disturbance
 metabolic disease
 drugs, eg digoxin

Normal maximum size of R and S waves (height in mm)				
Age	V1		V6	
	R	S	R	S
Birth	12	10	5	6
6 months	11	10	14	3
12 months	9	10	14	3
10 years	5	10	16	2
From: Heart Disease in Paediatrics. Jordan/Scott				

Q-WAVES

Normal in II; III; aVF; V5-6

Depth 2-3 mm

Pathological if greater than 4 mm (ie septal hypertrophy)

May be found in other leads in: anomalous coronary arteries
 hypertrophic obstructive cardiomyopathy
 transposition of great arteries (with opposite polarity).

Q-T INTERVAL

This is inversely proportional to the rate: $QT_c = QT \text{ measured} / RR \text{ interval}$

Normal value for QT_c should not exceed: 0.45 sec in infants <6 months birth
 0.425 sec at adolescence

Prolonged: Hypocalcaemia
 Myocarditis
 Long QT syndrome: Jervell & Lange-Neilsen syndrome
 Romano-Ward syndrome
 Head injuries or cerebrovascular episodes
 Diffuse myocardial disease
 Antiarrhythmics

Short Hypercalcaemia
 Digitalis effect

T-WAVE

Ventricular repolarisation

Normal: T inversion V4R / V1
 Amplitude is 25-30% of R wave
 <1yr: V5 - 11 mm; V6 - 7 mm
 >1yr: V5 - 14 mm; V6 - 9 mm
 Adolescence reduces amplitude

Peaked T-wave:	Hyperkalaemia LVH Cerebrovascular episode Post-MI
Flat T-wave:	Normal newborn Hypothyroidism Hypokalaemia Hyper/hypoglycaemia Peri/myocarditis Ischaemia Digoxin effect.

MEAN QRS AXIS

Vertical plane (limb leads)

Normal axis in vertical plane:	Birth	+135 (60 - 180)
	1 year:	+60 (10 - 100)
	10 years::	+65 (30 - 90)

Right axis deviation:	RVH left posterior hemiblock ostium secundum ASD (+ RBBB)
-----------------------	---

Left axis deviation:	LVH ostium primum ASD (+ RBBB) often in conduction defects.
----------------------	---

Horizontal plane (anterior chest leads)

Normal:	Transition at around V3
Clockwise rotation:	S greater than R in V4 = RA/RV hypertrophy
Anticlock rotation:	R greater than S in V2 = cardiac shift (eg pneumothorax).

LEFT VENTRICULAR HYPERTROPHY

Diagnosis:	SV1 + RV5 (40 mm (30 mm below 1 year)) ± prolonged QRS Flat T wave T wave inversion V5-V6 (LV strain) LBBB
------------	--

Causes LVH include:	Aortic stenosis Aortic regurgitation Hypertension Moderate VSD Hypertrophic obstructive cardiomyopathy Patent ductus arteriosus Mitral regurgitation
---------------------	--

RIGHT VENTRICULAR HYPERTROPHY

Diagnosis: RAD and RV1 greater than SV1
SV6 above maximum age: 10 mm (0-6 months)
7 mm (6-12 months)
5 mm (over 12 months)
Large R waves in V4R & V1 ± reduced amplitude V5, V6
T wave changes: upright in V1 / V4R (moderate)
inverted in V1 / V4R (severe)

Causes RVH include:

- Pulmonary stenosis/atresia
- Transposition of great arteries
- Pulmonary regurgitation
- Total anomalous pulmonary drainage
- Tricuspid regurgitation
- Fallot's tetralogy
- Pulmonary hypertension

BIVENTRICULAR HYPERTROPHY

Diagnosis: R + S greater than 50 mm V3-V4
LVH + Bifid R greater than 8 mm V1
RVH + LV strain
Q waves V3-V6 imply septal hypertrophy

TYPICAL CARDIOLOGICAL ECG ABNORMALITIES

PDA:	LVH > RVH; LA.
VSD:	LVH > RVH; ± RBBB; T inv LV leads
ASD:	
secundum	RAD; RBBB; ± increased PR; AF
primum	LAD; RBBB; BVH; RAH
Eisenmengers:	RVH; qRV4R / V1
Aortic stenosis:	LVH + Strain
Aortic regurgitation:	LVH
Coarctation:	
newborn	RVH
older	Normal or LVH ± strain; RBBB
Mitral regurgitation:	LVH; LAH
Pulmonary stenosis:	RVH; RAH
Ebstein anomaly:	Prolonged PR interval; gross RAH; RBBB
Fallot's tetralogy:	
newborn	Normal or T +ve V1
older	RVH; RAH
Pulmonary atresia:	RAH
Tricuspid atresia:	LAD; RAH; LVH

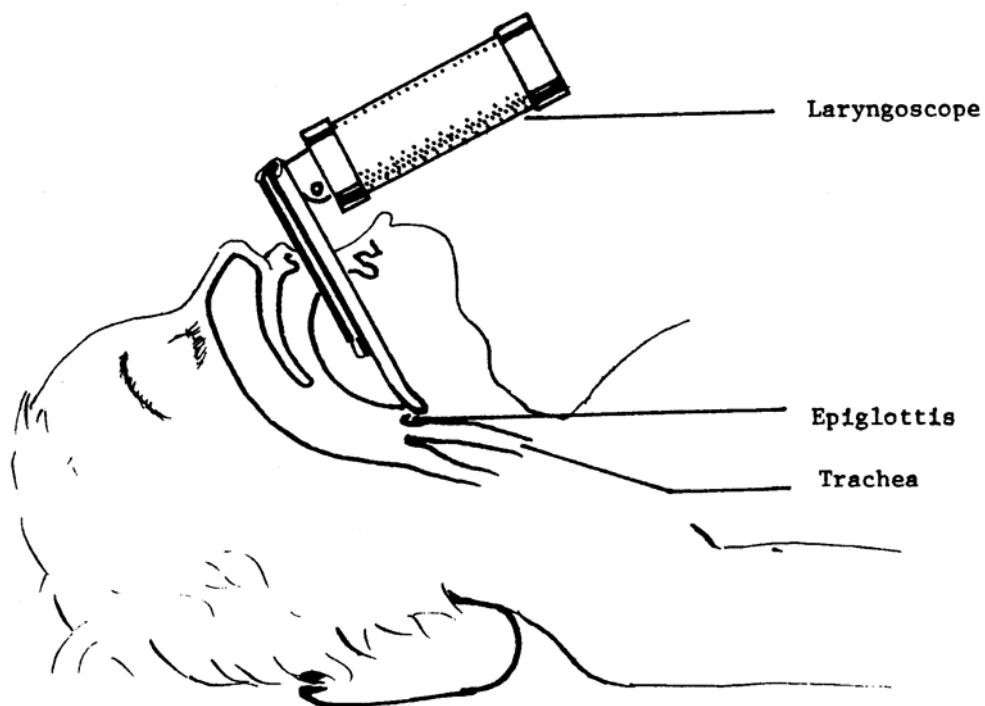
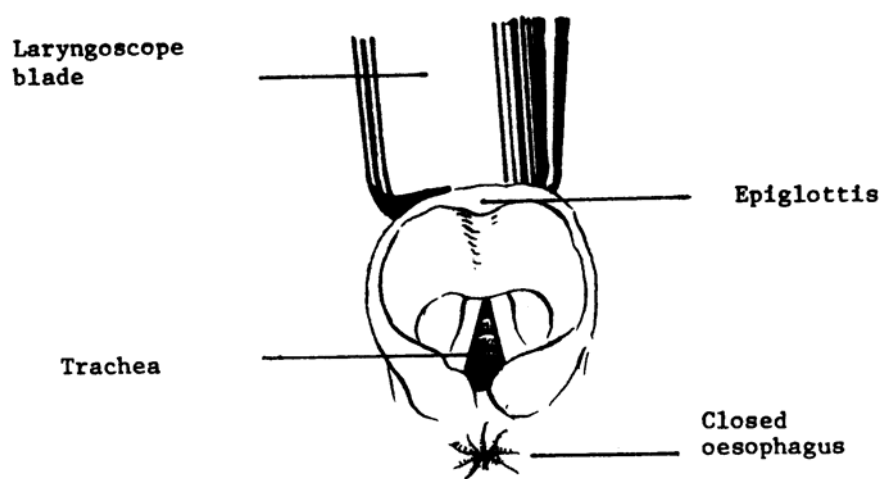
ENDOTRACHEAL INTUBATION

1. The child should be supine and lying flat. Do NOT put a pad under the shoulders or hyperextend the neck.
2. Hold the laryngoscope (with a small blade) in the left hand, and pass the blade along the tongue and anterior to (above) the epiglottis. Suck out any mucus. Under 6 months of age, use a straight bladed laryngoscope.
3. Ask an assistant to press the cricoid cartilage gently backwards towards the spine until you can see the vocal cords.
4. Insert an uncuffed endotracheal tube gently. Do NOT push it in too far. The tip can be felt in the neck if the tube is moved in and out a small distance. The tube should be strapped with the tip in the suprasternal notch. Auscultate to ensure that there is good air entry to both lungs. Suck out the tube.

Endotracheal tube (ETT) size	
Under 1 kg	2.5 mm
1-3.5 kg	3.0 mm
3.5-7 kg	3.5 mm
8-10 kg	4.0 mm
11-14 kg	4.5 mm
15-18 kg	5.0 mm
19-22 kg	5.5 mm
23-28 kg	6.0 mm
Over one year of age, the size of the ETT in mm = 4 + (age in years) / 4.	

Only uncuffed tubes are used in children before puberty. Always have handy a tube of the size above, and one of the size below the estimated size. Endotracheal tubes are usually too long when first supplied. Always cut them so that only about 2 cm protrudes from the mouth - otherwise you can easily push them in too far, and the extra length outside is liable to kink.

5. Before you intubate, always make sure that you have the right connection available to connect the endotracheal tube to the ventilating bag.
6. DO NOT VENTILATE A SMALL CHILD WITH TOO MUCH PRESSURE, or you will cause a pneumothorax. Watch the chest, and use the smallest amount of pressure that gives adequate movement.
7. Do not overventilate. Give about 20 breaths a minute (1 every 3 secs). Allow enough time for full expiration.



EOSINOPHILIA

ASYMPTOMATIC WITH AN EOSINOPHIL COUNT LESS THAN 1,000/CMM

Mild eosinophilia (up to 1000/cmm) is very common in children in developing countries, and there is a strong correlation between helminthic infestation and an eosinophilia of 5% or more of the total white cell count (Vines AP. An Epidemiological Sample Survey of TPNG. PHD, Port Moresby, 1970). These children can be treated with albendazole.

Upper limits of normal eosinophil count	
0-3 months	1000 eosinophils per cmm
3 m-2 yrs	850 eosinophils per cmm
2-14 years	600 eosinophils per cmm

SYMPTOMATIC, OR EOSINOPHIL COUNT OVER 1,000/CMM

1. Examine the stools for cysts, ova and parasites
2. Take a chest x-ray
3. In endemic areas, take three midnight blood films for microfilariae
4. Stop any drugs that may be causing eosinophilia.

DRUGS

Penicillin, streptomycin, chlorpromazine, PAS, nitrofurantoin, sulphonamides and other drugs may cause eosinophilia.

PULMONARY EOSINOPHILIA

This is eosinophilia with patchy opacities on chest x-ray. There are numerous causes of this syndrome:

- helminth migration through the lungs (eg ascaris or hookworm)
- drugs (eg streptomycin, PAS, nitrofurantoin or sulphonamides)
- tropical eosinophilia: an acute or chronic illness with mild fever, dry cough, malaise and dyspnoea. It is usually caused by filarial infection, but may be caused by toxocara
- polyarteritis nodosa
- allergic alveolitis.

PARASITIC INFESTATIONS

Penetration of tissues by helminth larvae may produce marked eosinophilia. There may be hepatomegaly. Causative parasites include *Ascaris lumbricoides*, *Toxocara canis* (visceral larva migrans), hookworm and *strongyloides*.

OTHER CAUSES OF EOSINOPHILIA

Other causes of eosinophilia are:

- allergy, which may be associated with asthma, hay fever, serum sickness, urticaria, food sensitivity, angioneurotic oedema, eczema, exfoliative dermatitis or scabies
- malignancy, leukaemia or lymphoma
- post-infectious rebound eosinophilia
- familial eosinophilia.

TREATMENT

If there are symptoms, or if the eosinophil count is over 1,000/cmm:

1. Review drug therapy. Withdraw possible causes.
2. Treat scabies.
3. If there are no pulmonary infiltrates, give albendazole. If this fails, diethylcarbamazine can be tried.
4. If there are pulmonary infiltrates, give albendazole and diethylcarbamazine. If severe symptoms persist, steroids may have to be given (exclude TB, and cover with isoniazid).
5. If strongyloides infection is likely, give thiabendazole (if available) 25 mg BD for 3 days.

EQUIPMENT

ORDERING

Standard equipment is usually ordered through Base Medical Stores or directly through the Hospital Boards. Equipment available through the Health Department is listed in the Medical Stores Catalogue that is revised regularly. Not infrequently, however, donations are received from service clubs, other organisations or individuals and this allows the purchase of replacement or “extra” equipment. It is important that these donations are used wisely.

It is vitally important when ordering equipment to remember that it must be functional and robust, and require little, if any, maintenance. Parts for repair need to be readily available, as should the personnel to repair it. As far as possible, equipment should be standardised to make replacement and repair as easy as possible (for example, it makes no sense to have six different brands of ultrasound machine in the country). It is best to deal with companies that already have sale arrangements with PNG. Buying direct from the manufacturer is likely to be much cheaper than buying through an agent. A good reliable and cheap source of equipment is ECHO-International Health Services Ltd., Ullswater Crescent, Couldson, Surrey CR5 2HR, UK (phone 0544 181 660 2220, fax 0544 181 668 0751). This organisation produces an excellent catalogue.

APNOEA ALARM

The Graseby Apnoea alarm MR10 is a reliable and simple battery-operated apnoea alarm. The “disposable” pneumatic sensors can, with care, be reused many times, but do not last indefinitely.

AURISCOPES

These are always in short supply, and theft is a problem. Many staff are happy to buy an auriscope for their own use - this has the advantage that it is then well cared for. If ten or more auriscopes are bought at any one time, for resale to staff, a discount is usually obtainable. A good auriscope for this purpose is the Minilux DOO.70.212 that accepts standard penlight batteries and costs about US\$60. They are obtainable from Surgical Manufacturing Co, 170-174 Abbotsford Street, North Melbourne, Victoria 3051, Australia. It is wise to buy some spare bulbs.

BILIRUBINOMETER

Most of the larger hospital laboratories should be able to provide bilirubin levels. American Optical market a bilirubinometer that measures total serum bilirubin on a drop of serum (collected in capillary tubes and centrifuged). It is cheapest to purchase it direct from American Optical Corporation, Buffalo, NY 14215, USA.

BLOOD WARMER

The Tuta Temperature Controlled Water Bath from Tuta Laboratories P/L, PO Box 166, Lane Cove, NSW 2066, Australia is particularly useful during exchange transfusions.

DEXTROSTIX

Dextrostix should not be regarded as “extra” equipment at all. It should be readily available from the Hospital Dispensary via Medical Stores. However, if there are shortages, they can be obtained from Miles Laboratories, PO BOX 203, Springvale, Victoria 3171 Australia, or ordered through a local pharmacy.

ELECTROCARDIOGRAM

ECG machines are now relatively inexpensive - and should be available through the Health Department. If considering purchasing a new or replacement machine, make sure it is compatible with the other machines in the hospital - in terms of ECG paper - and make sure you order paediatric leads (not essential - but they make life much easier!).

EMERGENCY TROLLEY

You should ensure that every ward has a well-equipped emergency trolley that contains oral airways, artery forceps, uncuffed endotracheal tubes, a wire introducer for tubes, a laryngoscope (with standard and infant blades), a metal sucker, sterile 8g and 12g nasogastric tubes (that can be used for endotracheal suction), adhesive plaster, lubricant jelly, scissors, cotton swabs, alcoholic skin cleaner, cutdown catheters, scalp vein needles, Dwellcaths, giving sets (for fluid and blood), disposable needles, sterile syringes, scalpel handles and blades, atraumatic suture needles and thread, a needle holder and ampoule files.

You can use two systems to ventilate an intubated child:

1. A Penlon Cardiff Inflating Bag (CIG No. TM 86) or a Laerdahl Neonatal Inflating Bag for babies, or an Airviva (CIG No. TM 25) or Laerdahl Paediatric Inflating Bag for older children, can be connected to the endotracheal tube with a Portex 15 mm endotracheal adaptor (Set No. 100/253/000 from Portex, Box 120, Carlingford, NSW 2118, Australia). This method is simple and safe, but does not permit the use of high inflating pressures or high oxygen concentrations.
2. The alternative is to use an Ohmeda-CIG paediatric anaesthetic circuit (DF 564, DF 558, OBM 373884, DF 559 and M 163) connected to the endotracheal tube with an endotracheal taper tip. This method allows precise ventilation with high pressures and 100% oxygen in children up to 20 kg weight, but it is dangerous in inexperienced hands. Make sure you cut a hole in the nipple on the rubber bag.

The trolley should contain ampoules of adrenaline, aminophylline, atropine, calcium, chlorpromazine, dexamethasone, 50% dextrose, diazepam, digoxin, dopamine, frusemide, hydrocortisone, isoprenaline, lignocaine 1% (10 mg/ml), paraldehyde, phenobarbitone, phenytoin, potassium chloride, sodium chloride 0.9%, sodium bicarbonate 8.4% and water for injection. There should be bottles of half strength Darrow's solution, 4.3% dextrose in 0.18% sodium chloride, 5% dextrose, 10% dextrose, Hartmann's solution, 20% mannitol and 0.9% sodium chloride.

EXCHANGE TRANSFUSION TRAY

Make sure you always have two or three on hand (PNG Catalogue No.5035).

HAND DRYERS

World Electric Hand Dryers are available from J D MacDonald Engineering Co P/L, PO Box 107, Glen Iris, Victoria 3146, Australia. They avoid the problem caused by frequent shortages of paper towels. The manual model is more reliable (you have to press a button to turn it on).

HEATERS FOR NEONATES IN COLD AREAS

The nursery for sick neonates should be kept at 27-30 °C. This can be done in two ways:

1. The first method is to heat the room with Speedie RH7 750w, 1100w or 1500w electric radiators mounted on the wall out of reach (Speedie Electrical Industries, 330 Reserve Road, Cheltenham, Victoria 3192, Australia). The operation of the radiators should be controlled by a Honeywell Sensor portable 10 amp room thermostat (or equivalent) from James McEwan and Co, 391 Bourke Street, Melbourne, Victoria 3000, Australia.

2. Alternatively, oil-filled electric heaters with thermostats can be used (eg The Dimplex E412 1000w panel heater from Don Stewart and Co., 513 Logan Road, Greenslopes, Queensland 4120, Australia. It can be wall mounted if necessary).

A Speedie 750w heater suspended from the ceiling on chains (it should NOT be mounted directly onto the ceiling) can be used to warm cold babies quickly, and to keep babies warm while procedures are being done.

Preterm babies can be nursed on a heated, water filled mattress manufactured by Kanthal Medical Heating, Aggelundavagen 2, S-175 62 Jarfalla, Sweden (Fax 468-7617580) - see Arch Dis Child 64:687-92,1989. An alternative method is to use a small electric blanket: the Riviera 37 x 27 cm blanket with ELV transformer is suitable. The baby should be well wrapped up with a gamgee cap on, lying on a well-covered electric blanket in a warmed nursery. This method is much cheaper and easier than using incubators and carries less risk of infection.

INTRAVENOUS EQUIPMENT

Needles, cannulae, bungs and long lines

Scalp vein needles should be used for most drips. However, when the drip is likely to be required for more than 24 hours, it is best to use an intravenous cannula. Size 24 is suitable for neonatal use, size 22 for infants and size 20 or 21 for older children. "Caps", "Stoppers" or "Bungs" are available when it is desired to give intravenous medication without having a continuous drip - but care must be taken in their use - sterile procedures should be used and they should be flushed through with sterile normal saline after each drug administration. There are occasions when it may be desirable to insert a "long line". 22 gauge and 19 gauge 8 inch Intracaths are suitable (one source is Deseret Pharmaceuticals Inc, 9450 South State Street, Sandy, Utah 84070, USA).

BURETTES

Standard burettes produce 15 drops per ml. Paediatric burettes produce 60 drops per ml. It is therefore vital that medical and nursing staff working in the Children's Services (including surgical services for children) are familiar with the different burettes and the calculations required for fluid administration:

Standard burette:	$\text{drops/min} \times 4 = \text{ml/hr}$
Paediatric burette:	$\text{drops/min} = \text{ml/hr}$

Infusion pumps

There are two types of infusion pump: the peristaltic pumps (eg IVAC) and the syringe pumps (eg Graseby). If considering ordering a peristaltic pump, it is important to order one which uses standard intravenous drip tubing, and not one which requires specially designed (expensive and hard to obtain) "dedicated" tubing. It is also important to be aware that some are calibrated to "drops per minute" and others to "mls per hour" - and confusion can easily arise. Syringe pumps are very reliable, but ideally require special tubing from the syringe to the cannula (though it is possible to improvise) and staff need to be familiar with the techniques of filling the syringes and dialling the settings.

NEBULISERS

Asthma can be treated with salbutamol from a nebuliser run on a gas flow of about 7 litre/min (Lancet 1:329,1981) or using an airpump (electrical or manual). Salbutamol given by inhalation is much more effective than by the oral route. Inspiron Mini-Neb nebulisers (No. OO2220) and paediatric oxygen masks (No. OO1476) with oxygen tubing (No. OO1503) can be obtained from Ramsay Surgical Limited, 182-206 Berkley Street, Carlton, Victoria 3053, Australia. The gas flow can be from:

- bottled oxygen (which is expensive)
- a Maymed electric air pump from Anaesthetic Supplies, 350 Victoria Street, West Melbourne, Victoria 3003

- a foot pump made for blowing up car tyres. These do not require electricity and can therefore be used in villages. They can be obtained in most garages or hardware stores. Keeler make a plastic hand or foot-operated air pump specifically for nebulising.

Note: **METERED DOSE INHALERS WITH SPACERS ARE A VERY EFFECTIVE ALTERNATIVE TO NEBULISERS** (see Asthma, p.41).

OXYGEN AND SUCTION TUBING

Clear plastic tubing can be purchased from hardware stores in most major towns. You can see through it, and it is less likely to kink or perish than rubber tubing. Use 1/2 inch tubing for oxygen lines and 3/8 inch tubing for suction. Pharma Plast polystyrene adaptors No. 646 and 648 are very useful connectors for oxygen and suction tubing; they are available from Mayven Medical P/L, 494 Rathdowne Street, North Carlton, Vic 3054, Australia.

PHOTOTHERAPY UNITS

Commercially available phototherapy units are convenient but expensive. Simple and effective units can be made by having 4 fluorescent strip lights fastened to a board that can be placed 48 cm above the baby. The tubes need to be replaced every three months because the emission of light of the appropriate wave length declines over time (see Neonates - Phototherapy, p.269).

PLASTIC SYRINGES AND 3-WAY STOPCOCKS

Disposable plastic syringes are now standard. Three-way taps are very useful for procedures such as thoracocentesis and are required for syringe pumps (two 2-way taps can be used but this is a very inconvenient arrangement open to error).

PULSE OXIMETERS

Pulse oximetry is a non-invasive and a convenient way of assessing arterial oxygen saturation either at a particular time or continuously. Both portable (eg Novamatrix) and “fixed” (eg Ohmeda and Nelcor) are suitable. Although easy to use, pulse oximeters are not foolproof and doctors using them should be familiar with their problems and limitations (Moyle 1996).

RECTAL BIOPSY

Noblett rectal biopsy forceps (with spare capsule, blades, gauge and block) can be bought from HA Taylor Surgical, 44 MacFarlan Street, South Yarra, Victoria 3141, Australia. They enable suction biopsy of the rectal mucosa without anaesthesia in cases of suspected Hirschsprung's disease (Campbell and Noblett 1969).

SPECIAL MILK FEEDS

It is very useful to have small stocks of Triglyde and Pregestimil or Ensure. Triglyde, a milk preparation with high levels of medium chain triglycerides, is useful in premature babies with fat malabsorption who fail to thrive on breast milk and in children with chylous ascites (usually due to abdominal tuberculosis). Pregestimil and Ensure are useful in children with severe malnutrition or chronic diarrhoea who have malabsorption of lactose and fat. Ensure is a high calorie (100 cal/100 ml) lactose-free milk substitute. Infasoy and Digestelact, Nutramigen and Portagen are also low lactose “milks”.

STETHOSCOPES

Nurses' lightweight stethoscopes (diaphragm only) can be obtained from JD Marshall Inc., PO Box 59446, Chicago, Illinois 60659, USA. An alternative source of stethoscopes is Medical Supplies N.Z Ltd., Corner Parumoana and Norrie Streets, Porirua, New Zealand - there is a discount for bulk orders of 10 or more.

SUCKERS

Regularly check that the suckers in your ward are working. Suction pressures should not exceed 200 mmHg (26 kPa). Blocked filters, leaking air seals and perished or kinked tubing are common problems that are easy to fix.

OTHER EQUIPMENT

Check regularly to see that all oxygen fittings are screwed up tightly - loose fittings mean that oxygen leaks into the air instead of going to the patient. Check that the ward fridge is defrosted regularly and that drugs are properly stored in it: see "How to Look After a Refrigerator" by J Elford from Healthlink Worldwide. Cityside, 40 Adler Street, London E1 1EE, UK. Website: www.healthlink.org.uk. Change the fluorescent tubes in your phototherapy unit every three months.

LOCAL SUPPLIERS

There are now several medical equipment suppliers in Papua New Guinea. It may be easier to order special equipment through these suppliers, but it is likely to be expensive.

REFERENCES

- Campbell PE, Noblett HR. J Pediatr Surg 4(4):410-415,1969. Experience with rectal suction biopsy in the diagnosis of Hirschsprung's disease.
- Dyke T, Lewis D, Heergaard W et al. J Trop Paediatr 41:196-201,1995. Predicting hypoxia in children with acute lower respiratory tract infection: a study in the Highlands of Papua New Guinea.
- *Elford J. How to look after a refrigerator. London: AHRTAG, 1980, Reprinted 1997.
- Moyle JBT. Arch Dis Child 74:77-80,1996. Uses and abuses of pulse oximetry.
- *Skeet M, Fear D. Care and safe use of hospital equipment. London: VSO Books, 1995.
- Smyth A, Carty H, Hart CA. Ann Trop Paediatr 18:31- 40,1998. Clinical predictors of hypoxaemia in children with pneumonia.
- Thilo EH, Park-Moore B, Berman ER, Carson BM. Am J Dis Child 145:1137-1140,1991. Oxygen saturation by pulse oximetry in healthy infants at an altitude of 1610 m (5280ft).
- Usen S, Weber M, Mulholland K et al. Br Med J 318:86-91,1999. Clinical predictors of hypoxaemia in Gambian children with acute lower respiratory tract infection: prospective cohort study.

*These books are available from AusAID-funded Hospital Management and Operations Improvement Project, Papua New Guinea.

EYES

SOME IMPORTANT RULES

- BEWARE THE UNILATERAL RED EYE: do NOT assume that it is due to conjunctivitis. Always exclude foreign body, corneal ulcer, iritis and glaucoma
- NEVER PAD A DISCHARGING EYE
- ALWAYS PAD AN EYE AFTER INSTILLING LOCAL ANAESTHETIC
- NEVER USE LOCAL STEROIDS UNLESS CORNEAL ULCERATION HAS BEEN EXCLUDED WITH FLUORESCEIN STAINING
- EVEN IF THE EYE LOOKS NORMAL, ALWAYS X-RAY THE ORBIT (AP AND LATERAL) IF THE HISTORY SUGGESTS INTRAOCULAR FOREIGN BODY
- A PENETRATING INJURY OF THE EYE IS AN OPHTHALMOLOGICAL EMERGENCY.

THE ACUTE RED EYE

	Conjunctivitis	Iritis	Glaucoma
Incidence	Very common	Common	Rare
Pain	Mild	Moderate	Severe
Discharge	Profuse	Moderate	Minimal
Visual acuity	Normal	Mild decrease	Marked decrease
Redness	Palpebral + bulbar	Around cornea	Around cornea
Pupil	Normal	Small \pm irregular	Semi-dilated, non-reactive
Pressure	Normal	Usually normal	Hard
Cornea	Clear	Hazy \pm ulcer	Cloudy
Treatment	Oxytetracycline or antibiotic eye ointment	Atropine 1% eye ointment + chloramphenicol eye ointment +, if no ulcer , 1% hydrocortisone eye ointment	Pilocarpine + acetazolamide + operation

ACID/ALKALI BURNS

Acids burns are not usually as severe as alkali burns. Thermal and acid burns usually show the full extent of the injury at presentation, but alkali burns may continue to progress for up to a week. Admit all alkali burns:

- irrigate the eye with sterile water or saline for 15 minutes and remove any particulate matter
- put in fluorescein stain to exclude corneal ulceration
- put in chloramphenicol eye ointment 6 hourly
- pad the eye
- advise complete rest for 24 hours
- follow up daily until the eye has healed.

If the burn is severe, also:

- apply 1% atropine 6 hourly
- apply 1% hydrocortisone 6 hourly (if no corneal ulcer) - and admit the child.

CONGENITAL CATARACT

Refer this to an ophthalmologist on his/her next visit to the region.

CONJUNCTIVITIS

Purulent

This is a very common condition with copious discharge, and redness of the conjunctiva on the inside of the lids as well as on the bulbar surface. Apply oxytetracycline or compound antibiotic eye ointment every 3 hours for 2 days, then every 6 hours for 3 days. Do NOT use chloramphenicol eye ointment routinely for conjunctivitis, as it is not effective for trachoma - which is common. Oxytetracycline is active against trachoma.

Persistent

Suspect trachoma (particularly if the conjunctivitis has failed to respond to chloramphenicol eye ointment). Trachoma causes photophobia, excess lacrimation, minor irritation and some exudate. Look for pale follicles 1-5 mm in diameter on the inside of the upper eyelid. Trachoma may go on to cause pannus (a vascularised scar of the upper cornea), entropion and blindness. Apply oxytetracycline eye ointment twice a day for one month. Refer to the ophthalmologist if no improvement.

Neonatal

(See p.252).

Severe conjunctivitis in the first 2 weeks of life may be gonococcal.

1. Wash out the pus from the eye.
2. Apply oxytetracycline, compound antibiotic, or chloramphenicol eye ointment every 6 hours for 5 days.
3. Give benzyl (crystalline) penicillin 125,000 units IM TID for 5 days. If no rapid improvement, change to ceftriaxone/cefotaxime if available, or add gentamicin if not.
4. Treat the mother and father and their sexual contacts for gonorrhoea.

CORNEAL ULCER (KERATITIS)

This is usually the result of an untreated foreign body or corneal injury. It may sometimes be due to a viral infection. The eye is red, watery and painful, with minimal discharge. There is fluorescein staining of the ulcer. There may be pus in the anterior chamber. Treat with oral chloramphenicol, and apply antibiotic eye ointment 3-4 times daily. Pad the eye, and review daily. If the ulcer is due to the cold sore virus, Herpes simplex, it has a characteristic “dendritic” branched appearance. This needs to be treated with idoxuridine (Stoxil) ointment or vidarbine (Zovirax) ointment.

DO NOT USE STEROIDS IN CORNEAL ULCERS OR KERATITIS.

EYELID LACERATION

The canaliculus may be involved in injuries to the inner third of the eyelids.

FLASH BURN

This is caused by exposure to ultraviolet light. Pain starts about 6 hours after exposure. Apply chloramphenicol eye ointment 6 hourly, pad the eye, and give paracetamol or aspirin for pain.

FOREIGN BODY

Conjunctival

Always evert the upper lid, as foreign bodies are often found on its posterior surface. Remove the foreign body with a moist cotton wool swab or swab stick.

Corneal

Apply local anaesthetic drops (0.5% amethocaine) and remove the foreign body with a moist cotton wool swab. Pad the eye for 6 hours. A small iris prolapse following perforating injury to the cornea may mimic a corneal foreign body.

Intra-ocular

Often caused by high-velocity fragments. There may be only a small sub-conjunctival haemorrhage. Even if the eye looks normal, always X-ray the orbit (AP and lateral) if the history suggests an intraocular foreign body. A penetrating injury of the eye is an OPHTHALMOLOGICAL EMERGENCY.

GLAUCOMA

Primary glaucoma is rare in children, as the globe is still growing. Any child with enlarged globes or with blue, hazy corneas needs to be referred to an ophthalmologist as soon as possible.

Secondary glaucoma is much more common, due to trauma or inflammation of the eye, or associated with systemic diseases such as arthritis, tuberculosis or leprosy.

A red, painful eye after trauma may be due to haemorrhage into the anterior chamber (traumatic hyphaema). Compare both eyes to check the clarity and depth of the anterior chamber. If blood is present, pad the eye and keep the child on bed rest for 24 hours, sedated if necessary. Even if the blood clears, there may be secondary bleeding. Give acetazolamide (Diamox) if the eye remains very painful.

When a painful red eye occurs in association with an underlying systemic disease, treat the disease appropriately. Check the pupils and exclude corneal ulcer. Treat the eye as for iritis. You may need to use acetazolamide (Diamox). Always ask for help (by phone if necessary) if you are not sure.

IRITIS

This is a common condition that causes quite severe pain, but only a moderate amount of exudate. There is reddening of the sclera around the cornea, and the pupil is small and irregular. There may be pus in the anterior chamber (hypopyon).

To treat iritis:

- apply 1% atropine eye drops 6 hourly until the pupil is dilated, then daily
- apply chloramphenicol eye ointment 6 hourly
- stain the cornea with fluorescein. If there is no ulcer, apply 1% hydrocortisone eye ointment 6 hourly.

STRABISMUS

Refer to an ophthalmologist as soon as possible, to exclude serious underlying conditions such as tumour, and to commence treatment for preservation of binocular vision.

TEAR DUCT OBSTRUCTION

Blockage of the tear duct with debris is common in neonates and produces the characteristic “sticky eye” and tearing. All that is required is to wash the eye out with water. Antibiotics are only indicated if the conjunctiva become inflamed (conjunctivitis).

More severe congenital obstruction is also relatively common in neonates. Treat infection with oxytetracycline eye ointment. Show the mother how to apply gentle upward pressure massaging over the nasolacrimal sac. Refer for probing if the eye still waters at 4 months of age.

TRACHOMA

Trachoma is an infection with *chlamydia trachomatis*. It may be non-blinding, but blinding trachoma occurs in hyperendemic areas with crowding and poor living standards. Children are the main reservoir of infection. Trachoma is a chronic follicular conjunctivitis causing corneal pannus, keratitis and scars. Blindness results from scarring of the eyelids which become turned inwards (entropion), so that the eyelashes abrade the cornea. Diagnosis is made from the clinical findings, with confirmation from serological tests (an immuno-fluorescent test for chlamydia antigen is now available).

Treatment is with topical tetracycline or erythromycin eye ointment TID for at least 5 weeks, or systemic erythromycin (15 mg/kg) or sulphamethoxazole (30 mg/kg; cotrimoxazole contains 80 mg of trimethoprim and 200 mg of sulphamethoxazole) daily for 3 weeks (tetracycline or doxycycline is used in adults). In blinding trachoma, one dose of doxycycline (5 mg/kg if over 20 kg) or sulphamethoxazole (60 mg/kg) should be given each month for at least 6 months after the initial intensive course to prevent reinfection.

XEROPHTHALMIA

Vitamin A is contained in green leafy vegetables and fruits. Deficiency causes night blindness, conjunctival damage with xerosis (dry wrinkles on the temporal quadrant), Bitot’s spots (foamy flecks) and pigmentation; and corneal damage with punctate keratopathy, corneal dullness and keratomalacia with perforation of the cornea. Vitamin A deficiency increases mortality from infectious diseases, particularly measles.

Treatment

For children over 1 year old, give Vitamin A 100,000 units if <10 kg, 200,000 units if >10 kg. A second dose is given the following day. A third dose is given after one week.

Prevention

In high risk areas, half the above doses can be given on a community-wide basis every 4-6 months. Papua New Guinea is currently in the process of introducing a Vitamin A supplementation programme. All children are scheduled to receive two doses, one at 6 months and the second at one year.

REFERENCES

- Galbraith JEK. Basic eye surgery: a manual for surgeons in developing countries, 1979.
Khaw P. ABC of eyes. BMJ Publishing Group, 3rd ed, 1999.
Sandford-Smith. Eye diseases in hot climates. Butterworth, 3rd ed, 1997.

FAMILY PLANNING

Family planning can make an enormous contribution to child health. Dr Maurice King has stressed the importance of family planning in countries where the infant mortality rate is falling: if fertility rates do not fall as mortality rates fall, the population may increase so much that the ecology of the area is destroyed and people die from lack of food and fuel (Demographic entrapment. *Trans R Soc Trop Med Hyg* 87 suppl 10:S23-28,1993). This phenomenon is already being seen in parts of Papua New Guinea.

POPULATION GROWTH IN PAPUA NEW GUINEA

The population of Papua New Guinea is currently growing at a rate of about 3.2% per year. This means the population will double in 25 years. A very large part of government expenditure will be spent on attempting to maintain existing levels of services for the increased population. Improving the level of services will be extremely difficult. Reducing population growth is vitally important to the future economic wellbeing of Papua New Guinea and indeed of the world (King M and Elliot C. To the point of farce: a Martian view of the Hardinian taboo - the silence that surrounds population control. *Br Med J* 315:1441-1443,1997). Many of the women in Papua New Guinea wish to have families smaller than those of previous generations (see below), and safe and effective means to allow them to space their children and control their family size should be readily available.

FAMILY SIZE

Large families have a higher incidence of:

- illness: families of eight have twice as much diarrhoea as families of three (Dingle JH. *Illness in the home*, Cleveland, Press of Western Reserve University, 1964)
- malnutrition: large families have been found to have more malnutrition in Africa (*Trans R Soc Trop Med Hyg* 62:2,164,1968), Thailand (Wray JD in *Rapid population growth*, Vol.2, Baltimore, Johns Hopkins Press, 1971) and India (*J Nutr Diet* 6:258;1969)
- growth retardation: in working class British families, the third or later child was 4 inches shorter at 5 years of age than his siblings (*Milbank Meml Fund Q Bull* 42:20, 1964)
- intellectual retardation: a number of American and British studies show that a single child has an average IQ of 105, while the sixth child has an average IQ of 90 (eg *Br J Prev Soc Med* 2:42-59, 1958)
- death: a study in India found an infant mortality of 117/1000 for the second child, and 206/1000 for the seventh to twelfth child (Wyon J B. *The Khanna Study*, Cambridge, Harvard Uni Press, 1971). Mortality in Britain also rises after the second or third child (*Lancet* 1:343, 1955).

BIRTH INTERVAL

There is good evidence that the time between successive babies in a family (the birth interval) is even more important than the total number of children (family size) (Kane P, 1985. *The impact of birth spacing on child health*. Evidence from the World Fertility Survey. University College Cardiff Press. Rutstein S. *Infant and child mortality: trends and demographic differentials*. World Fertility Survey Technical Bulletin 2001. Fredrick J, Adelstein P. *Influence of pregnancy spacing on outcome of pregnancy*. *Br Med J* Dec 29:753-756,1983).

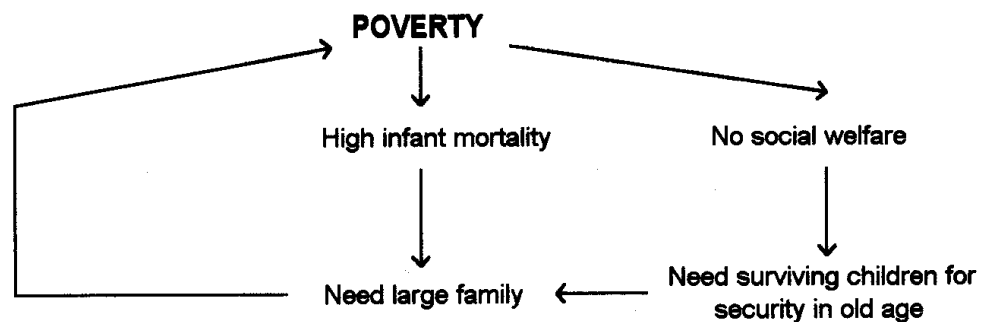
A short birth interval has been shown to be associated with high infant mortality in both America (*Am J Obs Gyn* 71:80, 1956) and India (Wray JD in *Rapid population growth*, Vol.2, Baltimore, Johns Hopkins Press, 1971). A short birth interval is also associated with increased infection in the family (Aaby P et al. *Lancet* 2:161,1983), malnutrition (*J Trop Ped* 15:76,1969) and lower intelligence (*Milbank Meml Fund Q Bull* 42:20,1964).

The baby in a family receives most attention from the mother who provides intellectual and emotional stimulation as well as physical care. If a family has a short birth interval, each child will receive less of this intensive mothering. Loss of this intellectual and emotional stimulation may be just as important to the child's wellbeing as the physical effects of a short birth interval.

BARRIERS TO ACCEPTANCE OF FAMILY PLANNING

People sometimes do not readily accept family planning when it is offered to them. Apart from traditional taboos and religious beliefs, there are economic and social reasons for this.

The governments of developing countries cannot afford social security pensions. Therefore, people need to have surviving children to look after them in their old age. Because of the high infant mortality, families have to be large to ensure that some children survive. Therefore, people are often reluctant to accept family planning. Countries that have had successful family planning programmes, such as Singapore and Japan, first reduced their infant mortality rate and provided a degree of social security.



FAMILY PLANNING IN PAPUA NEW GUINEA

The recent National Demographic Health Survey (DHSS 1996) indicated that whilst 72% of women of reproductive age group had some knowledge of family planning (traditional and modern), only 29% had ever used and 26% were currently using any contraceptive method. It is likely that the majority of these women were using traditional rather than modern methods (UNICEF figures for use of contraception are considerably lower at 4-7% - State of the World's Children).

The total fertility rate for the five years preceding the survey was 4.8 whilst the desired family size was 3.5.

In the detailed study in four provinces (Strengthening Reproductive Health Services UNFPA/WHO 1997), half of the women of reproductive age wanted no more children and yet were not using any form of contraception. More than 60% of the women in East Sepik reported birth intervals of less than 2 years.

DOCTORS AND FAMILY PLANNING

All doctors have a responsibility to discuss family planning with their patients and to offer advice on services available. Paediatricians as well as obstetricians should regard family planning as a key aspect of their work.

METHODS OF CONTRACEPTION

Whilst pills and condoms are readily available to educated urban users, they may not be available or appropriate for many of the rural people. The loop is available but associated with a relatively high incidence of side effects. Tubal ligation is a means of family completion rather than family spacing. Vasectomy is not widely practiced (though a vasectomy programme in Western province was

surprisingly well accepted). Depo-Provera (Medroxyprogesterone) has been widely used in Papua New Guinea as well as in other parts of the “third world”, in spite of criticism from well meaning but short sighted and uninformed Western pressure groups. It has a very good safety record (far safer than pregnancy!), is highly effective and generally well tolerated. It is generally reserved for parous women.

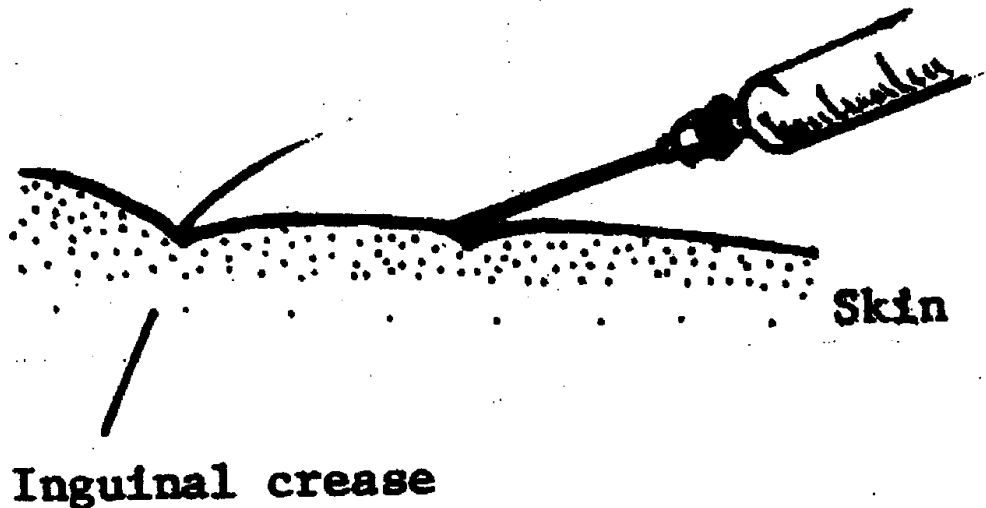
ADDITIONAL REFERENCE

Morley D and Lovel H. My name is Today: an illustrated discussion of child health, society and poverty in less developed countries. Institute of Child Health London. Macmillan. TALC 1986.

FEMORAL VEIN PUNCTURE

This should only be performed in children over 10 kg. Do not do a femoral puncture on a neonate. If you have difficulty obtaining blood in a small child, do a radial arterial puncture (p.34).

1. Have an assistant hold the child supine with the right hip extended and externally rotated.
2. Clean the skin with 70% alcohol (SVM).
3. Palpate the femoral artery just below the midpoint of a line joining the anterior superior iliac spine and the pubic tubercle. The vein lies just medial to the artery.
4. While still palpating the artery with your left index finger, push a 21 gauge needle on a 5 ml or 10 ml syringe through the skin at a very shallow angle at a point 2 cm below the inguinal crease. The vein is very close to the skin, unless the child is very fat. Aspirate gently and withdraw the needle slowly until blood enters the syringe.



5. After obtaining the blood and withdrawing the needle, have an assistant press firmly on the puncture site with a dry swab for 2 minutes.

Do NOT do a femoral puncture in a patient with nephrotic syndrome.

Try not to puncture the artery. If you do aspirate bright blood under pressure, aspirate the blood you need quickly, then withdraw the needle and apply firm pressure with a dry swab for 3 minutes. Keep a careful watch for subsequent haematoma formation (you may have to apply pressure for longer).

Do NOT touch the puncture site or needle with your fingers.

Do NOT put the needle in at too big an angle or go too deep. You can easily penetrate the hip joint and cause septic arthritis.

Do NOT push the needle in too far, or you may go above the inguinal ligament and into the peritoneal cavity.

FEVER

1. Find the cause of the fever, and treat the cause.
2. If you are in a malarious area, give antimalarials to EVERY child with a fever, even if there is another obvious cause.
3. Give paracetamol every 6 to 12 hours if the temperature is over 40 °C. Note that fever is a normal response to infection, and there is evidence that it reduces the morbidity and mortality of bacterial and viral infections: it is wrong to think that fever should always be abolished (see Pediatrics 66:720-4,1980; Am J Dis Child 134:176-81,1980; Pediatrics 74(Suppl):929-36,1984 and Am J Med (Aspirin Symposium) June 14, 1983:27-35). Paracetamol alone in adequate doses (10-15 mg/kg 6 hourly) is as effective as paracetamol and tepid sponging (Arch Dis Child 48:313-315,1973 and J Pediatr 77:824-9,1970).
4. Give extra fluids.
5. Tepid sponging may not be very effective - but it allows the mother to be involved in the care of her child.

REFERENCE

Agbolosu NB, Cuevas LE, Milligan P et al. Ann Trop Paeds 17(3):283-288, 1997. Efficacy of cool sponging versus paracetamol in reducing temperature in febrile children.

FEVER OF UNKNOWN ORIGIN

Exclude otitis media, pneumonia, early measles (there may be conjunctivitis, rhinorrhoea and cough), abscess, tonsillitis, diarrhoea, acute nephritis, (there may be oedema and haematuria), joint pains (see Arthritis, p.35-38) and osteomyelitis, pyomyositis or cellulitis (there may be a tender limb).

If the child is not very sick and has had fever for less than one week:

- do a blood slide, FBE, and a CXR and observe
- give antimalarials whilst waiting for the results of investigations.

If the child is very ill, or has had fever for more than one week:

- take specimens for a blood slide (URGENT), blood culture, FBE, Widal, LP, urine M & C, faeces culture for salmonella typhi (even if there is no diarrhoea) and shigella
- start IV chloramphenicol
- start treatment for severe malaria, but review this treatment when the result of the blood slide is available
- arrange for a chest x-ray and Mantoux. Take three gastric aspirates if there has been fever or cough for more than 2 weeks, a family history of TB, weight loss or haemoptysis. Biopsy any enlarged lymph node.

MALARIA

Check that antimalarials were given in the correct dose and were not vomited, and that the child did not have severe diarrhoea (give parenteral treatment for severe malaria in patients with vomiting or severe diarrhoea).

If *P.falciparum* is present despite oral antimalarials having been given properly, give treatment for Treatment Failure Malaria (p.199).

TB

Start full TB treatment if a 5u Mantoux is positive (if the child has had a BCG, a Mantoux over 15 mm is positive; if the child has not had a BCG, a Mantoux over 5 mm is positive).

If the child is still sick after 5-7 days of chloramphenicol, take three gastric aspirates (even if there is no other evidence of TB).

In an ill child with persistent fever despite adequate antibiotic therapy, the difficult decision may have to be taken to start TB treatment without definite evidence of TB. The TB Score Chart is likely to be helpful in this situation (see p.369-370).

TYPHOID

Typhoid may cause fever, headache, dry cough, myalgia, anorexia, abdominal pain, splenomegaly or a fleeting “rose spot” rash on the trunk. Although they may be constipated, children with typhoid often have diarrhoea (pea soup stools) that may contain blood (see Typhoid, p.373).

Give chloramphenicol for 3 weeks, and screen household contacts (culture their faeces and blood). Most patients take several days to respond to chloramphenicol.

URINARY TRACT INFECTION

This is very much under-diagnosed - but can have disastrous consequences, both short and long term. Collection of an uncontaminated sample of urine and its microscopy and culture should be a routine part of the investigation of children with unexplained fever. In most children it is possible to collect a clean catch urine (see p.379).

“HIDDEN” COLLECTION OF PUS OR MALIGNANT DISEASE

Don't forget the possibility of a “hidden” collection of pus or malignant disease.

FLUID AND ELECTROLYTE THERAPY

See also IV and Oral Fluids (p.397).

MAINTENANCE REQUIREMENTS

IV fluid: 4.3% dextrose in 0.18% saline.

Add to each litre: 1g (13 mEq) of potassium chloride (and 1 ampoule - 2 ml - of Intravite if IV maintenance and nil by mouth is to be continued for more than 3 days).

Amount:

0-10 kg: 100 ml/kg/day
 4 ml/kg/hr

11-20 kg: 1000 ml + 50 ml/kg/day for each kg above 10 kg
 40 ml + 2 ml/kg/hr

Above 20 kg: 1500 ml + 20 ml/kg/day for each kg above 20 kg
 60 ml + 1 ml/kg/hr

Examples:

7 kg child requires: 7×100 = 700 mls/day
 7×4 = 28 mls/hr

12 kg child requires: $1000 + (2 \times 50)$ = 1100 mls/day
 $40 + (2 \times 2)$ = 44 mls/hr

25 kg child requires: $1500 + (5 \times 20)$ = 1600 mls/day
 $60 + (5 \times 1)$ = 65 mls/hr

Water requirements are increased by fever and fluid loss.

Reduce the intake to two thirds or less with meningitis, cerebral malaria, coma, heart failure, or renal failure.

DEHYDRATION WITH SHOCK

IV fluid: 2.5% dextrose in half strength Darrow's solution.

Amount: 20 ml/kg fast. Repeat 20 ml/kg fast if still dehydrated. Then:

Under 5 kg:	25 ml/hour
5-9 kg:	50 ml/hour
10-14 kg:	75 ml/hour
>15 kg:	100 ml/hour

Don't forget that oral rehydration is highly effective in dehydrated children without shock (see Diarrhoea, p.103).

POST-OPERATIVE

Maintenance fluid: as above.

Replace gastric sspirate: give an equal volume of intravenous 0.9% sodium chloride with 1g KCl per litre.

PYLORIC STENOSIS

Pre-operative fluids if dehydrated.

Prolonged vomiting will cause hypochloraemic alkalosis due to the loss of HCl in gastric juice. Half strength Darrow's solution is not suitable as replacement fluid in this condition.

- check electrolytes if possible
- give 20 ml/kg of 0.9% sodium chloride IV fast. Repeat this if the child is shocked
- add 2 g (8 ml) KCl to a litre flask of 0.9% sodium chloride, and give 10 ml/kg/hour IV for 2 hours
- continue with 4.3% dextrose in 0.18% normal saline with 2 g KCl /litre at maintenance rates.

Note: It is most important to have the child adequately hydrated and in electrolyte balance before surgery. Surgery should not be done on an emergency basis in pyloric stenosis.

BURNS

See the section on Burns (p.60).

NEONATES

See the table under Neonates - Fluids and Feeds (p.250).

FLUID DISTRIBUTION

	Neonate	Child	Adult
Total water	750 ml/kg	650 ml/kg	550 ml/kg
Intracellular water	350 ml/kg	350 ml/kg	300 ml/kg
Extracellular water	400 ml/kg	300 ml/kg	250 ml/kg
Whole blood	90 ml/kg	80 ml/kg	75 ml/kg
Plasma	40 ml/kg	35 ml/kg	35 ml/kg

COMPOSITION OF FLUID LOSSES

	Na (mEq/l)	K (mEq/l)	Cl (mEq/l)
Gastric	20 - 80	5 - 20	100 - 150
Pancreatic	120 - 140	5 - 15	90 - 120
Small intestine	100 - 140	5 - 15	90 - 130
Bile	120 - 140	5 - 15	80 - 120
Ileostomy	45 - 135	3 - 15	20 - 115
Diarrhoea	10 - 90	10 - 80	10 - 110
Burns (+ protein 4 g%)	140	5	110

COMPOSITION OF IV FLUIDS

	mEq per litre					Dextrose
	Na	Cl	K	Lactate	Ca	
2.5% dextrose in half strength Darrow's	61	52	18	27	-	2.5%
4.3% dextrose in 0.18% sodium chloride	31	31	-	-	-	4.3%
Hartmann's solution	130	110	5	28	4	-
0.9% sodium chloride (normal saline)	150	150	-	-	-	-

SODIUM

1g NaCl contains 17 mEq Na and 17 mEq Cl. The number of mEq of sodium required to replace a deficit = $Wt (kg) \times 0.6 (140 - \text{serum Na})$. Normal saline contains 150 mEq of sodium per litre (give 7 ml of normal saline for each mEq of sodium required to replace the deficit). For example, for a 10 kg child with a serum sodium of 120 mEq/l, give $10 \times 0.6 (140-120) \times 7 = 840$ ml of 0.9% sodium chloride IV over 48 hours. Do not increase the serum sodium by more than 2 mEq/l every hour (and it is safer to increase it even slower than this).

POTASSIUM

The total body potassium deficit cannot be calculated from the serum potassium. 1 g of KCl contains 13.4 mEq K and 13.4 mEq Cl. The MAXIMUM safe rate of K infusion IV = 0.3 mEq/kg/hour (beware of K concentrations over 4 g/litre in IV fluid).

Eg: For an 8 kg child with hypokalaemia, with IV fluid at 25 ml/hour:
 $8 \times 0.3 = 2.4$ mEq K MAXIMUM in 25 ml
 $2.4 \text{ mEq in } 25 \text{ ml} = 2.4 \times 1000/25$ or 96 mEq/l. 96 mEq K = $96/13.4$ or 7g KCl per litre.
To be safe, add only half this amount (3.5 g) to each litre.

Oral potassium can be given as electrolyte mixture (see section below), eg to children on diuretics. 1,000,000u of potassium benzyl (crystalline) penicillin contains approximately 1 mEq K.

BLOOD GASES

If blood gases are available, the number of ml of 8.4% bicarbonate required to correct an acidosis is equal to: the base deficit \times wt (kg) / 3. It is usual to give half this calculated amount, and then repeat the blood gas estimation.

CALCIUM

10 ml 10% calcium gluconate = 4.5 mEq (2.2 mmol) Ca.

10 ml 10% calcium chloride = 14 mEq (7 mmol) Ca.

10 mEq calcium = 5 mmol.

The usual dose of 10% calcium gluconate (0.22 mmol/l) is 0.5 ml/kg IV slowly stat, then 5 ml/kg/day.

The usual dose of 10% calcium chloride (0.7 mmol/l) is 0.2 ml/kg IV slowly stat, then 2 ml/kg/day.

MAGNESIUM

The usual parenteral dose of 50% magnesium sulphate is 0.2 ml/kg IM daily (or BD in severe cases). Or add 5 ml 50% MgSO₄ to each litre of maintenance IV fluid. Magnesium can be given orally as electrolyte mixture (see p.230).

MOLES

No. millimoles = no. mEq / valence = mass (mg) / mol. wt.

ELECTROLYTE MIXTURE

This is given to children with severe malnutrition or chronic diarrhoea. Either give 5 ml TID or add 5 ml to each 240 ml of milk feed. Add 50g potassium chloride, 10g magnesium hydroxide (or 40g magnesium sulphate) and 2g zinc sulphate to 1 litre of water. Label clearly: SHAKE WELL BEFORE USE.

REFERENCES

- Schierhout G, Roberts I. BMJ 316:961-964, 1998. Fluid resuscitation with colloid or crystalloid solutions in critically ill patients: a systematic review of randomised trials.
- Winters RW. The body fluids in pediatrics, Little Brown.
- Advanced paediatric life support. The practical approach. 3rd ed. London: BMJ Books, 2001.

FOREIGN BODIES

EAR

Sedate the child with chloral hydrate 50 mg/kg (maximum 2g) or Vallergran (trimeprazine) 2-4 mg/kg oral. Attempt to remove the object using suction (cut the end off the suction catheter) and, if this fails, try irrigation. General anaesthesia may be required. You may be able to hook the foreign body out with a bent paper clip (but be careful not to push it in further).

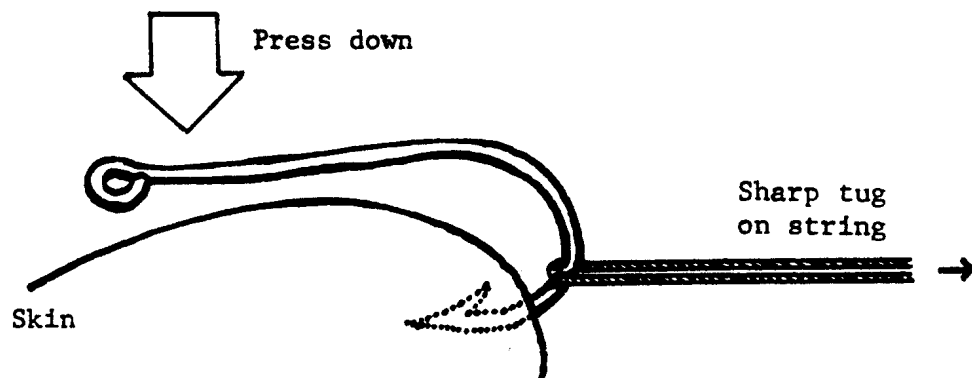
EYE

Foreign bodies often lodge in the upper recess (evert the upper lid). It may be necessary to give ketamine to an uncooperative child.

A corneal foreign body is best seen with lateral illumination. Use local anaesthesia in older children. Ketamine will be needed in most children under 8 years of age. Stain the eye with fluorescein to detect a residual corneal ulcer and, if you find one, apply chloramphenicol eye ointment QID for 3 days. Intraocular foreign body should be referred to an ophthalmologist urgently.

FISH HOOK

Loop a piece of string (or fishing line) around the bend of the hook where it enters the skin. Depress the eye end of the hook with your index finger to disengage the barb. Give a strong, sharp tug on the string to remove the hook. Tetanus toxoid should be given.



NOSE

Management is similar to the ear. Sedate the child with chloral hydrate or Vallergran and apply local anaesthetic with adrenaline. Try suction first, then irrigation. You may be able to hook the foreign body out with a bent paper clip (but be careful not to push it in further).

PHARYNX

Spray the area with local anaesthetic. Visualise the object with a spatula or laryngeal mirror and a good light. Remove it with forceps.

SOFT TISSUES

A foreign body may be very hard to find in the soft tissues at operation, even if they have been localised by x-ray. Use general anaesthesia and an Esmarch tourniquet. Do not forget to give tetanus toxoid.

SWALLOWED

Ingestion of a foreign body is commonest in the first year of life. If a foreign body lodges in the oesophagus, the child should be referred to a Base Hospital, where the object will be removed endoscopically. There is a danger of perforation and mediastinitis. Once in the stomach, most foreign bodies will pass safely; the exception is a very long hairpin which may lodge at the duodeno-jejunal flexure and perforate, so that a laparotomy is necessary.

X-rays of the head, neck, chest and abdomen may be needed. A swallowed object may lodge in the nasopharynx following vomiting.

Perform laparotomy for

- a long sharp object lodged at the duodeno-jejunal flexure
- a sharp object in the abdomen that does not progress for 14 days
- a blunt object in the abdomen that does not progress for 28 days.

TRACHEA, LARYNX OR BRONCHUS

There is acute onset of coughing, often with stridor or wheeze. An expiratory chest x-ray may show air-trapping (the film is darker on the side of the foreign body). Refer the child to a specialist surgeon for endoscopic removal of the foreign body (which may have to be done through a tracheostomy) under general anaesthesia. This should NEVER be attempted by an inexperienced doctor.

URINARY TRACT

These should always be removed. An open operation is usually necessary, although small objects in the bladder can sometimes be removed through a cystoscope.

FROG BREATHING

This technique of ventilation is only suitable for temporary use in babies under about 5 kg.

1. Insert a size 8 or 10 nasogastric tube through one nostril on free drainage. This is necessary to prevent overinflation of the stomach, with respiratory embarrassment.
2. Give nasopharyngeal oxygen at 2 litres per minute. The oxygen cannula should be inserted to a depth equal to the distance between the ala nasae (the side of the baby's nose) and the tragus (the front of the ear) - do not push it in any further than this, or it may go into the oesophagus.
3. With your left hand, lift up the baby's chin slightly, extend the neck and pinch the mouth shut. With your right hand, pinch the baby's nose for about 2 seconds, so that oxygen fills the lungs. Then release the nose for about 2 seconds and allow the elastic recoil of the lungs to empty them - be sure to allow adequate time for them to empty. You should be able to watch the chest expanding and deflating.
4. This technique should give about 12 to 15 breaths a minute. Ensure that the stomach does not inflate. If ventilation is inadequate, endotracheal intubation (p.113) is required.

GLUCOSE 6 PHOSPHATE DEHYDROGENASE DEFICIENCY (G6PD)

A high incidence of G6PD deficiency is related to the incidence of malaria (Nature 190:1120, 1961). In Papua New Guinea, many different variants of G6PD deficiency have been found. Fortunately only a few of these variants are associated with severe haemolysis. The diagnosis may be missed if G6PD deficiency is tested for immediately after a haemolytic crisis, because young red blood cells with relatively high enzyme levels predominate at that time.

Drugs that often cause haemolysis in G6PD deficient patients	Drugs that usually cause haemolysis only with severe G6PD deficiency, or in the presence of severe illness (eg infection)
ANALGESICS Acetanilide	ANALGESICS Acetylsalicylic acid Phenacetin
ANTIMALARIALS Pamaquine Pentaquine (Mepacrine) Primaquine Quinocide	ANTIMALARIALS Chloroquine Quinine Quinacrine (Atabrine)
SULPHONAMIDES Sulphanilamide N-Acetylsulphanilamide Sulphapyridine Sulphamethoxypyridazine	SULPHONAMIDES Sulphadiazine Sulphamerazine Sulphisoxazole (Gantrisin) Sulphathiazole
NITROFURANTOINS Furazolidone (Furoxone) Nitrofurazone (Furacin) Nitrofurantoin	MISCELLANEOUS Aniline Antazoline (Antistine) Ascorbic acid Chloramphenicol BAL Diphenhydramine Vitamin K (aqueous) Methylene blue Procaine amide Probenecid Naladixic acid (?)
SUPLHONES Sulphoxone (Dapsone)	
MISCELLANEOUS Naphthalene Phenylhydrazine Fava beans	INFECTIONS Respiratory viruses Infectious hepatitis Infectious mononucleosis Bacterial pneumonia Malaria
	DIABETIC ACIDOSIS

REFERENCE

Young G P. Med J Aust 1:876, 1974, G6PD deficiency in PNG.

HEAD CIRCUMFERENCE

See also Chronic Raised Intracranial Pressure (p.77).

Progressive enlargement of the head across the normal percentiles (see the chart on the next page) is better evidence of an abnormality than a single measurement showing a large head. Even so, a head circumference that is over the 98th percentile or under the 2nd percentile for age is likely to be abnormal, providing the child is not very tall or very short.

The occipito frontal circumference (OFC) is the maximum circumference of the head. The mean of three readings with a good quality tape measure is taken.

A LARGE HEAD (OVER 98TH PERCENTILE)

See Hydrocephalus, p.158

Check that the child's height is not over the 98th percentile too (a large child has a large head). Check that the parents do not have large heads. See also Chronic Raised Intracranial Pressure (p.77). An abnormally large head is usually due to hydrocephalus (a block to CSF flow) or a subdural collection. Premature or malnourished children often appear to have a large head. Rare causes are lead poisoning, space-occupying lesions (neoplasm, tuberculosis) or an increase in the amount of brain due to storage diseases (eg of mucopolysaccharide or lipid). Macrocephaly plus convulsions strongly suggests subdural effusion or haematoma.

Serial measurements of head circumference may be needed to confirm that percentile lines are being crossed (note that this is normal in premature infants). Arrange an ultrasound scan of the head if available. If not, transilluminate the skull: in total darkness, tightly apply a torch with a rubber adaptor to all areas of the skull. The area of illumination is normally less than 2.5 cm. Diffuse transillumination is seen with severe hydrocephalus, bilateral subdural effusions and the absence of cortical tissue. Asymmetrical transillumination suggests that there is a subdural effusion, porencephaly or unilateral ventricular enlargement. Subdural effusions of blood do not transilluminate. Take a skull x-ray. Do NOT do a lumbar puncture.

If hydrocephalus is confirmed treatment with acetazolamide (Diamox), frusemide and potassium chloride should be started immediately (see table p.159). Should this fail to control the increase in head circumference, shunting should be considered - though the results of this procedure in Papua New Guinea and in other "third world" situations are disappointing.

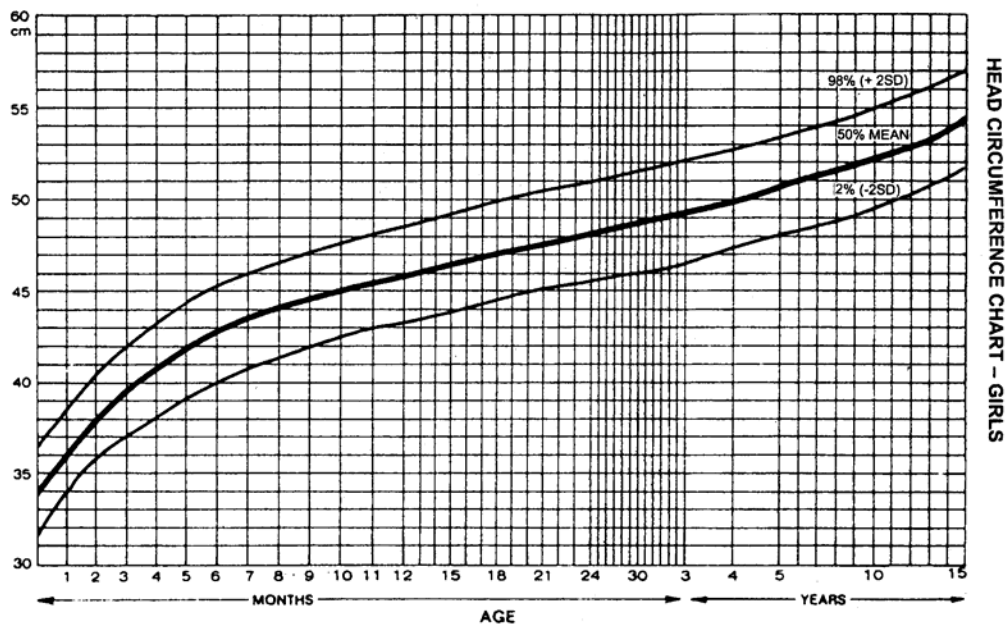
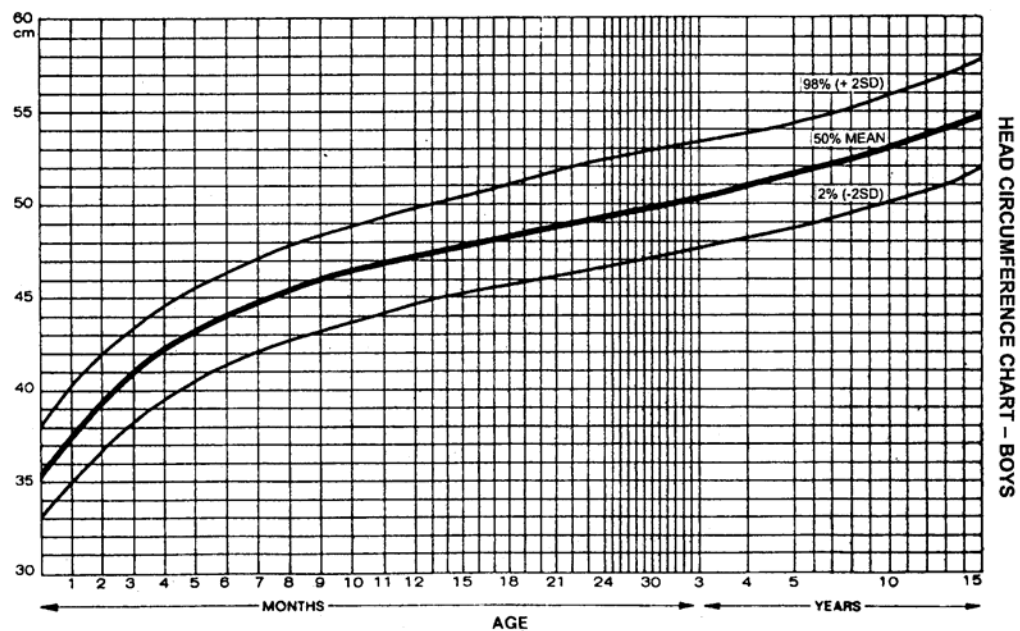
A SMALL HEAD (LESS THAN 2ND PERCENTILE)

Check that the child's height is not under the 2nd percentile (a small child has a small head). Check that the parents do not have small heads. Microcrania (small head) is usually due to failure of brain growth (microcephaly), but it may be due to craniostenosis (which prevents the head from expanding).

In microcephaly the child is usually retarded and the head has a normal shape (although the sutures may fuse early). Brain growth is retarded by a variety of congenital, metabolic and degenerative diseases, by hypoxia, and by either prenatal or postnatal infections. Intrauterine infections causing microcephaly can be remembered by the mnemonic TORCHES, which stands for Toxoplasmosis, Other, Rubella, Cytomegalovirus, Herpes simplex, Hepatitis and HIV, EB virus and Syphilis.

In craniostenosis the head is misshapen: the actual shape depends on which sutures have fused. The commonest situation is fusion of the sagittal suture, which causes a long, narrow head. Surgical treatment is difficult and not always effective even if it is performed before the age of 3 months.

To measure a child's head circumference, put the tape over the eyebrows, above the ears, and over the most prominent part of the back of the head (the occiput)



HEALTH CENTRE VISITS

In the rural areas of Papua New Guinea, health services are provided by community health workers (CHW) at aidposts, and by maternal and child health service staff patrolling from the supporting health centre.

Aidposts should have basic drugs such as crystalline penicillin, amoxycillin and antimalarials and surgical dressings, but they do not have inpatient facilities. The younger CHWs have Grade 10 education and have completed a two-year training course.

Health centres should have all the basic drugs, and should have inpatient facilities. The officer in charge (OIC) is likely to be a health extension officer (HEO). The younger HEOs have Grade 12 education and have completed a 4-year training programme and a 1-year residency covering both clinical and community health. The health centre will also have on its staff nursing sisters who have had training in maternal and child health (MCH) services (some may have postbasic paediatric or midwifery training), nursing orderlies, and CHWs. In general, each health centre is responsible for the supervision of its health subcentres (smaller health centres) and about 20 aidposts.

Whilst Papua New Guinea used to boast an effective and efficient rural health service, the situation now is, unfortunately, far from satisfactory. There are many reasons for this. Undoubtedly, one of the reasons is the lack of supervision and support at different levels.

Doctors working in hospitals have a responsibility to rural as well as to urban communities, which they should be able to honour by providing support to health centres and their staff by regular visits.

The purpose of these health centre visits is, first and foremost, to support the health staff. It should not be seen as “supervisory” in a paternalistic or “fault finding” way.

Aspects of the visit include:

- discussion of difficult clinical problems
- teaching - “inservice” - for the staff
- discussion of administrative problems, such as drug supply, and helping when possible, usually by taking matters up with the provincial health authorities
- discussing the work of the MCH services
- encouraging and assisting health centre staff to support the aidposts and CHWs for which they are responsible.

POINTS TO REMEMBER

1. Make sure that the OIC of the health centre is notified at least 2 weeks in advance of your visit.
2. Before you leave for the health centre, check at the provincial health office for any mail, medical supplies or government stores that are to go to the health centre. Pick up the Problem Book kept about the health centre at the provincial health office (suggest there should be one if there isn't).
3. When you arrive at the health centre, note whether the grounds are clean and tidy.
4. Have a private talk with the OIC about any staff discipline problems, medical supplies, transport for MCH clinics etc. Write the problems in the Problem Book.
5. Have a talk with the MCH staff. They have more direct involvement with the health of children than the other health centre staff, including the OIC. Ask about their transport, equipment, clinic attendance and any other problems.
6. See the patients at the health centre with the OIC and all the staff that are available:

TEACH AROUND THE PATIENTS YOU SEE TOGETHER - CONCENTRATE ON COMMON PROBLEMS AND MANAGERMENTS:

- a. check that staff are using standard treatments and drug doses
- b. check their dilutions of drugs
- c. emphasize the importance of the health record book

- d. check that every child is weighed, the weight plotted on the road to health chart, and appropriate treatment given
 - e. check that children are being appropriately immunised.
7. Check the medical store:
- a. is it clean and tidy?
 - b. the drugs should be arranged in the same order as on the health centre drug order form that goes to Base Medical Store, with the order number written on the shelf
 - c. are any items obviously overstocked?
 - d. check for shortages, in particular:
 - amodiaquine (infant Camoquin) tabs
 - chloramphenicol capsule, suspension, vial
 - chloroquine tab
 - Fansidar
 - digoxin paediatric elixir
 - penicillin vials, benzyl (crystalline)
 - amoxycillin tabs
 - drugs for severe and treatment failure malaria
 - e. record any shortages in the Problem Book.

Are there any problems with supplies? When did the OIC last order drugs? Medical supplies usually come from Base Medical Store, but important items can often be supplied from the provincial hospital.

8. Check the fridge:
- a. is it working properly?
 - b. is there enough kerosene/gas?
 - c. does it need defrosting?
 - d. are there enough vaccines?
 - e. make sure the BCG, Sabin, measles, triple antigen, hep B and tet tox vaccines are in the ordinary part of the fridge (and not in the freezer)

Record any shortages in the Problem Book.

9. Does any equipment need repair or replacement? Check the:
- a. BP machine
 - b. auriscope
 - c. sterilizer
 - d. sucker.
10. Check the dangerous drugs. See that the dangerous drug register is kept properly and sign that you have checked it.
11. Check the TB/leprosy register. Are patients coming for treatment? If not, what action has been taken?
12. Inspect the nutrition garden if there is one. Ask the OIC about his programme for health education talks to mothers, schools, councils etc. What MCH patrols are being done? Find out where they go, how many children they see, does the vaccine stay cold, and whether they have hanging scales to take that are in good order.
13. Before you leave, discuss with the OIC again what things you will try to do for him, and what you expect him to do. Be sure you keep your word.
14. When you return to your hospital, discuss any problems with the provincial health officer. Return the Problem Book.

Try to arrange for your visit to coincide occasionally with a community health worker (CHW) in-service course at the health centre. Assist in the teaching. The reputation and morale of these health workers is often poor - and yet these people treat the vast majority of the sick people in rural areas. Encourage health centres to take an active interest in the supervision and continuing education of their village-based community health workers, and reinforce this with your own participation.

A health centre visit will be most productive if you can stay overnight with the HEO. If this is not possible, at least try to have a meal or a cup of coffee with him. An informal chat during a meal or coffee break is often much more useful than the official part of your visit.

TEACHING HEALTH CENTRE STAFF

It is important to be very clear in your own mind about exactly what you want to teach paramedical workers. Teach the important things about the important diseases. For example, what do paramedical staff really have to know about pneumonia? They need to know which children need antibiotics and which children need to be referred for admission.

Teach:

1. Most children with cough do NOT need antibiotics
2. Children with cough and FAST BREATHING do need an antibiotic
3. Children with cough and CHEST INDRAWING need to be admitted (for 6-hourly benzyl penicillin).

Important things to teach all health workers about children

1. Two very important diseases:

Pneumonia:	<ul style="list-style-type: none"> • no fast breathing: no antibiotic • fast breathing: give amoxycillin for 5 days • chest indrawing: admit, give benzyl penicillin IM 6 hourly give chloramphenicol if there is cyanosis or poor feeding
Diarrhoea:	Look for DEHYDRATION (pale or cold hands and feet, slack skin, deep breathing): <ul style="list-style-type: none"> • no dehydration: outpatient and home oral fluids • some dehydration: outpatient ORT and review • severe dehydration: admit for IV therapy or ORT.
2. In any child with fever, give ANTIMALARIALS and look for:

Meningitis:	<ul style="list-style-type: none"> • is the child DROWSY (very sleepy)? • do a lumbar puncture if the child is drowsy or convulsing or very irritable.
-------------	--
3. Always look for the hidden diseases:

Malnutrition:	<ul style="list-style-type: none"> • weigh every child and mark the weight on the graph: <ul style="list-style-type: none"> ○ if the weight is 60 to 80% with a flat or falling weight curve but with no oedema, give outpatient nutrition education ○ if there is oedema OR the weight is less than 60%, admit
Anaemia:	<ul style="list-style-type: none"> • check if the tongue is pale on every child: <ul style="list-style-type: none"> ○ if the child is pale, give iron, antimalarials, folic acid and albendazole.
4. Teach the six nutrition rules:

When to start:	1. Start giving soft food as well as breast milk when your child is four months old. If you do not know his age, start when he can rollover
How often:	2. Feed your children four to six times a day
What to give:	3. Feed your child cooked and mashed peanuts, beans or fish each day.
	4. Add some coconut cream, dripping or margarine to the child's food
Other advice:	5. Continue to feed your child when he is sick - and give extra food after the sickness has finished.
	6. Mothers need to eat plenty when they are pregnant or breastfeeding.

Notes:

1. At the time of writing, a Health Centre Visit Report form is being developed. Make sure you become familiar with this form.
2. Doctors should be familiar with the 10-step checklist and use it as a basis for their teaching.
3. Doctors should always encourage the use of, and refer to, the Standard Treatment Book.

HEART DISEASE - CONGENITAL

Note: IT IS MOST IMPORTANT TO KEEP A CARDIAC REGISTER. Details should include the name and age of the child, the provisional diagnosis and, if possible, a contact address or telephone number.

THE FREQUENCY OF DIFFERENT TYPES OF CONGENITAL HEART DISEASE

	Infants	Children	Adults
Ventricular septal defect (VSD)	28%	24%	15%
Patent ductus arteriosus (PDA)	13%	15%	16%
Atrial septal defect (ASD)	10%	12%	16%
Coarctation aorta (CoAo)	9%	5%	8%
Transposition (TGV)	8%	5%	2%
Fallot's tetralogy	7%	11%	16%
Pulmonary stenosis (PS)	6%	11%	15%
Aortic stenosis (AS)	4%	7%	5%
All others	15%	10%	7%

The child's age at the onset of symptoms is very helpful in diagnosis.

Most lesions present with either cyanosis or congestive cardiac failure (CCF).

Children with lesions with reduced pulmonary blood flow (avascular lungs on chest x-ray) rarely develop CCF.

Day one:	Hypoplastic left heart (CCF)
First month:	Transposition of the great vessels (cyanosis) Coarctation of the aorta + ventricular septal defect (CCF)
After 1 month:	Fallot's tetralogy (cyanosis) Ventricular septal defect (CCF) Patent ductus arteriosus (CCF)
After infancy:	Pulmonary stenosis (fatigue, syncope) Aortic stenosis (fatigue, syncope) Atrial septal defect (CCF) Coarctation of the aorta (CCF, syncope)

DIAGNOSIS

Is the child cyanosed?

Are the lungs plethoric, normal or avascular on chest x-ray?

Acyanotic, no shunt, normal lung fields

Coarctation of the aorta, aortic stenosis: these lesions (with no pulmonary plethora) have to be distinguished from mild cases of the next group

- coarctation: reduced or absent femoral pulses, and an ejection systolic murmur (ESM) at the base and back
- aortic stenosis: an ejection murmur at the apex, in the neck and in the aortic area (with a thrill).

Acyanotic with L to R shunt (lungs normal or plethoric)

- Atrial and ventricular septal defects, patent ductus arteriosus: larger shunts show cardiomegaly and pulmonary plethora on chest x-ray. The lesion is inoperable if there is severe pulmonary hypertension with Eisenmenger's syndrome (cyanosis, clubbing, parasternal heave and a palpable 2nd heart sound).

- Ventricular septal defect: a pansystolic murmur and thrill at the lower left sternal edge.
- Patent ductus arteriosus: a machinery murmur with a thrill at the upper left sternum, with bounding pulses.
- Atrial septal defect: a fixed split of the 2nd heart sound, and an ejection systolic murmur (ESM) at the upper left sternal edge.

Acyanotic with avascular lungs

Pulmonary stenosis: an ejection systolic murmur and a thrill at the upper left sternal edge.

Cyanosis with pulmonary plethora

Transposition of the great vessels: cyanosis in first month of life. The chest x-ray may show pulmonary plethora (but it is often normal in the first week of life).

Cyanosis with avascular lungs

Fallot's tetralogy: an ESM and a thrill at the upper left sternal edge.

TREATMENT

Surgical

The only lesion which can be satisfactorily treated at the current time in Papua New Guinea within the normal surgical services is uncomplicated patent ductus arteriosus.

There is however a cardiac programme in place whereby patients with congenital and rheumatic heart disease are assessed firstly by a paediatrician with experience in paediatric cardiology, and subsequently by a visiting paediatric cardiologist in order to select children suitable for surgery by a visiting overseas cardiac surgical team. A few children may also be sent for surgery overseas - but this is extremely expensive. The team has to date operated on children with atrial septal defect, pulmonary stenosis, ventricular septal defect, and coarctation. Children with Fallot's tetralogy have been offered a two-stage procedure - a shunt procedure on the first occasion and a corrective procedure subsequently. Young adults have also received surgery for mitral valve disease.

Medical

Digoxin should only be used if heart failure is present. IM digoxin is poorly and erratically absorbed; digoxin should be given orally or by IV infusion (in a burette). Do NOT give digoxin to children with Fallot's tetralogy. Digoxin may not help CCF from left to right shunts (J Pediatr 92:867,1978) though in practice it is often given.

Oxygen is useful with cardiac failure or pneumonia. It is NOT effective for cyanosis due to a cardiac shunt (eg TGV or Fallot's tetralogy). Cyanosis abolished by giving oxygen is due to lung disease (although this may be secondary to CCF).

Frusemide: Give 1 mg/kg IM or oral once or twice a day for cardiac failure until it is controlled.

Other diuretics: Spironolactone is available from the Health Department and may be useful in combination with frusemide.

Captopril: May be obtained, sometimes through the Health Department, sometimes through local pharmacies. It is, however, very expensive.

Feeding: EBM may have to be given by NG tube. Give 15 ml/kg 3 hourly.

Infections: Be alert for pneumonia and subacute bacterial endocarditis (SBE). Give chloramphenicol for pneumonia, and penicillin and gentamicin for SBE. It is important to try to prevent SBE in patients with congenital heart disease. Give antibiotic prophylaxis just before any surgical or dental procedure (amoxycillin/ampicillin for "clean" procedures, ampicillin and gentamicin for "dirty" procedures).

REFERENCES

- Sengupta A, Auto J, Pawape G. PNG Med J 38(1):45-51, 1995. Unexplained pulmonary hypertension in children in the highlands of Papua New Guinea.
- Tefuarani N. PNG Med J 43(1-2):65-68,2000. A history of congenital heart disease surgery in PNG.

HEART DISEASE - INNOCENT (FUNCTIONAL) MURMURS

These are present in about 30% of children. They are accentuated by tachycardia (caused by fever, excitement or exercise), they often disappear on inspiration or with Valsalva manoeuvre, and they often vary with posture. A murmur heard only in diastole is never innocent.

PULMONARY SYSTOLIC MURMUR

This is a soft blowing high pitched ejection systolic murmur that is maximal at the second left intercostal space.

VIBRATORY MURMUR

This is a short midsystolic murmur like the buzzing of a bee that is heard at the lower left sternal edge. It is diminished by sitting up and extending the neck, and abolished when patients sit with their hands behind them and arch their back.

VENOUS HUM

This is a continuous murmur very like that of a patent ductus arteriosus, that is best heard in the supraclavicular fossa. It is diminished by compressing the internal jugular vein on that side, turning the head from the side to the midline, lying flat or by the Valsalva manoeuvre.

If there is any uncertainty about a murmur being innocent or not, arrangements should be made for the child to be assessed by the provincial or regional paediatrician.

HIV INFECTION

Infection with the human immunodeficiency virus (HIV) leads to progressive immunological dysfunction and will become one of the most important causes of immunodeficiency in children in PNG.

EPIDEMIOLOGY

The first case of HIV infection in PNG was identified in June 1987 and the first clinical case of acquired immune deficiency syndrome (AIDS) was reported to the Department of Health in March 1988. Since then the numbers have been increasing rapidly. As of December 2001, more than 5,000 HIV seropositive persons had been reported to the Health Department, including some 100 children, the majority of whom were below 18 months of age.

The age and sex distribution of infected adults is very similar to that seen in African countries. The majority of cases have been reported from National Capital District, but cases have been seen in nearly all provinces.

TRANSMISSION

The main route of transmission of HIV in adults is via sexual contact with an HIV infected individual. For infants and children, the most common mode of transmission is from mother to child - vertical transmission.

Modes of transmission of HIV in infants and children

Vertical transmission - from mother to child:	<ul style="list-style-type: none">• before birth, transplacental• during delivery• after birth, through breast milk
Contaminated blood or blood products	
Non-sterile needles or other skin-piercing instruments	
Sexual abuse	

Not all children born to HIV-infected mothers are infected with HIV and accurate estimates of the vertical transmission rates can only be obtained from prospective studies of cohorts of children born to infected mothers and who are then followed from birth. Current estimates of the rate of transmission range from 14% to 39% in different studies.

Breast milk does play a role in transmission of HIV from mother to baby, and transmission is increased when the mother has acquired the infection by blood transfusion during or soon after delivery. The resulting high virus load may lead to transmission of HIV via breast milk. However, it is important to realise that in Papua New Guinea where mortality from infectious diseases in infancy and childhood is high, the substantial protective benefits of breastfeeding outweigh the possible risk of transmission in most cases. Breastfeeding should therefore be encouraged in the majority of cases (see p.154).

Most affected infants and children acquire HIV infection from the mother, but it is also important to realise that even in countries with a relative high transmission rate (30-40%), the majority of the children born to HIV infected mothers are NOT infected.

CLINICAL FEATURES AND CASE DEFINITIONS

The clinical presentation of HIV infection in infants and children depends partly on the exposure to different infections. The spectrum of paediatric AIDS described in the classification of HIV infection in children developed by the United States Centers for Disease Control includes opportunistic infections, severe recurrent bacterial infections and malignancies. Most of the opportunistic infections cannot be diagnosed in Papua New Guinea, so this classification is not useful for the situation in PNG. In most non-industrialised countries, children present with non-specific signs and symptoms, such as failure to

thrive, chronic diarrhoea, chronic fever and cough and recurrent bacterial infections. The children with HIV infection seen in Papua New Guinea present mostly with severe diarrhoea, pneumonia and tuberculosis, failure to thrive and severe oral thrush.

Common manifestations of HIV infection in children

Weight loss or failure to thrive
Chronic fever
Enlargement of liver and spleen
Enlargement of parotid gland
Recurrent abscesses
Delayed development, encephalopathy, tone abnormalities
Oropharyngeal candidiasis

HIV infected children have an increased frequency not only of common paediatric infections, such as pneumonia and otitis media, but also of tuberculosis and chronic gastroenteritis. In addition, the symptoms common to many treatable diseases tend to be more persistent and severe in HIV infected infants. Moreover, HIV infected infants tend not to respond so well to treatment of these conditions and are more likely to suffer life-threatening complications.

The World Health Organization has developed guidelines for recognition of HIV infection in children. These may be used in situations where health workers suspect HIV infection and where testing is not available or where the child is too young for the test to be accurate. These guidelines may be helpful for clinical management of the child and to alert the health worker to possible needs of the mother for counselling and care.

Guidelines for recognising HIV infection in children

A diagnosis of HIV infection in children is made if the following are present:

- Any cardinal finding:
 - *Pneumocystis carinii* pneumonia (PCP), lymphoid interstitial pneumonitis, Kaposi's sarcoma, oropharyngeal candidiasis
 - Two or more characteristic findings:
 - recurrent infections, herpes zoster, cytomegalovirus infection, tuberculosis, neurological problems
 - One characteristic finding and two or more associated findings:
 - oral thrush, failure to thrive, skin rashes, fever longer than 1 month, diarrhoea longer than 14 days, generalised lymphadenopathy
 - Three or more associated findings and any epidemiological risk factor:
 - mother tested positive for HIV, sexual abuse, history of blood transfusion, use of contaminated needles or syringes
 - Two associated findings and laboratory evidence of HIV infection in the child.
-

A second clinical case definition, which is widely used was primarily developed for epidemiological purposes and includes a number of major and minor signs.

WHO clinical case definition for paediatric AIDS

Major signs:

- Weight loss or abnormally slow growth
- Chronic diarrhoea of more than 1 month duration
- Prolonged fever of more than 1 month duration

Minor signs:

- Generalised lymphadenopathy
- Oropharyngeal candidiasis
- Repeated common infections
- Persistent cough for more than 1 month
- Generalised dermatitis
- Confirmed maternal HIV infection

Paediatric AIDS is suspected in a child presenting with at least 2 major signs associated with 2 minor signs in the absence of known causes of immunosuppression, such as cancer, malnutrition or other recognised aetiologies.

This definition needs to be validated in different countries as it is rather non-specific and has a low positive predictive value. In PNG most children with tuberculosis would fulfil the criteria.

LABORATORY DIAGNOSIS

In PNG, tests to diagnose HIV infection are based on testing for HIV-specific IgG antibodies, not testing for the actual presence of the virus or viral antigens. As IgG antibodies cross the placenta, all children born to HIV infected mothers will test 'positive'. This does not mean that they are actually infected, but they may just be carrying maternal antibodies. These antibodies disappear over the first 18 months of life if the child is not infected, but they persist beyond that age if the child is infected with HIV. So in PNG a definite diagnosis of HIV infection can only be made when the child is 18 months old. Elsewhere, assays which test for the actual presence of virus, such as p24 antigen tests, virus culture and PCR, can be used to confirm the HIV status of a baby, but it has to be realised that these tests can be false negative and generally 2 consecutive positive tests are required to decide that a baby is HIV infected.

MANAGEMENT

The management of HIV infected babies differs greatly in different parts of the world, depending on available facilities. In PNG anti-retroviral drugs which are used in the industrialised world, are not available. The principles of management are summarised in the following table.

Management of HIV infected children and children born to HIV infected mothers

Treat infections as early as possible, using standard treatment guidelines

Maintain good nutrition:

- advise on breast feeding and weaning practices
- advise on feeding a child with a poor appetite

Regular growth monitoring

Oral rehydration therapy during diarrhoea episodes, to prevent dehydration

Immunise as usual

Early diagnosis and treatment of suspected tuberculosis for all family members

Pain relief when necessary

Prophylaxis for *Pneumocystis carinii* pneumonia

Treat the child as normal, playing etc.

Support for the family, especially for the mother.

PCP prophylaxis for children born to HIV infected mothers

Indications:	All children of HIV-infected mothers below 12 months of age who are not definitely free of infection.
Regimen:	Trimethoprim/sulfamethoxazol 0.6 ml syrup/kg daily.

HIV infection is a family disease and does not only affect the infected children and adults, but also the non-infected children and family members. Non-infected children have an increased mortality rate because of the deteriorating health of the mother. Families often face increased poverty and stress because adults are forced to leave their paid employment or may be unable to farm the land because of sickness. Women may be ill as well as caring for sick family members and looking after young children. The stigma which is often still attached to HIV infection means that the family requires a lot of support and part of this support needs to be provided by the health workers looking after the family.

PROGNOSIS

The prognosis depends on several factors, such as age of presentation, severity of AIDS diagnosis and the availability of health care and drugs to treat opportunistic infections. It seems that there are 2 patterns of disease progression to AIDS. During the first year of life, severe immunodeficiency associated with serious infections and encephalopathy seems to appear in approximately 20% of infected children. Most of these children die before they reach the age of 4 years. The remaining 80% of infected children have a slower progressive disease, similar to that observed in adults. Infected children born to mothers who have severe disease have a higher risk to develop the early severe form than infected children born to mothers with asymptomatic infection.

PREVENTION

At the time of writing, it seems probable that a preventative programme, using single doses of nevirapine to the mother during labour and to her baby in the first 24 hours of life will be introduced.

HIV INFECTION AND BREASTFEEDING

In arriving at a decision about breastfeeding for the infants of HIV-infected mothers, the following need to be considered:

- The risk of acquiring HIV through breastfeeding approximates 1 in 10
- Exclusive breastfeeding is associated with a much lower transmission rate than mixed breast/formula feeding
- Overall mortality in non-breastfed babies is likely to be considerably higher than in breastfed babies.

At present the best choice for most HIV-infected mothers in Papua New Guinea is to give exclusive breastfeeding for the first 4-6 months of life and to wean abruptly if this is practicable. There will be individual mothers for whom the best choice may be to formula feed - but it should be stressed that formula feeding does not mean bottle-feeding. Formula milk can be given with cup and spoon-feeding. Whatever method they chose, all HIV-infected mothers will need ongoing support and care.

STAGING IN CHILDREN

WHO has recently introduced a clinical staging system for HIV infection in children:

Clinical Stage 1

1. Asymptomatic
2. Generalised lymphadenopathy

Clinical Stage 2

3. Unexplained chronic diarrhoea
4. Severe persistent or recurrent candidiasis outside the normal period
5. Weight loss or failure to thrive
6. Persistent fever
7. Recurrent severe bacterial infections

Clinical Stage 3

8. AIDS-defining opportunistic infections
9. Severe failure to thrive
10. Progressive encephalopathy
11. Malignancy
12. Recurrent septicaemia or meningitis.

FURTHER READING

Two publications of The Australasian Society for HIV Medicine, published by the Medical Journal of Australia, 1997:

- Could it be HIV? The clinical recognition of HIV infection, 2nd edition, edited by G Stewart.
- Managing HIV, edited by G Stewart.

HIV series, published in The Lancet, 1996. Including:

- Paediatric HIV infection
- HIV as the cause of AIDS

Gibb D and Wara D. Paediatric HIV infection. AIDS 8 (suppl 1):S275-S283,1994.

REFERENCES

- Burns DN and Mofenson LM. Lancet 354 (suppl 11):1-6,1999. Paediatric HIV-1 infection.
- Coutsadis A, Pillay K, Spooner E et al. Lancet 354:471-475,1999. Influence of infant feeding patterns on early mother to child transmission of HIV-1 in Durban, South Africa: a prospective cohort study.
- Dabis F, Msellati P, Meda N et al. Lancet 353:786-792,1999. 6-month efficacy, tolerance and acceptability of a short regimen of oral zidovudine to reduce vertical transmission of HIV in breastfed children in Cote d'Ivoire and Burkina Faso: a double blind placebo controlled multicentre trial.
- Friesen H. PNG Med J 39(3):183-189,1996. Paediatric HIV infection.
- Guay LA, Musoke P, Fleming T et al. Lancet 354:795-802,1999. Intrapartum and neonatal single dose nevirapine compared with zidovudine for prevention of mother to child transmission of HIV-1 in Kampala. Uganda HIVNET 012 randomised trial.
- Latham MC, Preble EA. Br Med J 320:1656-1660,2000. Appropriate feeding methods for infants of HIV infected mothers in sub-Saharan Africa.
- Marseille E, Kahn JG, Mmiro F et al. Lancet 354:803-809,1999. Cost effectiveness of single dose nevirapine regimen for mothers and babies to decrease vertical HIV-1 transmission in sub-Saharan Africa.
- Mofenson LM. Lancet 353:766-767,1999. Short course zidovudine for prevention of perinatal infection. PNG Med J 39(3),1996. Focus Issue on HIV and AIDS.
- Ndati R, John G, Mbori-Ngacha D et al. JAMA 283:1167-1174,2000. Effect of breast feeding and formula feeding on transmission of HIV-1: a randomised clinical trial.
- Shaffer N, Chuachoowong R, Mock PA et al. Lancet 353:773-780,1999. Short course zidovudine for perinatal HIV-1 transmission in Bangkok, Thailand: a randomised controlled trial.
- Soderlund N, Zwi K, Kinghorn A, Gray G. Br Med J 318:1650-1655,1999. Prevention of vertical transmission of HIV: analysis of cost effectiveness of options available in South Africa.
- Wiktor SZ, Ekpini E, Karon JM et al. Lancet 353: 781-785,1999. Short course oral zidovudine for prevention of mother to child transmission of HIV-1 in Abidjan, Cote d'Ivoire: a randomised trial.

HOSPITALISATION

See also “Think about the child’s past and future health” and “Provide comprehensive care” in the Introduction (p.2)

ADMISSION

A minimum history is:

- age, weight, % weight, trend in weight-for-age (increasing, constant, falling)
- immunisation status
- presenting complaint and duration
- fever, cough, shortness of breath?
- diarrhoea, vomiting?
- drowsy, feeding poorly?
- been in malarious area?
- contact with TB?
- any previous admissions?
- position in family (eg fourth of five children).

DISCHARGE

1. Explain to the mother what was wrong with her child and why he or she got better.
2. Make sure that mother understands what to do at home. It is not always a good idea to give a village mother medicine to give to her child at home since it is often not always given.
3. Make sure that the child’s weight, diagnosis and admission number are recorded in the Health Record Book.
4. If you want the child to have treatment as an outpatient, write this in the book too, eg Infant Camoquin 1 tablet daily for three days.
5. If the child has no book, give one to him or her. If the book is at home, write the above information on a piece of paper for mother to keep with the book.
6. Check the child’s immunisation status, and give any vaccines that are due. If in doubt, vaccinate. Any child that is well enough to go home is well enough to be vaccinated (Note: Measles vaccination should be given at the time of admission if the child is due for it).

NOTES

In some areas, a high proportion of parents abscond with their child without telling staff they are going:

1. Ask each mother how her child is on the ward round each day. If she says the child is well (when he or she is still clearly ill), she is probably about to abscond. Discuss the importance of treatment with her.
2. Parents often say that a relative has died as an excuse for going home. Do not argue too much with them even if the child is still sick - they will probably leave anyway. Try to arrange alternative treatment. At least you have been warned of their departure!
3. Try to stop staff from scolding a mother who returns after absconding, no matter how frustrating this may be. She will not come back next time. A study in Goroka, showed that a major reason for mothers not bringing sick children to MCH clinics was that they were afraid of being scolded (they only brought their child if he was well).
4. If the child has pneumonia and you have taken a chest x-ray, always show it to the mother and explain that the white areas represent pus. This visible evidence often seems to be more convincing than other methods of persuasion.

5. If a child has meningitis, always show the cloudy CSF to the mother and explain that it should be clear water. Keep reminding her of what she saw.

Do not try to keep dying children in hospital unless the parents want to stay. Always tell parents if their child has untreatable disease - they will accept this very well and appreciate your frankness.

Do not exert excessive pressure if parents will not consent to a procedure being done on their child. This can be very difficult advice to follow, but it is most important that parents are not made to feel in the wrong when they are acting in what they see as the best interests of their child.

Always remember that scientific medicine is still “on trial” for many people in Papua New Guinea. We have to convince people that we have something worthwhile to offer; this is by no means taken as proven. If we fail to convince people, antagonise them or deceive them, they will not come to us in the future.

HYDROCEPHALUS

Hydrocephalus may be detected prenatally, at the time of birth or during infancy and childhood.

Congenital hydrocephalus is usually due either to a developmental abnormality of the CSF drainage system (lateral ventricles through Foramina of Munro to third ventricle, through Aqueduct of Sylvius to fourth ventricle and into subarachnoid space through the Foramina of Luschka and Magendie), or to intrauterine infection, resulting in either blockage of the drainage system or impairment of CSF absorption. Rarely it is due to overproduction of CSF by a choroid plexus papilloma.

Acquired hydrocephalus is most commonly secondary to meningitis, though it can also follow head injury, and can complicate space occupying lesions which compress the drainage system.

The availability of ultrasound examination has made the diagnosis of hydrocephalus relatively easy. Scanning through the anterior fontanelle is possible throughout the first year of life in most children and can be used at older ages if the sutures are sprung. If ultrasound is not available, transillumination is a substitute.

Hydrocephalus should be suspected when the child has a head circumference above the 98th centile (see Head Circumference, p.141) or when the head circumference is crossing the normal centiles (Note: It is quite possible for a child to have hydrocephalus with a head circumference within the normal range). Frequent serial measurements of head circumference are necessary to detect hydrocephalus in children at risk, and to determine the rate of progression of the hydrocephalus. The aim should be to diagnose hydrocephalus and commence treatment BEFORE the child develops neurological signs. By the time the classic “sunsetting” appearance is evident, there is almost certainly irreversible neurological damage.

TREATMENT

In deciding on treatment of children with hydrocephalus in PNG, it should be realised that whilst surgical treatment with a ventriculo-peritoneal shunt appears to be the most obvious choice, the results have been disappointing, with a high incidence of shunt failure and other complications. Children who have had shunts inserted require ready access to medical follow up in the event of shunt problems.

It is therefore sensible to start medical treatment as soon as the diagnosis is made. Surgery should then be considered if the hydrocephalus is rapidly progressive in spite of medical management, or if medical treatment fails to control a slow but abnormal increase in head circumference or ventricular dilatation.

Surgery should be instituted in the case of children with congenital hydrocephalus in whom a block is clearly demonstrated.

Medical treatment

The aim of treatment is to reduce the production of CSF. It consists of treatment with acetazolamide, frusemide and potassium chloride.

Criteria for medical treatment:

1. Head circumference which is increasing excessively and has crossed percentile lines.
2. Progressive ventricular enlargement documented by ultrasound.
3. Stable vital signs without lethargy, vomiting or apnoea (ie no signs of raised intracranial pressure).
4. Age more than 2 weeks and less than 1 year.
5. Normal renal function.

Treatment is given as per the following schedule:

- Acetazolamide (Diamox 250 mg tablets) commence at 25 mg/kg/day divided BD and increase to 100 mg/kg/day
- Frusemide (40 mg tabs) 1 mg/kg/day
- Potassium citrate (20 mmol/5 ml) 6 mmol/kg/BD

Drug	3-4.9 kg	5-9.9 kg	10-15 kg
Acetazolamide			
Day 1	¼ tab BD	½ tab BD	¾ tab BD
Day 2	½ tab BD	1 tab BD	1½ tab BD
Day 3	¾ tab BD	1½ tab BD	2¼ tab BD
Day 4	1 tab BD	2 tab BD	3 tab BD
Frusemide	¼ tab alt day	¼ tab daily	¼ tab daily
Potassium citrate	5 ml BD	10 ml BD	15 ml BD

The treatment is usually well tolerated, but children being treated may develop hypokalaemia and acidosis quickly if they develop diarrhoea.

Regular monitoring of head circumference is necessary. Should the increase in circumference be well controlled on treatment, this should be continued for 6 months, and then gradually withdrawn with ongoing monitoring. If head circumference treatment is not controlled, surgery is indicated.

Surgery

This consists of the insertion, under scrupulously sterile conditions, of a ventriculo-peritoneal shunt. The operation should be performed by an experienced surgeon. A number of different types of shunt are available but the choice in PNG is limited.

Complications of shunt procedures:

Short term: Infection

Longer term: Infection
Blockage
Disconnection
Displacement
Erosion of overlying skin
Shunt nephritis

Children who have had shunts inserted need:

1. regular review
2. ready access to paediatric/surgical services in the event of shunt problems
3. parents who are aware of the signs of shunt blockage such as fretful behaviour, poor feeding, headache and vomiting.

HYPERVENTILATION

Most cases of hyperventilation in children in Papua New Guinea are due to pneumonia or diarrhoea, and in these cases the cause will usually be obvious. A high fever may cause mild hyperventilation. Hysteria is a very rare cause of hyperventilation in Papua New Guinea.

If hyperventilation is not due to lung disease (usually pneumonia) or dehydration and acidosis (usually from diarrhoea), it is usually due to causes in the head - meningitis, cerebral malaria, encephalitis and (rarely) Reye's syndrome (p.83).

Do a lumbar puncture and a blood slide.

If the cause is still obscure, then the hyperventilation is probably due to acidosis (from causes other than dehydration). There are four important causes of unexplained acidosis:

1. Poisoning (check history: aspirin, aminophylline, cassava, angel's trumpet flowers).
Hyperventilation from poisoning is not always due to acidosis
2. Renal pathology - failure (check fundi, BP, urea) or renal tubular acidosis (see p.345)
3. Diabetes: there is a blood glucose or Dextrostix over 11 mmol/l (a result under 2.6 mmol/l suggests Reye's syndrome). Test the urine for sugar if the blood glucose or a Dextrostix cannot be done
4. Shock from low cardiac output (eg myocarditis): there will be cold hands and feet, with hypotension.

If the child looks ill and you cannot make a definite diagnosis, treat for meningitis (chloramphenicol) and cerebral malaria even if the lumbar puncture and blood slide are normal.

A summary of the causes of hyperventilation:

- It is usually due to pneumonia or diarrhoea
- There are four causes in the head: meningitis, cerebral malaria, encephalitis, and Reye's syndrome
- There are four causes of unexplained acidosis: poisoning, renal pathology, diabetes, and shock.

HYPOGLYCAEMIA

DEFINITION

There is no absolute cut-off point, but the generally accepted limit at all ages is a blood sugar level less than 2.6 mmol/l.

SIGNS AND SYMPTOMS

There is a progression of responses to hypoglycaemia, starting with release of counter-regulatory hormones, then an autonomic warning (sweatiness, tachycardia, pallor), then acute neuroglycopenic symptoms (headache, irritability, weakness), and then deteriorating cerebral function (confusion, coma, seizures). In repeated hypoglycaemia, the warning symptoms are often absent and the child progresses straight to deteriorating cerebral function.

In neonates, signs are non-specific - apnoeas, cyanosis, hypotonia, poor feeding, seizures.

Hypoglycaemia is frequent and often unsuspected in sick children. A visual dextrostix reading, glucometer reading, or blood sugar level should be done in any unwell/premature/small neonate, any seriously ill child, or in any child with an unexplained CNS deterioration.

CAUSES

Common

Malaria - particularly with falciparum, and after quinine, but often occurs before treatment
Gastroenteritis/dehydration
Any sick/premature/small-for-dates neonate
Advanced malnutrition
Other severe childhood illness
Infant of a diabetic mother.

Uncommon but important

Adrenal crisis - treat with dextrose and hydrocortisone

- Congenital adrenal hyperplasia - see p.286.
- Hypopituitarism - often with hypogonadism, other dysmorphisms

Hyperinsulinism - repeated hypoglycaemia with high dextrose need
Various inborn errors of metabolism - present in neonates, or in childhood after prolonged fasts.

TREATMENT

If the baby/child is conscious and able to drink and eat, the baby should be fed and the older child given a sweet drink followed by some more complex carbohydrate.

Otherwise, IV dextrose/glucose, given as a bolus of either 1 ml/kg of 50% or, if available, 5 ml/kg 10%. Check sugar level in 10-15 minutes. A glucose infusion is often necessary, of up to 10% dextrose (amounting to 4-8 mg/kg/min dextrose). Specialist advice should be sought if there is a dextrose infusion need of >10 mg/kg/min, or repeated unexplained hypoglycaemia.

HYPOTHYROIDISM

This is usually due to an absent or hypoplastic thyroid gland. Deficiencies of thyroid hormone synthetic enzymes (resulting in a goitre), hypopituitarism (with hormonal deficiencies), and autoimmune thyroiditis (presenting in later childhood) also occur. Congenital hypothyroidism occurs throughout the world with an incidence of about 1/4000 births.

Fewer than 1 in 3 affected children have obvious signs at birth, and signs become more marked with time. Affected children are lethargic and floppy, with a large protruding tongue, delayed development, constipation, bradycardia, and often a protuberant abdomen with an umbilical hernia, a hoarse cry, and puffy features. Prolonged neonatal jaundice is common and should raise suspicion. Later, myxoedematous features develop and growth failure and intellectual delay is profound. Children are short, and bone age is delayed (the lower femoral and upper tibial epiphyses should be present in normal children by birth, but their appearance is delayed in hypothyroidism).

In primary hypothyroidism (all gland problems), the serum thyroxine is low and TSH markedly elevated. Neonatal screening programmes in many countries have demonstrated that early thyroid hormone replacement is associated with good neurological outcome. Each month of delayed treatment from birth causes further neurological impairment. With no newborn screening as yet in PNG, it is important to have a high level of suspicion. Therapy should be commenced immediately the diagnosis is proved or highly suspected and blood for TFTs is taken. If blood cannot be collected or sent, treat anyway, and at a later stage stop treatment for one month and do the TFTs at the end of this month. Treatment should be begun immediately with thyroxine in a dose of 50 microgram/day (one half a tablet) if aged 0-6 months and 75microgram ($\frac{3}{4}$ tablet) if >6 months. After two weeks the dose can be dropped to a maintenance dose of 100 microgram/m² (generally 25-50 microgram (0-6 months), 50-75 microgram (6-12 months), 75-100 microgram (1-5 yr)). Dose is adjusted to maintain normal growth, clinical euthyroidism, and normal TFTs.

Endemic cretinism - see p.94.

IMMUNISATION

NOTES

Immunisation of children against infectious disease is a highly cost effective intervention. It is an integral part of Papua New Guinea Health Policy. It is everyone's duty to ensure that all children are vaccinated. The days when immunisation was left to the MCH services are long since gone. Every health facility and every health worker should ensure that children in their care are fully immunised.

The child's health record book should always be examined. If the child is due for vaccination, this should be given, unless there is a specific contraindication (the only common contraindication is a fever of $>38^{\circ}\text{C}$ when triple antigen, hepatitis B and pigbel should be deferred. Fever is NOT a contraindication for measles vaccination. The only vaccine contraindicated in children with HIV infection is BCG - and then only when the child has AIDS).

Immunisation should be regarded as part of the child's admission procedure. Failure to immunise the child may result in severe consequences. Hospital-acquired measles may be fatal, as may hospital-acquired whooping cough - particularly in a child who is already sick.

The Papua New Guinea Immunisation Policy (see chart) is, by generally accepted practice, very aggressive. TA and Sabin are given at monthly intervals from the age of one month (many countries use a schedule of 2, 4 and 6 months) and measles vaccine at 6 and 9 months (many countries give a single dose at a year or 15 months). THERE ARE SOUND PRACTICAL AND EPIDEMIOLOGICAL REASONS FOR THE PNG POLICY. The schedule should be followed as far as possible. On the other hand, immunisation policy is "opportunity based" and should be flexible.

Four doses of sabin vaccine are given. This is part of the Polio Eradication by 2000 strategy.

BCG, Sabin and hepatitis B should be given as soon after birth as possible. They should ideally be part of labour ward routine (giving the hepatitis B vaccine prevents perinatal hepatitis B transmission, and hence greatly reduces the possibility of the child becoming a chronic hepatitis carrier with its associated morbidity and mortality).

Doctors must be actively concerned about the maintenance of the COLD CHAIN. They must therefore ensure that the ward/health centre vaccine storage facilities are adequate and efficient.

NEW VACCINES. It is probable that at least one new vaccine - the Haemophilus influenzae type B conjugate vaccine will be introduced within the next 5 years. This will almost certainly be given together with TA.

IMMUNISATION CHART

Always check the expiry date on the ampoule or vial. Never use expired vaccine.

Keep all vaccines in the main compartment of the refrigerator (temperature 2-8 °C), not in the freezer. Only ice packs are kept in the freezer compartment.

Do not use alcohol or an alcohol swab to clean the skin before giving any injection. Use only cool boiled or distilled water for this purpose.

- On patrol:
- pack vaccines with ice packs in a vaccine carrier
 - if the ice packs have melted by the end of the day, all remaining vaccines, whether opened or not, should be discarded
 - if ice packs are still partly frozen by the end of the day, put all unopened vials back in the refrigerator and use these first at the next immunisation session. Write the date on the vials before returning them to the refrigerator.
 - if ice packs are still partly frozen, all opened vials should be discarded at the end of the day, whatever amount of vaccine they contain.

VACCINE	WHEN GIVEN	DOSE	ROUTE
BCG	As soon as possible after birth	0.05 ml	Intradermal into left upper arm
<p>BCG vaccine is best given at birth. Unvaccinated children between 1 and 5 years of age should also be given 1 dose of this vaccine, but use the child dose which is 0.1 ml instead of the infant dose. Only one dose is given per child, no booster doses are needed.</p> <p>When diluted, protect vaccine from heat and light and use within 6 hours. Discard reconstituted vaccine at the end of each immunisation session.</p>			
HEPATITIS B VACCINE	1 st dose: As soon as possible after birth 2 nd dose: At least one month after 1 st dose (with 1 st TA/OPV). 3 rd dose: At least two months after 2 nd dose (with 2 nd or 3 rd TA/OPV)	0.5 ml (10 mcg) “ “	Intramuscular into left thigh
<p>The first dose of Hepatitis B vaccine is best given at birth and every child must receive a full course of three doses. Unvaccinated children between 1 and 5 years of age should also be given 3 doses of this vaccine, but use the child dose which is 0.5ml (10 microgram) instead.</p> <p>At the end of each immunisation session return the opened Hepatitis B vial with the remaining vaccine to the refrigerator. It can be used within the next 5 days.</p>			
TRIPLE ANTIGEN (TA/DPT)	1 st dose: One month of age 2 nd dose: One month after 1 st dose 3 rd dose: One month after 2 nd dose	0.5 ml “ “	Intramuscular into right upper arm.
<p>Every child must receive a full course of three doses of DPT. At the end of each immunisation session return the opened TA vial with the remaining vaccine to the refrigerator. It can be used within the next 5 days.</p>			

VACCINE	WHEN GIVEN	DOSE	ROUTE
ORAL POLIO VACCINE (OPV/Sabin)	1 st dose: As soon as possible after birth 2 nd dose: At one month of age 3 rd dose: One month after 2 nd dose 4 th dose: One month after 3 rd dose	2 drops “ “ “	Orally at the back of the tongue
At the end of each immunisation session return the opened Polio vial with the remaining vaccine to the refrigerator. It can be used within the next 5 days.			
PIGBEL VACCINE	1 st dose: At one month of age 2 nd dose: One month after the 1 st dose 3 rd dose: One month after the 2 nd dose 4 th dose: First year community school 5 th dose: Last year community school	0.5 ml “ “ “ “	Intramuscular into right thigh
At the end of each immunisation session return the opened Pigbel vial with the remaining vaccine to the refrigerator. It can be used within the next 5 days.			
MEASLES VACCINE	1 st dose: At six months of age or as soon as possible thereafter 2 nd dose: At nine months of age or 3 months after the first dose	0.5 ml “	Subcutaneous into right upper arm.
When diluted, protect vaccine from heat and light and use within 6 hours. Discard reconstituted vaccine at the end of each immunisation session.			
TETANUS TOXOID	1 st dose: First year community school 2 nd dose: Last year community school 3 rd dose: During pregnancy (2 doses 4 weeks apart in the first pregnancy, 1 dose in each of the next pregnancies, maximum 5 doses)	0.5 ml “ “	Intramuscular into left upper arm.
At the end of each immunisation session return the opened TT vial with the remaining vaccine to the refrigerator. It can be used within the next 5 days.			

1. The above mentioned doses are recommended for the vaccines currently available in PNG, but in all cases the manufacturer's leaflet should be checked for the recommended doses.
2. Don't forget to check whether the patient's brothers and sisters and mother need immunisation also.
3. It is important to check the immunisation status of every child that you see in any clinic and to give any vaccines due immediately without referral to the immunisation clinic. The only vaccines ever to be withheld are Triple Antigen, Hepatitis B and Pigbel if the child has a fever of more than 38 °C. In such cases, the vaccine is only temporarily withheld while the fever persists. As soon as the child's temperature returns to normal, continue and complete the normal course of immunisation with these vaccines.
4. You must always immunise a child even though you may have to open a new vial for only one child. If necessary, order more vaccine to make sure you have sufficient for all children expected at the clinic, plus a reserve.

REFERENCES

- Aaby P, Samh B, Simonden F et al. *Br Med J* 311:481-485,1995. Non-specific beneficial effect of measles immunisation: analysis of mortality studies from developing countries.
- Combs BG, Lehmann D. *PNG Med J* 40(2):63-68,1997. Maternal Immunisation.
- Cutts FT, Steinglass R. *Br Med J* 316:765-767,1998. Should measles be eradicated?
- Cutts FT. Measles. In: *The immunological basis of immunisation*, Ch 7. Geneva: WHO, 1993.
- Eskola J, Anttila M. *Pediatr Infect Dis J* 18(6) 543-551,1999. Pneumococcal conjugate vaccines.
- Hall AJ, Cutts FT. *Br Med J* 307:1294-1295,1993. Lessons from measles vaccination in developing countries.
- Kristensen I, Aaby P, Jensen H. *Br Med J* 321:1435-1439,2000. Routine vaccination and child survival: follow up study in Guinea Bissau, West Africa.
- Linda D, Deeks J. *Br Med J* 321:931-933,2000. Effect of needle length on incidence of local reactions to routine immunisation in infants at 4 months: randomised controlled trial.
- Mulholland K, Hilton S, Adegbola R et al. *Lancet* 349:1191-1197,1997. Randomised trial of *Haemophilus influenzae* type B-tetanus protein conjugate vaccine for prevention of pneumonia and meningitis in Gambian infants.
- Semba RD, Munasir Z, Beeler J et al. *Lancet* 27:1330-1332,1995. Reduced seroconversion to measles in infants given vitamin A with measles vaccination. (Editorial commentary by Ross D, p. 1317-1318).
- Shann F. *PNG Med J* 43(1-2):24-29,2000 (in press). Immunisation: dramatic new evidence.
- Shann F. *Br Med J* 321:1423-1424,2000. Non-specific effects of vaccines in developing countries.
- Shann F, Steinhoff MC. *Lancet* 354(suppl 11):7-11,1999. Vaccines for children in rich and poor countries.
- World Health Organisation Report on the meeting on maternal and neonatal pneumococcal immunisation. Geneva: WHO, 1998.

INFANT FEEDING

See also Milk Feeds (p.230).

BREASTFEEDING

There is incontrovertible evidence that breastfeeding is the best way to feed babies:

- it is safe, simple and cheap
- breast milk is nutritionally designed specifically for the human infant
- breast milk is part of the baby's defence system, containing a battery of anti-infective substances, as well as living cells
- breast milk contains factors which optimise the growth of the developing human brain
- breastfeeding protects against the subsequent development of some "allergic" disorders (such as asthma)
- breastfeeding is a form of community family spacing.

ARTIFICIAL FEEDING

There is incontrovertible evidence that artificial feeding is dangerous, unless the mother has sufficient money, education and time to buy and prepare the milk cleanly and in the right strength. Every effort should be made to discourage artificial feeding. In particular, every effort should be made to discourage the use of feeding bottles, dummies and feeding cups. If artificial feeding is used, CUP AND SPOON feeding is the preferred method. Babies adapt quickly to this method.

The Baby Feed Supplies (Control) Act of 1977 and its amendment of 1984 aimed to protect Papua New Guinean children from the dangers associated with bottle feeding and to protect the practice of breastfeeding. Under this law:

1. Feeding bottles, teats, dummies and feeding cups can be obtained only from a registered pharmacy.
2. The mother or guardian must produce a prescription (authorisation).
3. Only a medical practitioner, HEO, nurse or nursing aide can write a prescription.
4. Before writing a prescription, the health worker must do the following:
 - a. Be satisfied it is in the baby's interest.
 - b. Instruct the mother or guardian how to clean the bottles properly (clean with a bottle brush and boil in clean water for 5 minutes or soak in sterilising solution).
 - c. Instruct the mother or guardian to keep the milk in a refrigerator if it is not immediately used.
 - d. Instruct the mother or guardian how to mix the milk in the correct strength.

Penalties (200K fine for a first offence and 500K for a second offence) can be imposed on health workers who do not follow these instructions, on unregistered outlets, or on registered outlets supplying bottles or cups without prescription.

This law has been only partially effective. A recent survey indicated that it is common for mothers to obtain feeding bottles and feeding cups without prescription from unregistered outlets. The Paediatric Society of Papua New Guinea is attempting through the Health Department to tighten the legislation and its implementation.

It is important that commercial premises selling bottles or feeding cups illegally should be reported to the Health Department Family Health Services and the Provincial Health Inspector.

The PNG Health Department has an Infant Feeding Policy which strongly advocates breastfeeding, urges the discouraging of bottle (and baby cup) feeding and indicates that bottle feeding should not be used in any health institution.

If a mother asks for a prescription for a bottle:

1. Try and find out why she wants to use a bottle.

2. Explain that bottle feeding is “second best” and puts the child at risk.
3. Encourage her to continue to breastfeed.
4. If she is going back to work suggest that she can express her milk for the baby and point out she is entitled to breastfeeding breaks (though facilities at most workplaces are non-existent or woefully inadequate). She should continue to breastfeed her baby before and after work.
5. If breastfeeding is not possible, discuss the other options and suggest that she use CUP and SPOON rather than a bottle.
6. Make sure (ideally by demonstration) that she knows how to make up the milk and the importance of cleaning all the utensils thoroughly.
7. If she is adopting the child, discuss the possibility of lactation induction (see p.182).
8. Encourage the mother to attend the clinic for regular weighing of her baby.

What milks are available?

1. Milks with vitamins and iron added: the usual ones are Lactogen, SMA, S26 and Enfamil. These are all effective; Lactogen is the cheapest.
2. Full cream milk without vitamins and iron: common brands are Farm Fresh, Sunshine, Anchor and Pacific. Vitamins and iron have to be given as well.

Vitamins: Pentavite is the cheapest. Other brands are Abdec and Poly Visol. Give 10 drops each day.

Iron: The Health Department supplies ferric ammonium citrate mixture and sometimes ferrous fumarate. Infant Fergon is the brand sold by chemists. Give 2 mg/kg elemental iron per day.

3. Do NOT use skim milk or low fat milks.

Which milk mixture to use

For babies artificially fed at home, the best milk to use is one containing added iron and vitamins. For babies artificially fed in hospital or health centres, it is best to use full cream milk (eg Sunshine, Pacific, Farm Fresh, Anchor) with 10 drops a day of vitamin mixture (eg Pentavite, Abdec) and 2 mg/kg/day elemental iron.

How to make up the milk

(See also p.231)

1. **For babies under 2 weeks old**

Use sugar-milk (Sunshine, Pacific or Anchor Instant milk): Mix 1 part by volume (eg cup or plastic medicine glass) of milk powder and half a part by volume of sugar with 6 parts by volume of cool, previously boiled water

2. **For babies aged 2 weeks or more**

Use full strength (Sunshine, Pacific or Anchor Instant milk): Mix 1 part by volume (eg cup or plastic medicine glass) of milk powder with 3 parts by volume of cool, previously boiled water .

If the milk cannot be kept cold in a refrigerator, only make enough milk for one feed at a time.

Milks used for special purposes

(See p.231)

Milk oil formula (MOF)

For malnourished children. Add 10 ml vegetable (eg peanut) oil, 2 heaped teaspoons of sugar and 5 ml of electrolyte mixture to 240 ml of full strength milk.

Low lactose milks (Digestelact or Nutramigen)

Used for children with diarrhoea caused by sugar (lactose) intolerance. Mix 1 part by volume of Digestelact or Nutramigen powder with 4 parts by volume of cool, previously boiled water.

Others

eg Infasoy, Pregestemil, Triglyde or Ensure (a high calorie, no lactose milk substitute) may be available. Follow the instructions on the tin.

EDUCATIONAL DIET

Start to give the baby mashed up food as well as milk by the age of 4 months. Feed the baby with a spoon. Breast milk by itself is not sufficient food for a baby after the age of 6 months. Breastfeeding should continue as long as culturally accepted. Even at 2 years of age, breast milk supplies a useful protein supplement. Malnutrition is often due to solid food being given too late and not often enough.

KEEP YOUR MESSAGE TO PARENTS SIMPLE:

1. Start giving solid food when your baby is 4 months old. If you do not know how old he is, start when he is getting his first tooth
2. Give:
 - a. local staple (eg kau kau) and dark green leaves
 - b. mix in a spoonful of dripping, vegetable oil or margarine
 - c. "as bin" (wing bean) or mashed up peanuts or some animal protein (eg fish, meat or chicken)
3. Try and give solid food to your child 4-6 times every day, including snacks, eg banana, avocado, coconut, pawpaw.

ARTIFICIAL FEEDING AT HOME

It is occasionally necessary for a baby to be fed artificially at home, eg if the child is adopted, or if the mother has died (but see Lactation Induction, p.182).

You must have the mother or guardian SHOWN how to make up a milk feed; do not just tell them how to do it. It is a good idea to have the notes that follow printed so that if the family is educated you can give them a copy AS WELL AS showing them what to do.

REFERENCES

- Cameron M. Manual on feeding infants and young children. 3rd ed. OUP, 1983.
- Friesen H et al. (Paediatric Society of Papua New Guinea) Survey to assess the current feeding practices in infants and children below 2 years of age in selected areas of Papua New Guinea. Report to Dept of Health 1996.
- Friesen H et al. Annals Trop Paed Int Child Health 18:209-215,1998. Infant feeding practices in Papua New Guinea.
- Friesen H, Vince J, Boas P, Danaya R. Bull WHO 77(3):271-274,1999. Protection of breastfeeding in Papua New Guinea.
- Helsing E. Breast-feeding in practice. OUP, 1982.
- Jelliffe DB, Jelliffe EFP. Human milk in the modern world. OUP, 1978.

This page can be copied and given to literate parents to take home (but SHOW them what to do first):

ARTIFICIAL FEEDING AT HOME

BREASTFEEDING IS BEST. Only use artificial feeding if you really cannot breastfeed. Use a cup and spoon, NOT A BOTTLE. In most cases, it is best to use a milk with iron and vitamins already added, such as Lactogen, S-26 or Enfamil.

How much milk to give:

Give babies as much as they want as often as they want. Most babies take 5-6 feeds every day.

A guide to how much milk to give with each feed is to multiply the baby's weight in kg by 30 (maximum 240 ml per feed). A baby weighing 5 kg would be offered $5 \times 30 = 150$ ml each feed 5 - 6 times a day.

As the baby takes as much milk per feed as he or she wants, there should always be a little milk left over after each feed. So if the baby finished all the feed, offer a little more next time.

How to make up the milk (for babies aged 2 weeks or more):

(Using Sunshine, Pacific or Anchor INSTANT milk).

Mix EXACTLY 1 part by volume (eg cup or plastic medicine glass) of milk powder with EXACTLY 3 parts by volume of cool, previously-boiled water. Do NOT pack the milk powder down tightly into the cup when measuring it out.

Weight of baby	Milk powder	Boiled water
2 kg	15 ml	45 ml
3 kg	20 ml	60 ml
4 kg	25 ml	75 ml
5 kg	30 ml	90 ml
6 kg	40 ml	120 ml
7 kg	45 ml	135 ml
8 kg	50 ml	150 ml
9 kg	50 ml	150 ml
10 kg	50 ml	150 ml

IF THE MILK CANNOT BE KEPT COLD IN A REFRIGERATOR, ONLY MAKE ENOUGH MILK FOR ONE FEED AT A TIME.

How to make a cup and spoon feed (baby aged 2 weeks or more):

(Using Sunshine, Pacific or Anchor INSTANT milk).

Boil water to make the feed. Let it cool.

Measure one part of milk powder to 3 parts of water.

Beat the milk with the spoon to dissolve the powder.

Feed the baby with the cup and spoon.

Do NOT keep milk from one feed to the next.

Wash the cup, spoon and plastic measuring glass very well - then boil them for 5 minutes in a covered pot (timing starts from when the water boils after putting in the utensils).

Store them in the empty covered pot.

IF USING LACTOGEN, S26, OR ENFAMIL - MAKE SURE YOU READ THE INSTRUCTIONS ON THE TIN.

SOLID FOOD

Start to give the baby mashed up foods as well as milk after the age of 4 months. Feed the baby with a spoon.

Locally available foods should be used. Start with mashed banana, pawpaw, kau kau, taro or other soft food. As the baby gets used to the taste of one food begin another food. By 8 months of age, the baby will usually hold a lump of food in his hand and chew on it. By 12 months of age, the baby should be eating the same kinds of food as the rest of the family. He or she should have at least 3 meals a day. Mix a spoonful of dripping with each feed.

It is important to give a mixed diet - one or more foods from each group at some time during the day:

Staple - kau kau, taro, yam, sago, rice or bread

Legumes and pulses - peas, beans (especially "as bin"), peanuts

Dark green leafy vegetables - aibika, pumpkin tops or sweet potato tops

Animal protein - meat, fish, eggs, soft insects (caterpillars, sago grubs)

Fruit or yellow vegetables - pawpaw, pumpkin, banana, pineapple, coconut

Oil, margarine, fat or dripping.

INTRAOSSEOUS INFUSION

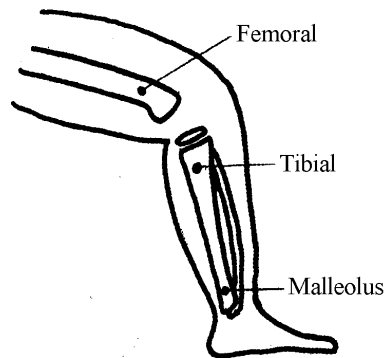
It is possible to give parenteral fluid rapidly into the bone marrow. In fact, the marrow cavity is an intravascular compartment, so the fluid is actually being given by intravenous infusion. This route is preferable to intraperitoneal infusion, since it is quicker and can be used in shocked patients.

Intraosseous infusion can be used for:

- circulatory collapse from severe dehydration, haemorrhage, burns, trauma and allergy.
Intraperitoneal fluid is not absorbed in shocked patients
- when no veins are available because of prior use, obesity, oedema or burns.

Viscous fluids such as whole blood often do not run in rapidly by this route, and may have to be pumped in. Ordinary IV fluids will run rapidly without pumping.

1. The child is held firmly, lying on one side.
2. Scrub your hands.
3. Choose the site for infusion. In children up to 2 years of age the usual sites are 2 cm below the tibial tuberosity (probably the easiest), or the junction between the middle and lower third of the femur. In children up to the age of 5 years, the medial malleolus of the tibia is suitable, whilst for all ages the superior iliac crest can be used (see Bone Marrow Aspiration, p.57).



4. Clean the skin around the puncture site with iodine.
5. If the child is conscious, infiltrate the skin, subcutaneous tissues and periosteum with 1% plain lignocaine.
6. If available, use the specially designed intraosseous infusion needle and stylet. If this (or a bone marrow biopsy needle) is not available, use a standard size 18 or 20 needle. Push the needle through the skin in a direction slightly away from the joint if the upper medial tibial or malleolar sites are being used. When the needle hits bone, rotate the needle back and forward, applying more pressure until there is a definite "give" with decreased resistance indicating that the needle has entered the bone marrow cavity.
7. If using a needle and stylet, remove the stylet. Aspirate a small amount of bone marrow to check the position of the needle. Then use a syringe to push in 5ml of infusion fluid to clear the end of the needle. Now connect up the giving set. You may find that the drip slows with time. In this case use a syringe to re-flush the needle.

REFERENCES

Practical procedures - circulation. Chap 23, p.221-232. In: Advanced paediatric life support. The practical approach. 3rd ed. London: BMJ Books, 2001.

INTRAVENOUS CATHETERS

See also Cut Down (p.97), Central Venous Catheterization (p.68) and Intraosseous Infusion (p.171).

SCALP VEIN NEEDLE

This is the normal route of intravenous infusion. The best sites are the back of the hand, the long saphenous vein on the medial side of the ankle, and scalp veins.

1. Put a firm tourniquet on the limb. Do not make it too tight - make sure you can still feel the radial pulse at the wrist, or the tibial pulse at the foot. If you are using a scalp vein, put a rubber band around the head, but make sure that it is not too tight.
2. Clean the skin thoroughly with 70% alcohol (SVM).
3. WITHOUT TOUCHING THE PUNCTURE SITE WITH YOUR FINGERS, carefully put the needle into the vein.
4. Tape the needle in and splint the limb.

DWELLCATH/CANNULA

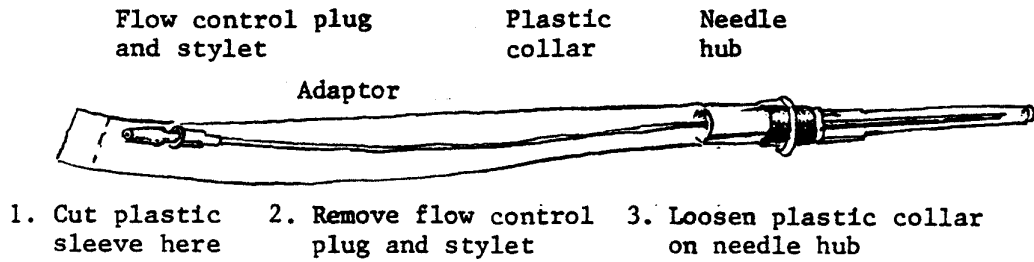
These are more expensive than scalp vein needles but last a lot longer. Caps or stoppers are available - but a cannula left in situ for more than 2 to 3 days is a potential site of infection.

INTRACATH

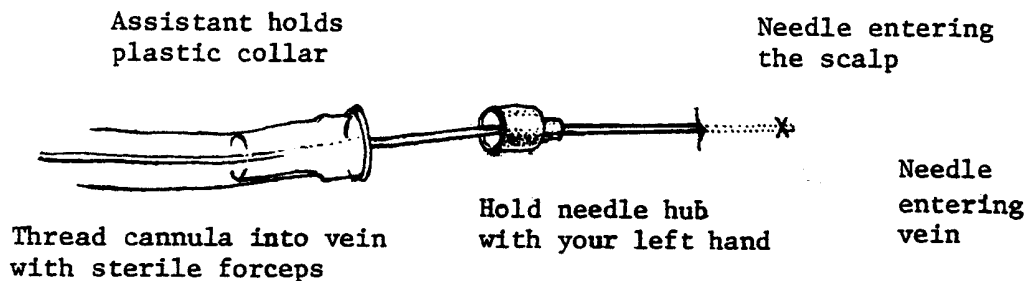
Intracaths are more expensive than scalp vein needles and cannulas. They cost about the same as a cutdown cannula. They should only be used in special situations: when intravenous fluids will be needed for a long time, or when irritant solutions (eg 10% dextrose and Aminofusion) are being infused. Because they are flexible and can be threaded into large proximal veins, Intracaths will often run for many days. They are certainly more convenient, and may in practice be cheaper than multiple scalp vein drips. They have a much lower incidence of sepsis than a cutdown, do not destroy the vein, and cost the same amount.

Good sites for insertion of an Intracath are the back of the hand, the long saphenous vein and the basilic vein on the medial side of the cubital fossa at the elbow. A particularly useful site for prolonged infusions in small children is a scalp vein, because a blue Intracath (22 gauge catheter) can often be threaded down to the external jugular or subclavian vein from this site:

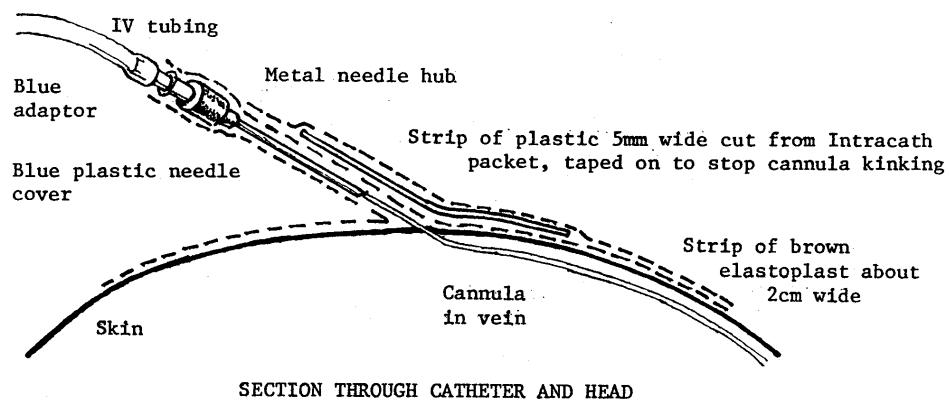
1. Have an assistant hold the child's arms and body, and another assistant hold the head.
2. Shave the scalp above and in front of the ear on one side. Clean the scalp with 70% alcohol (SVM).
3. Place a rubber band around the head just above the ears just tight enough to occlude the veins, but not tight enough to occlude the arteries.
4. Place a pair of sterile forceps where you can easily reach them with your right hand.
5. Take an eight inch blue Intracath (22 gauge catheter, Cat. No.3166, Deseret Pharmaceuticals Inc., 9450 South State Street, Sandy, Utah 84070, USA):
 - a. loosen the plastic collar on the needle hub
 - b. cut off the end of the plastic sleeve and remove the flow control plug and stylet.



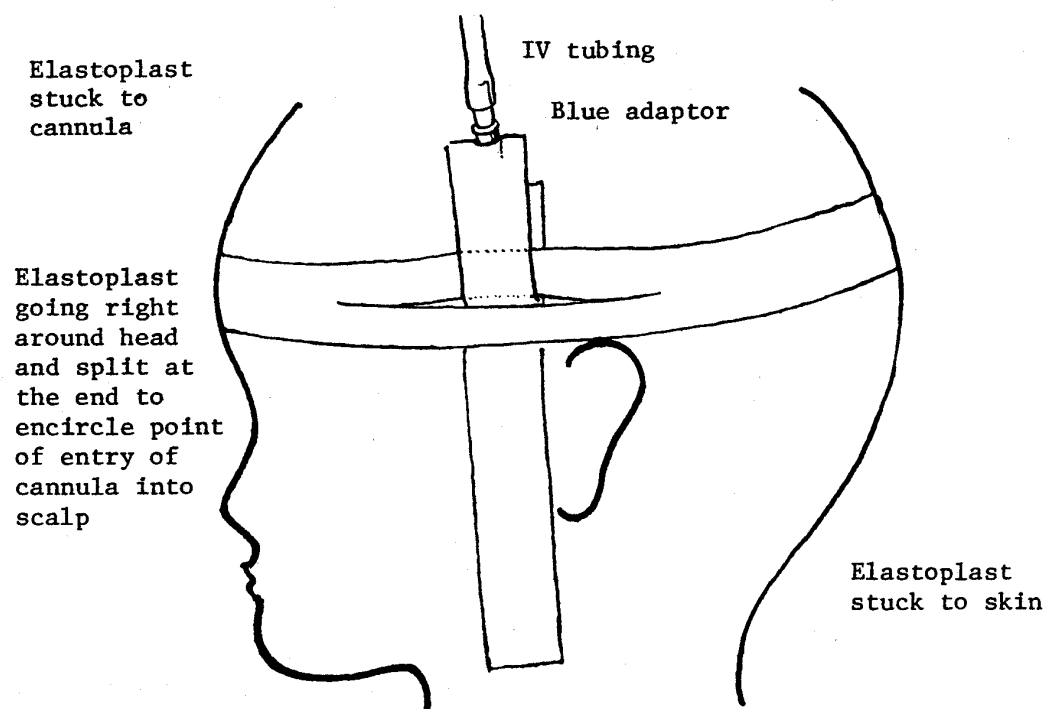
6. WITHOUT TOUCHING THE PUNCTURE SITE OR NEEDLE WITH YOUR FINGERS, carefully push the needle into the vein. Blood may not run back up the fine 22 gauge cannula, but you will often be able to see the scalp vein collapse as your needle enters it.
7. Hold the hub of the needle firmly in your left hand, and CAREFULLY pull the plastic collar back about 2 cm from the needle hub with your right hand. Get an assistant to hold the plastic collar in this position. Still holding the needle with your left hand, thread the cannula CAREFULLY down the vein using the sterile forceps in your right hand.



8. The cannula may stick after threading several centimetres. Try infusing fluid through it and threading GENTLY again. Do not push too hard, or you may push it through the wall of the vein.
9. Holding the cannula in place with the sterile forceps, carefully slide the needle back over the catheter and out of the scalp and push it firmly into the adaptor (do not kink the catheter inside the needle hub). Have an assistant press over the venepuncture site with a dry swab to prevent bleeding.
10. Place the needle cannula in the channel of the blue bevel cover (make SURE the catheter is in the channel) and snap the cover closed.
11. Strap the cannula in place. Put strips of Elastoplast 2 cm wide running from the blue adaptor over the needle hub and along the cannula onto the skin, one strip on each side of the catheter. Then cut a flat strip of plastic 5 mm wide from the Intracath packet, and tape it on to stop the cannula kinking.



12. Put a strip of brown Elastoplast 2 cm wide running from the forehead across the place the cannula enters the skin, to the occiput, and right around the head. Then split the last 15 cm in two, to encircle the point of entry of the cannula.



13. Although Intracaths will often run for many days (or even weeks), their use for long periods carries a high risk of septicaemia. An Intracath that has been in more than 24 hours **MUST** be removed from any child that develops an unexplained fever.

INTRAVENOUS PYELOGRAM

1. Give Coloxyl 50 mg tabs the night before.

Under 10 kg:	1 tab
10-20 kg:	2 tab
Over 20 kg:	4 tab

2. If a child weighs more than 10 kg, he should be fasted overnight. Do NOT fast small babies.
3. The patient voids, then a plain abdominal film is taken BEFORE any dye is injected.
4. SLOWLY inject 76% Urografin IV. Be prepared for an anaphylactic reaction (p.29).

Under 10 kg:	3 ml/kg (maximum 20 ml)
10 - 19 kg:	20 ml
20 - 29 kg:	30 ml
30 kg or more:	40 ml

5. It is not worth attempting to do an IVP if the urea is over 35 mmol/l. Abdominal compression is not required in children.
6. Take films of the renal areas 5 and 10 minutes after injecting the dye.
7. Take a further abdominal film after micturition at 15 minutes.

JAUNDICE IN CHILDREN - SUMMARY

HAEMOLYSIS OR HEPATITIS

Although testing the urine for bile usually distinguishes between haemolysis (no bile) and hepatitis (bile present), further tests are occasionally needed in difficult cases (eg when both are present together):

- urine for bile and urobilinogen
- Hb, reticulocytes
- serum bilirubin (direct and indirect)
- serum ALT.

Haemolysis is suggested by:

- a stable or falling Hb level with reticulocyte count 2% or more in the absence of blood loss
- a lack of bile in the urine
- increased urobilinogen in FRESH urine (but this may be normal)
- a raised total serum bilirubin (over 17 mmol/l) which is mainly due to an increase (over 12 mmol/l) in indirect or unconjugated bilirubin. There may be a small rise in direct or conjugated bilirubin (normal 2-5 mmol/l)
- an ALT less than 30 U/ml (in children aged 1-16 yrs).

Hepatitis is suggested by:

- the presence of bile in the urine
- a normal urinary urobilinogen, except in severe hepatitis
- a raised total serum bilirubin (over 17 mmol/l) with an increase in direct or conjugated bilirubin (normal 2-5 mmol/l) as well as indirect or unconjugated bilirubin (normal 3-12 mmol/l)
- an ALT of more than 30 U/ml (aged 1-16 yrs).

OBSTRUCTIVE JAUNDICE

This is rare in children, so it is not discussed here in detail. The urine contains bile with no urobilinogen, direct and indirect serum bilirubin are high, ALT is normal (under 40 U/ml) or moderately raised, and the serum alkaline phosphatase is raised. It is sometimes hard to distinguish between obstructive jaundice and hepatitis.

Obstructive jaundice does occur in neonates as a result of biliary atresia or more rare congenital causes of obstruction of the biliary system.

JAUNDICE IN CHILDREN - HAEMOLYSIS

See also Neonates - Jaundice (p.254)

In a jaundiced child, test the urine for bile:

- present = hepatitis
- absent = haemolysis

A JAUNDICED CHILD WITH NO BILE IN THE URINE HAS HAEMOLYSIS. Give a course of antimalarials (after taking blood slide).

TESTS

- Blood slide
- FBE and reticulocytes
- G6PD screen (but this may be falsely negative during a haemolytic crisis)
- Blood culture if the child looks sick
- Crossmatch packed cells if the Hb is less than 6 g/dl.

1. **Malaria**

Give a course of antimalarials. Give treatment for severe malaria if the child is very ill.

2. **Gram negative sepsis**

If the child looks sick, give IV chloramphenicol OR ampicillin and gentamicin without waiting for the blood culture result.

3. **Autoimmune haemolytic anaemia**

A positive Coombs test suggests the presence of auto-antibodies against red cells. Give prednisolone. Auto-antibodies may be secondary to malaria, sepsis, tuberculosis (NEJM 299:488,1978), lymphoma, penicillin or methyldopa.

PROGRESS

Monitor twice a week:

- Hb: it should rise.
- Reticulocytes: they should gradually fall.

A stable or falling Hb with a reticulocyte count of 2% or more suggests persistent haemolysis.

If haemolysis persists or recurs:

- Do a Hb electrophoresis for thalassaemia (which is common in malarious areas), and a G6PD screen (but this may be falsely negative during a haemolytic crisis). These tests should not be done for 3 months after a blood transfusion.

JAUNDICE IN CHILDREN - HEPATITIS

In a jaundiced child, test the urine for bile

- present = hepatitis
- absent = haemolysis

HEPATITIS

This usually presents as a jaundiced child with bile in the urine. Take a blood slide and give antimalarials for 3 days. Most children with hepatitis can be treated as outpatients. Only admit children who look ill.

If a child is admitted because he looks ill, do a:

- blood slide
- blood culture
- chest x-ray
- Hb, WCC, and reticulocyte count. A reticulocyte count of more than 2% suggests that the child has haemolysis as well as hepatitis.

Pneumonia

Pneumococcal pneumonia is a common cause of jaundice in Papua New Guinea particularly in older children and adults.

Septicaemia

This is a serious cause of jaundice from both hepatitis and haemolysis. There may be no fever. Any child with jaundice who looks ill should have a blood culture taken, and IV chloramphenicol OR ampicillin and gentamicin commenced. Do not wait for the result of the blood culture before starting antibiotics.

Malaria

Always treat any jaundiced patient with antimalarials. Patients may have another cause for jaundice in addition to having malaria. Malaria itself can cause hepatitis.

Viral hepatitis

This is a common cause of hepatitis, but the above causes must be excluded before making this diagnosis in a child that looks ill. There is a high incidence of G6PD deficiency in patients with viral hepatitis (these patients may have hepatitis AND haemolysis). Hepatitis A is spread by the faecal-oral route. It is endemic in Papua New Guinea and usually causes a mild illness - though it may also present with a fulminating course. A vaccine is available but is not currently routinely used in the country. Hepatitis B acquired perinatally or by blood/serum contamination in early infancy is likely to be associated with chronic carriage and an increased risk of cirrhosis and hepatoma in adult life. It should be prevented by immunisation (see p.163).

Amoebiasis

Amoebic liver abscess may cause mild jaundice. This is rare. There is usually fever, very painful hepatomegaly and leucocytosis. Chest X-ray may show distortion of the right diaphragm or a pleural effusion. Treatment is with metronidazole or tinidazole. If the abscess is very large or on the point of rupture, needle aspiration under ultrasound guidance is indicated. Other causes of a mass in the liver are pyogenic abscess, tuberculoma and hepatoma.

Drugs

Dapsone, rifampicin, isoniazid, pyrazinamide, chlorpromazine, phenytoin, halothane, methyl dopa, sulphonamides and hydrochlorothiazide may cause jaundice.

Chronic familial jaundice

This is common in some regions of developing countries. See PNG Med J 12:128-129,1969.

REFERENCES

Sapuri M, Babona D, Klufio CA, Vince JD. PNG Med J 34(4):234-237,1991. Hepatitis B surface and e antigen seropositivity in mothers and cord blood at Port Moresby General Hospital: implications for a control programme.

Nemba K, Babona D, Vince JD. Annals Trop Paed/Int Child Health 13:237-241,1993. Age specific prevalence of hepatitis B surface antigen in hospitalised children at Port Moresby, Papua New Guinea.

JOURNALS

Consider getting the following journals sent to you:

- The Papua New Guinea Medical Journal
- ARI News (a newsletter about acute respiratory infections), 85 Marylebone High Street, London W1M 3DE, England (free)
- Bulletin International Union Against Tuberculosis, 3 rue Georges Ville, 75116, Paris, France (quarterly, free to doctors in developing countries)
- Diarrhoea Dialogue, 85 Marylebone High Street, London W1M 3DE, England (quarterly, free)
- Pediatric Alert, PO Box 338, Newton Highlands, MA 02161, USA. A fortnightly 4 page summary of important paediatric articles
- Salubritas, 1015 Fifteenth Street, NW, Washington, DC 2000J, USA. Published by the American Public Health Association for doctors in the developing world (quarterly, free)
- TALC Newsletter, TALC, PO Box 49, St Albans, Herts AL1 5TX, UK. Fax: 054 41 727 846852. E-mail: talc@talcuk.org. Website: www.talcuk.org (occasional, free)
- The Lancet, 7 Adam Street, London WC2N 6AD, England. The best coverage of medicine in developing countries of the major medical journals
- Tropical Doctor, Academic Press Ltd., 24-28 Oval Road, London NW1 7DX, England (quarterly, US\$40 seamail)
- Current Opinion in Pediatrics, Rapid Science Publications, 2-6 Boundary Road London SE1 8HN, England. 6 issues per year covering the range of “western” paediatrics. Expensive (about 500 Kina/year) but reasonable value.
- Paediatrics Current Medical Literature Royal Society of Medicine. Carfax Publishing Company, PO Box 352, Cammeray, NSW 2062, Australia. About 75 Kina/year, reasonable value.

The medical library will send you photocopies of the Table of Contents of a selection of medical journals, so that you can ask them to send you copies of interesting articles (for example, you might ask to be sent the contents pages of Annals Trop Pediatr, Arch Dis Child, J Trop Paediatrics, Lancet, Brit Med J, J Pediatr, New Eng J Med and Pediatrics).

Although not readily available to everyone yet, access to the Internet allows access to Medline and to journal home pages. Several hospitals should already have Internet access. A very useful web site is www.healthinternetwork.net. This site gives free access to over 100 medical journals online.

LACTATION - DRUGS CROSSING INTO BREAST MILK

On rare occasions, drugs given to a mother may affect her breast-fed baby:

1. Drugs known to affect the baby:

- large amounts of alcohol
- narcotics (withdrawal symptoms)
- warfarin (if surgery on child)
- meprobamate
- phenytoin
- sulphonamides (G6PD haemolysis, kernicterus)
- heavy smoking
- high doses of aspirin
- diazepam
- phenobarbitone
- reserpine

Chloramphenicol, propranolol, isoniazid and metronidazole are present in high concentrations in breast milk, but are not usually harmful.

2. Potent drugs (even small amounts are dangerous in breast milk):

- antithyroid drugs (methimazole, thiouracil)
- cimetidine
- ergots
- iodides
- phenindione
- high doses of steroids
- bromocriptine
- cytotoxic drugs
- gold salts
- meprobamate
- tetracycline
- drugs causing G6PD haemolysis

3. If the mother has renal or hepatic failure, drugs excreted by these routes may accumulate in the plasma and be excreted in the breast milk.

A useful chart entitled “A Guide to Drugs in Breast Milk” by Dr M D Read can be obtained free of charge from Boehringer Ingelheim Ltd, Southern Industrial Estate, Bracknell, Berks RG12 4YS, England.

REFERENCES

Avery GS. Drug treatment, 2nd ed, Adis, 1980, p.113-117.

Busser J, Schultz J. Frontiers in Foetal Health 3(2):11-22 (readily available on Internet). Drugs in breast milk 2000.

Wilson JT. Drugs in breast milk, Adis, 1981.

LACTATION INDUCTION

It is often necessary to induce lactation in a mother who has absent or inadequate amounts of breast milk, or when a child has been adopted. It is much easier to induce lactation in a woman who has delivered a child but it is often possible to induce lactation in a nulliparous woman providing that she desires to breastfeed.

1. Prime the breast.

If the woman has no milk at all, give ethinyloestradiol 50 microgram tablet 3 times a day for 1 week. If ethinyloestradiol is not available, give medroxyprogesterone (Depo-Provera) 100 mg (2 ml) IM once. If the woman already has some breast milk, do not give ethinyloestradiol or medroxyprogesterone, but give metoclopramide or chlorpromazine straight away.

2. Stimulate prolactin production.

If the woman has lactated in the past or already has some breast milk, give metoclopramide (Maxolon) 10 mg 4 times a day OR chlorpromazine (Largactil) 25 mg 4 times a day until an adequate milk supply is established (usually within one week).

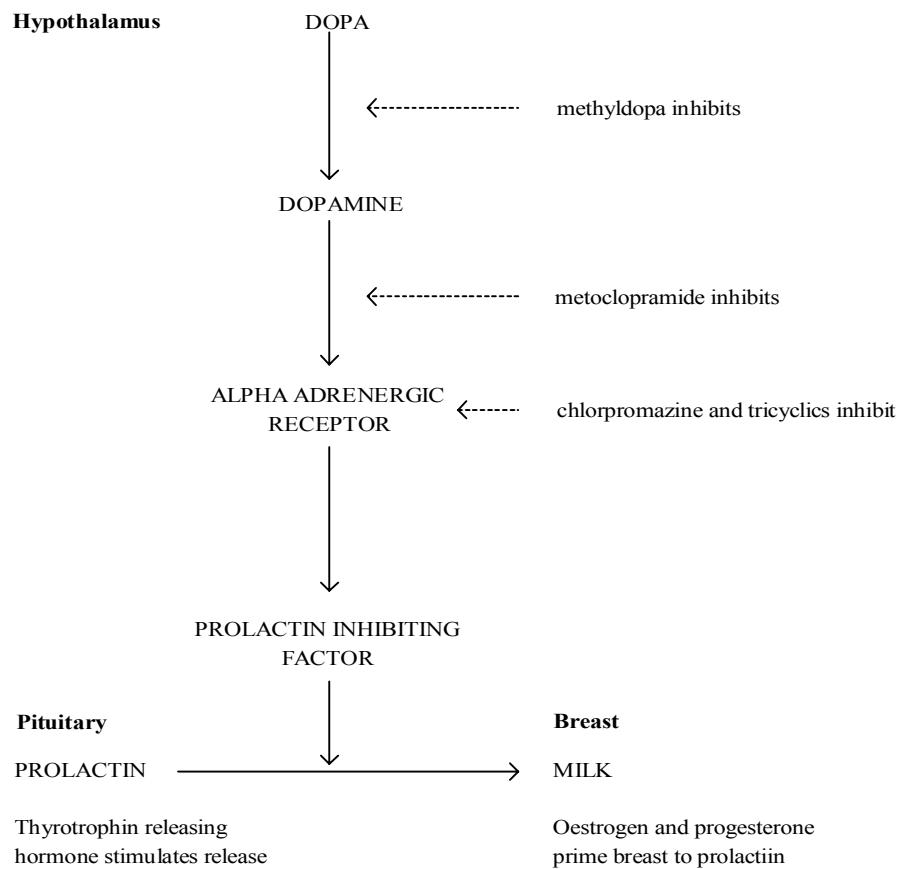
If the woman has never lactated and has no milk at all, give metoclopramide (Maxolon) 10 mg 4 times a day AND chlorpromazine (Largactil) 25 mg 4 times a day until an adequate milk supply is established. If there is not enough milk after 2 weeks of metoclopramide and chlorpromazine, add methyl dopa (Aldomet) 125 mg 4 times a day.

3. Frequent suckling.

At the time you start metoclopramide and/or chlorpromazine, encourage the mother to suckle the child AS MUCH AS POSSIBLE. This is an essential part of the procedure. Have the ward staff, other mothers and yourself encourage her as much as possible. Giving the mother frequent drinks of milk is good psychology.

Note: The above regimen is very intensive. Side effects such as drowsiness are common and may necessitate reducing the drug doses. Less intensive regimens are also successful. The key to the whole process of relactation is the desire of the mother to breastfeed. Support and encouragement are vital.

THE PHYSIOLOGY OF LACTATION



REFERENCES

- Abejiide OR et al. Ann Trop Paed/Int Child Health 1792:109-114,1997. Non-puerperal induced lactation in a Nigerian community. Case reports.
- Brown RE. Pediatrics 60:116-120,1977. Relactation.
- Kauppila A. Lancet 1:1175-77,1981. Metoclopramide and lactation.
- Nemba K. J Trop Paediatr 40(4):240-242,1994. Induced lactation: a study of 37 non-puerperal mothers.
- Ruis H. Br Med J 283:340-42,1981. Oxytocin and lactation.

LEPROSY

Leprosy is quite common in children, but rare under 2 years of age. It is often unrecognised, and 75% of cases regress spontaneously without treatment. Childhood leprosy usually responds rapidly to treatment and reactions are rare. Almost all cases in children are indeterminate or tuberculoid leprosy.

Leprosy bacilli are intracellular organisms. Host resistance therefore depends on cellular immunity and not on antibodies. Antibodies are made however, and are responsible for lepra reactions. Prolonged contact with a patient is NOT needed to contract leprosy. The incubation period is usually about 3 years (range 3 months to 40 years).

TYPES OF LEPROSY

Indeterminate

Indeterminate lesions are all flat lesions not showing typical tuberculoid features (clear-cut lesion with sensory loss of 2 or 3 modalities or superficial nerve enlargement).

When *M leprae* first invades the body, the small slightly pale skin patch of indeterminate leprosy develops. Usually cellular immunity destroys all the organisms and the patch disappears spontaneously. But the infection may develop into determinate leprosy: tuberculoid, borderline or lepromatous.

Tuberculoid (TT) - flat or raised

1. A clear-cut skin lesion with well-defined limits, PLUS
2. Loss of at least two of temperature, pain or light touch, OR enlargement of the superficial nerves supplying the area.

Tuberculoid leprosy occurs when cellular immunity is strong, but not strong enough to kill off all the leprosy bacilli. The intense inflammatory reaction prevents bacilli growing and spreading, but rapidly causes swelling, and damages the skin and nerves in which *M leprae* lives.

There are usually one or two hypopigmented lesions with clearly defined edges, small or large. The lesions may be flat or slightly raised, especially round the edges where the bacilli are active. Healing takes place from the centre, which may be flat and returning to normal colour. Skin smears are negative. There is a rapid response to treatment. Reactions are rare. The lepromin skin test is strongly positive.

Borderline (BB) - rare in children

In borderline leprosy, the host resistance is between that of the tuberculoid and lepromatous forms. Some leprosy bacilli are killed, but some spread from skin and nerves to bones, muscles, testes, etc.

There are multiple lesions of variable size and shape that may be flat or raised. The lesions are usually shiny and red, the edges slope upwards and the centres are flat. The skin lesions, but not the nerve lesions, are usually the same on each side of the body. Small firm nodules are common on the face. Skin smears are positive from the lesions, but negative from normal skin.

Lepromatous (LL) - very rare in children

Cellular immunity is poor. The bacilli multiply rapidly and spread to the eye, the mucous membranes of the nose, bones, muscles and testes. Lesions are widespread and symmetrical. There is little inflammatory reaction and local tissue damage at first, but later the large number of bacilli causes tissue necrosis and destruction.

SKIN AND NERVE LESIONS

Skin lesions

1. Hypopigmented macules.
2. Erythematous or hyperpigmented macules.
3. Infiltrated (raised) patches: usually erythematous or hyperpigmented. Desquamation comes with resolution.
4. Lichenoid lesions: pin-head papules on normal skin, macules or infiltrated plaques.
5. Papulonodular lesions: red, firm rounded elevations. Lesions of the nose, mouth and eyes are rare in children.

35% of skin lesions are single; 50% are found on the lower limb, 30% on the arm and forearm and 10% on the trunk.

Nerve involvement

1. The ulna, external popliteal and great auricular nerves are those that are most often enlarged.
2. Loss of sensation occurs. Temperature discrimination is usually affected first, followed by pain then light touch.
3. There is often pain or tenderness of the nerve trunk. Paralysis and deformities are rare in children.

DIAGNOSIS

1. Skin lesions with or without neural involvement

Diagnosis is made by:

- a. the appearance of the skin
- b. sensory loss in the skin lesions
- c. nerve enlargement, pain or tenderness.

2. Neural involvement only (an enlarged or tender peripheral nerve).

Definite sensory loss (without a history of trauma) is diagnostic of leprosy.

Pain or tenderness of a nerve trunk (without a history of trauma) is diagnostic of leprosy, especially if there is a history of contact.

An enlarged nerve unilaterally (without a history of trauma) is suggestive of leprosy. If there is a history of contact, start treatment. If there is no history of contact, review every 6 months.

Skin smear

Skin smears are negative in over 90% of cases in children. Infiltrated lesions are occasionally positive. Take smears from the child's lesions, ears and arms. Swab the skin with spirit, then pinch it up between finger and thumb to stop the blood flowing. Make a straight cut 2 mm deep and 5 mm long with a scalpel, then turn the blade quickly and scrape it inside the cut to get some juice (NOT blood) on the blade. Smear the juice on a clean glass slide labelled with the patient's name and the date. Juice from 6 or 8 sites can be smeared on one slide. Let the slide dry, then warm the back of the slide briefly over a flame until it is just too hot to hold on the back of your hand. Six or more organisms indicate lepromatous leprosy, 1-5 organisms indicate borderline leprosy, and no organisms are seen in tuberculoid leprosy.

The morphological index (MI) is the percentage of solid staining organisms on the slide. The MI falls from 25-75% at diagnosis to 0% after about 6 months of uninterrupted treatment. A stationary or rising MI indicates that the patient is not taking the drugs or that resistance has developed.

Biopsy

A FULL THICKNESS skin biopsy (down to fat) is sometimes helpful in diagnosis, and is very useful for correct classification. Biopsy across the edge of a suspected lesion. Fix the biopsy in FFA (40%

formaldehyde 10 ml, mercuric chloride 2 g, glacial acetic acid 3 ml, water to 100 ml) for 2 hours. Wash in water, then store in 70% alcohol. If FFA is not available, use normal saline.

The Lepromin (Mitsuda) Intradermal Test

This is not used in Papua New Guinea.

MANAGEMENT

1. Do a skin biopsy for suspected tuberculoid or borderline leprosy (which is most cases in children). Do a skin smear for suspected lepromatous or borderline leprosy. If the diagnosis is certain, start treatment as soon as you have taken the biopsy or smear.
2. Notify the Leprosy Control Officer of the case.
3. Make sure all the necessary paper work is completed. The family must understand:
 - a. that leprosy can be cured, PROVIDING treatment is taken regularly
 - b. how much medicine should be given each dose
 - c. how often medicine should be given
 - d. the importance of reporting quickly if nerve pain, anaesthesia, swelling or fever develop
 - e. that any change of address must be notified and treatment continued.
4. Examine all contacts (family and friends, schoolmates, those who live in the same house) for leprosy.

The treatment of leprosy was reviewed in Lancet 2:487,1988. Most children with leprosy have paucibacillary disease. They can be treated for just 6 months with daily unsupervised dapsone and monthly supervised rifampicin 15 mg/kg (maximum 600 mg). All treatment then stops and the patient remains under observation for 2 years.

Patients with multibacillary disease should continue with drug treatment until skin smears are negative (a minimum of 2 years). They are treated with daily unsupervised dapsone and clofazimine, and monthly supervised rifampicin 15 mg/kg (maximum 600 mg) and clofazimine 1 mg/kg. After treatment, observation should continue for 5 years.

Dapsone daily dose	
3-19 kg:	25 mg (half tab)
20-29 kg:	50 mg (1 tab)
30-49 kg:	75 mg (1.5 tab)
50 kg or more:	100 mg (2 tab)

Clofazimine dose	
Less than 10 kg:	50 mg (1 tab) every second day
10 kg or more:	50 mg (1 tab) every day

The treatment of lepromatous leprosy has improved greatly with the advent of combination chemotherapy - monotherapy with dapsone should no longer be used. Patients with lepromatous leprosy have deficient immunity to leprosy, and are very dependent on drug therapy. The treatment of lepromatous leprosy has been modified because of:

- a high incidence of resistance after 5 to 10 years' treatment of lepromatous leprosy with dapsone alone (Br Med J 2:914-915,1977)
- the persistence of viable dapsone-sensitive organisms despite full dapsone therapy.

In decreasing order of potency, bacteriocidal leprosy drugs are rifampicin, clofazimine, ethionamide and dapsone. Rifampicin is the most potent antileprosy drug. Thiacetazone, thiambutosine (DPT) and streptomycin are only bacteriostatic.

LEPRA REACTIONS

There are two forms of reaction. Both are rare in children.

Down-grading and reversal reactions

The reaction is due to a rapid change in the host-parasite relationship. It is usually seen in tuberculoid and borderline cases. Skin lesions increase in number, become inflamed and may ulcerate. Peripheral nerves may become tender and swollen, then acute paralysis develops. There may be oedema of the hands and feet.

In a down-grading reaction, the host resistance becomes weaker. New lesions appear. The lepromin reaction becomes weaker, and tuberculoid lesions change to borderline or lepromatous.

In a reversal reaction, the host resistance becomes stronger (this often occurs with treatment). Serial biopsies show fewer bacilli and increased lymphocytes, epithelial cells and giant cells. Borderline lesions change towards tuberculoid, and the lepromin reaction tends to be stronger.

Erythema nodosum leprosum (ENL)

This is caused by an antibody reacting with antigen from disintegrating bacilli. It usually occurs in lepromatous or borderline leprosy. There is little change in the leprosy lesions. Crops of tender red lumps appear every 5-7 days. There is often associated fever, oedema, neuralgia, arthralgia, acute paralysis, lymphadenitis, iritis, irido-cyclitis, epididymo-orchitis or proteinuria.

THE TREATMENT OF LEPRA REACTIONS

Do NOT reduce the dose of dapsone.

Thalidomide is rarely used (it is effective, but teratogenic).

A mild reaction (especially indeterminate or tuberculoid leprosy):

1. Advise REST - bed rest, splinting or sling.
2. Give ASPIRIN every 6 hours
3. Give CHLOROQUINE daily for 3 weeks (tablets 150 mg base)

Weight	1 st week	2 nd week	3 rd week
6-9 kg:	½ tab	¼ tab	¼ tab
10-19 kg:	1 tab	½ tab	¼ tab
20-29 kg:	1½ tab	1 tab	½ tab
30-39 kg:	2 tab	1 tab	½ tab
40-49 kg:	2½ tab	1½ tab	½ tab
50 kg or more:	3 tab	2 tab	1 tab

4. If there is no improvement, give CLOFAZIMINE 100 mg a day (every second day if under 10 kg), as well as dapsone. Up to 10 mg/kg of clofazimine can be given each day.

A severe reaction with acute neuritis, iritis or iridocyclitis

1. Give PREDNISOLONE 1 mg/kg/day in 3 divided doses. Reduce the dose gradually when the reaction is controlled. Prednisolone should ONLY be used for acute neuritis or iritis, and NOT for ENL alone.
2. Give CLOFAZIMINE 10 mg/kg/day as well as dapsone. When the reaction is controlled, reduce the dose gradually to 100 mg a day (or every second day in a child weighing less than 10 kg). When all signs of the reaction have gone, gradually withdraw clofazimine completely (leaving the patient still on maintenance dapsone).
3. Treat iridocyclitis with atropine drops and 1% hydrocortisone drops every 6 hours.

DAPSONE RESISTANCE

This is suggested by:

- no visible improvement after regular treatment for one year, or

- no reduction in the skin smear morphological index after regular treatment for one year, or
 - reappearance of solid rods on smears (relapse) despite regular treatment.
1. Take a skin smear each month for 3 months.
 2. Give supervised dapsone.
 3. If there is no improvement after 3 months:
 - a. arrange with the TB/Leprosy Section of the Health Department for a biopsy to be sent for culture and sensitivities (it may have to go overseas).
 - b. stop dapsone and give rifampicin 10 mg/kg/day for 12 weeks, and clofazimine 100 mg daily indefinitely.

REFERENCES

- Bagshawe A. Trans Roy Soc Trop Med Hyg 83:121-7,1989. Epidemiology of leprosy.
- Browne SG. Leprosy in children. Geneva: WHO, 1976.
- Editorial. Lancet 2:487-488,1988. Chemotherapy of leprosy.
- Jopling WH. Handbook of leprosy. 3rd ed. London: Heinemann, 1984.

LIMP

See also Arthritis, pp.35-38.

NEVER IGNORE A LIMP, OR PAIN IN THE HIP OR LEG. IT MUST ALWAYS BE REGARDED AS PATHOLOGICAL.

ACUTE LIMP

Pain in the knee is often due to disease of the hip.

X-ray the appropriate area. Consider pyogenic arthritis, osteomyelitis, cellulitis, TB, fracture, Perthes' disease, scurvy, rheumatic fever, malignancy and irritable hip. Any febrile child with a painful hip should get chloramphenicol and be referred for aspiration or exploration of the hip.

ACUTE AND CHRONIC LIMP

All ages:	trauma, osteomyelitis, pyomiositis, TB or pyogenic arthritis malignant disease (leukaemia, neuroblastoma, sarcoma)
1-2 years:	cerebral palsy, CDH, spiral crack fracture tibia
2-5 years:	monoarticular synovitis, mild spastic hemiplegia
5-10 years	irritable hip (aspirate to exclude suppuration if the child is febrile), Perthes' disease (osteochondritis: 80% of cases occur in boys, 10% are bilateral, and X-ray shows flattening of the femoral head with a normal acetabulum; cf TB in which the acetabulum is involved). Refer the child to a surgeon
10-15 years:	slipped upper femoral epiphysis - take a lateral x-ray of both hips and compare the 2 sides.

Note: The diagnosis of TB of the hip is often missed until irreversible damage has occurred. Always consider the possibility of TB in all age groups.

A LIMP AFTER AN INJECTION

It is quite common to see a child who has had an injection in the buttock for an acute febrile illness, and subsequently developed a weak leg on the side of the injection. Is the weakness due to damage to the sciatic nerve from the injection; or was the febrile illness actually polio, which caused the flaccid paralysis? (This is unlikely now - but still a possibility.)

Ask, did the paralysis come on immediately after the injection (sciatic nerve damage), or did it come on several days later (polio)? Polio usually causes wasting of the quadriceps and buttock as well as the calf muscles, the knee jerk is reduced on the affected side, there is no sensory loss, and the child may not be able to walk on the affected leg at all. Sciatic nerve damage does not cause wasting of the quadriceps or buttock, the knee jerks are equal, there is anaesthesia to light touch and pinprick on the lateral side of the foot, and the child can walk - but with the characteristic gait of a patient with foot-drop.

An injection given to a child with early poliomyelitis may precipitate paralysis in that limb. This is one of a number of good reasons for avoiding the indiscriminate use of injections in patients with non-specific febrile illness.

LIVER BIOPSY

This is a dangerous procedure, and should only be performed with adequate indications. These are very rare in childhood. The best results are obtained by using a spring-loaded modified Trucut needle with ultrasound guidance.

**LIVER BIOPSY MUST ONLY BE PERFORMED BY EXPERIENCED STAFF.
AND IT SHOULD BE DONE UNDER ULTRASOUND GUIDANCE IF POSSIBLE.**

Do a prothrombin time. If this is 2 seconds or more longer than the control, give vitamin K, 1.0 mg/kg IM and wait 24 hours. Do not do the biopsy if the prothrombin time is still prolonged. The platelet count should be over 100,000. Crossmatch one unit of blood.

Drain any ascites before doing a liver biopsy. Ensure that your biopsy needle is SHARP.

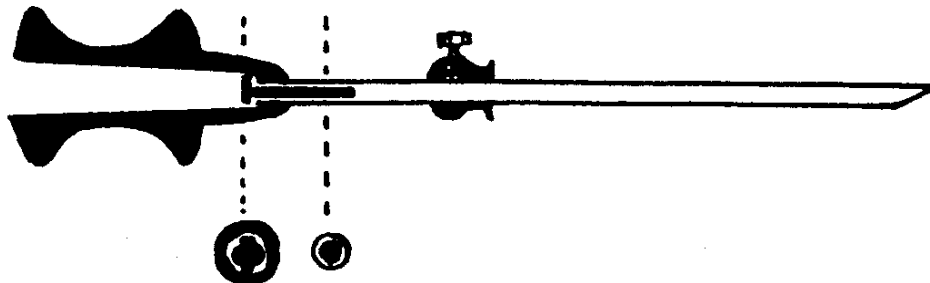
Over 4 cm liver: use the subcostal route

Under 4 cm liver:

- infant: give IV diazepam just before the procedure, and have an assistant compress the chest at the end of expiration to arrest respiration. Use the intercostal route
 - older child: use the intercostal route under relaxant general anaesthesia, unless the child is old enough to co-operate fully (about 12 years or more).
1. The child lies supine with the right side at the edge of the bed. The right arm is held above the head.
 2. Scrub your hands and put on sterile gloves.
 3. Subcostal: puncture below and to the right of the xiphoid process in the midclavicular line. Aim towards the left shoulder.
 4. Intercostal: puncture at the point of maximum dullness (8-10th intercostal space) in the mid axillary line. This technique involves puncture of the pleural cavity and must ONLY be used if full expiration can be maintained throughout the procedure.
 5. Clean the skin with iodine in a 10 cm radius around the proposed puncture site. Inject 2% plain lignocaine through a 23 gauge needle into skin, subcutaneous tissue, pleura, peritoneum and liver capsule. Make a nick in the skin with a scalpel blade.

MENGHINI “ONE SECOND” NEEDLE BIOPSY

1. Place the nail in the shaft of the needle with the flat end away from the point. Attach a 5 ml syringe to the needle. Draw up 2 ml of sterile saline into the syringe.



Longitudinal section of the Menghini liver biopsy needle. Note the nail in the shaft of the needle.

2. Push the needle perpendicularly down to, but not through, the intercostal muscle. Inject 1 ml of saline to clear the needle of any fragments. Commence and maintain firm aspiration.
3. With the chest maintained in expiration, QUICKLY push the needle into the liver, then remove it. Do not rotate the needle. This part of the procedure should take only one second.

4. Put the needle into a bottle of 10% formalin, and gently inject a little of the remaining saline from the syringe to free the biopsy.

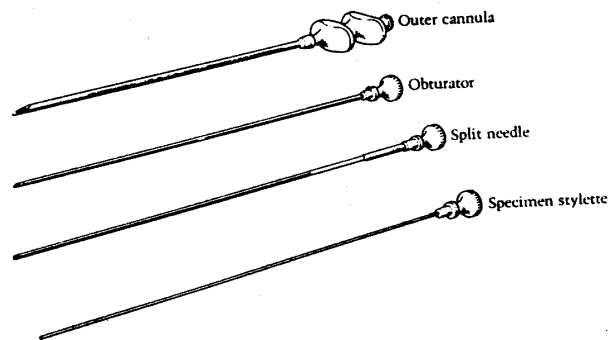
TRUCUT NEEDLE BIOPSY

This method is more dangerous than the Menghini technique (particularly with an uncooperative patient), but it has a higher success rate and usually yields a better biopsy specimen. It is an improved version of the Vim-Silverman needle.

1. Push the needle into the liver with the chest held in expiration.
2. While holding the main shaft of the needle still, rapidly advance the cutting blade so that it excises the fragment of liver that is resting in the slot on the needle (or release the trigger if a spring-loaded Trucut biopsy gun is being used).
3. Remove the needle, pull back the cutting blade, and carefully remove the biopsy into a bottle of 10% formalin.

VIM-SILVERMAN NEEDLE BIOPSY

This method is more dangerous than the Menghini technique, but has a higher success rate with a fibrotic liver. Have the chest held firmly in expiration.



1. Introduce the trocar and cannula into the liver.
2. Remove the trocar. Holding the cannula firmly in one hand, push the split needle in rapidly until its head hits the head of the cannula.
3. Hold the split needle stationary and advance the cannula until its tip is level with that of the longer split needle inside the liver. Rotate the cannula and split needle.
4. Remove the split needle and cannula together. The biopsy is held between the two halves of the split needle, and can be freed under 10% formalin.

OBSERVATION

The main risk from liver biopsy is haemorrhage. The pulse rate must be taken hourly for 24 hours after the biopsy. You must be notified if it rises. Be prepared to give the crossmatched blood. There may be some right chest and shoulder tip pain.

REFERENCE

Sherlock S. Diseases of the liver and biliary system. Blackwell.

LUMBAR PUNCTURE

1. Have the patient held in the CORRECT position by an assistant:
 - a. lie the patient on the side with the back and hips flexed so that the knees are bent up onto the abdomen
 - b. the assistant should hold the child by the shoulders and NOT by the head. Strong flexion of the neck in a small child can cause respiratory obstruction and death
 - c. the child's back should be at the edge of the bed
 - d. the child's back must be vertical - very often the child is rolled slightly onto the front, and this makes your job much more difficult.

Note: Ensuring that the child is firmly held in the correct position is essential to success.

2. Scrub your hands. Use sterile gloves, if available.
3. Clean the whole of the lower back and the tips of your left index and middle fingers (that you will use to feel the child's back) with iodine.
4. Find the L3-4 or L4-5 space, at or just above a line joining the two iliac crests. A common mistake is to choose too low a space in small children. Do NOT inject local anaesthetic in babies or children under 20 kg.
5. Use a sterile 21 or 23 gauge disposable needle. Use a 21 gauge scalp vein needle with all but 2 cm of the tubing cut off for babies. DO NOT TOUCH THE SHAFT OF THE NEEDLE WITH YOUR FINGERS. Keep the needle EXACTLY HORIZONTAL and SLOWLY push it in, aiming towards the umbilicus.
6. Push the needle in, in VERY SMALL stages of about 1 mm each. Wait AT LEAST 15 seconds between each stage, and turn the needle a quarter of a turn each way on its long axis (in case the end is blocked).
7. It is easy to push the needle in too far in small children. Go in SLOWLY, and WAIT a long time between each time you push it in. The CSF often comes out very slowly in small children, so you can be in the subarachnoid space without knowing it if you do not wait long enough.
8. Take 1 ml of CSF into each of two labelled bottles. Send the CSF for microscopy, culture, protein and sugar. Laboratory examination of CSF is dealt with on p.70.
9. Take out the needle, lie the child on his front, clean off the iodine, and stick on a small dry swab with a piece of Elastoplast.
10. If you get a traumatic tap, treat the child for meningitis and repeat the lumbar puncture even more carefully 2 days later.

Common causes for a failed or traumatic LP are:

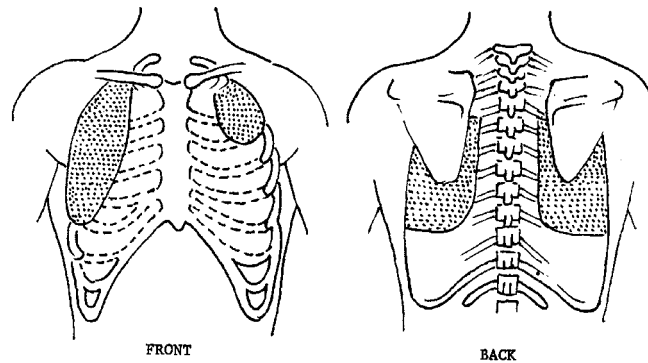
- the child was not held correctly (it is YOUR job to see this is done properly)
- the puncture was made too low
- the needle was not in the exact midline of the spine and not kept exactly horizontal
- the needle was pushed in too fast and too far without waiting long enough between each stage.

LUNG ASPIRATION

Lung aspiration is not a routine procedure. However, properly performed, it is comparatively safe: death is uncommon from the procedure, pneumothorax requiring drainage occurs approximately once in every 400 aspirations, and transient haemoptysis once in every 130 aspirations.

When reliable bacteriological services are available, lung aspiration is a justifiable diagnostic procedure in children with very severe pneumonia not responding to standard antibiotic therapy. It may also be useful in the diagnosis of pulmonary tuberculosis

1. Take a chest x-ray with AP and right lateral views (or left lateral if the signs are on the left). Aspirate ONLY if there are chest x-ray changes or clear clinical localising signs (crepitations, bronchial breathing or dullness).
2. Consider the lung as upper, middle or lower zones. Aspirate the affected zone from either the front or the back (but not from the side), depending on the lateral x-ray and whether there are more crepitations at the front or back.
3. Aspirate ONLY in the dark areas of the diagram:



4. Have oxygen and a chest drain available.
5. Have an assistant hold the child sitting up with hands over the head.
6. Clean the skin with iodine.
7. Use a 23 gauge needle on a 10 ml syringe.
8. Draw up 1 ml of sterile saline or culture medium.
9. Have another assistant grasp the child's chest FIRMLY in expiration just before you aspirate. It is most important that the child be held firmly in expiration, because the needle may tear the pleura if the child breathes while the needle is in the lung.
10. Push the needle quickly into the chest to the required depth. Go just above the rib.
11. Inject 0.5 ml saline into the lung. With the needle in the same position, exert maximal suction. Then slowly withdraw the needle over 2 or 3 seconds while still exerting strong suction.
12. Inoculate the saline in the syringe onto the appropriate culture media.
13. Observe the child closely for the next hour for signs of pneumothorax (increasing tachypnoea and cyanosis). There may be transient haemoptysis.
14. Take an AP chest x-ray later in the day or next day to check for pneumothorax.

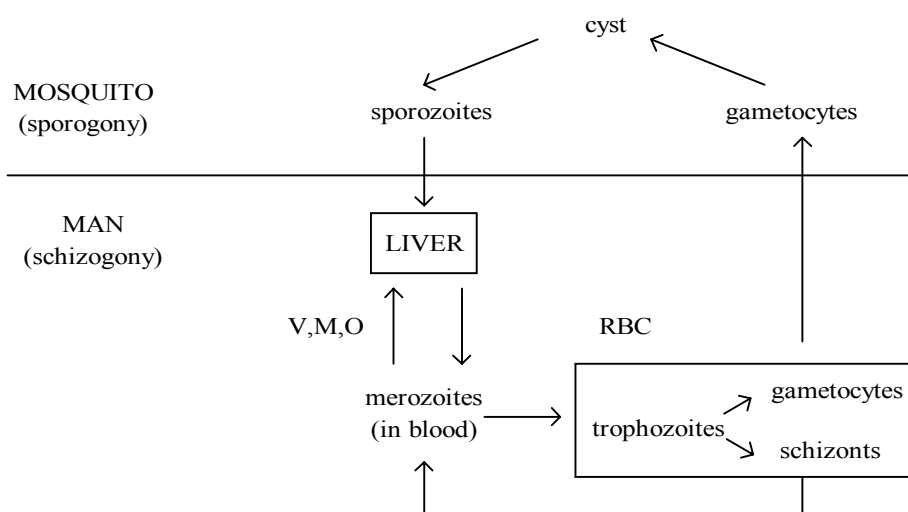
REFERENCES

- Shann F. Lancet 2:537-541,1984. Aetiology of pneumonia.
Silverman M. Arch Dis Child 52:925-931,1977. Aetiology of pneumonia.
Weech AA. Am J Dis Child 122:277,1971. Lung puncture.

MALARIA - SUMMARY

Note: In view of the significant problems posed by chloroquine resistant malaria and the serious side effects of quinine treatment the Health Department has decided to change the standard management protocols. A single dose of Fansidar is added to the 3-day course of chloroquine or infant Camoquin (amodiaquine) for the treatment of uncomplicated malaria (Treatment A). A 7-day course of Artemisinin derivatives (artemether IMI and oral artesunate) with a single dose of Fansidar on the 3rd day of treatment is used for the treatment of severe malaria (Treatment B) and of treatment failure malaria (Treatment C). The use of a single gameticidal dose of primaquine has been abandoned. Since it will take time for the introduction of the new protocols, both the old and the new are given here. Doctors must be familiar with both.

SUMMARY OF LIFE CYCLE OF MALARIA



SUMMARY OF ANTIMALARIAL DRUGS

	Resistance	Liver phase	Schizonto-cidal	Gameto-cidal	Causal proph.
Schizontocides					
4AQ (chloroq, amod)	F, V	No	Yes	Not F	No
Quinine	F	No	Yes	Not F	No
Fansidar	F, V	Weak	Yes	No	Yes
Proguanil (Paludrine)	F, V, M, O	No	Weak	No	Yes
Pyrimethamine (Daraprim)	F, V, M, O	No	Weak	No	Yes
Artemisinin + derivatives	?Nil	No	Yes	?	?
Mefloquine	?	No	Yes	No	Yes
Exoerythrocytic (liver)					
8AQ (primaquine)	No	Yes	Weak	Yes	Yes

F = falciparum, V = vivax, M = malariae, O = ovale. 4AQ, 8AQ = 4 and 8 aminoquinoline

SUMMARY OF TREATMENT OF MALARIA

Children with malaria are diagnosed into one of three groups:

Uncomplicated malaria

Children who are febrile but who are not very sick (very sick being defined as a child who has an indication for admission - see Paediatric Rules, p.286). These children are treated with chloroquine or infant Camoquin and a single dose of Fansidar, as per Treatment A1 (new regimen) or as per Treatment A2 (old regimen).

Note: A single dose of primaquine has been given as a gametocidal as part of routine treatment of malaria in Papua New Guinea. There is no convincing evidence that it is effective in reducing malaria transmission and it has been discontinued.

Severe malaria

Children who are febrile and who are very sick - ie have any of the indications for admission (see Paediatric Rules, p.286) or who are unconscious or convulsing (the latter situation loosely categorised as 'cerebral malaria' - though the WHO definition specifies the presence of impaired consciousness for more than one hour after the fit). These children are treated with Treatment B1 or B2. They are also usually treated for meningitis with chloramphenicol until this diagnosis has been excluded by a CSF examination.

Note: In the old regimen, parenteral quinine is followed by three days of oral quinine - plus Fansidar and primaquine. Thus, total duration of quinine therapy is not 3 days - but may be 4-7 days. This aspect of treatment has often been overlooked.

Treatment failure malaria

Children who have not responded to a correctly administered, swallowed and appropriate treatment course, or who are deteriorating after starting treatment for uncomplicated malaria, or who have another attack of malaria within one month of completing a standard course of chloroquine or infant Camoquin. Treat these children as per Treatment C1 or C2.

SUMMARY OF THE IMMEDIATE MANAGEMENT OF THE COMPLICATIONS OF SEVERE MALARIA

1. Weigh the child.
2. Check the airway and lie the child on the side.
3. Do a blood slide, FBE, dextrostix, U+E, culture and cross match.
4. Start 10% dextrose IV.
5. Start treatment as per Treatment B1 or B2.
6. Assess hydration clinically, urine SG and output (catheter if necessary).
7. Decide fluid requirements (coma regimen in STB).
8. If the temperature is over 40 °C, give paracetamol, tepid sponge, fan.
9. Check the fundi: if there is no papilloedema, do an LP.
10. Give anticonvulsants, antibiotics and vitamin K if necessary.
11. Give a blood transfusion if the Hb is 6 g/dl or less.

Coma - maintain the airway, nurse on the side, turn 2 hourly, exclude other causes of coma (meningitis, hypoglycaemia) and avoid harmful treatments (steroids, heparin).

Hyperpyrexia - give paracetamol, tepid sponge and fan.

Convulsions - maintain the airway, give paraldehyde and then phenobarbitone, check the blood glucose (do a dextrostix).

Severe anaemia (Hb 6 g/dl or less) - give a blood transfusion and frusemide.

Haemoglobinuria - alkalize the urine if this is very severe.

Acute pulmonary oedema - prop up at 45°, and give oxygen and frusemide, stop IV fluids (this complication is less common in children than in adults).

Acute renal failure - exclude dehydration, give 5 ml/kg saline over 20 min. If there is still no urine, give frusemide 2 mg/kg, then 4 mg/kg, then 8 mg/kg IV at hourly intervals. If there is still no urine, give dopamine 2.5 microgram/kg/min via central line for 6 hours. If the child is still oliguric, start peritoneal dialysis.

Spontaneous bleeding - transfuse fresh whole blood, and give vitamin K.

Metabolic acidosis - exclude hypoglycaemia, hypovolaemia and sepsis.

MALARIA - TREATMENT AND COMPLICATIONS

REMEMBER: fever in children is frequently NOT due to malaria, even in highly malarious areas. Treat for malaria, but look for other causes of fever.

Daily parasite density counts may be useful in severe malaria, or if you suspect resistance. Note that amodiaquine and chloroquine do not kill the gametocytes of *P falciparum* - only the persistence of *P falciparum* trophozoites shows resistance. Distinction must be made between trophozoites and gametocytes. Record the daily trophozoite count in the patient's notes.

UNCOMPLICATED MALARIA TREATMENT

Treatment A1 (New)

Give amodiaquine (Infant Camoquin) or chloroquine (Nivaquine) tablets daily for 3 days. Give a single dose of Fansidar on day 1 of treatment.

Uncomplicated malaria: Treatment Chart A1 (New)			
Weight (kg)	Amodiaquine 100 mg tab Daily for 3 days	Chloroquine 150 mg tab Daily for 3 days	Fansidar Single dose
3 - 5.9	½ tab	¼ tab	¼ tab
6 - 9.9	1 tab	½ tab	½ tab
10 - 14.9	1½ tab	1 tab	1 tab
15 - 18.9	2 tab	1 tab	1 tab
19 - 27.9	-	1½ tab	1½ tab
28 - 36.9	-	2 tab	2 tab
37 - 49.9	-	3 tab	2½ tab

Treatment A2 (Previous but without the single dose of primaquine))

Give amodiaquine (Infant Camoquin) or chloroquine (Nivaquine) tablets orally for 3 days.

Uncomplicated malaria: Treatment Chart A2 (Previous)		
Weight (kg)	Amodiaquine 100 mg tab Daily for 3 days	Chloroquine 150 mg tab Daily for 3 days
3 - 5.9	½ tab	¼ tab
6 - 9.9	1 tab	½ tab
10 - 14.9	1½ tab	1 tab
15 - 18.9	2 tab	1 tab
19 - 27.9	-	1½ tab
28 - 36.9	-	2 tab
37 - 49.9	-	3 tab

SEVERE (COMPLICATED) MALARIA - INCLUDING CEREBRAL MALARIA

Take a blood slide and do a lumbar puncture and dextrostix. Give oxygen. Put up a 4.3% dextrose in 0.18% normal saline (dextrose-saline) drip. If the patient is fitting, stop the fit with paraldehyde or diazepam, and commence treatment with phenobarbitone (loading dose IMI 15 mg/kg as per STB). If the patient is hypoglycaemic, give intravenous 10% dextrose 5 ml/kg.

- Cloudy CSF (or if you decide against an LP or are unsuccessful or get a bloody tap): give treatment as per Table B1 (New) or Table B2 (Previous), and treat for meningitis with IM chloramphenicol.
- Clear CSF (see Coma, p.82): treat for cerebral malaria (the CSF may contain a few cells with a slightly raised protein). Give treatment as per Table B1 (New) or B2 (Previous). Do daily parasite density counts. Restrict the fluid intake to two-thirds maintenance (Coma regime as per STB). Monitor BP, hydration, blood sugar (eg dextrostix), urine output, Hb, urea and electrolytes. Hypoglycaemia is common in severe malaria (New Engl J Med 319:1040-7,1988).

Treatment B1 (New)

IM Artemether (80 mg/ml):

Give IM once daily until child improves (3.2 mg/kg day 1, 1.6 mg/kg thereafter).

Note: The dose on the first day is different from the dose on the following days.

When the child has improved and can take oral treatment, give

Oral Artesunate (tab 50 mg):

Give once daily to complete a total of 7 days (2.5 mg/kg).

Give a single dose of Fansidar on day 3 of treatment or on the first day of oral treatment if this is after day 3.

Severe malaria: Treatment Chart B1 (New)				
Weight (kg)	IMI Artemether Day 1	IMI Artemether Day 2-7	Oral Artesunate Day 2-7	Oral Fansidar Single dose Day 3
3 - 5.9	0.25 ml	0.25 ml	¼ tab	¼ tab
6 - 12.9	0.5 ml	0.25 ml	½ tab	½ tab
13 - 18.9	0.75 ml	0.5 ml	¾ tab	1 tab
19 - 24.9	1 ml	0.5 ml	1 tab	1½ tab
25 - 30.9	1.25 ml	0.75 ml	1½ tab	2 tab
31 - 36.9	1.5 ml	0.75 ml	1½ tab	2 tab
37 - 43.9	1.75 ml	1 ml	2 tab	2½ tab
>44	2 ml	1 ml	2 tab	2½ tab

The dose of Artemether on day 1 is bigger than the dose for the following days.
 Change to oral treatment as soon as the patient can take it.
 Continue treatment for 7 days.
 Give a single dose of Fansidar on day 3 of treatment or on the first day of oral treatment if this is after day 3.

Treatment B2 (Previous but without the single dose of primaquine)

Severe malaria: Treatment Chart B2 (Previous)			
Weight (kg)	IMI Quinine Twice daily	Oral Quinine 3 times daily	Fansidar Single dose
3 - 3.9	0.5 ml	¼ tab	¼ tab
4 - 5.9	1 ml	¼ tab	¼ tab
6 - 9.9	1.5 ml	¼ tab	½ tab
10 - 14.9	2 ml	½ tab	1 tab
15 - 19.9	3 ml	½ tab	1 tab
20 - 24.9	4 ml	1 tab	1½ tab
25 - 29.9	5 ml	1 tab	1½ tab
30 - 39.9	6 ml	1½ tab	2 tab
Quinine:	IMI Quinine (120 mg/2 ml): Twice daily until child improves, then Oral Quinine (300 mg tab): 3 times daily for 3 days.		
Fansidar:	Single dose on first day of oral treatment.		

TREATMENT FAILURE MALARIA

This is defined as the persistence of *P falciparum* trophozoites for 7 days, or recurrence within 4 weeks, after an **adequate, supervised** and appropriate course of treatment for uncomplicated malaria (either New, A1, or Previous, A2). Do daily trophozoite counts if you suspect treatment failure (likely to be the result of drug resistance):

- you must be certain the child did not vomit or spit out the drugs.

(Note: Resistance to amodiaquine or chloroquine is mainly a problem with *P falciparum*, but cases of resistant vivax are becoming more common and *P falciparum* resistant to Fansidar is also well established).

Treat with oral Artesunate and single dose Fansidar (Chart C1, New regimen) or with quinine and Fansidar (C2, Previous regimen).

Treatment C1 (New)

Oral Artesunate (tab 50 mg):

Give once daily for 7 days (5 mg/kg day 1, 2.5 mg/kg thereafter).

Note: The dose of Artesunate on day 1 is bigger than the dose on the following days.

Oral Fansidar:

Give a single dose on day 3 of treatment.

Treatment failure malaria: Treatment Chart C1 (New)			
Weight (kg)	Oral Artesunate Day 1 Single dose	Oral Artesunate Day 2-7 Once daily	Oral Fansidar Day 3 Single dose
4 - 5.9	½ tab	¼ tab	¼ tab
6 - 8.9	¾ tab	½ tab	½ tab
9 - 12.5	1 tab	½ tab	1 tab
12.6 - 18.5	1½ tab	¾ tab	1 tab
18.6 - 24.9	2 tab	1 tab	1½ tab
25 - 31.9	2½ tab	1½ tab	2 tab
32 - 37.5	3 tab	1½ tab	2 tab
37.6 - 50	4 tab	2 tab	2½ tab
If you do not have any Artesunate, use Quinine and Fansidar:			
Quinine (tab 300 mg):	Give oral 3 times daily for 3 days (see Treatment C2)		
Fansidar:	Give a single dose on the first day of treatment.		

Treatment C2 (Previous but without the single dose of primaquine)

Treatment failure malaria: Treatment Chart C2 (Previous)		
Weight (kg)	Oral Quinine 3 times daily	Fansidar Single dose
3 - 3.9	¼ tab	¼ tab
4 - 5.9	¼ tab	¼ tab
6 - 9.9	¼ tab	½ tab
10 - 14.9	½ tab	1 tab
15 - 19.9	½ tab	1 tab
20 - 24.9	1 tab	1½ tab
25 - 29.9	1 tab	1½ tab
30 - 39.9	1½ tab	2 tab
Oral quinine (300 mg tab):	Give 3 times daily for 3 days	
Fansidar:	Give single dose on day 1 of treatment	

COMPLICATIONS OF ACUTE MALARIA

Hyperpyrexia (temperature over 40 °C)

Hyperpyrexia may cause convulsions. It may be associated with cerebral malaria. Give antimalarials, paracetamol, cool sponge and fan.

Anaemia

In acute malaria, the anaemia is usually normochromic and normocytic. However, it is frequently hypochromic and microcytic due to an associated iron deficiency anaemia; the red blood cells show polychromasia and there is a raised reticulocyte count indicating the haemolytic element of the anaemia. A haemoglobin of 6 g/dl or less is an indication for urgent blood transfusion. Give 20 ml/kg of packed cells. To prevent precipitating heart failure, give IV frusemide (Lasix) once the blood transfusion has started. Do NOT give frusemide if the child has diarrhoea or is dehydrated.

Hypoglycaemia

Hypoglycaemia is common in children with severe malaria, and is associated with a high mortality (Lancet 1:708-711,1987). Do a dextrostix and give 5 ml/kg 10% dextrose (or 1 ml/kg 50% dextrose) IV over 15 min if the blood glucose is less than 40 mg% (2.2 mmol/l).

Dehydration

This is often caused by severe vomiting and diarrhoea. Correct the dehydration with half strength Darrow's solution IV.

Algid malaria

This is severe malaria associated with shock. It is often caused by gram negative sepsis (as well as malaria). The child is usually dehydrated from severe diarrhoea or vomiting or both. The child may also have cerebral malaria. Commence treatment for severe malaria immediately (Treatment B1 or B2). Take blood and urine cultures, then give IV chloramphenicol, or ampicillin and gentamicin. Give 20 ml/kg of normal saline fast, then half strength Darrow's solution.

Blackwater fever

Severe intravascular haemolysis causes methaemalbumin in plasma and haemoglobinuria, which makes the urine dark. Haemoglobinuria has a number of causes (see p.382), but is called blackwater fever when caused by *P. falciparum* infection. Blackwater fever is probably due to the production of auto-antibodies in response to immunological changes in parasitized red blood cells. It may occur more often when quinine is used. It may also occur with G6PD deficiency. Give treatment for severe malaria (Treatment B1 or B2), keep a careful record of the child's fluid balance (do NOT overhydrate) and, if anuria develops, consider starting peritoneal dialysis. Give a blood transfusion if the Hb is 6 g/dl or less. Exchange transfusion may be needed in severe cases.

Liver failure

A slight degree of jaundice due to haemolysis is quite common in severe acute malaria. Occasionally, however, there is progressive jaundice and liver failure due to liver cell damage.

MALARIA PROPHYLAXIS

Prevention of mosquito bites

The incidence of malaria can be reduced by lowering the risk of mosquito bites: wearing clothes that cover the arms and legs at night, using insect repellants (including pyrethrin burning coils at night), covering windows and doorways with wire-netting and using residual insecticide sprays in dwellings. Mosquito nets have been shown to be effective in Papua New Guinea and are more effective if they are impregnated with the insecticide permethrin.

Drugs for prophylaxis

No drug destroys the sporozoites, therefore true prophylaxis is impossible. Proguanil and pyrimethamine kill the pre-erythrocytic forms of *P. falciparum* and they therefore prevent initiation of the erythrocytic cycle. This is sometimes called “causal prophylaxis”. For a review of antimalarials, see *Antimicrob Agents Chemother* 32:953-61, 1988.

The indications for prophylaxis in children are:

- as part of the treatment of anaemia (3 months)
- tropical splenomegaly syndrome
- severe malnutrition.

From a practical point of view, it is easiest to treat children in these groups with chloroquine or infant Camoquin 5 mg/kg weekly. Breakthrough malaria would then be treated as chloroquine resistant/treatment failure malaria. In particular cases, it might be necessary to advise other prophylaxis in addition to or instead of chloroquine.

Paediatricians and medical officers are sometimes asked to give advice to parents of non-immune children travelling to areas of high malaria transmission. As a general rule, parents of expatriate children should be encouraged to follow the advice provided by the health authorities of their own countries. Alternatively, the WHO recommendations currently in force can be followed (at present this is weekly mefloquine).

There are worries about the use of mefloquine - though the risks may have been overstated. Fansidar has been used but may not be effective. For children aged 10 years or older, tetracycline or doxycycline may well be effective. Proguanil is another alternative, though probably less effective. For children older than 5 years, Maloprim (pyrimethamine and dapsone) has been used, but there are worries about its safety. It is definitely contraindicated in children less than 5 years of age (Maloprim may cause methaemoglobinemia).

Chloroquine or infant Camoquin should be taken in addition to Maloprim to provide some protection against vivax malaria.

NOTES ON DRUGS USED TO TREAT OR PREVENT MALARIA

Artemisinin and its derivatives

Artemisinin (qinghaosu) and its derivatives. Qinghaosu - a herbal product from Qinghao or sweet wormwood - has been used in China for thousands of years. A number of compounds derived from Qinghao has been isolated and are now available for use. They include:

- artemisinin, the parent compound
- dihydroartemisinin
- artesunate, metabolised to dihydroartemisinin
- artemether, metabolised to dihydroartemisinin.

Dihydroartemisinin is available in oral and suppository form.

Artesunate is available in oral and parenteral form - but the latter is complicated to use.

Artemether is available in oral and parenteral form. It is soluble in oil and, as an intramuscular injection, has been used successfully, easily and safely, including in a WHO collaborative study in PNG. It is also available in suppositories.

The Health Department has decided to opt for artemether IMI and artesunate tablets. Other preparations are available through the private sector. Doctors must be familiar with the correct doses. Artemisin or artemether suppositories may become available in the future for the treatment of children with severe malaria.

Chloroquine and amodiaquine

1. A treatment course of either drug is 25 mg/kg over 3 days.
2. The usual oral dose is 10 mg/kg daily for 3 days.
3. Prophylactic chloroquine in the correct dose does NOT cause retinopathy - even after many years.

4. Prophylactic amodiaquine has been associated with agranulocytosis in adults (Lancet 1:411-414,1986). The risk may be as high as 1/2000, so it is no longer recommended for prophylaxis in adults.

Quinine

With the introduction of artemether, quinine is likely to become a reserve drug for use in severe malaria if artemether is not available or if artemether resistance develops. It will, however, still be used for severe malaria in the first trimester of pregnancy.

If used in children, quinine is given 12 hourly IM until the patient improves. Oral quinine is then given for 3 days and a dose of Fansidar given on the first day of oral treatment.

Occasionally, quinine is given intravenously. The dose (10-15 mg/kg/dose) is given in 10 ml/kg of 5% or 10% dextrose over 4 hours.

Fansidar

Because of the high levels of chloroquine and amodiaquine resistance in Papua New Guinea, Fansidar is now part of the treatment for uncomplicated malaria, together with amodiaquine or chloroquine.

Fansidar (pyrimethamine and sulphadoxine) kills the malarial parasites in a similar way to that in which cotrimoxazole (Septrin) kills bacteria, by blocking steps in the synthesis of purines.

Fansidar takes some time to act and should not be used alone. It has a half-life of 4 to 8 days. Fansidar has been associated with agranulocytosis and also causes Stevens-Johnson syndrome (exfoliative dermatitis); the incidence may be as high as 1/5000 (MMWR 34:185-195,1985). Other side effects of Fansidar include less severe skin rashes, nausea, vomiting, thrombocytopenia, megaloblastic or haemolytic anaemia, methaemoglobinaemia and jaundice.

Maloprim

Maloprim contains dapsone (a sulphone) and pyrimethamine. It is slow-acting, and has been used successfully for the prevention of falciparum malaria in chloroquine-resistant areas. It is not preventative for vivax, so chloroquine is needed as well. Maloprim is no longer recommended because of the risk of agranulocytosis. The incidence of this complication when Maloprim is given twice a week may be as high as 1/2500 (Br Med J 282:988,1981) though it appears to be considerably less when given once weekly. It has been associated in Papua New Guinea with a fixed drug eruption.

Primaquine

Primaquine has been used as a gametocidal as a strategy to prevent transmission. Its benefit for this purpose has not been proven and it has been removed from the new standard treatment regimens.

To treat the exoerythrocytic (liver) phase of P vivax, malariae or ovale, give 0.25 mg/kg oral for 14 days. Nausea and abdominal discomfort are very common. Haemolysis may occur in G6PD deficient patients. Cyanosis may occur due to methaemoglobinaemia.

Mefloquine

Mefloquine is a derivative of quinine, and has a long half-life. It is active against many strains of P falciparum that are resistant to other drugs, but some resistant strains have already been reported. Mefloquine is expensive and a high incidence of side effects, particularly vomiting in children and neuropsychiatric problems in adults, have been reported, though these have probably been over-emphasised. It is the WHO's currently recommended prophylactic for travellers to Papua New Guinea.

Other drugs

Tetracycline or doxycycline are effective prophylactic agents. They are given daily, as is proguanil.

PARASITE DENSITY

This is expressed as the number of parasites seen per number of high powered fields (HPF) examined, eg 150/10 means 150 parasites were seen in a total of 10 HPF (ie 15 parasites per HPF).

- Under 10 parasites per HPF - mild infection
- 10 - 50 parasites per HPF - moderate infection
- Over 50 parasites per HPF - severe infection

Serial parasite densities are useful in monitoring the progress of an individual patient. If 5% of a patient's red blood cells are parasitised, the prognosis is poor, and recovery is unusual if 10% or more of red cells are parasitised

REFERENCES

- Allen SJ, O'Donnell A, Alexander NDE, Clegg JB. *QJ Med* 89:779-788,1996. Severe malaria in children in Papua New Guinea.
- Allen SJ, Raiko A, O'Donnell A, Alexander NDE et al. *Arch Dis Child Fetal Neonatal Ed* 79:F135-F140,1998. Causes of preterm delivery and intrauterine growth retardation in a malaria endemic region of Papua New Guinea.
- Berkley J, Mwarumba S, Bramham K et al. *Trans Roy Soc Trop Med Hyg* 93:283-286,1999. Bacteraemia complicating severe malaria in children.
- Bojang KA, Palmer A, Boele van Hensbroek M et al. *Trans Roy Soc Trop Med Hyg* 91(5):557-561,1997. Management of severe malarial anaemia in Gambian children.
- Bojang KA, Van Hensbroek MB, Palmer A et al. *Ann Trop Paeds* 17:355-359,1997. Predictors of mortality in Gambian children with severe malarial anaemia.
- Crane GG. *PNG Med J* 20:6-14,1977. Tropical splenomegaly.
- Crawley J, Waruiru C, Mithwani S et al. *Lancet* 355:701-706,2000. Effect of phenobarbital on seizure frequency and mortality in childhood cerebral malaria: a randomised controlled intervention study.
- Editorial. *Lancet* 1:511-512,1988. Mosquito control and malaria.
- Krogstad DJ. *New Engl J Med* 320:1538-40,1561,1988. Prophylaxis and treatment.
- Marsh K, Forster D, Waruiru C et al. *New Engl J Med* 332(21):1339-1404,1995. Indicators of life threatening malaria in African children. (Editorial commentary by Taylor TE and Molyneux ME, p.1441-1442).
- PNG Med J* 35(4):227-318,1992. Focus issue on malaria.
- Seaton RA, Trevett AJ, et al. *Ann Trop Med Parasitol* 92(2):133-139,1998. Randomised comparison of intramuscular artemether and intravenous quinine in adult Melanesian patients with severe or complicated *Plasmodium falciparum* malaria in Papua New Guinea.
- Shankar AH, Genton B, Semba RD et al. *Lancet* 354:203-209,1999. Effect of vitamin A supplementation on morbidity due to *Plasmodium falciparum* in young children in Papua New Guinea: a randomised trial.
- Shann F. *J Pediatr* 106:506-510,1985. Quinine in children.
- Vince JD. *PNG Med J* 35:258-263,1992. Malaria in children in Papua New Guinea.
- Warrell DA. *New Engl J Med* 306:313-19,1982. Dexamethasone harmful in cerebral malaria.
- White NJ. *New Engl J Med* 335(11):800-806,1996. The treatment of malaria.
- White NJ. *Lancet* 1:708-711,1987. Hypoglycaemia in children with malaria.
- WHO. The management of severe and complicated malaria. A practical handbook, 1991.
- WHO. *Trans Roy Soc Trop Med Hyg* 94(Supplement 1),2000. Severe *falciparum* malaria.

MALIGNANT DISEASE

GENERAL PRINCIPLES

1. Malignant disease in children in countries such as PNG is not rare, but it is swamped by infectious diseases.
2. Children often present at a late stage of the disease.
3. Anti cancer treatment is usually very unpleasant, may be fatal and is expensive.
4. Some malignant diseases of children (in particular, Burkitt's tumour, Wilm's tumour, and acute lymphoblastic leukaemia type L1) have a surprisingly good prognosis if treated early. An aggressive approach to the treatment of children with early presentation of these malignancies is therefore followed in some (but not all) base hospitals.
5. It is often better to alleviate suffering than to attempt a cure. If the children have advanced disease or have a malignancy with a very poor prognosis, the doctor's role is to:
 - a. provide adequate analgesia and sedation
 - b. ensure adequate nursing care
 - c. ensure adequate bowel and bladder function
 - d. help prepare the parents and family for the child's death.

Parents of a dying child may wish to use alternative or traditional medicines. They will also probably want their church pastor or priest to be involved. At the end, they will probably want to take the child home to die in familiar surroundings with the support of their family.

It is most important that the doctor be available to explain the problems and to provide support. The parents' and family's wishes and beliefs must be respected at all times.

CLINICAL PRESENTATION OF CHILDREN WITH MALIGNANT DISEASE

1. General:
 - weight loss
 - recurrent fevers
 - failure to respond to normal treatment
2. Specific:
 - masses, swellings
 - anaemia, bleeding
 - signs of a space occupying lesion.

DIFFERENCES BETWEEN CHILDHOOD AND ADULT MALIGNANCIES

Some malignancies are specific to childhood:

- Nephroblastoma
- Neuroblastoma
- Retinoblastoma
- Medulloblastoma
- Hepatoblastoma

Some types of haematological and lymphoid malignancies are more common in children than adults. For example, acute lymphoblastic leukaemia type L1 and Burkitt's tumour.

Childhood carcinomas are uncommon. Most tumours are deep rather than superficial.

BURKITT'S TUMOUR

This is a common tumour in malarious areas in parts of equatorial Africa and Papua New Guinea. Most cases occur in children 3-12 years old. The usual presentation is as a facial tumour, but it may present as an abdominal tumour, as paraplegia or in other ways. Generalised lymphadenopathy is rare.

Biopsy material should always be sent for histology before starting treatment, because other childhood tumours may mimic Burkitt's lymphoma.

There is often a dramatic response to chemotherapy. Because symptoms often disappear rapidly, patients often default from treatment and then relapse. The importance of having the full course of treatment must be carefully explained before chemotherapy is begun.

It is also very important to warn the parents that the treatment is very hard for the child - and that they may become very sick - and perhaps even die with the treatment. The child's hair is likely to fall out, he/she is likely to vomit and is likely to feel very unwell. However, without treatment the child will die in a short space of time.

It is very important for doctors and other health workers to realise that Burkitt's tumour, though it is in theory a curable disease, is a very rapidly progressive form of cancer. Any delay at all in diagnosis and treatment is likely to adversely affect the outcome. Every effort should be made to get a biopsy done and treatment started within 2 to 3 days of admission.

Treatment

Consists of:

1. Allopurinol 10-20 mg/kg/day ($\frac{1}{2}$ tab TID for those <20 kg and 1 tab TID for those >20 kg). This is started 24 hours before starting chemotherapy and continued for at least a week.
2. Intravenous chemotherapy
 - a. If there is likely to be delay in diagnosis, give:
 - cyclophosphamide 30 mg/kg
 - b. If the diagnosis is confirmed, give quadruple therapy:
 - cyclophosphamide 30 mg/kg
 - vincristine 0.04 mg/kg
 - methotrexate 0.8 mg/kg
 - adriamycin 0.5 mg/kg

Intravenous quadruple chemotherapy is given every 3 to 4 weeks, depending on WCC and platelet count for a total of 6 doses if possible.

3. Antiemetic. Give metoclopramide (Maxolon) 0.1-0.2 mg/kg every six hours as required. This is very important. It should not be accepted that children on chemotherapy inevitably vomit.
4. Adequate intravenous fluids. Keep an intravenous line with 4.3% dextrose saline running at least maintenance rate to ensure adequate urine output.
5. Intrathecal methotrexate
 - a. Do a lumbar puncture
 - b. Collect 15 drops of CSF for bacteriology and cytology (if available)
 - c. Gently inject methotrexate 10 mg if child < than 5 years, 15 mg if child > than 5 years.

Intrathecal methotrexate is given with the first four courses of intravenous chemotherapy.

Monitoring

Haemoglobin, WCC and platelet count should be checked before each course of chemotherapy. If the haemoglobin is less than 6 g/dl, packed cells should be given. If the WCC is less than 3,000, or the platelet count less than 100,000/dl chemotherapy should be delayed until the counts have risen. Prednisolone in a dose of 1 mg/kg/day will help to raise the WCC.

Prophylaxis

In the parts of PNG where Burkitt's tumour is most common, tuberculosis is also very common. It is therefore sensible to have the patients on prophylactic INAH (5 mg/kg/day) for the duration of treatment. If the chemotherapy reduces the WCC dramatically, it is also wise to have the patients on prophylactic cotrimoxazole (4-5 mg/kg/trimethoprim, 20-25 mg/kg sulphamethoxazole daily).

Radiotherapy

This has little, if any, part to play in curing the disease but can be used to reduce tumour mass or to treat local symptoms (eg to hasten recovery after laminectomy for spinal cord compression).

LEUKAEMIA

There are different patterns in tropical and temperate climates.

Acute myeloid leukaemia (AML) appears to be more common than acute lymphoblastic (ALL).

Common ALL that peaks at 3-5 years does not seem to occur in PNG.

Chloroma-type myeloid leukaemia appears to be relatively common.

Presentation is often late because of masking of signs and symptoms by commonly occurring problems.

Clinical presentation

Bleeding, anaemia, and recurrent infection

Enlarged spleen and lymph nodes

Non specific - for example, PUO.

Histological classification

Acute lymphoblastic, acute non-lymphoblastic, other non specified

FAB classification: ALL 1-3

M1-M7 classification on cell markers (immunophenotype).

Treatment

Acute lymphoblastic leukaemia FAB L1

ALL FAB1 is potentially curable. The five year survival in Western countries is 70% or more with treatment.

- 1. Remission induction**
Vincristine 1.5 mg/m² IV weekly for 6 weeks
Prednisolone 40 mg/m² daily oral for 6 weeks
FBC and bone marrow at 6 weeks
- 2. Consolidation**
Continue above, with addition of doxorubicin (Adriamycin) 20 mg/m² or daunorubicin 20 mg/m² weekly for a further 6 weeks
- 3. CNS prophylaxis**
Intrathecal methotrexate 12mg/m² twice a week for 5 doses after bone marrow remission
- 4. Maintenance**
Methotrexate 20mg/m² IV weekly, or 6-m mercaptopurine 50 mg/m² oral daily for 2 years.

Acute lymphoblastic leukaemia - other types

Although the prognosis is less favourable, it may be worth attempting a cure.

ALL FAB L3 is equivalent to haematogenous Burkitt's tumour - use the Burkitt's treatment regimen.

Acute myeloblastic leukaemia

AML prognosis is poor, and it may be kinder not to attempt treatment in PNG. Treatment in the developed world involves very aggressive chemotherapy and bone marrow transplantation.

NEPHROBLASTOMA (WILM'S TUMOUR)

Wilm's tumour is a curable disease if it is diagnosed in Stage 1 and treated appropriately (surgery and relatively inexpensive chemotherapy is available in PNG).

Clinical presentation

The most frequent presentation is with an asymptomatic abdominal mass which has often been discovered "accidentally".

Wilm's tumour is also associated with aniridia, hemihypertrophy, genito-urinary abnormalities, renal abnormalities (the WAGR syndrome), neurofibromatosis and Beckwith Weidemann syndrome.

The mean age of presentation is 3½ years.

Macroscopic haematuria is uncommon, but microscopic haematuria is present in 30%.

Staging

- Stage 1: Limited to kidney (and completely resected)
- Stage 2: Extends beyond kidney but completely resected
- Stage 3: Residual tumour confined to abdomen (includes spillage)
- Stage 4: Haematogenous metastases
- Stage 5: Bilateral renal involvement.

Histology

The degree of differentiation affects prognosis.

Treatment

- Stage 1 and 2: Surgical excision and chemotherapy
 - Vincristine 0.04 mg/kg IV
 - Actinomycin D 75 microgram/kg IVChemotherapy is given before surgery, at 6 weeks post operatively, and then 3 months later (Stage 1) or at 3, 6, 9, 12 and 15 months later (for Stage 2).
- Stage 3 and 4: Surgical excision with more aggressive chemotherapy and radiotherapy.

Prognosis

- Stage 1: 90-95% cure. Treatment should be immediate.
- Stage 2: With favourable histology, has an 80% chance of cure.

CHILDREN SUSPECTED OF HAVING WILM'S TUMOUR DESERVE EMERGENCY MANAGEMENT.

NEUROBLASTOMA

- Can arise at any site containing cells derived from neural crest.
- Majority arise in abdomen - particularly adrenal gland.
- Abdominal tumours may present with or without pain, anorexia, vomiting weight loss and a mass.
- May present with mediastinal compression, dyspnoea and cough.
- May present with proptosis.
- Bone pain and anaemia.
- Spinal cord compression.
- Rarely profuse diarrhoea due to production of VIP.
- Rarely opsoclonus-myoclonus syndrome of cerebellar ataxia (Dancing Eyes Syndrome).
- Elevated levels of catecholamine metabolites in urine.

Staging

Stage 1: Confined to organ or structure of origin
Stage 2: Extending beyond organ but not across midline \pm regional node involvement
Stage 3: Extending across midline \pm regional nodes bilaterally
Stage 4: Remote disease
Stage 4S: As in 1 or 2 except for remote disease in liver skin or bone marrow.

Treatment

Surgical excision for Stage 1 and 2.
Chemotherapy \pm radiotherapy depending on age and stage.
Many centres in PNG will elect not to treat neuroblastoma.

Prognosis

Often presents at late stage.
Overall prognosis poor.
Cure may be achieved in early stages.
Spontaneous remission occurs in a high proportion of children less than 1 year old.

RETINOBLASTOMA

60% sporadic, usually unilateral.
40% hereditary, often bilateral involvement.
Retinoblastoma tumour suppressor gene (located on chromosome 13) product is deficient.
In the inherited form, 11-20% develop further primary malignancy in childhood or adult life.

Clinical presentation

Earliest presentation as “Cat’s Eye” reflex.
Visual disturbance, strabismus and nystagmus.
Proptosis.
Extra ocular spread - destroyed orbit, extension along optic nerve.
Fungating mass from orbit.

Treatment

Surgical removal of tumour \pm radiotherapy, chemotherapy.
Palliative enucleation \pm radiotherapy, chemotherapy in advanced cases.

Prognosis

If diagnosed and treated early, and if sporadic form and unilateral tumour, the prognosis is good, with 90% cure.
However, since most cases present late in PNG, the prognosis is unfortunately poor.

OTHER MALIGNANCIES

Lymphomas other than Burkitt’s

These present in a number of ways - but usually as mass lesions or lymphadenopathy. Both Hodgkin’s disease and some favourable histological types of non-Hodgkin’s (non-Burkitt’s) lymphomas are associated with relatively good prognoses. COMP (cyclophosphamide, vincristine, methotrexate and prednisolone) is a commonly used regimen.

Sarcomas

Soft tissue sarcomas include rhabdomyosarcoma (head and neck; vagina, vulva and bladder) and Kaposi's sarcoma. Bone sarcomas include osteosarcoma and Ewing's sarcoma (which can mimic chronic osteomyelitis).

Liver tumours

Hepatoblastoma and hepatoma.

Germ cell tumours

Embryonal sarcoma, and teratoma (sacroccocygeal teratoma).

Central nervous system tumours

These are underdiagnosed in PNG. Gliomas account for 75% of childhood brain tumours in USA. 50-60% of childhood cerebral tumours are infratentorial, and 75% occur in the midline. There is a high incidence of astrocytoma and medulloblastoma in children, and craniopharyngioma is much more common than in adults.

Cerebral tumours present with raised intracranial pressure, focal neurological signs, and endocrine disturbance (especially with craniopharyngioma).

NOTE: Because TB is common in PNG, it is worth starting anti-tuberculous treatment in any child who presents with signs suggesting a CNS tumour.

REFERENCE

Clezy JKA, Martin WMC. Cancer management of the neoplasms in Papua New Guinea - a handbook for doctors. Department of Health, 1990. Rather old, but still useful.

MALNUTRITION - INTRODUCTION

Malnutrition is common in Papua New Guinea. Around one in three children in some parts of the country have a weight lower than the international standard. It is important to note that malnutrition is of major importance, not only because of the classic malnutrition syndromes, but also because of its major effects on immune function and susceptibility to infection, learning ability, stamina and so on. By these indirect effects on body function, malnutrition makes a considerable contribution to the overall high mortality of children in Papua New Guinea. (The effects of malnutrition on intellect are discussed in Grantham-McGregor SM. The effect of malnutrition on mental development. In: Waterlow JC. Protein energy malnutrition. Edward Arnold, 1992).

We do not know enough about how measurements such as weight, height, arm circumference and head circumference correlate with morbidity and mortality. Two prospective longitudinal studies have been published that correlate mortality with age, height and weight in children in developing countries: Lancet 1:1247-1250, 1978 (weight-for-age only) and Am J Clin Nutr 33:1836-45, 1980. These and other studies suggest that there is a good correlation between weight-for-age and mortality and between mid-upper arm circumference and mortality. (The relationships between mortality and anthropometric/nutritional indices are discussed in Malnutrition and mortality, chap 18. In: Waterlow JC. Protein energy malnutrition. Edward Arnold, 1992).

CLASSIFICATION AND DEFINITION

Weight-for-age

The standard method of assessing nutritional status is to express the weight of the child as a percentage of the expected weight at that age (weight-for-age). The expected weight is arbitrarily defined as the median weight (50th percentile) for American children of that age (Harvard Standard Weight), and this is called the 100% weight-for-age of children in Papua New Guinea and other "third world" countries. Children over 80% of their expected weight-for-age are said to be well nourished. The 80% line on the Papua New Guinea weight-for-age chart approximates the 3rd centile of the Harvard Standard Weight. Children 60% to 80% of the expected weight have moderate malnutrition, and those under 60% have severe malnutrition (marasmus). One study in India showed that, on average, child mortality doubled with each 10% decline below 80% of the Harvard Standard Weight (Lancet 1:1247-1250, 1978). But in Bangladeshi children, mortality did not increase until weight-for-age was less than 70% of the Harvard Standard Weight (Am J Clin Nutr 33:1836, 1980). The presence of oedema due to malnutrition indicates severe malnutrition (kwashiorkor) whatever the weight.

The pattern of growth should be considered as well as weight-for-age. A child with a weight that is static or falling for three months or more has malnutrition, even if his or her weight-for-age is over 80%. A child who is steadily gaining weight and who remains at a constant percentage weight-for-age probably does not have malnutrition. However, the importance of the pattern of growth has been challenged by a study in Bangladesh that found that weight-for-age was a better predictor of mortality than change in weight or change in weight-for-age (Br Med J 298:1607-11, 1989).

Change in weight provides a fairly sensitive indication of the adequacy of recent food intake. The weight chart in a child's health record book provides an invaluable tool for following the pattern of growth. The book is essential to proper childcare and every effort should be made to see that all children have a book and a plastic bag to keep it in.

Mid upper arm circumference

Weight-for-age is not an ideal measurement of nutritional status. The age of many children is not known, and counting the number of teeth present then adding six to get the age in months (J Pediatr 64:97, 1964) gives only an approximate estimate of age. Children are often weighed inaccurately, and the weight plotted incorrectly on the graph. Measurement of mid upper arm circumference (MUAC) gives a reasonable assessment of nutritional status in children one to five years of age (Lancet 1:758, 1974 and Lancet 1:87-89, 1987). During this period, the MUAC stays relatively constant, and children with a circumference of less than 14 cm are considered to be malnourished (In Papua New Guinea, 13.5 cm is

taken as the cut off point). MUAC reflects long-term nutritional status, while weight reflects recent change. MUAC may be preferable to weight-for-age in the monitoring of growth in primary health care (see Lancet 2:1337-1338,1985 and Lancet 2:725-728,1987).

Stunting and wasting

Children who are below 80% of the Harvard Standard Weight may be too light because they are very short (stunted, low height-for-age), because they are very thin (wasted, low weight-for-height) or because they are both stunted and wasted. Stunting suggests chronic mild malnutrition or genetic short stature, while wasting suggests recent severe malnutrition. To decide if an underweight child is stunted or wasted, height has to be measured in addition to age and weight. In Bangladeshi children, weight-for-age was the best index of mortality, height-for-age correlated less well, and weight-for-height was the weakest index of mortality (Am J Clin Nutr 33:1836-1845,1980). Similar results have been obtained in children in Papua New Guinea.

REDUCING MALNUTRITION

What can be done to improve nutrition (see Lancet 1:334-336,1988)? Supplementary feeding programmes are expensive, often impracticable, and of questionable benefit (Nutrition Reviews 9:278-280,1978). Control of intestinal parasites may be useful in selected groups (Lancet 2:108-110,1977). Maternal malnutrition causes low birthweight babies (Am J Clin Nutr 28:1223,1975) that fail to thrive (Lancet 2:178,1978). Perhaps more emphasis should be put on improving the nutrition of pregnant and lactating women and in attending to the other antenatal factors that predispose to low birthweight. (Interestingly there is now good evidence that intrauterine growth retardation predisposes to the subsequent development of diabetes and cardiovascular disease in adult life (Barker DJP. Fetal and infant origins of adult disease. BMJ publications 1992). Thus, the benefits of improving antenatal health may have long-term as well as short-term benefits). In the long term, improved nutrition will probably depend on general socio-economic change, the effect of nutrition education and improved female literacy (Lancet 2:487-488,1987).

Nutrition education

The short- term effects of nutrition education should not be overestimated. Salfeld (PNG Med J 17:179-182,1974) showed little change in the nutritional status of a rural community after a year of intensive nutrition education. Care must be taken not to blame malnutrition on the "ignorance" of mothers: if 30% of children are malnourished, then 70% are well fed. Traditional feeding practices may not be perfect, but they have evolved over many generations, and care must be taken that suggested changes to them are soundly based (see Trop Doctor 6:37-42,1976).

Attempts to improve the nutritional status of a community will be most effective if there is close co-operation between the departments of health, agriculture and education, and involve international agencies, rather than separate and often conflicting campaigns by different government and non-governmental agencies. There should be close co-operation between medical staff and the provincial nutritionist. The main emphasis ought to be on education of the community, rather than individual families in hospital.

Nutrition education messages are often far too complicated to be understood by village mothers, and adequate distinction is not made between what should be taught to village people and what should be taught to health workers. For example, teaching about the three food groups is appropriate for nurses, but may not be appropriate for village people. Suggestions must be practical: posters suggesting a protein intake of cheese or a leg of ham are not helpful. Keep your message relevant: people do not have to be told to eat their staple diet (eg rice or sweet potato). Local beliefs about nutrition must be taken into account; for example, for a discussion of beliefs about nutrition in the Papua New Guinea Highlands, see PNG Med J 22:65-71,1979.

What SHOULD be taught?

When to start solid food

Babies fed only on breast milk grow well for 4-6 four months. Therefore, it is recommended that solid food be introduced at four months of age. A recent study showed that a large percentage of Papua New

Guinean mothers introduce solids before the age of 4 months (Friesen et al Ann Trop Paed 1998). Weaning foods in the village situation are often grossly contaminated with bacteria, and may be just as dangerous in this respect as artificial milk feeds (Lancet 1:136-138,1978). If we encourage the introduction of solids at four months of age, we must also encourage their careful preparation, which is difficult in a village setting. Proper studies are needed of the best time to introduce solids to village infants. For community education purposes at present, it is suggested that if a child's age is unknown, solids should be introduced when the child starts to roll over. Encourage mothers to breast feed until their baby is at least two years old. An effective contraceptive should be used once weaning starts. The contraceptive value of breastfeeding is lost when solid foods are introduced.

How often to feed solids

Children have a high energy requirement per kilogram of body weight because of their requirements for growth. Many staples, such as sweet potato, are very bulky, so that a large volume has to be consumed for a given energy intake. If they have only one or two meals a day, children cannot consume a large enough volume of the staple to satisfy their energy requirements. They need 3-4 meals a day with snacks in between, and oil or fat (compact calories) added to the staple to increase the calorie density. The estimated calorie requirements of infants have recently been reduced (Lancet 1:1161,1988).

What to grow in the garden

Wing beans ("as bin"), peanuts and pandanas nuts have a high fat and protein content. Fat is a concentrated source of energy, so that if wing beans or nuts are added to a diet of sweet potato, kumu (greens) and breast milk, the energy content can be substantially increased.

What to buy in the store

If money is available, tinned dripping (tin gris) or margarine gives the largest amount of energy per kina, and it is in a concentrated form (as fat). If the major dietary deficiency is energy rather than protein (Trop Doctor 7:28-32,1977), then dripping may be better value than tinned fish or tinned meat.

Family planning

The birth interval between successive children in a family is a major determinant of their nutritional status. Parents should be strongly encouraged to space their children at least three years apart. As soon as weaning starts, the parents should be encouraged to use a modern contraceptive method to prevent the mother becoming pregnant again.

MALNUTRITION - MANAGEMENT - SUMMARY

MODERATE MALNUTRITION

Moderate malnutrition

Weight 60-80% with a flat or falling weight chart and no oedema

Treat as outpatient.

Advise parents: give one or two of the 6 Nutrition Messages below. Use a flipchart on nutrition if you have one.

Check and treat for diseases. Treat for worms, anaemia, chronic diarrhoea and other infections if present. Rule out resistant malaria and TB.

Admit only if

- other illness present
 - or no improvement after 1 month.
-

SEVERE MALNUTRITION

Severe malnutrition

Weight below 60% and flat or falling weight chart OR there is oedema

Admit.

Treat infection and anaemia (Rule out resistant malaria and TB).

Fatten the child:

- if he will eat, give him food
- if he will not eat, give him MOF.

Dietary education: discuss nutrition messages (Nutrition Unit).

Family planning: discuss available methods.

Drugs: vitamin A, multiple vitamin liquid, folic acid, electrolyte mixture, albendazole, tinidazole, cotrimoxazole, antimalarials, measles and other vaccines (if due or overdue).

THE SIX NUTRITION MESSAGES

1. Start giving soft food as well as breast milk when your child is 4 months old. If you do not know his age, start when he can roll over.
2. Add extra coconut cream, dripping or margarine to the child's food.
3. Feed your children 4-6 times a day.
4. Feed your children cooked and mashed peanuts, beans or fish every day.
5. Continue to feed your child when he is sick and give extra food after sickness.
6. Eat plenty when pregnant or breastfeeding.

MALNUTRITION - DIAGNOSIS

MEASURING AND RECORDING THE WEIGHT

1. WEIGH the child carefully on the most accurate scales available. Zero the scales first.
2. Assess the AGE as accurately as possible, and record it in the notes. NB: If the child has 19 teeth or less, then the approximate age (in months) = number of teeth + 6.
3. Using the WEIGHT CHART, mark the weight in the column above the child's age.
4. Compare the weight with the previous weights.

DECIDING THE CHILD'S NUTRITIONAL STATUS

1. If the weight is **above 80%** and increasing, then the child is well nourished. If the weight is **between 60-80% and increasing**, then the child is also **well nourished**:
 - a. Praise the parents for their well fed child
 - b. If he is over 4 months old, check that he is being given food as well as breast milk
 - c. Use this opportunity to discuss family spacing, if appropriate.
2. If the weight is **60 - 80% and not increasing** (flat or falling weight curve) and the child has **no oedema**, the child has **moderate malnutrition**:
 - a. Give dietary education (p.211)
 - b. Admit if other illness is present or the child does not improve after 1 month.
3. If the weight is below 60% and not increasing, or there is oedema and a flat or falling weight curve, this is severe malnutrition:
 - a. ADMIT the child.

MID UPPER ARM CIRCUMFERENCE (MUAC)

If the child is older than 12 months and the MUAC is less than 13.5 cm, then he or she is likely to be malnourished. If possible, check the weight and plot it on a growth chart.

Risk factors for dying are: previous attendance, young age, low weight-for-age, oedema, hepatomegaly, tuberculosis, large family, single parent family, and a history of the death of a sibling.

MALNUTRITION - INPATIENT TREATMENT

ADMIT:

1. Children with moderate malnutrition (weight over 60-80% with no oedema and a flat or falling weight chart) if not improving after 1 month of outpatient treatment
2. Children with severe malnutrition (weight under 60% - marasmus, OR there is oedema - kwashiorkor - with a flat or falling weight chart).

PROCEDURE OF ADMISSION

1. Take a good history. Information should include:
 - a. The occupation of father
 - b. The age and number of siblings
 - c. The housing conditions and number of persons in the house
 - d. How long the child has not been eating
 - e. What the child has eaten in past 24 hours
 - f. Whether the child is still breast feeding
 - g. What foods are available and culturally acceptable to the family
 - h. Any recent illnesses
 - i. The immunisation history
 - j. Any possible contacts with tuberculosis
 - k. Whether the mother is pregnant
 - l. The mother's interest in or use of family planning
 - m. The family social history, smoking, drinking, gambling etc.
2. Weigh the child and record the weight on the weight chart.
3. Put previous weights, if they are available from the MCH book, on the weight chart to show the growth curve.
4. Measure the child's height and mid upper arm circumference.
5. Assess the severity and type of malnutrition, noting the degree of wasting of fat and muscle, oedema, hair changes, skin changes, misery, apathy and appetite.
6. Assess the child's hydration.
7. Examine conjunctivae and cornea for signs of Vitamin A deficiency (xerosis, Bitot's spots, keratomalacia).
8. Note the presence and severity of pallor.
9. Note the size of the spleen and liver.
10. Look for signs of infection of the skin, chest, mouth and ears.
11. Record the child's temperature - be aware of the possibility of hypothermia.
12. Look for signs of heart failure. Children with kwashiorkor have a reduced amount of heart muscle (Arch Dis Child 63:1359-62,1988).
13. Do some routine tests:
 - a. Mantoux
 - b. Chest x-ray
 - c. Hb and film
 - d. Stool for parasites (especially Strongyloides) if the child has oedema
 - e. Urine for protein if oedema is present
 - f. Urine for microscopy and culture
 - g. Stools for sugar and culture if diarrhoea present
 - h. Blood slide for malaria if febrile

- i. Blood for liver function tests if oedema is present
 - j. Fasting gastric aspirates for AFB if tuberculosis likely
 - k. Mantoux and chest x-ray household contacts if tuberculosis likely
 - l. Lumbar puncture, blood culture and urine culture if the child looks ill, whether he is febrile or not
 - m. Blood sugar, to detect hypoglycaemia, as well as lumbar puncture and blood slide for malaria, if child has a convulsion.
14. Think of the possibility of HIV infection. If you think the child may have HIV infection, take blood from mother and child for HIV antibody tests - but only after you have explained what you are doing and with mother's permission.

FEEDING

1. If the child will eat, give breast milk plus a well-balanced high protein diet (see below).
If the child will not eat, insert a nasogastric tube and give milk oil formula (MOF) (see p.231).
If the child has diarrhoea, do NOT give MOF but treat as below (see IF THE CHILD HAS DIARRHOEA, p.217).
2. Multiply child's weight in kg by 30 to obtain number of mls of milk to give each feed (maximum 240 ml/feed), eg a child weighing 7 kg needs $7 \times 30 = 210$ ml/feed.
3. Give 6 or 7 feeds a day of milk. This will provide about 6 g of protein/kg/day and about 200 Cal/kg/day.
4. Encourage the child to eat: good foods to give are mashed ripe banana, bread or wheat meal with margarine, ground-up roasted or boiled peanuts, sweet potato, rice etc. As the child's appetite returns, decrease the milk oil formula (MOF) to 3 times a day and increase the amount of food given.

Feeding programme	
6 am	Meal plus MOF
9 am	Snack plus MOF
Noon	Meal plus MOF
3 pm	Snack plus MOF
6 pm	Meal plus MOF
9 pm	Snack plus MOF

Meals should include:

- A staple (kau kau, taro, yam, sago or rice)
- plus some meat or fish
- plus dark green leaves (aibika, pumpkin tips or sweet potato tops)
- plus coconut cream, dripping or margarine if available.

Snacks:

- high-protein biscuits, peanut paste, peanut balls, banana, pawpaw, egg or milk balls.

To make peanut balls:

- mix together 2 cups of mashed sweet potato (or taro, yam or sago) and a quarter of a cup of peanut paste. Roll into balls of about one teaspoonful and leave to dry.

To make milk balls:

- mix together 6 tablespoons of milk powder, 1 tablespoon of sugar and 1 tablespoon of cocoa. Then mix in 3 tablespoons of water. Roll into balls of about one teaspoonful and leave to dry.

Note: If a good diet is not possible or practical, give milk feeds 4-6 times daily.

DRUGS

1. Give oral vitamin A immediately.

under 1 year:	½ concentrated capsule or tablet (100,000 units)
1 year or more:	1 concentrated capsule or tablet (200,000 units)

Give 2 more doses, the second on the second day and the third after one week.

- Multiple vitamin liquid daily (check instructions on the container).
- Folic acid 1 tablet daily.
- Albendazole (must be crushed or chewed)

Weight	Dose	No oedema	Oedema
5-9.9 kg	1 tab	Stat dose	Daily for 3 days
10 kg or more	2 tab	Stat dose	Daily for 3 days

If you have no albendazole, use mebendazole (see p.393).

- Tinidazole: give once daily for 3 days (see p.396).
 - Cotrimoxazole (Septrin) twice a day for 7 days (see p.391).
- Note: Use ampicillin/amoxycillin (or benzyl penicillin) and gentamicin (or streptomycin) if the child has severe kwashiorkor or severe marasmus (see pp.389-396).
- Check for enlarged spleen and ask about recurrent fevers. Treat for resistant malaria (TFM) if present (see p.199). Otherwise, treat for uncomplicated malaria (see p.197). Give weekly prophylaxis for up to 3 months (if in a malarial area) or till the child is no longer malnourished (see p.201).
 - Give measles and other vaccines if the child is due or overdue for them (see p.163).
 - Nystatin 1 ml 3 times a day or Gentian Violet 2 times a day if the child has thrush.
 - Electrolyte mixture (zinc sulphate, magnesium sulphate and potassium chloride) 5 ml daily. This can be added to milk during preparation. See p.230).

ACCOMPANYING INFECTIONS AND CONDITIONS

- Treat associated infection (eg otitis media, pneumonia, scabies and other skin infections).
- Treat anaemia, hypothermia and hypoglycaemia.
- Give a full course of antimalarials.
- Give IM or IV ampicillin and gentamicin if the child looks ill, is hypothermic (less than 36 °C) or is febrile from an unexplained cause. Obtain specimens of CSF, urine and blood for culture first.
- Give a blood transfusion if the haemoglobin is below 6 g/dl. Give frusemide 1 mg/kg IV with the transfusion, unless the child is dehydrated.
- Check for tuberculosis - use the TB score chart (and CXR). Note that failure to increase weight on nutritional treatment will increase the TB score. If the TB score is 7 or more, start TB treatment.
- HIV infection is a rapidly increasing problem in Papua New Guinea. The clinical presentation can easily be mistaken for malnutrition and its associated immunodeficiency state. Children with advanced AIDS may be seronegative.

IF THE CHILD HAS DIARRHOEA

DO NOT GIVE MILK OIL FORMULA (MOF).

Mild diarrhoea (no dehydration)

Test the stools for sugar (p.106):

- If there is no sugar in stool: give breast feeds and full strength full cream milk (p.231) with Electrolyte Mixture (p.230) 5 ml added to each 240 ml of milk, but NO oil. Change to MOF when the diarrhoea has stopped. Continue feeding the child with solid food if he will take it.
- If sugar is present in the stool: stop breast feeds for 2-3 days and give a lactose-free milk (eg Digestelact, Ensure, Pregestimil, Infasoy, or Nutramigen, if available, see p.231) with Electrolyte Mixture (p.230) 5 ml added to each 240 ml of milk, but NO oil. Make sure the mother keeps her breasts expressed and make sure that baby goes back to breastfeeding as soon as the diarrhoea has stopped. Continue feeding the child solid food if he will take it.

Severe diarrhoea (with dehydration)

- give half strength Darrow's solution (see p.133)
- give Electrolyte Mixture 5 ml TID (p.230)
- give nystatin 1 ml orally QID if oral thrush is present
- give tinidazole orally daily for 3 days or metronidazole (Flagyl) orally TID for 5 days
- test the stools for sugar (p.106). If there is no sugar in the stool, continue breast feeds, and GRADUALLY introduce MOF once the diarrhoea has stopped. If sugar is present in the stool, stop breast feeds and give Digestelact (or other lactose-free milk) 30 mg/kg (maximum 240 ml/feed) 3 hourly 7 times a day. Sometimes, diarrhoea will persist despite these measures. Stop all oral feeds and give IV feeding using Aminofusin and dextrose until the diarrhoea stops, then give Pregestimil or Ensure milk if available, or other lactose-free milk if not (p.231).

NURSING CARE

1. Keep the child warm, and away from open windows, doors and fans. In colder highlands regions, an electric blanket is often needed (see p.119).
2. Warm the milk feeds before giving them by tube.
3. Involve the mother in the care and feeding of her child.
4. The child should sleep beside the mother at night to keep warm, and be carried around by the mother in the day time to prevent hypostatic pneumonia.
5. Keep careful records of food and fluid intake and fluid loss (diarrhoea, vomiting).
6. Keep the child away from other children with infections as far as is possible.

FAMILY PLANNING

If one child in the family is already malnourished, and the mother has another baby, then there will be less food for each child. Discuss with the mother (and if possible the father), the benefits of family planning and suggest she attend the family planning clinic.

For family planning tell them about:

- condoms
- the injection (medroxyprogesterone)
- the pill
- the loop
- the ovulation method.

For family completion tubal ligation (or vasectomy) is available.

If they wish to accept a method arrange this for them.

EDUCATION

Education of the mother and family on nutrition, child care and family planning is an essential part of the treatment of a malnourished child. If this is not done during the child's stay in hospital, hospital treatment is likely to be an expensive waste of time. Advice given to the family must be based on a knowledge of the socio-economic and cultural background of the family and community. It is important

to involve the mother as much as possible in the nutrition of her child whilst in the hospital. A nutrition unit is the best place for this, so that she can be involved in the food preparation, and learn the value of a balanced but simple and affordable diet. Home visiting and follow-up after discharge are very important.

Nutrition education must be kept SIMPLE and PRACTICAL. Do not try to teach uneducated parents detailed information on nutrition that only health workers need to know.

There are SIX basic nutrition messages to tell parents:

1. START GIVING SOLID FOOD AS WELL AS BREAST MILK WHEN YOUR CHILD IS FOUR MONTHS OLD. IF YOU DO NOT KNOW HOW OLD HE IS, START WHEN HE STARTS TO ROLL OVER
2. FEED YOUR CHILDREN 4 - 6 TIMES EVERY DAY
3. ADD SOME COCONUT CREAM, DRIPPING OR MARGARINE TO THE CHILD'S FOOD
4. FEED YOUR CHILDREN COOKED AND MASHED PEANUTS OR BEANS OR FISH EVERY DAY
5. CONTINUE TO FEED YOUR CHILD WHEN HE IS SICK AND GIVE EXTRA FOOD AFTER SICKNESS
6. EAT PLENTY WHEN PREGNANT OR BREASTFEEDING.

REFERENCES

- Ahmed T, Ali M, Ullah MM et al. Lancet 353:1919-1922,1999. Mortality in severely malnourished children with diarrhoea and use of a standardised management protocol. (Editorial commentary by Waterlow J, p.1142).
- Berkowitz FE. Am J Dis Child 138:551-556,1984. Bacteraemia in malnutrition.
- Biddulph J. PNG Med J 15:259-262,1972. Management of severe malnutrition.
- Editorial. Lancet 2:1337-1338,1985. The role of growth monitoring.
- Gibson RS. Lancet 355:2008-2009,2000. Zinc supplementation for infants.
- Humphrey JH, Rice AL. Lancet 356:422-424,2000. Vitamin A supplementation of young infants.
- Lepage P. Lancet 1:1458-1461,1987. Bacteraemia common in febrile outpatients.
- Morley D, Woodland M. See how they grow. London: Macmillan, 1979.
- Peltola H. Pediatr Infect Dis J 7:488-92,1988. Intestinal parasites in Somalia.
- PNG Med J 31(2):87-145,1988. Focus issue on nutrition.
- Shankar AH, Genton B, Semba RD et al. Lancet 354:203-208,1999. Effect of Vitamin A supplementation on morbidity due to Plasmodium falciparum in young children in Papua New Guinea: a randomised trial.
- Topley JM. Arch Dis Child 63:842-4,1988. HIV infection in Zimbabwe.
- Umeta M, West CE, Haidar J et al. Lancet 355:2021-2026,2000. Zinc supplementation and stunted infants in Ethiopia: a randomised controlled trial.
- Waterlow JC. Protein energy malnutrition. Edward Arnold, 1992. Available from TALC.
- WHO. Measuring change in nutritional status. Geneva, 1983.
- WHO. Management of severe malnutrition: a manual for physicians and other senior health workers. Geneva, 1999.

MALNUTRITION - OUTPATIENT TREATMENT

Children with **moderate malnutrition** (underweight) ie **weight 60-80%** and **no oedema** and **a flat or falling weight curve**.

1. Discuss the problem with the parents:
 - a. Try to find the reason for the malnutrition, eg
 - i. soft food was started later than 4 months of age
 - ii. not enough high energy and protein foods given
 - iii. not enough food for the family
 - iv. only one or two meals of solid food each day
 - v. too much 'rubbish' food (sweet biscuits, cheese pops, lollywater)
 - vi. the child has a chronic infection (gastroenteritis, malaria, tuberculosis, urinary infection).
 - b. Discuss nutrition and family planning with the parents (use a flipchart if you have one).
Encourage the mother to:
 - i. continue breast feeding
 - ii. give food 4-6 times a day
 - iii. add coconut cream, dripping or margarine to the child's food
 - iv. grow peanuts and beans for the child to eat
 - v. commence a suitable family planning method and arrange this for her if she and her husband are willing.
2. Give a single dose of albendazole (see p.389).
3. Examine for any infection or anaemia: treat if present.
4. Ask about chronic diarrhoea (more than 1 week) and treat if present (see p.103).
5. Check for enlarged spleen and ask about recurrent fevers.
Treat for resistant malaria (TFM) if present (see p.199).
Otherwise, give a 3-day course of infant Camoquin (see p.389).
Give weekly prophylaxis for up to 3 months (if in a malarial area) or till the child is no longer malnourished (see p.389).
6. Check for tuberculosis by performing a TB score (see p.370). Commence treatment if indicated.
7. Refer to Nutrition Unit, if available, and arrange for regular follow up to check progress.

IF THERE IS NO WEIGHT GAIN AFTER 1 MONTH:

- Suggest admission to hospital or health centre.

IF MOTHER REFUSES ADMISSION:

1. Give as much of the inpatient investigation and treatment as you can.
2. Teach the mother one or two important nutrition messages and give a demonstration, if possible.
3. Arrange for the MCH sisters to make home visits.

Malnutrition: outpatient treatment - summary

STEP 1:	Discuss with the parents: more food more often/family planning (Use a flipchart if you have one)
STEP 2:	Albendazole
STEP 3:	Examine for any infection or anaemia: treat if present
STEP 4:	Treat for chronic diarrhoea if present
STEP 5:	Treat for resistant malaria if present
STEP 6:	Do TB score

MEASLES

1. Treat as an outpatient where possible.
2. Admit children with measles to hospital if they look very sick, or if they are malnourished, or if they have one or more of the following complications:
 - a. pneumonia
 - b. a dark staining rash
 - c. severe diarrhoea
 - d. stridor
 - e. convulsions
 - f. severe oral thrush
 - g. difficulty with drinkingDark staining of the skin suggests severe measles.
3. Because measles is a viral illness, treatment with antibiotics is not helpful in most cases, unless there is pneumonia or otitis media.

TREATMENT

1. Treat fever over 38 °C with paracetamol.
2. Give antimalarials if the child is febrile.
3. Give extra fluids if the child has diarrhoea.
4. Treat conjunctivitis with antibiotic eye ointment if there is pus in the eyes.
5. Treat pneumonia and otitis media if present.
6. Give 2 doses of Vitamin A on consecutive days. Give A 100,000 IU by mouth to children less than 12 months old, and 200,000 IU to children over 12 months old (Lancet 1:1076-8,1987).
7. Give measles vaccine to all the child's siblings who are between 3 months and 5 years of age and who have not been vaccinated. If you are not sure if the child has measles or not, give him measles vaccine as well (Note: measles vaccine given between 3-5 months is an extra dose. The 6 months dose should still be given).
8. Make sure the other children in the ward have had measles vaccination. If they have not and they are between the ages of 3 months and 5 years, give the vaccine (Note: Hospital acquired measles has a higher mortality than community acquired measles).
9. Treat oral thrush if present.

Measles has an incubation period of 7-14 days, and is infectious from two days before the onset of the rash. About one case in every 1,000 gets encephalitis (these children are drowsy, and have convulsions, focal neurological signs and a high CSF protein).

Measles may cause an exacerbation of tuberculosis. Measles has a bad effect on nutrition. After an attack of measles a child's nutrition must be monitored and nutrition education given if necessary. The high mortality from measles in developing countries is usually attributed to malnutrition and young age at the time of infection, but it has been suggested that outbreaks where many cases occur together (clustering) are more important (Br Med J 296:1225-8,1988). An increased perinatal mortality has been reported in children of mothers exposed to measles during pregnancy (Lancet 1:516-519,1988).

Koplick spots are not always easy to see. On dark skin, the rash is easily missed by an inexperienced observer. Use oblique lighting and look for raised lesions on a dull skin.

MEASLES CAN BE PREVENTED BY IMMUNISATION.

There is now overwhelming evidence that measles vaccination reduces mortality (Pediatr Infect Dis J 8:197-200,1989). If measles does occur in vaccinated children, the disease is milder than in unvaccinated children (J Infect Dis 154:858-63,1986). The Papua New Guinea immunisation policy is to

give 2 doses of standard titre Schwarz strain live attenuated measles vaccine at 6 months and at 9 months. It is vital that all unimmunised children admitted to hospital for whatever reason should be vaccinated as part of admission procedure (there are no contraindications).

Papua New Guinea has the dubious distinction of having the highest incidence of subacute sclerosing panencephalitis (SSPE) ever reported. Reasons for this are probably multiple but include the young age of affected children (see p.358).

Every effort must be made to immunise at every opportunity.

REFERENCES

- Editorial. *Lancet* 1:1067-8,1987. Vitamin A for measles.
Rev Inf Dis 10:451-99,1988. Symposium on crowding and mortality from measles.
Aaby P, Samh B, Simonden F et al. *Br Med J* 311:481-485,1995. Non-specific beneficial effect of measles immunisation: analysis of mortality studies from developing countries.
Cutts FT, Steinglass R. *Br Med J* 316:765-767,1998. Should measles be eradicated?
Cutts FT. Measles. In: *The immunological basis of immunisation*, ch 7. Geneva: WHO, 1993.
D'Souza RM, D'Souza R. *Cochrane Database Review* (1),CD001479,2002.
Duke T, Mgone C. *Lancet* 2002 (in press). Measles; not just another exanthem.
Hall AJ, Cutts FT. *Br Med J* 307:1294-1295,1993. Lessons from measles vaccination in developing countries.
Semba RD, Munasir Z, Beeler J et al. *Lancet* 27:1330-1332,1995. Reduced seroconversion to measles in infants given vitamin A with measles vaccination. (Editorial commentary by Ross D, p.1317-1318). WHO. Recommended strategies for the prevention and control of Communicable Diseases . WHO Geneva.

MENINGITIS

See also Tuberculosis, (p.367); Neonates - Meningitis (p.262); Meningitis - Cryptococcal (p.227); and Cerebrospinal Fluid Examination (p.70).

DIAGNOSIS

If meningitis is a possibility, a lumbar puncture **MUST** be done **UNLESS** there is evidence of papilloedema or the child is comatose or desperately ill. If you cannot obtain CSF, or if it is blood stained, or if it is unsafe to do an LP, treat the child for both meningitis and cerebral malaria (p.197).

- if the CSF is cloudy, start treatment immediately
- if the CSF looks clear, obtain the results of the microscopic examination before concluding that the child does not have meningitis. If the child is sick and you cannot get an immediate microscopic examination, treat for meningitis and cerebral malaria (p.197) until you get the result.

TREATMENT

Antibiotics

Chloramphenicol is still the drug of choice in Papua New Guinea. There is no evidence that outcome with third generation cephalosporins is better than that with chloramphenicol (providing the bacteria is sensitive) and they are very expensive. Chloramphenicol given alone is as effective, if not more so, than a combination of chloramphenicol and crystalline penicillin. Intramuscular chloramphenicol is highly effective, and forms the basis of standard management. In hospital, chloramphenicol is often given intravenously.

Unfortunately, chloramphenicol resistant *Haemophilus influenzae* currently accounts for between 20-30% of *Haemophilus* isolates in Goroka and Port Moresby. In hospital practice where the diagnosis of bacterial meningitis can be confirmed by CSF examination it is reasonable to commence children less than 1 year of age on a third generation cephalosporin if available. Ceftriaxone is the most convenient (50mg/kg BD for 10 days). Chloramphenicol can be substituted if the *Haemophilus* is sensitive or with pneumococcal or meningococcal meningitis.

Hopefully, the introduction and use of the *Haemophilus influenzae* type B vaccine will greatly reduce the incidence of *Haemophilus influenzae* infections as has occurred in many other countries of the world.

So far, pneumococci in Papua New Guinea have remained sensitive to chloramphenicol, but multidrug resistant organisms are causing major problems in some countries.

Give chloramphenicol for 14 days:

Give chloramphenicol 25 mg/kg (maximum 500 mg in children less than 30 kg) 6 hourly for 14 days. Start by giving intramuscular or intravenous chloramphenicol, adding 4 ml sterile water to each 1 gram vial (which gives 1 gram in 4.75 ml).

Weight	No. of ml	Weight	No. of ml
3-4.9 kg*	½ ml	15-19.9 kg	2 ml
5-6.9 kg	¾ ml	20-29.9 kg	2½ ml
7-9.9 kg	1 ml	30-49.9 kg	3 ml
10-14.9 kg	1½ ml	Adult	4¾ ml
*for babies less than 1 month old, see Neonates - Drug Doses, p.245			

When the patient is afebrile and looks well (usually after 3 to 5 days), change to oral chloramphenicol every 6 hours, except in those under 3 months and in severely malnourished children in whom absorption of oral chloramphenicol is unpredictable (absorption depends on the hydrolysis of chloramphenicol palmitate by a gut lipase - which may be deficient in the very young and the malnourished).

Weight	Oral chloramphenicol
3-4.9 kg	4 ml suspension
5-6.9 kg	6 ml suspension
7-9.9 kg	8 ml suspension
10-14.9 kg	12 ml suspension or 1 capsule
15-19.9 kg	15 ml suspension or 1 capsule
20-49.9 kg	2 capsules
Adults	4 capsules
If the child vomits up the oral chloramphenicol or will not take it, change back to giving intramuscular chloramphenicol.	
GIVE CHLORAMPHENICOL FOR 14 DAYS.	

If you do not have any chloramphenicol (or ceftriaxone), give benzyl (crystalline) penicillin 1,000,000 units vial diluted with 2 ml sterile water 0.2 ml/kg (maximum 4 ml) IV or IM every 6 hours plus probenecid.

Antimalarials

If the results of CSF examination confirm meningitis and if the blood slide is negative for MPS, treat the child as for uncomplicated malaria.

If the results are not available or not clear cut, treat the child for both meningitis and severe (cerebral) malaria (Treatment B, p.197).

Anticonvulsants

1. Stop the fit.

If the child convulses, it is most important to stop the convulsion (because it will further increase intracranial pressure and cerebral oedema). Stop the convulsion with IM paraldehyde or IV or rectal diazepam.

2. Prevent fits.

Children older than 2 years who have had a convulsion and all children under 2 years (irrespective of whether or not they have had a convulsion) are treated with phenobarbitone (two out of three children under the age of 2 years with meningitis are likely to have a fit during the course of the illness). A loading dose of 15 mg/kg is given IM and then daily maintenance of 5 mg/kg until the child has recovered. In those children who have had repeated convulsions, phenobarbitone is continued.

In general, phenytoin should not be used in combination with chloramphenicol. However, in practice, it is often useful when phenobarbitone is ineffective in controlling seizures.

Fluids and feeding

There is a high risk of cerebral oedema if patients with meningitis are overhydrated (Arch Dis Child 60:963-966,1985), so that IV fluids are best avoided in meningitis unless they can be closely monitored. Therefore, most children with meningitis in Papua New Guinea are probably best managed WITHOUT an IV drip (Med J Aust 1:577-78,1981; Lancet 2:681-684,1985). In a shocked or dehydrated child, IV fluids and IV chloramphenicol should be given.

In a child who is not feeding after 24-48 hours of antibiotic therapy, it will be necessary to give nasogastric feeding as per the "coma" regimen in the STB. Give EBM or FSM 4 times a day:

	NG milk or ORS if dehydrated (ml QID)	IV fluids if dehydrated or shocked (ml per hour)
3-5 kg	100	20
6-9 kg	150	25
10-14 kg	200	50
15-19 kg	250	75
20-29 kg	300	75
30-49 kg	350	75

Steroids

The use of steroids in bacterial meningitis is still controversial. There is evidence that steroids given before the start of treatment of *Haemophilus meningitis* with a third generation cephalosporin reduce the incidence of sequelae. The evidence in relation to pneumococcal meningitis and to the use of other antibiotics is not clear (New Engl J Med 319:1012-4, 1988). There are increased risks of using steroids in countries where tuberculous and parasitic meningitis and brain abscess are common, and where diagnosis is often imprecise. Steroids should not be given routinely to children with bacterial meningitis in Papua New Guinea (Trop Doctor 26(2):91-92, 1996).

Blood transfusion

Transfuse packed cells if the child has a haemoglobin less than 6 g/dl.

Nursing care

This is, of course, of vital importance. Regular attention to the child's position, clearing of airways and monitoring of fluid balance may make the difference between recovery and demise.

ASSESS PROGRESS

Note each day:

- the child's temperature
- the conscious state, fontanelle tension, occurrence of convulsions, focal neurological signs, head circumference (twice weekly)
- the state of hydration, circulation and urine output.

REVIEW THE CULTURE AND SENSITIVITIES

Always continue chloramphenicol for 14 days, unless the organism is resistant. Alternative antibiotics are ampicillin or a third generation cephalosporin (Ceftriaxone or Cefotaxime are the best but Ceftazidime is also effective). Aminoglycosides (streptomycin, kanamycin, gentamicin) should NOT be used alone to treat meningitis because they penetrate poorly into the CSF. However the combination of an aminoglycoside and a penicillin (Crystapen/Ampicillin and Gentamicin) may be used in neonates and in situations where chloramphenicol is not possible or appropriate and where a third generation cephalosporin is not available.

Treat the intimate contacts of meningococcal meningitis with rifampicin 20 mg/kg (max 600 mg) for 2 days.

SUBDURAL EFFUSION

Measure the head circumference twice a week in all children with meningitis. Subdural effusion is particularly likely in meningitis due to *Haemophilus influenzae* and in children under 2 years of age. In a series in Papua New Guinea, subdural effusion was found in 20% of children aged less than one year. Suspect it if:

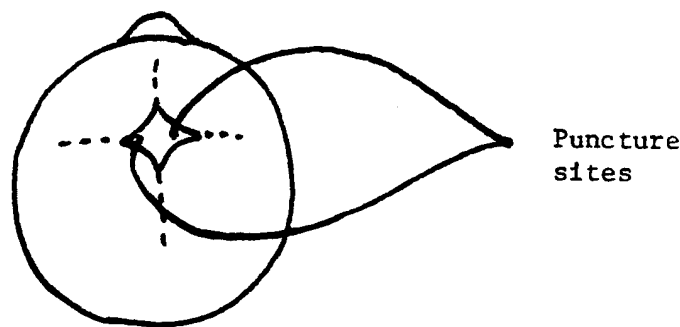
- fever persists more than 3 days
- vomiting persists or recurs

- feeding difficulty appears
- the head circumference increases
- papilloedema occurs
- focal neurological signs appear
- the CSF protein rises
- or there is persistent or increased bulging of the fontanelle.

The diagnosis is confirmed by ultrasound or by asymmetrical illumination with supratentorial transillumination of the skull (use a flashlight with a rubber adaptor tightly applied to the skull in a dark room).

Treatment is by subdural puncture:

1. Shave the anterior half of the scalp and swab with iodine. Glove and drape. Have the child's head held firmly.
2. Scrub your hands and put on sterile gloves and a cap and mask.
3. Use a 19 or 20 gauge short bevel needle. Puncture the scalp obliquely at the extreme lateral corner of the anterior fontanelle at least 3 cm from the midline, and advance the needle until a feeling of resistance is overcome at a depth of 0.5 to 1 cm. **DO NOT GO DEEPER THAN THIS.** Allow up to a maximum of 15 ml of fluid to drain. **DO NOT ASPIRATE FLUID.** The needle may be rotated, but should not be moved from side to side.
4. Repeat the puncture on the other side.
5. The puncture may have to be repeated on several different days. 15 ml per side may be removed each day.



REFERENCES

- Biddulph J. PNG Med J 11;23-29,1968. Purulent meningitis.
- Duke T, Mokela D, Frank D et al. Ann Trop Paediatr 22(2):145-157,2002. Management of meningitis in children with oral fluid restriction or intravenous fluid at maintenance volumes: a randomized trial.
- Gratten M. PNG Med J 28:233-240,1984. Aetiology of meningitis in PNG.
- Shann F. Med J Aust 1:577-78,1981. Treatment of meningitis without IV fluid.
- Shann F. New Engl J Med 313:410-414,1985. Intramuscular chloramphenicol.
- Shann F. Arch Dis Child 60:963-966,1985. Hyponatraemia in meningitis.
- Shann F. Lancet 2:681-684,1985. IM chloramphenicol for meningitis.
- Tefuarani N, Vince JD. Annals Trop Paed Int Child Health 12:375-383,1992. Purulent meningitis in children; outcome using a standard management regimen with chloramphenicol.
- Vince JD. Trop Doctor 26(2):91-92,1996. Bacterial meningitis in developing countries (letter).

MENINGITIS - CRYPTOCOCCAL

Cryptococcus neoformans (*Torula histolytica*) is a yeast-like fungus that causes a lymphocytic meningitis with multiple brain abscesses. There are two varieties, *C. neoformans* var *gattii* and *C. neoformans* var *neoformans*. Var *gattii* tends to affect young healthy adults and older children with apparently intact immune function and is the form currently prevalent in Papua New Guinea. Var *neoformans* is more commonly associated with immune deficiency such as HIV infection or steroid treatment.

Cryptococcal meningitis is often misdiagnosed as TB meningitis, and should be included in the differential diagnosis of chronic meningitis. It should be suspected whenever a child being treated for TB meningitis does not respond to treatment.

There is gradual onset of headache, vomiting, malaise and weight loss. Fever and visual disturbances may occur.

DIAGNOSIS

Every patient treated for TB meningitis should have CSF examined for cryptococcus by indian ink preparation and, if available, cryptococcal serology (cryptococcal antigen latex agglutination test). Any patient who does not respond to treatment for TB meningitis should have a repeat lumbar puncture. The CSF should be examined with indian ink, cultured for cryptococcus, if possible, and a specimen sent for a cryptococcal antigen latex agglutination test.

TREATMENT

1. **Amphotericin.** Give amphotericin B by IV infusion each day. Use 5% dextrose and NOT normal saline (the drug is incompatible with normal saline). Start with 0.5 mg/kg/day and increase gradually to 1 mg/kg/day over 1 week. Monitor urea, potassium and Hb each week. Amphotericin has a relatively long half-life, and if response to treatment is good, alternate day infusions of 1.5 mg/kg can be given.

Amphotericin causes thrombophlebitis. Intravenous cannulae should be checked and changed regularly. A small dose of heparin (1 unit/ml) in the infusion helps to prevent thrombophlebitis. Since amphotericin is light sensitive, the infusion should be covered.

Side effects such as chills, fever and rigors are common. They can be minimised by giving antihistamines (such as promethazine) or small doses of intravenous hydrocortisone. Hypokalaemia due to renal tubular dysfunction is common. Patients may require potassium supplementation.

2. **5-fluorocytosine.** Give 5-fluorocytosine 100 mg/kg/day in 4 divided doses orally.
3. **Steroids.** There is now good evidence that the use of steroids (at an equivalent daily dose of 100 mg hydrocortisone) prevents visual deterioration in patients with *Cryptococcal neoformans* var *gattii* meningitis.
4. **Other antifungal agents.** There is no evidence to indicate that the results of using the newer Azole drugs (fluconazole and itraconazole) are any better than those using combined amphotericin B/5-fluorocytosine.
5. **Other treatments.** Pharmaceutical measures to reduce raised intracranial pressure (mannitol, high dose steroids, acetazolamide) may be used, but there is no convincing evidence of benefit.
6. **Nutritional support.** Patients with chronic meningitis are often malnourished and need nutritional support.

How long should treatment be continued?

Treatment should be continued for a minimum of 6 weeks and a minimum total dose of 30-35 mg amphotericin/kg.

Indian ink stain should be negative before discharge - but other CSF parameters and serum cryptococcal antigen levels do not appear to be good predictors of relapse in PNG.

If the child relapses:

- try to arrange for the organism to be sent to Australia for sensitivity testing
- give amphotericin B and fluorocytosine for a minimum of another 10 weeks, and until the CSF cryptococcal antigen titre is less than 1/16.

REFERENCES

- Brown N, Trevett A. PNG Med J 37(3):161-165,1994. Cryptococcal meningitis in Papua New Guinea: a reminder.
- Laursen IF, Trevett AJ, Lalloo DG et al. Trans Roy Soc Trop Med Hyg 90:57-60,1996. Meningitis caused by *Cryptococcus neoformans* var.*gattii* and var. *neoformans* in Papua New Guinea.
- Randolph AG, Cook DC, Gonzales CA, Andrew M. BMJ 316:969-974,1998. Benefit of heparin in peripheral, venous and arterial catheters: systematic review and meta-analysis of randomised controlled trials (no benefit of heparin flush).
- Seaton RA. PNG Med J 39(1) 67-73,1997. Clinical practice. The management of cryptococcal meningitis in Papua New Guinea.
- Seaton A, Hamilton AJ, Hay RJ, Warrell DA. Trans Roy Soc Trop Med Hyg 90:508-512,1996. Exposure to *Cryptococcus neoformans* var.*gattii* - a seroepidemiological study.

MICTURATING CYSTOURETHROGRAM

1. Give Coloxyl 50 mg tab the night before:

Under 10 kg:	1 tab
10-20 kg:	2 tab
Over 20 kg:	4 tab
2. If the child is over 10 kg, he or she should be fasted overnight.
3. Take a plain film of the abdomen.
4. In males, inject 2 ml of xylocaine jelly into the urethra. With full sterile precautions, pass a urethral catheter into the bladder (p.377). Send a sample of the catheter urine for micro and culture.
5. Using a mixture of 76% Urografin and saline, fill the bladder until the patient wants to micturate:

Infant:	20 ml Urografin + 80 ml saline
Child:	40 ml Urografin + 160 ml saline
Adult:	60 ml Urografin + 250 ml saline
6. Use 12" x 10" (30 cm x 25 cm) film. Take slightly oblique views in males (so that the shadow of the penis is to one side):
 - a. take a film while filling the bladder
 - b. take a film during micturition
 - c. take a post-micturition film.

MILK FEEDS

See also Infant Feeding (p.167) and Neonates - Milk Feeds (p.264).

BABIES LESS THAN 28 DAYS OF AGE

(See p.264)

Breast milk.

If breast milk is unavailable: use sugar-milk.

With diarrhoea: use sugar-water, ORS or low lactose milk (eg Digestelact, Nutramigen).

With chronic diarrhoea that persists on Digestelact or Nutramigen: use Pregelstimil.

CHILDREN OVER 28 DAYS OF AGE

Breast milk.

If breast milk is unavailable: use full strength full cream milk (p.231).

Malnourished children WITHOUT DIARRHOEA: use milk oil formula (MOF).

Well nourished children with diarrhoea: use breast milk plus sugar-water or ORS.

Malnourished children with diarrhoea: breast milk plus ORS, plus low lactose milk if available, full strength full cream milk if not.

Children with confirmed lactose intolerance: stop breast feeds for 2-3 days.

- in well nourished children, give sugar-water or ORS
- in malnourished children, give a low lactose milk such as Digestelact, Nutramigen or Ensure (a lactose free/high calorie milk substitute).

With chronic diarrhoea that persists on Digestelact or Nutramigen: use Pregelstimil or Ensure (see p.106).

ELECTROLYTE MIXTURE

This is given to children with severe malnutrition or chronic diarrhoea.

Either give 5 ml TID, or add 5 ml to each 240 ml of milk feed. For a discussion of the role of zinc deficiency in malnutrition, see Pediatrics 83:532-8,1989.

To make electrolyte solution:

Add 50 g potassium chloride, 10 g magnesium hydroxide and 2 g zinc sulphate to 1 litre of water. Label clearly: SHAKE WELL BEFORE USE.

MILK MIXTURES

1. Full strength full-cream milk (FSM or FSS, eg Sunshine or Pacific Instant Milk)

	One feed	Ward use
Milk powder (instant)	One (1) 50 ml measuring cup	One (1) big cupful
Cool, previously boiled water	Three* (3) 50 ml measuring cups	Three* (3) big cupfuls
*If using non-instant powder use four cupfuls of water instead of three		

2. Milk oil formula (MOF)
 - a. Make full strength full-cream milk as above
 - b. Add vegetable oil and sugar as below:

	One feed	Ward use
Vegetable oil	10 ml	50 ml
Sugar	2 heaped teaspoons	1 heaped 50 ml cup

3. Half strength Sunshine milk (sugar-milk)

	One or two feeds	Ward use
Milk powder (instant)	One (1) 50 ml measuring cup	One (1) big cupful
Sugar	Half (½) 50 ml measuring cup	Half (½) big cupful
Cool, previously boiled water	Six** (6) 50 ml measuring cups	Six** (6) big cupfuls
**If using non-instant powder use seven cupfuls of water instead of six		

4. Digestelact (or other lactose-free milk)

	One feed
Digestelact powder	One (1) 50 ml measuring cup
Cool, previously boiled water	Four (4) 50 ml measuring cups
Check instructions on the individual cans	

GENERAL RULES

1. Whenever you use these milk mixtures, give multiple vitamin liquid each day.
2. If you cannot keep the milk cold in a refrigerator, only make enough milk for one feed at a time.
3. Try to stimulate mother's milk supply with chlorpromazine (see p.182):
 - a. if mother's milk is drying up or
 - b. if the mother is adopting and not lactating.
4. If you have to give artificial feeds, always use a glass with a steel spoon, because they are easier to keep clean. Never use a baby bottle, medicine dropper or feeding cup with a spout.

REMEMBER THAT BREAST MILK IS BEST FOR BABIES AND THAT ARTIFICIAL MILK FEEDS PUT THE BABY AT RISK FOR DIARRHOEA AND OTHER SERIOUS INFECTIONS.

MYCOBACTERIUM ULCERANS

This is a chronic ulcer with undermined edges that usually occurs on the buttocks or legs (buruli ulcer). It is caused by *M ulcerans* (the Bairnsdale Bacillus). Isolated cases have been reported from throughout coastal Papua New Guinea and from many parts of Africa. The average age of patients is 9 years (range 6 weeks to 50 years).

The commonest form develops as a papule, blind boil or subcutaneous lump. After a few weeks the lesion exudes sero-gelatinous material and leaves an ulcer with irregular extensively undermined edges and a necrotic base. On occasions, quite extensive surrounding oedema with swelling and induration may occur. Metastatic spread has been described in Zaire but not in Papua New Guinea, where lesions are in continuity. Lesions are "cold" unless there is secondary infection. Acid-fast *M ulcerans* can be found in smears from necrotic areas or from the undermined edges, but not from enlarged local lymph nodes. *M ulcerans* infection does not cause Mantoux conversion, but a specific skin test is available. Biopsy can assist diagnosis.

The differential diagnosis includes staphylococcal carbuncle (which is hot and painful), tropical ulcer (less undermined), tuberculous ulcer (rare, with different histology and bacteriology), malignancy, sporotrichosis (yeasts on smears, a different biopsy appearance, and growth on Sabouraud's medium), injection abscess, burns and pyodema gangrenosum.

TREATMENT

Drug therapy is unsatisfactory. The best regimen may be streptomycin, dapsone and cycloserine or rifampicin. *M ulcerans* is sometimes sensitive to ethionamide. Clofazimine is often used, but a controlled trial has shown it to be ineffective (Lancet 2:873,1973). Until further evidence is available, children with *M ulcerans* should be treated with streptomycin, isoniazid and dapsone until the ulcers have healed.

Surgery has traditionally involved radical excision, but current practice is to excise only necrotic tissue. Secondary infection should be treated with saline dressings, irrigation and systemic antibiotics before surgery.

Temperatures of 40 °C inhibit growth of the organism, and local heat can be applied by having a standard lamp shining just above the exposed ulcer.

BCG immunisation probably provides partial protection against *M ulcerans* infection.

REFERENCES

- Thangaraj HS, Evans MRW, Wansbrough-Jones MH. Trans Roy Soc Trop Med Hyg 93:337-340,1999. Mycobacterium ulcerans disease; Buruli ulcer.
Van der Werf TS, Van der Graaf WTA, Tappero JW, Asiedu K. Lancet 354:1013-1018,1999. Mycobacterium ulcerans infection.

MYELOGRAM

Do not attempt this if the child has raised intracranial pressure.

1. Perform a CAREFUL lumbar puncture (p.192). This may be difficult to do if there is a spinal block and a lumbar puncture has been performed recently. Cisternal puncture (p.80) may then be necessary. For this reason, NEVER do a lumbar puncture in a child with a suspected cord lesion until the time of the myelogram.

Send the CSF for micro, culture, protein, glucose, AFB and indian ink. It is important to be sure that the lumbar puncture needle is correctly placed, to avoid injecting dye outside the subarachnoid space.

2. Inject iophendylate (Myodil), then remove the lumbar puncture needle:

Infant:	3 ml
Child:	6 ml
Adult:	9 ml

3. Turn the patient prone. Always keep the head EXTENDED. Myodil is heavier than CSF, and can be made to run up and down the spinal cord by tilting the head or feet down. NEVER allow Myodil to enter the head.
4. If an abnormal area of the spine is encountered as the Myodil is moved along it:
 - a. fill the region with Myodil as much as possible by tilting the table
 - b. take a PA and lateral.
5. If an obstruction is encountered:
 - a. take a PA and lateral immediately
 - b. increase the head down tilt and wait 2 minutes, hoping the Myodil will run into the obstructed area to give a better outline. If it does, repeat the PA and lateral films
 - c. if the Myodil passes the obstruction, tilt the feet down to outline the upper end of the block. Repeat the PA and lateral films.
6. It is not necessary to remove the Myodil after you have finished the examination.

NEONATES - ADMISSION

ADMISSION TO THE SPECIAL CARE NURSERY (SCN)

Infants should be admitted to the SCN only when there are sound medical indications. It is not in the interests of mother and child to be separated unnecessarily and the simpler procedures such as IM antibiotics and phototherapy are possible on the postnatal ward.

The following babies should be admitted straight to SCN:

1. Birth weight <2 kg or gestation <36 weeks.
2. Severe perinatal asphyxia or severe birth trauma (Apgar < 7 @ 5 min).
3. Respiratory difficulty (resp rate >60, grunting, or central cyanosis) at 5 min.
4. Serious congenital abnormality likely to threaten immediate survival.
5. Infants of diabetic mothers.

The following babies need urgent assessment within an hour of birth. The mother should be encouraged to feed these babies whilst waiting. The decision to admit often depends on ability to feed, and on the facilities available at the time.

1. Birth weight 2.0 - 2.2 kg or 36 weeks gestational age.
2. Maternal sedation with pethidine requiring reversal with naloxone in infants.
3. Prolonged rupture of membranes (>24 hours) or chorioamnionitis.
4. Macrosomic appearance or >4 kg.
5. Thick meconium not in respiratory distress (admit directly if in respiratory distress).

If assessment within an hour is not possible and the child looks sick, he/she should be admitted straight to SCN.

Admit infants from the postnatal ward straight to SCN if they have:

1. Respiratory distress (respiratory rate >60/min, indrawing, grunting or blue).
2. Apnoeic or cyanotic attacks.
3. Convulsions.
4. Jaundice requiring exchange transfusion.
5. Hypoglycaemia (dextrostix <2.5 mmol/l or clinically symptomatic: floppy, poor response to stimulation, too sleepy to feed) requiring dextrose infusion.
6. Major feeding problem with vomiting and/or abdominal distension, or requiring supplementary feeds more than once consecutively.
7. Hypothermia (temperature <36 °C).
8. Suspicion of serious infection.

Babies with the following conditions require observation at a level that will usually be possible on postnatal wards:

1. Babies born before arrival (if well, give Tetanus Ig if mother not immunised. See p.240).
2. Jaundice.
3. Babies born to mothers with hyperthyroidism.
4. Non-life threatening congenital abnormalities.
Twins (try to keep together: if one needs admission, admit both).

Babies born to mothers on medication that may affect the newborn.

Minor infections (cord, superficial skin infection).

Signs of congenital syphilis (Note: if the mother is VDRL +ve and the baby is completely normal give 0.5 ml of benzathine penicillin. There is no need for further review).

These are only guidelines and any sick infant that medical or nursing staff are concerned about, should be referred at their discretion.

PROCEDURE FOR BABIES WHO REQUIRE ADMISSION

Preparation

The following equipment should be available and ready to use immediately:

1. Cot with blankets.
2. Oxygen with tubing and feeding tube.
3. Intravenous infusion apparatus: cannula, tape, 10% dextrose infusion, EDTA, plain, and blood culture bottles, syringe and needles, dextrostix.

Procedures

Rough or unnecessary handling and cooling are detrimental to the baby's condition. Inadequate monitoring and indecisive management are also detrimental. Therefore, aim to obtain the maximum information with the minimum amount of disturbance.

1. Measurement: All babies should be weighed. It only takes a few seconds.
2. Respiratory support: When required, this must take priority over other procedures. The infant should be settled with a nasopharyngeal oxygen tube and the flow rate should be gradually increased until the baby is pink to maximum of 1 litre/min.
3. Temperature: This should be recorded.
4. Dextrostix: This should be performed within one hour of birth. Take blood from heel stab or when blood taken for other investigations.

The following procedures may also be required urgently:

1. Intravenous infusion
 - a. set up early if dextrose or antibiotics required. VLBW infants should receive dextrose within 2 hours of birth if oral feeding contraindicated.
2. NG tube
 - a. gastric aspirate required for culture
 - b. tracheo-oesophageal fistula suspected
 - c. required for early enteral feeding
 - d. abdominal distension (free drainage), for example, if on oxygen.
3. Blood sampling
 - a. if starting on antibiotics, a blood culture is useful to guide when to stop treatment and ensure pathogenic organisms are covered.
4. Chest x-ray
 - a. if very severe respiratory distress (eg still cyanosed on oxygen)
 - b. suspicion of pneumothorax or congenital diaphragmatic hernia
 - c. respiratory distress not improving after 4 hours.

When the infant has been evaluated and the appropriate procedures defined, the procedures should be performed as efficiently as possible. Monitoring should then proceed with minimal disturbance to the infant.

NEONATES - APNOEA

Neonates often have periodic breathing, with spells of rapid breathing for about 20 seconds alternating with cessation of breathing for about 10 seconds. Apnoea is said to occur if the baby stops breathing for more than 20 seconds (though symptoms may occur before this). The baby may develop bradycardia, cyanosis and hypotonia. Apnoea usually occurs in very small babies, and in those with a PDA or respiratory distress. Attacks may occur frequently for 2 weeks or more before gradually becoming less frequent. An untreated attack may cause death.

CAUSES

Causes of apnoea in neonates:

1. Airways obstruction due to excessive flexion or extension of the neck.
2. Sepsis - meningitis, septicaemia.
3. Hypoxia - asphyxia, RDS, pneumonia, anaemia, reflux/inhalation, cardiac failure, PDA.
4. Cerebral - meningitis, convulsions (may be sub-clinical), intraventricular haemorrhage.
5. Metabolic - hypoglycaemia, hypocalcaemia, hyponatraemia.
6. Physical factors - hyperthermia, hypothermia, excess handling or suction.
7. Drugs - maternal sedation.

In some small premature babies, apnoea occurs without any apparent precipitating factor. However, babies with apnoea must always be treated for sepsis, and meningitis excluded by lumbar puncture.

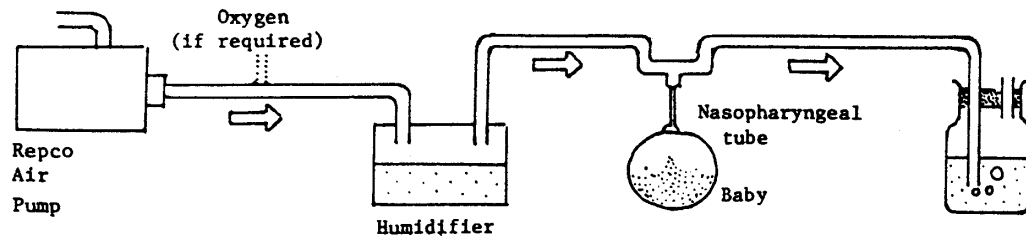
TREATMENT

1. Ensure correct positioning of the head (the neutral position - neck not flexed and not extended).
2. Treat any of the above precipitating factors.
3. Do a lumbar puncture
 - a. clear: give ampicillin and gentamicin (see p.244, 245)
 - b. cloudy: treat as per neonatal meningitis (see p. 262).
4. Do a Hb (transfuse if Hb less than 10 g/dl) and a dextrostix (give IV dextrose if the blood glucose is less than 2.5 mmol/l, but still look for the other causes of apnoea).
5. Treat cardiac failure with frusemide, digoxin and fluid restriction. If there is a patent ductus arteriosus, consider attempting to close it with indomethacin suspension (see p.267).
6. See that the baby is not too hot or too cold.
7. Handle the baby as little as possible.
8. Monitor respiration with an apnoea alarm (eg MR-10 Graseby) if you have one.
9. Give oxygen to correct hypoxia, if it is present. If there is no clinical hypoxia, but apnoea is frequent and severe, it is reasonable to try giving intranasal oxygen at 0.25 litre/min. Do NOT give more than this, as hyperoxia makes apnoea worse and may cause blindness due to retrolental fibroplasia. Stop the oxygen if it has no effect.
10. As soon as attacks occur, stimulate the baby by flicking the sole of the baby's foot with your finger. If this does not work, BRIEFLY BUT GENTLY suck out the baby's nose and pharynx. These stimuli will usually make the baby cry and breathe.
11. If the baby still does not breathe, ventilate him with a face mask and bag, or by frog breathing (but this may make the baby blind if you have to do it many times with 100% oxygen). If you ventilate the baby by these means, insert a nasogastric tube and make sure that the stomach does not become

distended with air or oxygen. The best way to ventilate is by passing an endotracheal tube, but this should only be attempted by an experienced person.

If apnoea is frequent and severe despite the above measures:

12. Aminophylline often reduces apnoeic attacks, however overdose of this drug is very dangerous and it should be used very cautiously. Toxicity causes tachycardia, arrhythmias, vomiting and convulsions. The pulse rate should be taken before each dose is due, and no aminophylline given if the pulse is over 180. Subsequent doses should be reduced. Aminophylline elixir has 25 mg/5 ml. Give a loading dose of 1.25 ml/kg, and then maintenance doses of 0.5 ml/kg every 12 hours by NG tube. Alternatively, the aminophylline can be given intravenously: dilute 2 ml of intravenous aminophylline (250 mg/10 ml amp) with 8 ml of sterile water. Give 1.25 ml/kg of the dilute solution IV SLOWLY over 60 minutes as a loading dose, then 0.5 ml/kg slowly IV every 12 hours.
13. If aminophylline is not effective, nasal continuous positive airways pressure (CPAP) is an option. However, it is time consuming and if it is to be done at all it must be done well. A suitable circuit using an air pump (producing about 6 litre/min) and a size 3.0 endotracheal tube passed through the nose so that the tip is in the pharynx is shown below. Good connections and a leak-free humidifier is essential. The tube needs regular suction to keep it patent. The mouth and other nostril are not closed.



REFERENCES

- | | |
|----------------|---|
| Apnoea: | J Pediatr 90:342-347,1977
Lancet 1:987-988,1977 |
| Nasal CPAP: | Pediatrics 56:218-223,1976
Lancet 2:853,1976 |
| Aminophylline: | Arch Dis Child 54:190-193,1979
Pediatrics 96:99-103,1980 |

NEONATES - ASPHYXIA

DEFINITION

An Apgar score of <7 at 5 minutes.

	Apgar score		
	0	1	2
Appearance	Blue or pale	Baby pink, blue extremities	Completely pink
Pulse	Absent	Below 100	Over 100
Grimace*	No response	Grimace	Cry
Activity	Limp	Some flexion limbs	Active
Respiration	Nil	Hypoventilation, poor cry	Good, strong cry
*Give a light slap to the soles of the feet			

Apgar score	Infant's condition	Physiological group
7 - 10	Vigorous	Normal
3 - 6	Some depression	Primary apnoea
0 - 2	Severe depression	Terminal apnoea

The Apgar score at one minute gives an indication of the need for resuscitative measures. The change in Apgar between 1 and 5 minutes and within the first 15 minutes after delivery have been shown to be reasonable predictors of outcome.

Although a considerable amount is now known about the pathophysiology of perinatal asphyxia, the outcome for babies with severe perinatal asphyxia remains poor even in the centres of excellence.

MANAGEMENT

In Papua New Guinea, the management of babies with asphyxia is based on high quality supportive care:

1. Oxygen. In the absence of continuous oxygen saturation monitoring, it is reasonable to give nasopharyngeal oxygen (0.5 litre/min) until the baby recovers. If monitoring is available, oxygen is given as appropriate.
2. Thermal control. Baby's body temperature should be kept in the normal range of 36.5-37.2 °C (sometimes the babies become hyperpyrexia).
3. Correction of shock. If peripheral perfusion is poor, it is reasonable to give 20 ml/kg of normal saline initially. If perfusion remains poor, the use of dopamine should be considered.
4. Fluid balance. Give IV fluids at 2/3 maintenance. Use 10% dextrose.
5. Monitor blood glucose with dextrostix and do not let it fall below 2.2 mmol. Avoid hyperglycaemia (keep blood glucose below 8 mmol).
6. Prevent/control convulsions. In severely asphyxiated babies, it is reasonable to give "prophylactic" phenobarbitone. In less severely affected babies, phenobarbitone should be given when there is any suspicion of actual or impending convulsions (phenobarbitone loading dose 20 mg/kg IMI or 10 mg/kg slowly IVI, then 5 mg/kg daily orally).
7. Treat hypocalcaemia if it occurs (or more practically, if the baby has uncontrollable fitting with a normal dextrostix).

Notes

1. Corticosteroids should not be used, and although many paediatricians use mannitol, there is no evidence for its effectiveness.
2. Babies with severe asphyxia may appear to settle relatively quickly after the resuscitation - but there is likely to be a deterioration after 6-12 hours or so as cerebral oedema develops.

REFERENCES

- Oswyn G, Vince J, Friesen H. PNG Med J 43(1-2):110-120,2000. Perinatal asphyxia in Port Moresby General Hospital: a study of incidence, risk factors and outcome.
- Saugstad OD, Rootwelt T, Aalen O. Pediatrics 102(1):e1,1998. Resuscitation of asphyxiated newborn infants with room air or oxygen: an international controlled trial, The Resair 2 study.
- Saugstad OD. Resuscitation of newborn infants with room air or oxygen. Semin Neonatol 6(3):233-239,2001. Review.
- Tarnow-Mordi WO. Lancet 352:341-342,1998. Room air or oxygen for asphyxiated babies? Editorial.

NEONATES - BORN BEFORE ARRIVAL (BBA)

If these babies are sick or weigh under 2.2 kg they should be admitted to the neonatal nursery, if one is available.

1. Give vitamin K (phytomenadione) 1 mg. 1 mg/0.5 ml or 1 mg/ml (NOT 10 mg/ml) IM once.
2. Apply crystal violet (gentian violet) to the cord.
3. Apply (oxy)tetracycline (terramycin) eye ointment to both eyes once.
4. If the mother has not been immunised against tetanus during the pregnancy give tetanus immunoglobulin 60 units IM.
5. Give the baby Sabin, hepatitis B and BCG vaccines.

If the baby:

- a. is not sucking well
- b. is not gaining weight
- c. has vomiting or abdominal distension
- d. is febrile or hypothermic
- e. is jaundiced
- or
- f. has any other signs of possible infection



TREAT THE BABY WITH ANTIBIOTICS FOR NEONATAL SEPSIS

NEONATES - CONVULSIONS

Seizures occur in approximately 0.5% of newborn infants. They are often subtle and may manifest themselves in a great variety of different ways. Convulsions may primarily affect muscular tone and give rise to brief periods of clonic extension of the body, tonic or clonic movement of any part of the body, cyclical movement, doggy paddling or simply episodes of apnoea, jitteriness, tremors, facial twitching, repetitive eye opening, nystagmus, abnormal cries or even sudden chewing, swallowing or yawning movements. Vasomotor skin changes have also been attributed to convulsions in the neonate. Therefore, it is essential to have a high index of suspicion in order to diagnose fits in this age group.

AETIOLOGY

1. Primary cerebral
 - a. Asphyxia
 - b. Meningitis
 - c. Intraventricular haemorrhage
 - d. Congenital infection
 - e. Structural brain abnormality
2. Electrolyte disturbances
 - a. Hyponatraemia
 - b. Hypocalcaemia
 - c. Hypomagnasaemia
3. Metabolic
 - a. Hypoglycaemia
 - b. Kernicterus
 - c. Pyridoxine deficiency
 - d. Inborn error of metabolism.
4. Drug withdrawal
 - a. Narcotics
 - b. Anticonvulsants

Document

1. Maternal drug history.
2. Maternal illness during pregnancy.
3. Foetal wellbeing, eg growth, liquor, foetal movements.
4. Complication of labour, eg. foetal distress.
5. Condition of infant at birth/resuscitation.

INVESTIGATIONS

Depending on availability:

1. Urgent
 - a. dextrostix
 - b. serum sodium, calcium, glucose, bilirubin (if jaundiced)
 - c. magnesium only if calcium low
 - d. full infection screen including lumbar puncture
2. Non-urgent
 - a. ultrasound scan of head

TREATMENT

1. Ensure clear airway (beware - overzealous suction can precipitate a vasovagal attack. Posture is more important than suction).
2. Prevent ongoing metabolic damage:
 - a. Give oxygen
 - b. Treat hypoglycaemia if present. If dextrostix <3 mmol/l or if you have no dextrostix, give 5 ml/kg of 10% dextrose IV (and continue with 10% dextrose infusion).
3. Stop the fit:
 - a. Paraldehyde. Stat dose 0.1 ml/kg IM. Dissolves plastic: only draw up into syringe when ready to inject and give immediately
 - b. Diazepam. Initial dose 0.25 mg/kg. Diazepam has a long half-life which is variable, but especially long in preterm infant (40-400 hours). Frequent repeated doses will give rise to very high levels. The infant may become apnoeic as a result.

Sometimes neonatal fits are refractory to treatment and in these cases repeated or larger doses may be required to terminate the fits.

Respiratory suppression is more likely to occur if phenobarbitone has also been given - so be prepared.

4. Treat any identified cause of the fit without delay.
5. If no cause is identified and the fit is difficult to control, it is worth giving:
 - a. 10% calcium gluconate 1 ml/kg over 10-15 minutes
 - b. 50% magnesium sulphate 0.2 ml/kg IM stat
 - c. pyridoxine 20-25 mg IV or oral stat.
6. Prevent further fits. Give "prophylactic" anticonvulsant:
 - a. Loading dose
 - i. First line: Phenobarbitone 10 mg/kg slowly IV. Repeat dose if fits not controlled. Alternatively, 15-20 mg/kg IM
 - ii. Second line: Phenytoin 20 mg/kg IV. NB: Give over 1 hour.
 - b. Maintenance treatment
 - i. Start 12 hours after loading doses
 - Phenobarbitone 3-6 mg/kg/day, daily
 - Phenytoin 5 mg/kg/day in 12 hourly doses. The dose may have to be increased after the first few weeks because of an increasing rate of elimination.
 - ii. Maintenance treatment can be stopped after a few days in babies whose fits have been of short duration, or in whom a treated metabolic cause was found. It should be continued long term for babies with:
 - severe birth asphyxia
 - meningitis
 - fits very difficult to control
 - evidence of neurological damage.

FOLLOW-UP

Babies with abnormal neurological movements and fits need follow-up, including developmental assessment, hearing testing, measurement of head circumference and adjustment of anticonvulsant therapy as required. Pertussis and measles immunisations may be given as normal.

NEONATES - DRUG DILUTIONS

Copy out the chart below, and put it up where the injections are prepared in your nursery. These dilutions differ from those used for older children and adults. **Always check the dilutions and concentrations.**

BABIES - DRUG DILUTIONS	
AMINOPHYLLINE	250 mg/10 ml amp: Take 2 ml of this and add 8 ml sterile water. Store in a fridge for up to 7 days.
AMPICILLIN or amoxycillin	250 mg vial: add 1.0 ml sterile water. 500 mg vial: add 2.0 ml sterile water. Store in a fridge for up to 24 hours.
CEFOTAXIME	500 mg vial: add 2 ml sterile water (250 mg/ml). 1G vial: add 4 ml sterile water. 2G vial: add 8 ml sterile water
CEFTAZIDINE	250 mg vial: add 1 ml sterile water (250 mg/ml). 500 mg vial: add 2 ml sterile water. 1G vial: add 4 ml sterile water. 2G vial: add 8 ml sterile water.
CEFTRIAZONE	1G vial: add 10 ml sterile water.
CHLORAMPHENICOL	1G vial: add 4 ml sterile water . Store in a fridge for up to 7 days.
CLOXACILLIN	250 mg vial: add 1.5 ml sterile water.
DIAZEPAM	10 mg/2 ml amp: add 8 ml sterile water. Use immediately. Discard any unused portion.
GENTAMICIN	20 mg/2 ml amp: no dilution (10 mg/ml). 80 mg/2 ml amp: add 6 ml sterile water. Store in a fridge for up to 7 days.
PENICILLIN Benzathine	2,400,000 units: add 5 mls sterile water
PENICILLIN Benzyl (crystapen)	1,000,000 unit vial: add 2 ml sterile water. Store in a fridge for up to 7 days.
PETHIDINE	50 mg in 1 ml amp: add 4 ml sterile water (10 mg/ml)
PHENOBARBITONE	200 mg/ml amp: add 3 ml sterile water (50 mg/ml) MIX VERY WELL. Store in fridge for up to 7 days
STREPTOMYCIN	1G vial: add 4.5 ml sterile water (NB: DILUTE SOLN). Store in fridge for up to 21 days.

NEONATES - DRUG DOSES

**NOTE: ALWAYS CHECK THE DRUG YOU ARE GOING TO USE IS THE SAME TYPE AND STRENGTH AS THE ONE LISTED ON THE TABLE.
ALSO CHECK CONCENTRATIONS AND VOLUMES AFTER ADDITION OF WATER FOR INJECTION.**

Neonatal drug doses	Weight (kg):		1.0-1.4	1.5-1.9	2.0-2.4	2.5-2.9	3.0-3.4	3.5-3.9
AMINOPHYLLINE. For apnoea. Omit dose if pulse over 180			Diluted	Diluted	Diluted	Diluted	Diluted	Diluted
➤ Amp 250 mg/10 ml: dilute 2 ml of this with 8 ml sterile water								
• initial loading dose: IV into burette/syringe driver (6 mg/kg, 1.2 ml/kg)	ml	1.5	2.0	3.0	4.0	5.0	6.0	
• maintenance dose: 12 hourly IV in burette/syringe driver (2.5 mg/kg, 0.5 ml/kg)	ml	0.6	0.9	1.1	1.4	1.6	1.9	
➤ Elixir 25 mg/5 ml								
• initial loading dose (6 mg/kg or 1.2 ml/kg)	ml	1.5	2.0	3.0	4.0	5.0	6.0	
• maintenance dose: 12 hourly (2.5 mg/kg or 0.5 ml/kg)	ml	0.6	0.9	1.1	1.4	1.6	1.9	
AMODIAQUINE (Infant Camoquin) Tab 100 mg. Daily for 3 days. Oral	tab	¹ / ₈	¹ / ₈	¹ / ₄	¹ / ₄	¹ / ₄	¹ / ₄	
AMOXYCILLIN. 250 mg vial: add 1 ml sterile water.								
High doses for meningitis/severe infection. 50 mg/kg/dose IV or IM								
• under 1 week of age: 12 hourly	ml	0.2	0.3	0.4	0.5	0.6	0.7	
• over 1 week of age: 6 hourly	ml	0.2	0.3	0.4	0.5	0.6	0.7	
AMPICILLIN. 250 mg vial: add 1 ml sterile water or 500 mg vial: add 2 ml sterile water								
High dose for meningitis/severe infection 50 mg/kg/dose (0.2 ml/kg)								
• under 1 week of age: IM or IV 12 hourly	ml	0.2	0.3	0.4	0.5	0.6	0.7	
• over 1 week of age: IM or IV 8 hourly	ml	0.2	0.3	0.4	0.5	0.6	0.7	
BICARBONATE (Sodium) 8.4% 1-2 ml/kg slow IV	ml	1.5	2.0	3.0	4.0	5.0	6.0	
CALCIUM GLUCONATE 10% 1 ml/kg slow IV	ml	1.0	1.5	2.0	2.5	3.0	3.5	
CEFOTAXIME. 50 mg/kg/dose IV			Dilute as per manufacturer’s instruction					
• under 1 week of age Preterm: 12 hourly	ml							
• under 1 week of age Term: 8 hourly	ml							
• over 1 week of age: 6 hourly	ml							
CEFTAZIDIME. 50 mg/kg/dose IV or IM			Dilute as per manufacturer’s instruction					
• under 1 week of age: 12 hourly	ml							
• over 1 week of age: 6 hourly	ml							
CEFTRIAXONE. 50 mg/kg/dose IV or IM			Dilute as per manufacturer’s instruction					
• under 1 week of age: once daily	ml							
• over 1 week of age: 12 hourly	ml							

Neonatal drug doses	Weight (kg):		1.0-1.4	1.5-1.9	2.0-2.4	2.5-2.9	3.0-3.4	3.5-3.9
CHLORAMPHENICOL. 1g vial: add 4 ml sterile water. 25 mg/kg/dose (0.1 ml/kg)								
➤ Under 2.2 kg or jaundiced:								
• first week of life: daily IM or IV	ml	0.1	0.2	0.2	0.3	0.3	0.4	
• second week of life: 12 hourly IM or IV	ml	0.1	0.2	0.2	0.3	0.3	0.4	
• after second week of life: 8 hourly IM or IV	ml	0.1	0.2	0.2	0.3	0.3	0.4	
➤ Over 2.2 kg, not jaundiced:								
• first week of life: 12 hourly IM or IV	ml	0.1	0.2	0.2	0.3	0.3	0.4	
• after first week of life: 8 hourly IM or IV	ml	0.1	0.2	0.2	0.3	0.3	0.4	
CHLORPROMAZINE for tetanus:								
• amp 50 mg/2 ml double dose stat, then 5 mg/kg (0.2 ml/kg) IM 12 hourly	ml	0.2	0.3	0.4	0.5	0.6	0.7	
• syrup 25 mg/5 ml, 5 mg/kg (1 ml/kg) oral 12 hourly	ml	1.5	2.0	2.5	3.0	3.5	4.0	
• tab 25 mg, 5 mg/kg oral 12 hourly	tab	¼	¼	½	½	¾	¾	
CLOXACILLIN. 250 mg vial: add 1.5 ml sterile water. 25 mg/kg/dose (0.15 ml/kg)								
• first week of life: 12 hourly	ml	0.2	0.3	0.3	0.4	0.5	0.6	
• after first week of life: 6 hourly	ml	0.2	0.3	0.3	0.4	0.5	0.6	
DEXTROSE								
• 10% 5 ml/kg IV	ml	6.0	9.0	12.0	14.0	16.0	19.0	
• 50% 1-2 ml/kg slow IV	ml	1.0	1.5	2.0	2.5	3.0	3.5	
DIAZEPAM. 10 mg/2 ml (add 2 ml sterile water)								
• for fitting, 0.25 mg/kg (0.1 ml/kg) IV	ml	Diluted 0.1	Diluted 0.2	Diluted 0.2	Diluted 0.3	Diluted 0.3	Diluted 0.4	
DIGOXIN. 6 hourly for 3 doses, then daily:								
• amp 50 microgram/2 ml infant (not 250 microgram/ml). 7.5 microgram/kg/dose (0.3 ml/kg/dose) IM or IV	ml	0.4	0.5	0.7	0.8	1.0	1.1	
• elixir 50 microgram/ml. 10 microgram/kg/dose (0.2 ml/kg/dose) oral	ml	0.2	0.3	0.4	0.5	0.6	0.7	
FRUSEMIDE. Amp 20 mg/2 ml. 2 mg/kg (0.2 ml/kg) IM or IV	ml	0.2	0.3	0.4	0.5	0.6	0.7	
GENTAMICIN. Amp 20 mg/2 ml (or 80 mg/2 ml with 6 ml sterile water added) IV or IM								
➤ Less than 36 weeks gestational age:								
• first week of life: 3.5 mg/kg once daily	ml	0.4	0.6	0.8	1.0	1.1	1.3	
• after first week of life: 7.5 mg/kg once daily	ml	0.9	1.3	1.7	2.1	2.4	2.8	
➤ More than 36 weeks gestational age:								
• first week of life: 5 mg/kg once daily	ml	0.6	0.9	1.1	1.4	1.6	1.9	
• after first week of life: 7.5 mg/kg once daily	ml	0.9	1.3	1.7	2.1	2.4	2.8	
ISONIAZID (INAH) 100 mg tab. 5 mg/kg/day, twice a week, oral	tab	¼	¼	¼	½	½	½	
MAGNESIUM SULPHATE. 50% amp. 0.2 ml/kg, IM daily	ml	0.2	0.3	0.4	0.5	0.6	0.7	
METRONIDAZOLE flask. 100 mg/20 ml, 5 mg/ml, 7.5 mg/kg/dose (1.5 ml/kg) IV	ml	2.0	2.5	3.5	4.0	5.0	5.5	

Neonatal drug doses	Weight (kg):		1.0-1.4	1.5-1.9	2.0-2.4	2.5-2.9	3.0-3.4	3.5-3.9
NALOXONE (Narcan) 0.1 mg/kg IV or IM*								
• amp 0.4 mg/ml (400 microgram/ml)	ml		0.25	0.25	0.5	0.5	0.75	0.75
*Note dose now recommended is much higher than in previous edition.								
PARALDEHYDE amp. 5 ml, 0.2 ml/kg IM	ml		0.2	0.3	0.4	0.5	0.6	0.7
PENICILLIN Benzathine. 2,400,000 units vial: add 5 ml sterile water. IM once only	ml		0.5	0.5	0.5	1.0	1.0	1.0
PENICILLIN Benzyl (Crystapen). 600 mg (1 Megaunit) vial: add 2 ml sterile water. 30 mg/kg/dose IV or IM								
• under 1 week of age: 12 hourly	ml		0.1	0.2	0.2	0.3	0.3	0.4
• over 1 week of age: 6 hourly	ml		0.1	0.2	0.2	0.3	0.3	0.4
PHENOBARBITONE			Diluted	Diluted	Diluted	Diluted	Diluted	Diluted
➤ Stat: 15-20 mg/kg as either:								
• amp 200 mg/ml (add 3 ml sterile water) 0.4 ml/kg IM	ml		0.5	0.7	0.9	1.1	1.3	1.5
• or tab 30 mg oral	tab		$\frac{3}{4}$	1.0	$1\frac{1}{2}$	$1\frac{1}{2}$	2.0	$2\frac{1}{2}$
➤ Then maintenance 5 mg/kg/day: 30 mg tab once daily, oral	tab		$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$
PHENYTOIN vial 250 mg/5 ml, susp 25 mg/1 ml								
➤ Loading dose: 15-20 mg/kg (0.4 ml/kg) IV over 1 hour (for status epilepticus only)	ml		0.5	0.7	0.9	1.1	1.3	1.5
➤ Maintenance:								
• 2 mg/kg/dose under one week old prem: 12 hourly 0.08 ml/kg oral	ml		0.1	0.1	0.2	0.2	0.3	0.3
• 4 mg/kg/dose under one week old term: 12 hourly 0.16 ml/kg oral	ml		0.2	0.3	0.4	0.4	0.5	0.6
• 4 mg/kg/dose over one week old: 8 hourly 0.16 ml/kg/dose oral	ml		0.2	0.3	0.4	0.4	0.5	0.6
QUININE amp. 120 mg/2ml IM 12 hourly. 10 mg/kg/dose (0.17 ml/kg)	ml		0.2	0.3	0.4	0.5	0.6	0.7
STREPTOMYCIN vial								
1 g: add 4.5 ml sterile water. 20 mg/kg/day. IM once a day	ml		0.1	0.2	0.2	0.3	0.3	0.4
TETANUS Immunoglobulin amp. 60 units/ml or 250 units/3.8 ml								
• prophylaxis, IM	ml		1.0	1.0	1.0	1.0	1.0	1.0
• treatment, IM	ml		3.8	3.8	3.8	3.8	3.8	3.8
VITAMIN K (Phytomenadione, Konakion) amp. 1mg/0.5 ml IM	ml		0.5	0.5	0.5	0.5	0.5	0.5

NEONATES - EXCHANGE TRANSFUSION

Indications

1. Rapidly rising or high SBR, particularly in the presence of acidosis or other metabolic problem
2. Partial exchange with FFP for polycythaemia.

Explain the procedure, indications and risks to parents and get written consent.

Take 2-3 ml clotted blood from the baby AND 5 ml clotted blood from the mother for group and cross-match. You will need 180 ml/kg of blood for the exchange. A bag of whole blood contains about 400-450 ml. A bag of packed cells contains 150-250 ml. This can be made up to "whole blood" with FFP (each bag of FFP contains 150-250 ml). Remember to give antimalarials after doing an exchange.

HOW TO CROSS-MATCH FOR EXCHANGE

Always use FRESH whole blood - it must not be more than 6 days old.

This protocol is based on the fact that that Rh negative patients are very rare in Papua New Guinea.

1. If mother and baby are the same blood group
 - OR the mother is AB
 - OR the baby is OTHEN
 - use blood of the same group as the baby
 - and cross-match the donor cells against the mother's serum.
2. If the mother is blood group O, and the baby A, B or AB
 - OR the mother is A, and the baby is B or AB
 - OR mother is B, and baby A or ABTHEN
 - use blood of the same group as the baby
 - and cross-match the donor cells against the baby's serum.
3. If you do not have FRESH blood of the same blood group as the baby's, THEN
 - use FRESH group O blood
 - and cross-match the donor cells against the mother's serum.

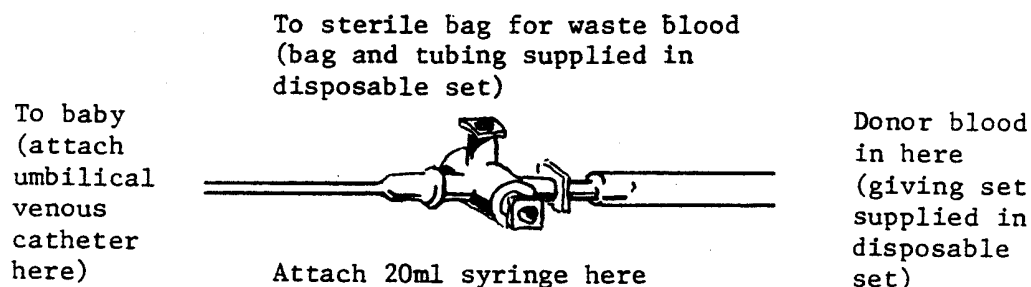
You are advised to seek advice from the Director of Blood Bank at PMGH (phone 3248294) if:

1. the baby has had a previous transfusion
2. the baby is over 4 weeks old
3. the mother is an expatriate (Rh typing necessary)
4. or Rh negative patients are common in your area.

HOW TO DO AN EXCHANGE TRANSFUSION

1. Warm the donor blood by immersing it in water at 37 °C; or use a temperature-controlled water bath. NEVER warm blood under a hot tap.
2. Keep the baby warm. Wrap the baby's head and limbs in a blanket. Gently strap the baby to a well padded wooden cross. It is good to use an overhead heater if you have one when doing an exchange on very small babies.
3. Tape a stethoscope to the baby's chest, so that your assistant can monitor the baby's pulse rate.
4. Using a sterile technique, insert an umbilical venous catheter (see p.277) from an exchange transfusion set. Use the small catheter if the baby is under 2 kg. Do not leave the umbilical catheter open to the air (because of the danger of air embolus).

5. Connect the 4-way tap from an exchange transfusion set. The handle points to the port that is open. It can be moved by turning the syringe in a clockwise direction. Adjust the position of the catheter until blood can be withdrawn freely (but withdraw gently, or you will block the catheter).



6. If the baby weighs less than 2 kg, exchange 10 ml at a time. If the baby weighs 2 kg or more, exchange 20 ml at a time.
7. Take out the first 20 ml of baby's blood (10 ml in small baby). Put some of the baby's blood in an EDTA bottle for the pre-exchange Hb, and some into a plain bottle for the pre-exchange bilirubin.
8. The order of exchange is:
 - a. remove exactly 20 ml (or 10 ml if the baby weighs less than 2 kg) of blood from the baby
 - b. turn the syringe clockwise a quarter turn, and squirt the blood into the waste bag
 - c. turn the syringe clockwise a quarter turn, and draw up exactly 20 ml (10 ml) of donor blood
 - d. turn the syringe clockwise a half turn, and slowly inject the donor blood into the baby.

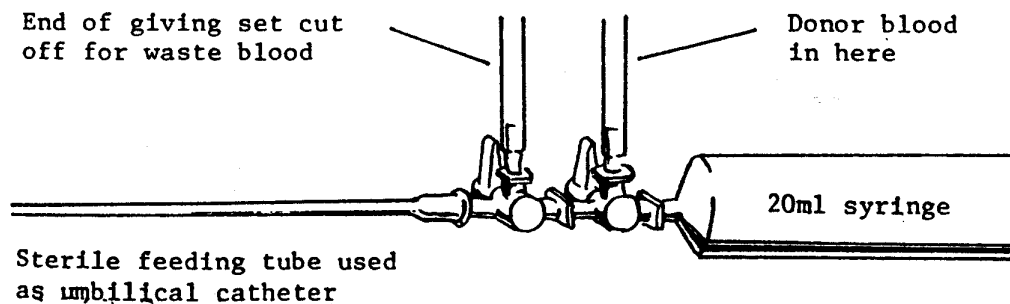
Whenever there is a pause in the exchange, leave the catheter full of donor blood to prevent clotting. Agitate the donor blood every 15 minutes to keep the cells and plasma mixed.
9. Exchange a total of 180 ml/kg.
10. After each step, you must call out the volume of blood withdrawn or injected. Your assistant must enter these volumes on a record sheet, and keep cumulative totals. After each 100 ml of exchange (50 ml if the baby weighs less than 2 kg) check the baby's pulse and respiration rates and enter them on the record sheet. If the pulse rate is over 150/min or the baby is very restless, give 1 ml of 10% calcium gluconate IV by injecting it through the rubber port on the side of the 4-way tap into the 20 ml syringe. Mix it with 20 ml (or 10 ml) donor blood and inject it SLOWLY over 2 minutes. Some paediatricians give calcium routinely after every 100 ml of an exchange, providing the pulse rate is over 120/minute.
11. At the end of the exchange, keep some of the last aliquot of blood from the baby for a Hb and bilirubin.
12. Remove the catheter slowly and apply pressure on the umbilicus. Put a light dressing on the umbilicus. Ask the nurse to watch for bleeding. Untie the baby.
13. Give a stat dose of infant Camoquin to the baby orally, or, if the baby is very ill give IM quinine stat and then amodiaquine daily for 3 days.
14. Record details of the exchange transfusion in the baby's history.
15. Give ampicillin and gentamicin for 5 days.
16. Go and see the mother.

A LIST OF THE EQUIPMENT NEEDED FOR AN EXCHANGE TRANSFUSION

Copy this list and pin it up in your nursery so that the nurses know what to have ready:

1. Cut down tray (sterile towels, bowls, syringes, needles, forceps, scapel, scissors, silk thread)
2. Large bowl to warm blood to 37 (C (NOT hotter) or Blood Warmer
3. Scapel blade
4. Elastoplast
5. Gown, mask, sterile gloves
6. 2 Hb bottles, 2 plain bottles (for bilirubin)
7. Oxygen and catheter
8. Suction and catheter
9. Cross-shaped splint and bandages
10. Blankets
11. Drip stand
12. A good light
13. Exchange Transfusion Tray (Pharmaseal Laboratories, Glendale, CA 91201, USA).

The Exchange Transfusion Tray is expensive (US\$40). If it is not available, you will need 2 three-way taps, a 20 ml syringe, a sterile plastic feeding tube (5 gauge if the baby weighs less than 2 kg, 8 gauge if the baby weighs 2 kg or more), a blood giving set, an ordinary giving set (cut the bottom 3 feet off and use it to discard the waste blood into a bag or bowl), a 5 ml syringe, 10 ml of 10% calcium gluconate, a bowl of sterile saline to rinse out the syringe, and a bag or bowl for the discarded blood.



Exchange Transfusion Using Two Three-Way Taps

NEONATES - FLUIDS AND FEEDS

FEEDING

Breast feed whenever possible. If not, use EBM if available or HSSM if not.

Cup and spoon or NG 3 hourly feed volumes

Birth weight (kg)	Day							
	1	2	3	4	5	6	7	8
1.5-1.9	10	15	20	25	30	40	45	50
2.0-2.4	15	20	30	35	40	50	55	65
2.5-2.9	20	25	35	40	50	60	70	75
3.0-3.4	20	30	40	50	60	70	70	75
3.5-4.0	25	35	45	60	70	80	80	80

FLUIDS

Intravenous fluid regime (ml/kg/day)				
Day	Weight (kg)			
	<1	1.0-1.5	1.5-2.5	>2.5
1	100	80	60	60
2	120	100	90	90
3	150	130	120	110
4	180	150	150	130
5	200	180	170	150
6	200	180	170	150
7	200	180	170	150
8-13	200	180	170	150
14-20	180	160	150	150
21-27	160	160	150	150
>28	160	150	150	150

Always use the birth weight or current weight - whichever is greater - to calculate fluids.

Usual fluid regimen

	Low birth weight	Full term >2.5kg
Day 1	>1kg: 10% dextrose <1kg: 5% dextrose	10% dextrose
Day 2	10% dextrose + 0.18% saline*	4.3% dextrose/saline
Day 3 onwards	10% dextrose + 0.18% saline* with potassium	4.3% dextrose/saline with potassium

*10% dextrose + 0.18% saline: Add 10 ml 50% dextrose to 90 ml 4.3% dextrose/0.18% NaCl.

Potassium: Add 1g KCl to each litre when urine output well established.

Normal K⁺ levels in newborn are relatively high in the first 10 days (3.5-7.0 mmol/l).

Potassium requirements 2-3 mmol/kg/day after day 2. Requirements increased by high urine flow rates, diuretics, high prostaglandin levels and hyperaldosteronism.

NEONATES - INFECTION (NEONATAL SEPSIS)

Infection in the newborn baby may have originated before, during or after birth.

PREDISPOSING FACTORS TO INFECTION

1. Low birth weight
2. Prematurity
3. Prolonged rupture of membranes (more than 24 hours)
4. Babies with congenital abnormalities
5. Protracted or difficult labour
6. Babies who have been handled a great deal and who have tubes inserted (eg umbilical venous catheter).
7. Maternal fever

DIAGNOSIS OF INFECTION

This is often difficult especially in babies of low birth weight. The symptoms may be vague and non-specific. The following symptoms are suggestive of an infection:

- | | |
|------------------------|--------------------------------|
| • Poor colour | • High or low body temperature |
| • Lethargy/inactivity | • Jaundice |
| • Hypotonia | • Cyanotic attacks |
| • Irritability | • Respiratory distress |
| • Poor suck | • Poor weight gain |
| • Vomiting | • Purpuric spots |
| • Abdominal distension | |

The nursing staff will describe babies with a mixture of these signs and symptoms as having “Gone off”: Listen to them, and always consider infection as a likely cause of the problem.

EXAMINATION OF A BABY WITH SUSPECTED INFECTION

1. Look for signs of infection. Check the eyes, the umbilicus, the nails and the skin. There may be septic spots to be found.
2. Examine the joints and limbs. Tenderness may indicate an underlying osteomyelitis or osteoarthritis. Examine the chest for signs of pneumonia.
3. Examine the fontanelle for signs of raised intracranial pressure, although this is not always detectable in babies with neonatal meningitis.
4. Examine the abdomen for distension and tenderness. Auscultate for bowel sounds. Examine the stool for the presence of blood.

INVESTIGATIONS

(Dependent on facilities available.)

1. Swab any suspicious lesion (eg skin, umbilicus or nails).
2. Blood cultures. Taken from a peripheral vein. Clean skin with betadine. Use a scalp vein “butterfly” needle and syringe.
3. White cell count. The neutrophil count is often low (below $2000 \times 10^9/l$) when a newborn baby has an infection.

4. Urine. Collect urine by having a sterile pot ready. Tap over the supra-pubic area for 3 minutes. This will often cause the child to void spontaneously and a clean catch urine can be collected. If unsuccessful a supra-pubic aspiration of urine must be performed.
5. CSF examination. Meningitis is possible with minimal signs but LP should be reserved for significant signs of infection rather than as a routine screening test.
6. Chest x-ray if respiratory distress.
7. Abdominal x-ray if abdominal distension is noted.

TREATMENT (GENERAL)

Treatment should be commenced at the first suspicion of infection. Blood and other appropriate cultures should be taken, if possible, prior to treatment. Because the symptoms and signs of infection are non-specific, some babies who are not infected will inevitably be treated with antibiotics.

Antibiotics can be stopped after 48-72 hours, if infection is subsequently felt to be an unlikely cause of the baby's symptoms and signs, if there is a rapid improvement and bacteriological cultures are negative.

Ampicillin/amoxycillin and gentamicin are currently used for confirmed or suspected neonatal infection. These antibiotics will cover both gram negative and Streptococcal infection. When Staphylococcal infection is suspected, cloxacillin should be given instead of ampicillin.

TREATMENT OF SPECIFIC BACTERIAL INFECTIONS

Meningitis

See separate section, p.262.

Urinary infections

Ideally, treatment should be started after a clean catch urine specimen has been collected or a supra-pubic aspiration has been performed, although treatment should not be delayed by more than 4 hours. In the first week of life, gentamicin is the antibiotic of choice for an acute infection. Cotrimoxazole should be avoided in preterm or jaundiced infants during the first two weeks of life. Subsequently, prophylaxis can be given as a single night time dose of cotrimoxazole (1 ml/kg/dose of 40 mg trimethoprim in 5 ml) and this should be continued until both reflux and obstruction have been excluded.

A renal ultrasound scan is useful to exclude obstruction as this can easily be organised within a few days. Although neonatal urinary tract infection is often contracted from bacteraemia/septicaemia, it is still advisable to organise an MCU as an outpatient after a delay of six weeks on prophylaxis (see UTI, p.378).

Conjunctivitis

First 24 hours: could be gonococcus. Wash the eyes to clear as much pus as possible. Oxytetracycline eye ointment QID for 5 days. Benzyl penicillin IM TID for 5 days. If no rapid improvement, change to ceftriaxone/cefotaxime if available, or add gentamicin if not. Treat both mother and father.

After 24 hours: the most likely organisms are Staphylococcus and E coli, though gonococcus is still possible. Do a swab, gram stain and culture, if possible. Treat with oxytetracycline eye ointment QID for 5 days. Give parenteral antibiotics (ampicillin and gentamicin) if there is any deterioration or if there is conjunctival or lid oedema.

After 1 week: could be chlamydial infection. The baby should have oxytetracycline eye ointment and erythromycin orally for 3 weeks to prevent pneumonitis. The parents should be referred back to the consultant obstetrician for investigation and treatment.

Necrotising enterocolitis

See separate section (p.265) for diagnosis and management. Currently, the most widely used antibiotic regimen for this condition is penicillin, gentamicin and metronidazole, administered intravenously.

DURATION OF TREATMENT

1. Treat confirmed infections in a baby that responds rapidly for 10 days.
2. Treat suspected infections in a baby that responds rapidly for 7 days.
3. If there is a slow recovery treat for 14 days.
4. Treat babies with neonatal meningitis for 21 days.

BABIES OF VDRL +VE MOTHERS

If no signs or symptoms of intrauterine infection:

Give infant: <2.5kg: 120,000 units benzathine penicillin IM stat
>2.5kg: 240,000 units benzathine penicillin.

If signs of intrauterine infection:

- blisters or rash especially on palms or soles
- hepatosplenomegaly
- petechiae or bruising
- early onset or prolonged jaundice

Give benzathine penicillin stat and benzyl penicillin (Crystapen):

<2.5kg: 125,000 units IMI BD for 10 days

>2.5kg: 250,000 units IMI BD for 10 days

Check both parents have been treated.

PROLONGED RUPTURE OF MEMBRANES (PROM) - MORE THAN 24 HOURS BEFORE DELIVERY

- IF:**
- a. the baby is well
 - b. the baby is >36 weeks gestation
 - c. the mother is well
 - d. there is no obvious chorioamnionitis (no foul smelling liquor)
 - Treat with ampicillin and gentamicin and stop after 48 hours if the baby remains well.

- IF:**
- a. the baby is sick
 - b. the baby is <36 weeks gestation
 - c. the mother is sick
 - d. there is obvious chorioamnionitis
 - Take blood culture and blood slide
 - Treat with ampicillin and gentamicin for a minimum of 5 days
 - Treat for longer (see above) if there is a slow response or if cultures are positive.

REFERENCES

Bang AT, Bang RA, Beitule SB et al. Lancet 354:1955-1961,1999. Effect of home-based neonatal care and management of sepsis on neonatal mortality: field trial in rural India.
Frank D, Duke T. PNG Med J 43(1-2):121-126,2000. Congenital syphilis in Goroka Base Hospital: incidence, clinical features and risk factors for mortality.
Taha TE, Biggar RJ, Broadhead RL et al. Br Med J 315: 216-219,1997. Effect of cleansing the birth canal with antiseptic solution on maternal and newborn morbidity and mortality in Malawi: clinical trial.

NEONATES - JAUNDICE

Routinely measure only the total serum bilirubin (SBR). Measure the direct and indirect bilirubin only if jaundice persists beyond 14 days of age, or if the stools are pale.

Healthy baby 2.2 kg or more		
Total bilirubin (mmol/l)	Under 48 hours	Over 48 hours
170-240	Phototherapy	-
240-320	Phototherapy	Phototherapy
320-425	Exchange	Phototherapy
>425	Exchange	Exchange
Cease phototherapy when SBR under 240		

Sick baby 2.2 kg or more OR healthy baby under 2.2 kg		
Total bilirubin (mmol/l)	Under 48 hours	Over 48 hours
170-240	Phototherapy	Phototherapy
240-320	Exchange	Phototherapy
>320	Exchange	Exchange
Cease phototherapy when SBR under 155		

Sick baby under 2.2 kg		
Total bilirubin (mmol/l)	Under 48 hours	Over 48 hours
85-170	Phototherapy	-
170-240	Exchange	Phototherapy
>240	Exchange	Exchange
Cease phototherapy when SBR under 140		

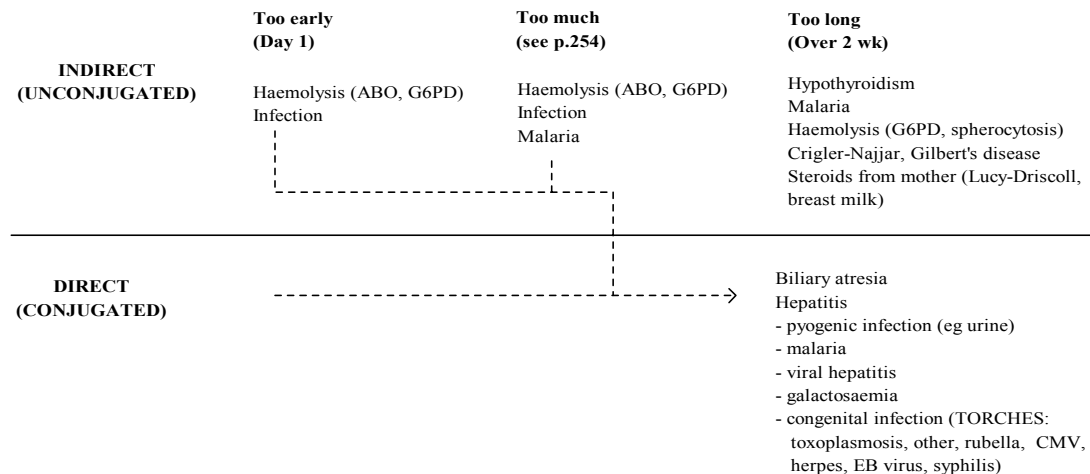
Notes:

1. Neonates may be jaundiced because of infection. Consider cultures of blood, urine and possibly CSF (if indicated). If the baby is not sucking well or if there is any other reason to think there may be infection commence treatment with ampicillin and gentamicin for at least 5 days.
2. Babies under phototherapy require additional fluids. A full term healthy neonate will take care of this by sucking more from the breast. For small or sick babies it is important to give the extra fluids required - either by cup and spoon or by nasogastric tube (give an additional 20% of the daily fluid requirement).
3. Measure the SBR at least daily until the day after stopping phototherapy (longer if the SBR rises again).
4. If the SBR appears to be rising rapidly start phototherapy sooner than indicated above and repeat the test.
5. The levels of bilirubin stated here for exchange transfusion are higher than those normally specified in developed countries. This is because of the higher risk associated with exchange transfusion outside specialist neonatal units and because Rh disease (which causes rapid haemolysis) is rare in Papua New Guinea. In Rh disease, you should do an exchange transfusion at lower bilirubin levels. Furthermore, bilirubin levels appear to be higher in healthy non-Caucasian than in Caucasian babies (Ann Trop Paediatr 5:127-30,1985).

ICTEROMETER

The icterometer is a transparent perspex strip with 5 shades of yellow painted on one side representing increasing degrees of jaundice. The perspex strip is pressed against the neonate's gum (this is much more accurate than pressing it on the skin of the nose) and the nearest colour is matched. It is useful to determine which babies should have a serum bilirubin assay. It should only be washed in water, not detergent or alcohol.

CLASSIFICATION OF CAUSES OF NEONATAL JAUNDICE



NEONATES - LOW BIRTH WEIGHT

DEFINITION

A low birth weight (LBW) baby is now universally defined as one that weighs less than 2500 g at birth. Babies between 1000 and 1500 g are called very low birth weight (VLBW) and those less than 1000 g extremely low birth weight (ELBW).

INTRODUCTION

Considerable expenditure and resources are used in affluent countries to provide neonatal intensive care. Babies as small as 700 grams are expected to survive. Such resources are not available in Papua New Guinea and it is unrealistic to expect all but a very few ELBW babies to survive. What is available, however, is the knowledge and resources to provide a high level of basic care for VLBW and LBW babies and good outcomes are achieved without the use of expensive high technology, but with skilled and dedicated nursing and medical attention.

CLASSIFICATION OF LOW BIRTH WEIGHT BABIES

Low birth weight babies include 2 groups:

Immature or preterm - gestation less than 37 weeks

The problems associated with immaturity are:

1. Respiratory distress syndrome
2. Apnoeic attacks
3. Feeding difficulties
4. Intraventricular haemorrhage
5. Jaundice
6. Infections
7. Hypothermia.

Small-for-dates - underweight for the period of gestation

The problems associated with small-for-dates babies are:

1. Intra-uterine malnutrition
2. Intra-uterine hypoxia and birth asphyxia
3. Meconium aspiration
4. Hypoglycaemia
5. Hypothermia
6. Infections.

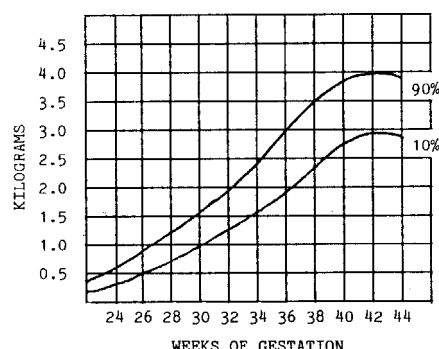
CRITERIA

Gestational age

Score the baby on the criteria indicated on the rapid assessment table.

Neonates - gestational age: rapid assessment																	
<p>SKIN OPACITY - Inspection of trunk when baby is quiet</p> <ol style="list-style-type: none"> 1. Numerous veins, tributaries and venules clearly seen 2. Veins and tributaries seen 3. Few large blood vessels clearly seen 4. Few or no large blood vessels indistinctly seen <p>BREAST SIZE - Pick up breast tissue between finger and thumb</p> <ol style="list-style-type: none"> 1. <0.5 cm on both sides 2. 0.5-1 cm palpable on one or both sides 3. >1 cm palpable on one or both sides <p>NIPPLE FORMATION - Inspection</p> <ol style="list-style-type: none"> 1. Nipple barely visible, no areola 2. Nipple well defined, areola present but not raised 3. Nipple well defined, edge of areola raised above the skin <p>SCALP HAIR - Inspection</p> <ol style="list-style-type: none"> 1. Fine hair, woolly or fuzzy, individual strands difficult to distinguish 2. Hair coarse and silky. Each hair appears as a single strand <p>EAR FIRMNESS - Palpation and fold upper pinna: assess more “mature” if 2 ears different</p> <ol style="list-style-type: none"> 1. No cartilage in the antitragus 2. Cartilage in the antitragus 3. Cartilage in the antihelix 4. Cartilage in edge of pinna <p>FINGER NAILS - Scratch nail on examiner</p> <ol style="list-style-type: none"> 1. Nails do not reach fingertips 2. Nails reach fingertips 3. Nails reach or pass fingertips, firm edge scratches <p>PLANTAR SKIN CREASES - Inspect only broad creases, ignore if disappear when stretched from toes to heel</p> <ol style="list-style-type: none"> 1. No skin creases 2. Anterior transverse creases only 3. Occasional creases over anterior 2/3 4. Whole sole covered with creases (including heel) 																	
Score	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
Weeks	27	28	29	30	31	32	33	34	35	36	37	38	38	39	40	41	42
Finnstom O. Acta Paed Scand 66: 601-04,1977. White PL Clin Paed 19:319-21,1980																	

Weight for gestational age



Classify the baby

Low birth weight: less than 2.5 kg

Preterm: less than 37 weeks gestation

Small for gestational age (SGA): below the 10% line on the graph

Birth weight	Gestational age <37 wks	Gestational age >37 wks
Below 10%	Preterm, small for dates	Term, small for dates
10% - 90%	Preterm	Normal
Over 90%	Preterm, large for dates	Term, large for dates

MANAGEMENT OF LOW BIRTH WEIGHT BABIES

There are four major principles of looking after LBW babies. They are the same the world over - but how they are put into practice depends on the resources available. These principles are:

1. Keep the baby pink
 2. Keep the baby warm
 3. Keep the baby fed and watered
 4. Prevent infection
-
1. Give vitamin K 1 mg after birth.
 2. Keep the baby warm. THIS IS THE MOST IMPORTANT ASPECT OF THE MANAGEMENT OF LOW BIRTH WEIGHT BABIES. A temperature that seems quite hot to an adult is actually VERY COLD for a small baby. Humidicribs are generally unnecessary, they require regular maintenance and they are often dangerous as sources of infection. The nursery for low birth weight babies should be a small, well-insulated room kept at a temperature of between 27 °C and 30 °C. It is necessary in the highlands areas to have wall or ceiling heaters to maintain this temperature - and it should be remembered that even on the coast the temperature falls well below 27 degrees at night. Premature babies should be kept well wrapped up with bonnets on their heads. Kangaroo care - nursing the baby between the mother's breasts - is a highly effective way of ensuring the baby is kept warm - but if this is not practicable the baby should be nursed on a heated, water filled mattress (Arch Dis Child 64:687-92,1989) or an electric blanket with transformer (see p.119).
 3. Handle the baby as little as possible. However, when the baby no longer needs oxygen, consider the option of "kangaroo care". The mother is encouraged to "kangaroo nurse" her baby naked (except for a nappy) against the skin between her breasts for as much of the day as possible and during the night when it gets cooler. There is nothing to stop the mother walking outside with the baby "kangaroo'ed" during the day. Kangaroo care is not always an easy concept for the mothers or the staff to adapt to - but dramatic reduction in mortality of VLBW babies has been documented in many countries. It reduces the risk of reflux and aspiration, reduces apnoea and infection and cuts down the length of hospital stay. It also encourages bonding between mother and baby, and improves lactation ("Current knowledge of Kangaroo mother intervention". Current Opinion in Pediatrics 8:108-112,1996).

4. Always be alert for signs of infection in a baby. ANY BABY WITH RESPIRATORY DISTRESS SHOULD BE GIVEN AMPICILLIN AND GENTAMICIN (as well as oxygen and other appropriate management).
5. Oxygen is only necessary if the baby is cyanosed or having respiratory difficulties. Give intranasal oxygen by inserting a 5 or 8 FG catheter into the baby's nose to a depth equal to the distance from the side of the nose (ala nasi) to the front of the ear (tragus). The catheter should be removed and cleaned at least twice a day. Oxygen from a cylinder is very dry: bubbling it through a bottle of water helps (change the water every day), but it is much better to use a heated humidifier (eg Hudson humidifier set at 100% oxygen). Give oxygen at the lowest flow rate required to make the baby pink (probably about 0.5 litre/min, not more than 1.0 litre/min). Put a size 5 FG nasogastric tube into the stomach through the other nostril and leave it open to prevent distension of the stomach and intestine.
6. Prevent apnoea: apnoea is common in those less than 32 weeks (see Neonates - Apnoea, p.236). Put these infants on an apnoea monitor, if available, and commence aminophylline.
7. Feeding:
 - a. If the baby is active and sucks well, breast feed on demand.
 - b. If the baby does not feed well from the breast, try feeding expressed breast milk with a cup and spoon. Do NOT bottle feed. If the baby is too weak to feed with a cup and spoon, feed by nasogastric catheter.
 - c. Use size 5 FG for babies <2200 g, and 8 FG for babies >2200 g.
 - d. Change the intragastric polythene feeding catheter twice a week.
 - e. Feed the baby full strength expressed breast milk by tube.
 - f. If expressed breast milk is not available, use sugar-milk (eg Sunshine or Anchor milk powder 1 part by volume, sugar half part, boiled and cooled water 6 parts) for the first 4 weeks of life, and then change to full strength full cream milk (eg Sunshine milk powder 1 part by volume, boiled and cooled water 3 parts). Give vitamin mixture (eg Pentavite or Abdec) 10 drops a day. Never give more than 250 ml/kg/day of artificial milk.
 - g. The amount of milk to give every 3 HOURS is shown in the table:

Birth weight (kg)	Day of life							
	1	2	3	4	5	6	7	>8
1.0-1.4	8	10	15	20	25	30	30	35
1.5-1.9	10	15	20	25	30	40	45	50
2.0-2.4	15	20	30	35	40	50	55	65
2.5-2.9	20	25	35	40	50	60	70	75
3.0-3.4	20	30	40	50	60	70	70	75
3.5-3.9	25	35	45	60	70	80	80	80

These quantities are a guide only. Some low birth weight babies will need more.

- h. Feed the bigger babies 3 hourly. The smaller babies (ELBW and VLBW) may not tolerate this amount and may require 2 hourly or hourly feeding with smaller volumes.
- i. Feed the baby before he/she is 2 hours old: this reduces jaundice and hypoglycaemia.
- j. When the baby is sucking well and gaining weight, the quantity of complement should be reduced so that full breast feeding is achieved as soon as possible. Many babies will be sucking well by the age of one week or less, and it is then not necessary to keep on increasing the complement.
- k. Some very premature neonates have fat malabsorption. This is not usually a problem if breast milk is used. However, if breast milk is not available and if the baby fails to gain weight on sugar-milk feeds, try giving a milk with a high medium chain triglyceride content such as Triglyde or Pregestimil. Start with Triglyde 1/7, and increase gradually to 1/4 over 3 days.
8. Prevent hypoglycaemia: this is common in low birth weight babies, particularly babies who are small for gestational age. A dextrostix should be done on all small-for-gestational-age babies at 2, 6, 12, 24, 36 and 48 hours of age. If the dextrostix is less than 2.5 mmol, send blood to the laboratory for a blood glucose so that the diagnosis is confirmed later:

If the baby has a dextrostix <2.5 mmol and has symptoms such as apnoea, convulsions, coma, bradycardia or hypotension, give IV 10% dextrose 5 ml/kg immediately, and then give the baby 8-12

mg/kg/min of glucose by a combination of IV and oral feeding. 2.4 ml/kg/hr of 10% dextrose IV gives 4 mg/kg/min of glucose, and 1.5 ml/kg/hour of milk orally gives 4 mg/kg/min of glucose

If the baby has a dextrostix <2.5 mmol but no symptoms, give the next milk feed immediately, by NG tube if necessary, and check the dextrostix one hour later.

9. Iron supplements. Low birth weight babies will become iron deficient during infancy unless they are given extra iron. If the mother can afford it, ask her to buy some Fergon Elixir (iron as ferrous gluconate) and give 20 drops each day until the child is taking solids. Alternatively, a hospital pharmacy can make up a solution of ferrous sulphate containing 200 mg/30 ml. Give 1 ml/kg/day (2 mg/kg of elemental iron). If you feel that the mother will not be able to give daily oral iron to her child, ask her to bring her baby back for an injection of 2 ml of Imferon when he is 4 months old.
10. Discharge. The baby is ready to go home when fully breast fed and gaining weight. There is no particular weight that has to be reached.
11. Vaccination. BCG vaccine, Sabin and Hepatitis B should have been given before discharge.
12. Follow-up. Ensure that the mother knows when and where to attend the MCH clinic or the Neonatal Follow-up clinic. Give the baby a Clinic Book. It is important to try and ensure that the babies discharged under 2 kg are seen and weighed within a week or two of discharge, so that feeding problems (admittedly rare) can be picked up early. Low birth weight babies have a higher risk of dying from infection in the first year of life than normal babies, although the risk may not be as high as thought (Pediatrics 81:807-11,1988).

NOTE THE TWO MOST IMPORTANT WAYS OF PREVENTING CROSS INFECTION IN THE NURSERY:

1. USING BREAST MILK (part of the baby's immune system)
2. ENFORCING STRICT HANDWASHING PROCEDURES FOR EVERYONE INVOLVED.

REFERENCES

- Allen SJ, Raiko A, O'Donnell A et al. Arch Dis Child Fetal Neonatal Ed 79:F135-140,1998. Causes of preterm delivery and intrauterine growth retardation in a malaria endemic region of PNG.
- Brown N. PNG Med J 39(1):12-15,1996. How should very low birthweight babies best be managed in Papua New Guinea?
- Charpak N, Ruiz-Pelaez JG, Figueroa Z, Charpak Y. Pediatrics 100:682-688,1997. Kangaroo mother versus traditional care for newborn infants <2000 g. A randomised controlled trial.
- Doyle LD. Lancet 350:1721-1722,1997. Kangaroo mother care. Editorial.
- McMaster P, Vince J. J Trop Paediatr 45:57-61,1999. Neonatal care in Port Moresby: a 19-year review.
- McMaster P, Haina T, Vince J. Trop Doctor 30(3):136-138,1999. Kangaroo care in Port Moresby.
- Peters HP, Vince JD, Friesen H. J Trop Paediatr 47:17-23,2000. Low birth weight in a Papua New Guinea Highlands hospital.
- Saugstad OD. Clin Perinatol 25(3):741-756,1998. Resuscitation in room air, or oxygen supplementation?
- Simkiss DE. J Trop Pediatrics 45:192-193,1999. Kangaroo mother care. Editorial.
- Vince JD. Med J Aust 159:78-80,1993. Neonatology: constraints, triumphs and challenges in contemporary Papua New Guinea.

NEONATES - MECONIUM ASPIRATION

Meconium aspiration is a potentially fatal condition which, in some cases, may be prevented by appropriate delivery room management.

The paediatrician or medical officer should be notified in good time and be present at delivery.

When meconium is present at delivery, always suction the mouth, nose and nasopharynx as soon as the head is delivered and before delivery of shoulders (immediately after head in breech). This is to clear meconium before the baby gasps.

If the meconium is thin and the baby cries immediately, no further action is required.

If the meconium is thick and the baby cries immediately, observe within next 2 hours:

1. if no respiratory distress, admit to postnatal ward
2. if developing respiratory distress, admit to SCN

If the baby does not cry, move baby quickly to resuscitation area.

If you are confident at intubation, check for meconium aspiration by direct laryngoscopy.

When meconium is present in trachea:

1. Either intubate with a large suction cannula (size 10 FG) and apply suction directly to trachea or intubate with an ETT tube and attach suction to ETT whilst withdrawing it. When meconium is very thick it will not pass through ET tube or suction catheter but can be removed by extubation and the application of suction simultaneously. The plug of meconium will be removed with tube or suction catheter.
2. When trachea and cords are clear, resuscitate in the normal way.

If you are not confident at intubation, start resuscitation with bag and mask.

Remember that babies who aspirate meconium are also asphyxiated and therefore lung inflation should not be delayed more than 1-2 minutes.

Before delivery, check suction no greater than 100 mmHg (15 kPa) when suction tubing fully occluded.

Transfer baby to SCN if:

1. meconium is visualised below cords
2. baby is tachypnoeic or has chest recession
3. there is any significant risk of infection.

On arrival in Nursery, commence oxygen if indicated:

1. NBM
2. Antibiotics
3. IV fluid.

Watch for complications:

1. Pneumothorax
2. Respiratory failure
3. Persistent pulmonary hypertension.

REFERENCE

Moodley et al. Trop Doctor 28(1) 31-34,1998. Intrapartum amnio-infusion for meconium stained liquor in developing countries.

NEONATES - MENINGITIS

See also Lumbar Puncture (p.192) and Cerebrospinal Fluid - Examination (p.70).

Symptoms and signs of neonatal meningitis are non-specific. Early diagnosis depends on a high index of suspicion and a low threshold for lumbar puncture.

A lumbar puncture should be performed in any baby who:

1. is not sucking well
2. is vomiting or has abdominal distension
3. is febrile or hypothermic
4. is having apnoea
5. is unduly irritable
6. is unduly lethargic
7. looks unwell
8. is fitting
9. has a bulging fontanelle.

If the CSF is normal: give ampicillin and gentamicin for 5 days.

If the CSF is unobtainable, blood stained or has an abnormal cell count:

1. give ampicillin and gentamicin
2. repeat the LP in 1-2 days.

If the CSF is suggestive of meningitis:

1. treat with ampicillin and gentamicin initially. Modify this if necessary on the basis of microscopy and culture.
2. treatment should be continued for 3 weeks.

TREATMENT

Antibiotics

The treatment of neonatal meningitis is unsatisfactory, and the mortality is 30-60%. It is often due to infection with gram negative organisms, and the antibiotics active against them (eg aminoglycosides) cross poorly into the CSF or are toxic to neonates (eg chloramphenicol). Ampicillin plus a third generation cephalosporin such as cefotaxime is now often used to treat neonatal meningitis, but this combination is very expensive, and may not be much better than ampicillin plus gentamicin.

Anticonvulsants

Phenobarbitone 15-20 mg/kg IM or 10 mg/kg slowly IV stat, then 5 mg/kg/day as prophylaxis. Paraldehyde to stop fits (or diazepam - but beware of the combination of phenobarbitone and diazepam - which may cause respiratory arrest). Phenytoin if no control (see Convulsions, p.88).

Restrict total fluid

Restrict intake to 60 ml/kg/day (80 ml/kg/day if having phototherapy).

REVIEW

Review the antibiotic treatment when culture results available

Ampicillin plus gentamicin is the best available treatment for *E coli*, *Streptococcus faecalis*, *Proteus* or *Listeria meningitis*. The combination is often synergistic against these organisms. Penicillin and gentamicin should be used for Group B streptococcus meningitis.

Staphylococcus aureus meningitis should be treated with cloxacillin and gentamicin. Penicillin is the treatment of choice for pneumococcal or meningococcal meningitis.

Chloramphenicol is the best antibiotic for *Haemophilus meningitis*, since it is bactericidal against this organism. Unfortunately, chloramphenicol resistant *H influenzae* is becoming common. Third generation cephalosporin can be used if available. Chloramphenicol is only bacteriostatic against *E coli* and *Proteus* and should NOT be used to treat meningitis caused by these organisms. Never give streptomycin, kanamycin or gentamicin with chloramphenicol (Lancet 2:210,1978).

NEONATES - MILK FEEDS

See also p.167, p.230 and p.259.

BREAST MILK

All doctors and nurses working with babies and children must be committed to breast feeding as by far the best way of feeding.

Health institutions should follow the prerequisites of the Baby Friendly Hospital Initiative.

Bottle feeding should not be practiced in the nursery or hospital wards.

Nursing staff should be familiar with the common breast feeding problems and see it as their responsibility to help mothers (particularly the young primigravid mothers) who are experiencing some difficulty with feeding.

Lactation can be assisted with the judicious use of Maxolon (metoclopramide) or chlorpromazine (see p.182).

Babies should usually be breast fed.

Premature babies who are too preterm to suck should be given expressed breast milk (EBM) by nasogastric tube or by cup and spoon.

Babies should be fed sugar-milk ONLY if breast milk is not available.

Special circumstances

1. If a neonate develops mild diarrhoea:
 - a. stop all milk feeds and give sugar-water until the diarrhoea stops. If the diarrhoea lasts more than 24 hours, treat as for severe diarrhoea.
2. If a neonate develops severe diarrhoea:
 - a. stop all oral feeds immediately
 - b. give IV fluids (see p.104)
 - c. when diarrhoea has completely stopped for at least 24 hours, gradually introduce breast feeding or EBM. If this is not tolerated it may (rarely) be necessary to use a lactose free milk such as Nutramigen or Pregestimil.
3. Very occasionally a baby may not tolerate either breast milk or a low lactose formula. These babies will require a period of intravenous alimentation.
4. Maternal drug ingestion:
 - a. there are very few absolute contraindications to breast feeding. Cytotoxics, warfarin and immune suppressants would be definite indications for use of cup and spoon feeding with sugar-milk or a formula feed.

Copy out this chart and put it up on the wall in your nursery kitchen:

SUGAR-MILK

Sunshine or Anchor milk powder x 1, Water x 6, Sugar (sucrose) x 0.5

SUGAR-WATER

Large mug water, heaped teaspoon sugar (or 2 heaped teaspoons glucose)

DIGESTELACT

Digestelact powder x 1, Water x 7, Glucose (Glucodin) x 1. If no glucose, use sugar x 0.5.

NUTRAMIGEN OR PREGESTIMIL OR TRIGLYDE

Start with: Milk powder x 1 (Nutramigen or Pregestimil or Triglyde 1/7), Water x 7

Gradually increase up to: Milk powder x 1, (Nutramigen or Pregestimil or Triglyde 1/4), Water x 4

NEONATES - NECROTISING ENTEROCOLITIS

PREDISPOSING FACTORS

1. Prematurity
2. Perinatal asphyxia
3. Hypotension
4. Respiratory distress
5. Early formula feeding
6. Congenital heart disease
7. Hirschsprung's disease
8. Umbilical artery catheterisation

CLINICAL FEATURES

Onset:	Usually 2 - 21 days of age
General symptoms:	Lethargy/hypotonia Apnoea Shock/hypotension
Specific symptoms:	Bile stained aspirates or vomit Abdominal distension/tenderness/rigidity Blood in stool. Visible/occult
Severe or fulminant disease:	Oedema of abdominal wall Sclerema Generalised bleeding tendency

INVESTIGATIONS

Full infection screen (see infection chapter, p.251).
Platelet count and clotting studies, if available: DIC is common in this condition with low platelet count.
Abdominal x-ray, supine and erect/lateral decubitus. Gaseous distension, oedematous loops, frothy appearance of bowel lumen, fluid levels, pneumatosis cystoides intestinalis (bubbles of gas in the bowel wall - pathognomonic), gas in portal vein. Free air in abdominal cavity: look for visualisation of ligamentum teres on the supine film.

DIAGNOSIS

This is easy in severe cases with pneumatosis cystoides intestinalis. Milder cases are difficult to differentiate from simple ileus or poor gut motility. It is wise to stop enteral feeds at the first sign of problems. When in doubt, it is better to over treat.

MANAGEMENT

1. Stop enteral feed.
2. Aspirate stomach content and then leave on free nasogastric drainage with 3 hourly aspirations.
3. Correct shock/hypotension with colloid or FFP.
4. Institute IV maintenance fluids and consider TPN in well established cases.
5. Commence antibiotic treatment with penicillin, gentamicin and metronidazole.
6. Avoid repeated abdominal palpation as this may precipitate a perforation.
7. Discuss with surgical colleagues.
8. Re-introduction of enteral feeds will vary according to the severity of the enterocolitis. Certainly, feeding cannot recommence until the abdominal distension has subsided and aspirates have

diminished. In well established cases, it is usual for enteral feeding to be precluded for at least a week.

LONG TERM COMPLICATIONS

1. Malabsorption secondary to a short bowel following surgical resection.
2. Strictures which may occur several years after original episodes.

NEONATES - PATENT DUCTUS ARTERIOSUS

The ductus arteriosus joining the aorta to the pulmonary artery frequently remains patent in small premature babies. There may be a large left to right shunt across the ductus, which causes severe cardiac failure. This may be life threatening in premature babies with severe lung disease (eg pneumonia or RDS) or apnoea of prematurity. A persistent ductus predisposes the baby to chronic lung disease by increasing oxygen dependence. Treat the cardiac failure by restricting fluid intake and giving digoxin and hydrochlorothiazide in the hope that the ductus will close spontaneously (note that frusemide may keep the ductus open - New Engl J Med 308:743-748,1983).

DIAGNOSIS

1. Heart murmur systolic (59%) or continuous (30%) and heard maximally below the left clavicle - but in 11% there is no audible murmur (Note the loudness of the murmur is not a reliable guide to the severity of the problem).
2. Bounding pulses, easily palpable dorsalis pedis pulse, wide pulse pressure and hyperactive precordium with tachycardia.
3. Increasing respiratory distress.
4. Pallor (with a normal Hb).
5. Apnoea.
6. Hepatomegaly if in heart failure.
7. CXR shows large heart and pulmonary plethora.

TREATMENT

1. Fluid restriction. Limit to 90-120 ml/kg/24hrs - and give 20% less than previous day.
2. Diuretic. Use chlorothiazide 20 mg/kg once or twice daily rather than frusemide (which may promote PDA).

This treatment may be sufficient but if the ductus persists:

1. Indomethacin. Indomethacin (Indocid) is a prostaglandin antagonist. It has been found that prostaglandins keep the ductus open, and that indomethacin will close the ductus in many premature babies. It is most likely to be effective if the baby is less than 12 days old. Indomethacin has no place in the treatment of PDA after the neonatal period.
2. Indomethacin suspension (0.3 mg in 1 ml):
 - Indocid capsule powder 25 mg
 - Cpd. tragacanth powder 3.3 g
 - Syrup BP 75 ml
 - Distilled water to 83 ml
 - Store in fridge for up to 7 days. Shake well before use.

Give 0.5 ml/kg (0.15 mg/kg) orally or by NG tube. The ductus usually closes after a few hours, but it may reopen again. This dose should be repeated twice a day for up to four days if there is no response. Complications are transient renal failure, vomiting, abdominal distension and cyanosis.

Indomethacin should NOT be given to all premature babies with PDA: the ductus usually closes spontaneously. Only use it for a large ductus causing cardiac failure despite fluid restriction, digoxin and hydrochlorothiazide, particularly if this exacerbates respiratory distress or apnoea.

REFERENCES

- Archer N. Arch Dis Child 69:529,1993. Patent ductus arteriosus in the newborn.
Friedman WF. New Engl J Med 295:526-529,1976. Indomethacin and PDA.
Green TP. New Engl J Med 308:643-748,1983. Frusemide promotes PDA.
Harnick E. Lancet 2:245,1977. Problems with indomethacin for PDA.
Heyman MA. New Engl J Med 295:530-533,1976. Indomethacin and PDA.
Merritt TA. J Pediatr 93:639-646,1978. PDA: ligation v indomethacin.

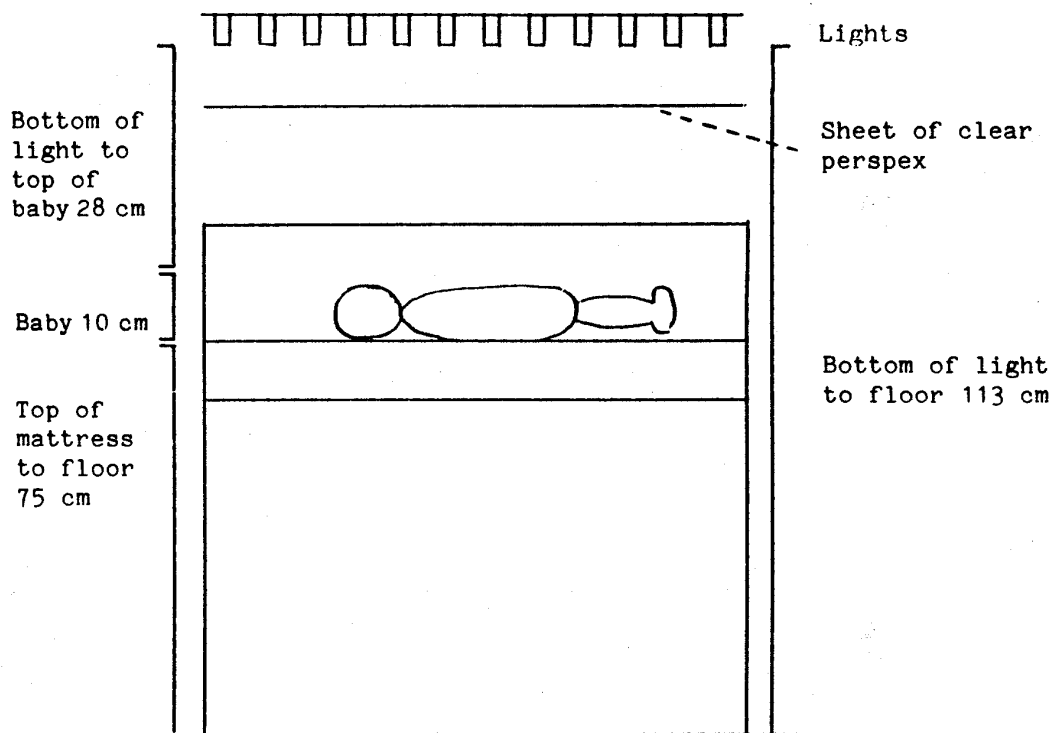
NEONATES - PHOTOTHERAPY

See also Neonates - Jaundice (p.254).

Bright blue light (wavelength 400-500nm) breaks down unconjugated bilirubin in the skin of a jaundiced baby. It can therefore be used to treat neonatal jaundice.

Phototherapy systems are available commercially but are expensive. An efficient unit can be simply constructed by mounting 6 or more ordinary 20 watt fluorescent tubes on a frame. It is important that the bottom of the light tubes should be 28cms above the surface of the baby's body. It is also important that there is space between the lamps to allow for ventilation, and that there is a sheet of clear perspex between the lamps and the baby (in case the lamps explode). The frame may be fixed to a wall, or to a separate stand, so that cots can be pushed underneath. Alternatively, a mobile unit can be made.

Since the emission of blue light from normal fluorescent light tubes deteriorates with time, THE TUBES MUST BE CHANGED EVERY THREE MONTHS. It is a good idea to mark the date the tubes are to be changed on the phototherapy unit.



Phototherapy unit for use with standard nursery cot. The unit can be attached to the wall, or have legs of its own (long enough for a cot to be pushed under it).

Phototherapy is a specific form of treatment. It is not simply a question of "putting the baby under the light". The following points must be noted:

1. Babies receiving phototherapy should be nursed naked except for a pad over the eyes (to protect the neonatal retina).

2. The baby's temperature must be carefully monitored. In the highlands babies may easily become hypothermic, unless nursed in a warm room (27-30 °C). Small babies may require an electric blanket. Even on the coast, the night time temperature may fall to levels that are dangerously low for small babies. On the other hand, babies may also become overheated under the phototherapy lamps and it is important that there be adequate ventilation.
3. Since it causes increased insensible water loss, babies under phototherapy require extra fluids. As a general guideline, babies should receive at least 120% of their normal daily requirements. Whilst an otherwise healthy full term baby may adjust his/her own fluid intake, accurate fluid balance is particularly important in low birth weight babies, or those who are not feeding vigorously.
4. Phototherapy may cause diarrhoea in some babies. Fluid balance becomes even more important in this situation.
5. Babies on phototherapy may not appear as jaundiced as they actually are. Serum bilirubin estimations should be done daily until the day after stopping phototherapy.
6. Phototherapy may make the baby drowsy. In this circumstance, it may be difficult to exclude neonatal sepsis. If in any doubt whatsoever, treat the baby with antibiotics for sepsis.

NEONATES - RESPIRATORY DISTRESS

Respiratory distress is present if the baby has at least two of the following:

1. Respiratory rate >60/min
2. Rib indrawing
3. Grunting
4. Nasal flaring
5. Cyanosis.

BUT a neonate may have respiratory illness without any specific signs - no tachypnoea, rib indrawing, grunting, cough, nasal discharge, nasal flaring or cyanosis.

- GIVE AMPICILLIN (OR AMOXYCILLIN) AND GENTAMICIN TO ANY BABY WITH RESPIRATORY DISTRESS
- KEEP THE BABY WARM
- EXCLUDE HYPOGLYCAEMIA.

1. **Pneumonia.** Any baby with respiratory distress must be treated for pneumonia even if you think there is another cause. Give IM or IV ampicillin (or amoxycillin) and gentamycin.
2. **Meconium aspiration.** Occurs mainly in term babies or small-for-dates babies. The clinical picture is of an asphyxiated baby with hyper-inflated chest and respiratory distress from the time of birth. There is often interstitial emphysema, pneumomediastinum or pneumothorax (see Neonates - Meconium Aspiration, p.261 and Neonates - Asphyxia, p.238).
3. **Pneumothorax.** This may occur spontaneously or secondary to meconium aspiration, respiratory distress syndrome or staphylococcal pneumonia. It may also complicate over-vigorous resuscitation. It is very difficult to diagnose clinically and can only be excluded by taking a chest x-ray. If the pneumothorax is small and not under tension, it will usually resolve with nasopharyngeal oxygen at 0.5 litre/min. If the pneumothorax is large or under tension it will require underwater seal drainage. In an emergency the pneumothorax can be aspirated with a syringe and needle. A large bore Dwellcath can be used and a standard drip tube, with the "bottom" end inserted into the catheter and the "top" end in a bowl of water below the level of the baby forms a readily available emergency underwater seal drain.
4. **CNS depression.** A sleepy baby, slow to breathe, cyanosed, often with rib indrawing, and a history of sedation to the mother prior to delivery. Give naloxone. If there is a response, observe the baby carefully over the next hour, as the effects of the naloxone wear off before those of the pethidine.
5. **Cardiac disease.** May cause cyanosis and tachypnoea and may be very difficult to distinguish from pneumonia, even with a chest x-ray. The commonest lesions presenting at different ages are:
 - 1st day of life: hypoplastic left ventricle (CCF)
 - 1st week of life: transposition (cyanosis) or coarctation of aorta ± VSD (CCF)
 - >1month: VSD (CCF), Fallot's tetralogy (cyanosis) or patent ductus (CCF)

Unlike cyanosis due to lung disease, cyanosis due to an intracardiac shunt will not be abolished with intranasal oxygen. Digoxin may help CCF (do not give digoxin to a child with Fallot's). Indomethacin may be used to close a PDA that is causing CCF- see p.267.
6. **Diaphragmatic hernia.** Herniation of the abdominal contents into the thorax (usually on the left side). There is gross rib indrawing, cyanosis, poor chest movements and sometimes a scaphoid abdomen. The apex beat is felt on the right side. Chest x-ray may show the heart on the right side (ensure correct labelling of the film) and bowel in the thorax (note that in a very early film the bowel may not contain any air). Pass a nasogastric tube and give oxygen by nasopharyngeal catheter. Do not use bag and mask or frog breathing ventilation (this will blow up the bowel with oxygen and make the respiratory distress worse). If ventilation is required, intubate.
7. **Oesophageal atresia** with tracheo-oesophageal fistula (TOF). Suggested by polyhydramnios and a mucousy baby with respiratory distress especially related to feeding, relieved by pharyngeal suction. The diagnosis is confirmed if a STIFF catheter will not pass to the stomach. Look for associated congenital abnormalities (Vertebral, Ano-rectal, Cardiac, Tracheo-oesophageal, Renal, and Limb - the VACTERL association). Whilst waiting for surgery, nurse the baby prone, suck out the mouth frequently, and give IV fluids, with nil by mouth. All babies born following a pregnancy with

polyhydramnios should have a 5 FG catheter passed into the stomach immediately after delivery and the aspirate checked with litmus paper.

8. **Respiratory distress syndrome** - RDS (hyaline membrane disease, surfactant deficiency). This is associated with pre-term delivery, asphyxia, maternal diabetes, Caesarean section and APH. Give oxygen and ampicillin and gentamycin. RDS cannot be reliably distinguished from neonatal pneumonia. RDS is relatively uncommon in PNG, probably because most preterm PNG babies are also small for dates. Avoidance of perinatal hypoxaemia and postnatal hypothermia limits the frequency and severity of RDS.

MANAGEMENT

1. **Give oxygen.** Use a 5 or 8 FG feeding tube. Measure the distance from the anterior nares to the tragus of the ear. Insert the end of the tube this distance - which will bring the tip into the nasopharynx, but not the oesophagus. Start at a flow rate of 0.25 litre/min, and increase to a maximum of 0.5 litre/min. **GIVE ONLY ENOUGH OXYGEN TO KEEP THE BABY PINK.** Excess oxygen causes permanent retinopathy. Vagally mediated bradycardia may result from stimulation by the catheter tip of the nasopharynx. If this happens, pull the catheter back. A stomach distended with oxygen can be decompressed by placing a 5 FG feeding tube through the other nostril.
2. **Check the dextrostix.** Give 5 ml/kg IV dextrose if less than 2.5 mmol/l, but still check for other causes of respiratory distress. If you suspect hypoglycaemia but there is no dextrostix, give a bolus of 5 ml/kg 10% dextrose IV.
3. **Antibiotics.** Always give ampicillin (or amoxycillin) and gentamicin for at least 5 days.
4. **Maintain fluid balance.** Give IV fluids if the baby is sick enough to require oxygen. Nasogastric feeds can be gradually introduced as the respiratory distress improves.
5. **Take a chest x-ray.** To exclude pneumothorax, diaphragmatic hernia.
6. **Other treatment.** Give digoxin if there is cardiac failure. Consider giving indomethacin if there is a PDA (see p.267).

NEONATES - RESUSCITATION

Most babies do not require active resuscitation. It is normal for some fluid (lung fluid squeezed from the lungs during delivery) to drain from the mouth and nose. Suction is not required.

However a small number of babies will need resuscitation - and it is important to:

1. have advanced warning of their arrival, if possible (eg if there is foetal distress - including meconium staining, if the baby is known to be preterm or small for dates, if there is breech presentation or if there are twins)
2. have fully operational resuscitation equipment available.

It is best to arrive at the resuscitation before the baby does!!

It is also best to have some assistance available. Two pairs of hands are better than one.

1. **Gently suck out the nose and mouth when the head is delivered.** If there is thick meconium staining, this is the most important part of management (see Neonates - Meconium Aspiration, p.261).
2. **Transfer the baby to a flat surface** as soon as the cord has been clamped and cut.
3. **Quickly dry the baby with a towel** and then wrap the baby in a cloth (unless you have an overhead heater). IT IS MOST IMPORTANT TO KEEP THE BABY WARM AT ALL TIMES.
4. **Assess the baby's condition.** The Apgar score at one minute after complete delivery is traditionally used - but in practice the score is given in retrospect and decisions are based on a quick assessment of colour, respiratory effort, heart rate and muscle tone or activity.

Apgar score			
	0	1	2
Appearance	Blue or pale	Baby pink, blue extremities	Completely pink
Pulse	Absent	Below 100	Over 100
Grimace*	No response	Grimace	Cry
Activity	Limp	Some flexion limbs	Active
Respiration	Nil	Hypoventilation, poor cry	Good, strong cry
*Give a light slap to the soles of the feet			

Apgar score	Infant's condition	Physiological group
7 - 10	Vigorous	Normal
3 - 6	Some depression	Primary apnoea
0 - 2	Severe depression	Terminal apnoea

The changes in Apgar between 1 and 5 minutes and within the first 15 minutes after delivery have been shown to be reasonable predictors of outcome.

If the baby is blue, has irregular or inadequate respiration and a heart rate of 60/min or less (an Apgar score of 3-6) he is probably in primary apnoea.

1. Stimulate by gentle oral or nasal suction.
2. If this is not effective in establishing spontaneous respiration, gently flick the baby's feet.
3. If this is still not effective commence ventilation with bag and mask. Ventilation should certainly be commenced if there is no spontaneous respiration by 2 minutes. It is usual, but not essential, to use oxygen. Recent reports suggest that ventilation with air may be at least as effective as ventilation with oxygen. Make sure that the neck is in the neutral position and not hyper-extended, that the chin is lifted and that the mask is of an appropriate size and fits properly. If ventilation with bag and mask (or frog breathing) does not result in spontaneous respiration within 1-2 minutes then the baby should be intubated if possible.

If the baby is blue or white, apnoeic, and with a heart rate less than 60/min or absent (an Apgar score of <3) he is probably in terminal apnoea, and requires urgent ventilation.

1. Quickly but gently suck out the nose, pharynx and mouth if necessary.
2. Give bag and mask ventilation for a few breaths.
3. Intubate the baby if you can do so competently. Use a 2.5 mm ET tube for babies less than 2 kg, a 3.0 mm tube for those 3-4 kg and a 3.5 mm tube for bigger babies.
4. If you are unable to intubate competently continue with bag and mask ventilation (bag and mask ventilation with the baby's airway correctly positioned and using the appropriate bag and correct size mask is very efficient in most instances).
5. The first few inflations of the bag should be long (2-3 seconds). This helps to establish functional residual capacity. Thereafter, ventilate rhythmically about 30 times per minute with inspiratory time about 1 second. Always check for chest movement and auscultate for breath sounds. Unequal ventilation in an intubated baby usually means that the tube is in the right main bronchus and should be withdrawn.
6. If, after 30 seconds of adequate ventilation (assessed by observing chest movement) the heartbeat remains less than 60/min with no signs of acceleration, give external cardiac massage (depress the middle third of the sternum one third of the depth of the chest at a rate of 90-100/min. If you have an assistant the most effective method is to encircle the chest with both hands and depress the sternum with both thumbs). Give 3 chest compressions to one breath.
7. If there is no improvement, adrenaline 1 in 10,000, 0.1ml/kg may be given via the endotracheal tube or intravenously after obtaining venous access (usually through the umbilical vein).
8. If the heart rate still remains low, acidosis is likely. Give 8.4% sodium bicarbonate 1 ml/kg intravenously. Bicarbonate and adrenaline are usually not appropriate if the baby has no heart beat.
9. If there is still no improvement, check the dextrostix if possible. If the recording is less than 2 mmol/l, or if you have no dextrostix, give 5 ml/kg 10% dextrose intravenously.
10. If there is no response to resuscitation by 10 minutes or if there is no spontaneous respiration by 30 minutes, it is extremely unlikely that further effort will result in a good outcome, and resuscitation should be stopped.
11. Note: If there is a good response to resuscitation, but no spontaneous respiration, and if the mother has had pethidine within 8 hrs of delivery, give naloxone, 0.1 mg/kg IM. It is important to observe these babies over the next few hours, as the effects of naloxone may wear off before those of the pethidine.

REFERENCE

Resuscitation at birth. Chap 7. In: Advanced paediatric life support. The practical approach. 3rd ed. London: BMJ Books, 2001.

NEONATES - TETANUS

Neonatal tetanus is a serious disease with a very high mortality. It is prevented by aseptic cord care after delivery, and if the mother has had two injections of tetanus toxoid, at least one of them during the pregnancy (this was discovered in Papua New Guinea, see Schofield. Br Med J 2:785-789,1961).

The average time in hospital is 40 days; muscle rigidity lasts for 5-7 weeks.

1. Keep the baby warm.
2. Avoid all unnecessary handling and noise.
3. If the spasms are severe, give intranasal oxygen.
4. Give paraldehyde 0.2 ml/kg IM stat on admission to provide initial sedation.
5. So that the diazepam, tetanus immunoglobulin and fluid can be given IV, insert an IV drip of 4.3% dextrose in 0.18% sodium chloride (or 10% dextrose in 0.18% sodium chloride, if it is available) with 1.5 g KCl per litre, and run it at 6 ml/kg/hour using a burette.
6. Give slow intravenous injections of DIAZEPAM to abolish muscular spasms and rigidity. Give 2.5 mg (0.5 ml of 10 mg/2 ml) IV every 5 minutes. Up to 30 mg may be required in some babies. Ensure that there is no extravasation of diazepam into the subcutaneous tissues, or necrosis may occur. Inject the diazepam into the IV tubing as close to the IV cannula as possible. Have the IV fluid running in fast as you inject the diazepam. Diazepam is absorbed onto plastic tubing, so the drug must NOT be given by IV infusion. See p.360 for a discussion of the pharmacology of diazepam.
7. Pass a nasogastric tube.
8. Give maintenance sedation: diazepam 1-2 mg/kg by NG tube every 12 hours, and chlorpromazine 5 mg/kg by NG tube every 12 hours.
9. Additional slow intravenous injections of 2.5 mg of diazepam may be given for frequent, severe spasms. In severe tetanus, the baby dies because the amount of sedation required to control spasms causes severe respiratory depression.
10. Hiccups can be treated with chlorpromazine 2.5 mg IV.
11. Give tetanus immunoglobulin (human):
 - a. If it is available, use a 2,000 unit ampoule of intravenous human immunoglobulin. Perform a careful lumbar puncture, and remove 2 ml of CSF for microscopy, culture, protein and glucose. Then SLOWLY inject 3 ml (about 200 units) of immunoglobulin intrathecally through the LP needle. Infiltrate 3 or 4 ml of immunoglobulin around the umbilicus. Give the remainder of the 2000 unit ampoule plus another 2000 unit ampoule IV at 10 ml/kg/hour
 - b. If the IV preparation is not available, use 250 unit ampoules of human immunoglobulin for IM use. Give 375 unit (1.5 ampoule) IM stat. Infiltrate 125 unit (0.5 ampoule) around the umbilicus. Give another 250 unit (1 ampoule) IM the following day. Do NOT give the human IM preparation intrathecally.
12. Give dexamethasone 2 mg (0.5 ml) IM 12 hourly for 5 days.
13. Give benzyl (crystalline) penicillin and gentamicin for 7 days.
14. Clean the umbilicus thoroughly with hydrogen peroxide and a dry swab on a stick, then apply crystal (gentian) violet twice a day.
15. Severe spasms have usually been controlled after 2 to 3 days. Gradually introduce nasogastric milk feeds. Start with EBM 3 ml/kg/hour, increasing by 1 ml/kg/hour each day up to 10 ml/kg/hour, then change to 30 ml/kg 3 hourly. If the feeds are tolerated, remove the IV drip.
16. Reduce the dose of diazepam by 10% every third day; do NOT reduce it too rapidly.
17. Make sure the baby has had BCG, hepatitis B and Sabin vaccine and the first dose of triple antigen before discharge.

REFERENCES

- Galazka AM. Indian J Pediatr 52:329-341,1985. Control of neonatal tetanus.
- Khoo BH. Arch Dis Child 53:737-739,1978. High dose diazepam.
- Sanders RKM. Lancet 1:974-977,1977. Intrathecal ATS (horse) for tetanus.
- Sanders RKM. Trop Doctor 7:99-104,1977. Tetanus: successful treatment.
- Schofield F. Br Med J 2:785-789,1961. Neonatal tetanus in Papua New Guinea.

NEONATES - UMBILICAL VEIN CATHETERISATION

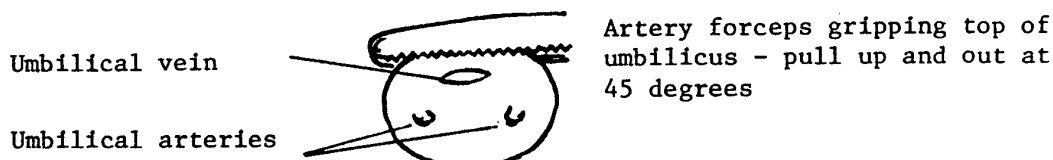
INDICATIONS

1. In an emergency, especially in the resuscitation of the newborn, the umbilical vein provides rapid access to the vascular space for drugs and fluids.
2. It is useful for large volumes of blood to be collected - such as in exchange transfusion.
3. It can be used when it is not possible to get an IV line established peripherally. It should be removed as soon as practicable (eg when a peripheral line has been established following restoration of fluid volume via the umbilical vein).

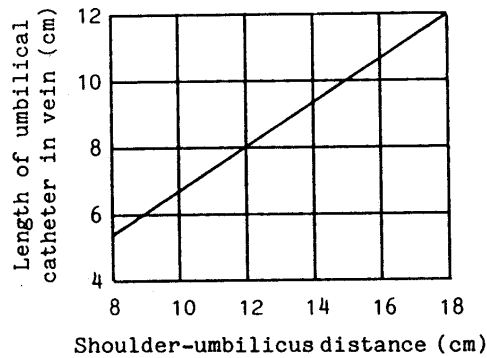
PROCEDURE

Measure shoulder to umbilicus distance before starting:

1. Have an assistant hold the supine baby's trunk and legs.
2. Put on sterile gloves.
3. Clean the abdomen with an iodine containing cleaning solution.
4. Slice the cord across with a sterile scalpel blade 0.5 cm from the base.
5. Pick up the umbilicus at the superior edge with a pair of artery forceps. Identify the umbilical vessels. The vein is usually superior and central (12 o'clock), and looks like a slit. The two arteries are at 4 and 8 o'clock and usually stand out as small white protrusions, the lumen not always being obvious.



6. Fill the catheter with normal saline. Use an 8 FG feeding tube for babies >2 kg, and a 5 FG for those <2 kg.
7. Carefully remove any clot from the vein with a pair of fine artery forceps, and holding the catheter near the tip with artery forceps insert the catheter into the vein. If you cannot see the vein, it can often be entered by gently pushing (but not forcing) the catheter into the funnel-shaped depression in the centre of the cord. The vein runs just under the skin up towards the baby's head, so point the catheter in this direction, NOT perpendicularly. The catheter usually threads in easily. Do NOT insert the catheter too far, just until blood flows back freely (see the graph below relating shoulder-umbilical measurement to inserted length).



From: Dunn P. Arch Dis Child 41:69, 1966.

Occasionally, the umbilical vein is kinked and the catheter “sticks” at about 1-2 cm. Gentle traction on the cord usually relieves this obstruction. Sometimes, there appears to be obstruction after a few centimeters. This is most likely at the porta hepatis. Gentle manipulation of the cord may help, but it may be necessary to withdraw the catheter a little and try again.

8. Connect the catheter to the exchange transfusion set or to the IV giving set. NEVER leave the catheter open to the air, because fatal air embolus can occur.
9. Strap the catheter in firmly. If the catheter falls out, the baby can die from blood loss very quickly.
10. DO NOT LEAVE THE CATHETER IN LONGER THAN NECESSARY (eg 48 hours). Always use a peripheral vein for IV infusion if you can. Prolonged umbilical vein catheterisation often leads to thrombosis and infection.
11. To remove the catheter, pull it out and apply pressure above the umbilicus by picking it up between thumb and index finger. Wait 2 minutes, then release the pressure and watch for bleeding. If there is no bleeding, put on a firm, dry gauze dressing. Ask the nurse to watch closely for bleeding for the next hour.

Note: When umbilical catheterisation is performed in the first 24 hours, a purse string suture can be applied.

NEPHROTIC SYNDROME

See also Oedema, p.282.

In general, nephrotic syndrome has a much worse prognosis in Papua New Guinea and in other developing countries than in Western countries. Minimal change disease is much less common with a large proportion of cases being focal proliferative glomerulonephritis, diffuse proliferative glomerulonephritis, membranous glomerulonephritis or amyloid. Many cases of nephrotic syndrome are probably due to an abnormal reaction to *P. malariae* or possibly to other forms of malaria and other infectious diseases.

In one series in Papua New Guinea, only about 20% of cases appeared to make a full recovery, and even this figure may be an overstatement because of poor follow up. By comparison, children in the United Kingdom with nephrotic syndrome more often have minimal change disease, and over 60% of cases make a full recovery.

Children with nephrotic syndrome and with microscopic haematuria or an elevated serum urea and creatinine are rather unlikely to respond to steroids - the mainstay of treatment for minimal change disease.

MANAGEMENT

1. Do a microurine, serum creatinine, blood slide, CXR and Mantoux. Ask about a family history of TB.
2. Give prednisolone 1 mg/kg BD for 4 weeks. If there is a response, reduce the dose gradually over 3 months, but keep proteinuria to no more than a trace. Discontinue prednisolone if there is no response after 4 weeks. Tail off over 2 weeks.
3. Give isoniazid while giving steroids.
4. Give a high protein diet. Do not give cow's milk, because of the high sodium content.
5. Treat for malaria (Treatment A, p.197) then give weekly prophylaxis (p.201).
6. Give phenoxymethyl penicillin (penicillin V) 250 mg BD to prevent pneumococcal infection.
7. Give frusemide and spironolactone only if there is massive oedema causing severe discomfort.
8. IV albumin can be given if there is circulatory collapse due to hypovolaemia. If there is severe respiratory embarrassment from pleural or peritoneal effusion, IV albumin can be given together with frusemide.

Cyclophosphamide (3 mg/kg/day for 8 weeks - with INAH cover) has been used in frequently relapsing steroid responsive nephrotic syndrome. Steroids are gradually reduced during the cyclophosphamide therapy. It is important to note that it is unlikely to be of any benefit if there is no response to steroid - and may be potentially fatal in a "tropical" environment. Levamisole (2.5 mg/kg second daily in addition to steroids) has also been used.

Low doses of ACE inhibitors (eg 2.5 mg/day enalapril) have been shown to be of benefit, and if available, should be tried in patients not responding to steroids and in relapsing patients.

REFERENCES

- Arneil GC. *Indian J Pediatr* 49:23-27,1983. Nephrotic syndrome in children.
Kincaid-Smith P. *Aust NZ J Med* 1:419,1971. Nephrotic syndrome in PNG.
Powell KC. *Aust NZ J Med* 4:363-373,1971. Nephrotic syndrome in PNG.
Saber MS. *Indian J Pediatr* 52:67-71,1985. Steroid-resistant nephrotic syndrome.

NORMAL VALUES

LABORATORY

Investigation	Sample	Normal value (child)
Alpha fetoprotein	2ml clotted blood	Absent
Bilirubin	2 ml clotted blood	2 - 17 micromol/l
Blood for exchange transfusion:		
• baby	1 - 2 ml clotted blood	
• mother	4 - 5 ml clotted blood	
Blood for transfusion	1 - 2 ml clotted blood	
Calcium	2.5 ml clotted blood	2.25 - 2.75 mmol/l
Inorganic phosphate		0.8 - 1.5 mmol/l
CSF		See CSF Examination (p.70)
Full blood examination (FBE):	2 ml EDTA	
• haemoglobin		11 - 15 g/dl
• PCV		33 - 45%
• MCHC		32 - 36 g/dl
• MCV		75 - 90 f L
• MCH		24 - 32 pg
• WCC		5 - 15 x 10 ^{9/l}
o neutrophils		5 - 15 x 10 ^{9/l}
o lymphocytes		5 - 15 x 10 ^{9/l}
o eosinophils		0.3 (3%)
o basophils		<1%
o monocytes		0.4 (3%)
o platelets		150 - 450,000/cu mm
o reticulocytes		<2%
Glucose	2.5 ml fluoride bottle	3.6 - 6.4 mmol/l
G6PD	6 drops on filter paper	Negative
Hb Electrophoresis	2.5 ml EDTA bottle	HbA2 <3%, HbF <2%
Liver function tests:	2 - 5 ml clotted blood	
• AST		5 - 50 u/l
• ALT		5 - 40 u/l
• alkaline phosphatase		100 - 300 IU
• total protein		60 - 85 g/l
• albumen		30 - 46 g/l
• globulin		30 - 45 g/l
Renal function tests (+ electrolytes):	2 - 5 ml clotted blood	
• sodium		135 - 146 mmol/l
• potassium		3.5 - 5.5 mmol/l
• chloride		95 - 107 mmol/l
• urea		1.3 - 6.6 mmol/l
• creatinine		0.01 - 0.06 mmol/l
• bicarbonate (CO ₂)		21 - 30 mmol/l
Thyroid function tests:	2 ml clotted blood	
• Free T4		0.75 - 1.85 ng/dl
• TSH		0.32 - 5.0 micro IU/ml
Urine		See Urine tests (p.382)
Widal	2 - 5 ml clotted blood	Titre >160 - very suggestive Four fold rise in titre - diagnostic

Note: The values listed above are those of children 2-4 years of age. Several of the above values vary with age. For most investigations the variation with age is small, but in some cases, for example neonatal haematological indices and glucose, values differ significantly. For detailed information, consult one of the major paediatric texts or books.

TEETH

Approximate age in months = 6 plus number of teeth (provided there are less than 18 teeth)
All 20 primary or deciduous teeth are usually present by 2½ years.

VITAL SIGNS

Age	Pulse rate (per minute)	Respiration (per minute)	Blood pressure (mmHg)
At birth	120-140	45	90/60
6 mth - 3 yr	110	30	90/60
4 yr - 7 yr	95	25	95/70
8 yr - 10 yr	85	20	100/70
11 yr - 12 yr	82	20	105/75

WEIGHT

A normal child should:

1. **double the birth weight by 5 months**
2. **triple the birth weight by 1 year.**

OEDEMA

See also Ascites (p.39, 40).

COMMON CAUSES OF OEDEMA OF LEGS AND FACE - SUMMARY

Urine protein -/+	Urine protein ++/++++
Kwashiorkor - clinical diagnosis	Nephrotic syndrome
Hookworm or strongyloides	CCF - exclude clinically
Acute nephritis - haematuria	
CCF - exclude clinically	

Oedema is quite a common finding in children in Papua New Guinea. It is important to have a rational approach to the investigation of these children.

Sometimes the children do not appear ill and they have a negative urine test for protein. It is sensible to treat these children with antihelminthics, antimalarials and to encourage a good diet. The oedema is often due to hypoproteinaemia due to loss of protein from the bowel secondary to Strongyloides infection or as a reaction to hookworm (which does not have to be heavy enough to cause anaemia). Malaria may be a contributing factor. Unusual causes of oedema include chloramphenicol, angioneurotic oedema, pertussis (face) and glandular fever (face).

TESTS

1. Urine for protein and blood
2. Faeces for cysts, ova and parasites
3. FBE
4. Blood slide for malaria parasites
5. Blood for urea, creatinine, electrolytes, protein and albumin
6. Take the blood pressure and examine the fundi.

The crucial test is for urine protein.

URINE PROTEIN ++/++++

Nephrotic syndrome

Urine protein +++++, with oedema of the face (worse in the morning) and legs. See p.279.

Cardiac failure

Urine protein ++, with oedema of the legs (that is worse in afternoon), tachycardia, hepatomegaly and a high JVP. Do a chest x-ray and an ECG. Give digoxin, diuretics and aneurine (thiamine) 5 mg tablet BD if no specific cause is found. See Cardiomegaly (p.66).

URINE PROTEIN NEGATIVE OR +

Kwashiorkor

Clinical diagnosis: pale sparse hair, flaking skin, apathy, underweight, oedema. See Malnutrition (p.210).

Acute nephritis

Haematuria (rather than proteinuria), hypertension (diastolic pressure over 90), CCF, oedema, fever:

- Give amoxycillin TID for 10 days, or give a stat dose of benzathine penicillin. Treat scabies (nephritis may be caused by secondary infection by streptococci).

Hypertension:

- restrict fluids
- IM or IV hydralazine 0.1-0.2 mg/kg (repeat if necessary)
- oral hydralazine 1.2-1 mg BD
- methyldopa 5-10 mg/kg QID oral
- frusemide 2 mg/kg IM once, followed by hydrochlorothiazide 1-2 mg/kg oral BD
- if available, captopril oral 0.1-1.0 mg/kg 6 hourly.

Cardiac failure or severe oedema:

- restrict fluids
- IM or IV frusemide, then oral hydrochlorothiazide.

Fluid restriction means giving the previous day's urine output, plus the fluid requirements in anuria. Monitor urea, Na, K.

Fluid requirements in anuria	
0 - 9 kg	30 ml/kg/day
10 - 11 kg	325 ml/day
12 - 15 kg	375 ml/day
16 - 23 kg	475 ml/day
24 - 36 kg	525 ml/day
37 kg or more	700 ml/day

Give the fluid as 10% dextrose oral or IV.

Hookworm or strongyloides

If the above diagnoses have been excluded, the oedema is probably caused by protein losing enteropathy from strongyloides or possibly hookworm infestation. There is a rapid response to albendazole or thiabendazole, a high protein diet and antimalarials. Treat anaemia if present.

Cardiac failure

This can, of course, present without proteinuria. There is a raised JVP, tachycardia and hepatomegaly. Do chest x-ray and ECG. Give digoxin, diuretics and aneurine (thiamine) 5 mg tablet BD if no specific cause found. See Cardiomegaly (p.66).

Chronic renal failure

This condition is not uncommon in Papua New Guinea. It may present with respiratory distress due to acidosis, with headaches or fitting due to hypertension, with tetany due to hypocalcaemia or with oedema.

Chronic liver disease

Chronic liver failure is rare in Papua New Guinean children.

REFERENCES

- Kincaid-Smith P. Aust NZ J Med 1:419,1971. Nephrotic syndrome in PNG.
Powell KC. Aust NZ J Med 4:363-373,1971. Nephrotic syndrome in PNG.

OSTEOMYELITIS, PYOMYOSITIS AND SEPTIC ARTHRITIS

OSTEOMYELITIS AND SEPTIC ARTHRITIS

Osteomyelitis and septic arthritis are both serious infections. Both present with fever, toxicity and severe pain.

In **osteomyelitis**, the affected limb which is hot and usually swollen, is exquisitely tender - and the child will not allow it to be touched. The ends of the long bones are most commonly affected - but it is important to remember osteomyelitis as a cause of bone pain and fever in other sites. A diagnosis of cellulitis should only be made with great caution - it is safest to regard a child with a hot swollen tender limb as having osteomyelitis. A very rapid response to antibiotic therapy will favour a diagnosis of cellulitis.

In **septic arthritis**, the child will not allow the affected joint to be moved.

Early diagnosis and treatment with intravenous antibiotics within the first 24 hours may prevent the need for surgery. Surgery is indicated if:

1. the history is longer than 48 hours
2. there is severe swelling of the limb or joint
3. there is no improvement within 24 hours of starting antibiotics.

Investigations

FBE

Blood culture (if available)

Culture of pus (or joint aspirate).

Note: X rays of the bone are not helpful in making the diagnosis in acute osteomyelitis.

Treatment

It is best to give antibiotics intravenously or intramuscularly until the child's symptoms improve. The most likely causative organism is *Staphylococcus aureus* but *Haemophilus influenzae* and *Salmonella* species are well recognised causes. In hospitals, it is reasonable to start treatment with cloxacillin and chloramphenicol until results of culture are known. If culture is not available or is negative, it is reasonable to give cloxacillin in older children and to give both cloxacillin and chloramphenicol in children less than 1 year.

Antibiotics should be continued for a minimum of 6 weeks.

Blood transfusion of packed cells should be given if the haemoglobin is less than 6 g/dl.

PYOMYOSITIS

The abscess involves only muscle, and not bone. It is a relatively common condition in Papua New Guinean children. Common sites include the quadriceps and the rectus muscles.

Treatment

Incision and drainage is usually necessary.

Antibiotics (usually cloxacillin or chloramphenicol) are given for a week.

REFERENCE

Reid IS. PNG Med J 13:59-60,1970. Pyomyositis.

OTITIS MEDIA

ACUTE

A red ear drum, OR pus discharging from the ear for less than 2 weeks.

1. Give amoxycillin TID for 5 days.
2. If the child is febrile, give antimalarials.
3. Look carefully for signs of meningitis, and do a lumbar puncture if you are in any doubt.

CHRONIC

One or both ears discharging pus for more than 2 weeks. The ear will only heal when it is dry.

Dry the ear

A number of techniques for drying the ear have been described. The Standard Treatment Book recommendation is the toilet paper spear method. This is something which can easily be taught to the mother, and which is safe and effective.

1. Use a piece of toilet paper.
2. Twist lightly from one corner to form a tissue paper spear.
3. Break off the tip which is too small.
4. Break off most of the paper so that there is a small blunt spear - about 4 cm long.
5. Get mother to steady the child's head while you pull the tip of the ear upwards and backwards to straighten the ear canal.
6. GENTLY push and twist the tissue into the ear canal until it stops (usually about 2 cm).
7. Leave in place for about 2 minutes to absorb the pus.
8. GENTLY remove the tissue spear.
9. If soaked with pus, repeat with more spears until the ear is dry.

Give the mother some toilet paper to take home. Tell her to dry the ear with toilet paper at least 4 times a day. This treatment should continue until the ear stays dry. It usually takes about a week. The child should not go swimming for one month.

Boric acid in alcohol ear drops can be used after ear toilet. For the first few occasions that it is applied it is painful.

Show mother how to put in the drops.

1. Lie the child on the side on mother's lap.
2. Put 1 or 2 drops in the ear canal.
3. Press twice on the flap of the skin in front of the ear canal to help push the ear drops through the perforation.
4. Supply one bottle of the ear drops for the mother to use at home.

REFERENCES

- Browning GG. Br Med J 287:1024,1983. Chronic otitis: antibiotics ineffective.
Eason RJ. New Zealand Med J 99:812-815,1986. Chronic otitis media.
Peever MV. Med J Aust 1(Suppl):1-4,1980. Successful assault on ear disease.

PAEDIATRIC RULES

1. IMMUNISE. Always check the health record book and immunise if the child is due or overdue for it.
 - a. There is no contraindication to giving measles vaccine
 - b. TA, Hepatitis B and Pigbel vaccines should not be given if the child has a fever above 38 °C. They should be given when the temperature has settled
 - c. Check the child's brothers and sisters and immunise them as well if they are not immunised up to date
 - d. Do not send a child who is due for immunisation away just to save vaccine just because he is the only one
 - e. If the mother is pregnant, she needs Tetanus Toxoid. Check that she is going to Antenatal Clinic.
2. ADMIT children who have any of the following:
 - a. intercostal recession - chest indrawing
 - b. dehydration
 - c. convulsion with fever
 - d. fever and not sucking
 - e. drowsiness or confusion
 - f. continued abdominal pain and vomiting
 - g. oedema (swelling)
 - h. weight less than 60% line, and flat or falling weight curve
 - i. MUAC less than 12.5 cm
 - j. sudden onset of paralysis
 - k. swelling of limb or joint
 - l. under 6 months with whooping cough
 - m. stridor (noisy breathing)
 - n. snakebite
 - o. swallowed poison
 - p. passing blood in the urine
 - q. vomiting blood
 - r. passing a lot of blood in the stool
 - s. history of unconsciousness after head injury
 - t. suspicious injuries which do not fit the history.

Also admit neonates with any sign of infection.
3. WEIGH. Always weigh the child. Give the correct dose of medicine for this weight. Plot the weight on the weight chart in the health record book.
4. REFER to hospital:
 - a. **Urgent referral:**
 - i. Babies born with an imperforate anus
 - ii. Babies with bile stained vomiting
 - iii. Babies with frequent vomiting and lots of saliva in the first few hours of life
 - iv. Babies less than 4 weeks old with meningitis, severe jaundice (orange) or those with sepsis who are not improving after 2 days of treatment
 - v. Babies with ambiguous genitalia (not sure whether the baby is a boy or a girl)
 - vi. Children with conjunctivitis who are not improving after 2 days of treatment
 - vii. Children with meningitis who are not improving after 2 days of treatment
 - viii. Children in coma
 - ix. Children with fever, tenderness and swelling of a limb or a joint which does not improve after 2 days of treatment
 - x. Children passing blood in the urine with or without oedema, who do not improve after 2 days of treatment
 - xi. Children with Kwashiorkor
 - xii. Children with a distended, tender abdomen
 - xiii. Children with sudden onset of paralysis
 - xiv. Children with polyuria (passing a lot of urine), dehydration and sweet smelling breath.

- b. **Non-urgent - but important referral:**
 - i. Children with slow development or who are poorly responsive and who have an umbilical hernia
 - ii. Children with a persistent heart murmur
 - iii. Malnourished children who do not respond to treatment for malnutrition
 - iv. Other children not responding to standard treatment.
- 5. Amoxycillin or procaine penicillin (procillin) must **only** be given to outpatients for the following diagnoses:
 - a. mild pneumonia (see p.311)
 - b. otitis media (see p.285)
 - c. skin sores (see p.350)
 - d. sudden swelling of lymph glands
 - e. tonsillitis (see p.355).

Note: “Strong cough”, “big cough” or “productive cough” are **not** reasons for giving amoxycillin or procillin **unless** the child has **fast breathing** or one of the above conditions.
- 6. Avoid giving injections unless the child is moderately or severely sick. Intramuscular injections to children less than 2 years old should be given on the upper and outer part of the thigh.
- 7. Do not give single doses of antibiotics. Do not give single doses of antimalarials for fever.

PAEDIATRIC SURVEILLANCE UNIT

The Papua New Guinea Paediatric Surveillance Unit was established in 1996. It is a combined project between the Paediatric Society of Papua New Guinea, responsible for determining the conditions under surveillance, and for the follow up of reported cases, and HOPE Worldwide (PNG), responsible for the administration of the Unit.

The aim of the unit is to obtain information relating to conditions occurring in Papua New Guinean children which, though not among the leading causes of death and morbidity, are nevertheless of importance.

All paediatricians, and a number of other doctors and health workers throughout the country actively participate in the Unit's programme. Each month the Unit sends out a card listing the diseases under surveillance to the participants, who return the card indicating if they have seen any children affected during the previous month. The paediatrician responsible for a particular condition then communicates with the reporter in order to obtain further information about the cases seen. This allows a central database on that condition to be built up.

There are currently about 40 participants reporting to the Unit.

Initially the diseases under surveillance were:

1. Insulin dependent diabetes mellitus
2. Paediatric HIV infection and AIDS
3. Congenital hypothyroidism
4. Thalassaemia
5. Renal tubular acidosis
6. Neurologic endemic cretinism
7. Paediatric malignancies.

A number of other conditions have been added, and, as adequate information has been obtained, some of the conditions have been deleted. Acute flaccid paralysis was one of the conditions added, and the Surveillance Unit worked closely with the Health Department Surveillance Unit in the successful programme to document the eradication of polio.

The Unit has provided important information on a number of conditions since its inception. Of particular note perhaps has been the information on paediatric malignancy, HIV/AIDS, subacute sclerosing panencephalitis and renal tubular acidosis.

The latest condition to be added is pigbel. It is anticipated that surveillance of this condition will help to provide a rational basis for decisions relating to pigbel vaccination.

Diseases currently under surveillance are:

1. Insulin dependent diabetes mellitus
2. Congenital hypothyroidism
3. Renal tubular acidosis
4. Neurologic endemic cretinism
5. Subacute sclerosing panencephalitis
6. Acute flaccid paralysis
7. Pigbel

PARALYSIS - ACUTE

An acute presentation with paralysis is not uncommon in Papua New Guinea. It is important to determine whether the paralysis is of **UPPER OR LOWER MOTOR NEURONE TYPE**.

Classically, the signs of **upper motor neurone** paralysis are hypertonia with hyperreflexia (spastic paralysis), upgoing plantars, and, if due to spinal cord compression, loss of sensation and impaired bowel and bladder function.

The signs of **lower motor neurone** paralysis are hypotonia, hyporeflexia (flaccid paralysis) downgoing or non-reacting plantars and no objective sensory loss. However, it should be remembered that in the early stages of spinal cord compression, which is normally associated with upper motor neurone paralysis, patients may present with hyporeflexia and downgoing plantars (spinal shock).

Cerebellar lesions cause hypotonia with decreased reflexes, but there is ataxia and nystagmus.

Disorders of the motor end plate (myaesthesia gravis) and of the muscles (the muscular dystrophies) should be considered in the differential diagnosis of hypotonic paralysis but there is usually a chronic presentation.

Hypokalaemia may also cause flaccid paralysis.

ACUTE FLACCID PARALYSIS

The child with acute flaccid paralysis presents the clinician with the need to make urgent decisions. All children with acute flaccid paralysis must be notified immediately to the provincial and national disease control officers, and to the Paediatric Surveillance Unit, as part of the disease surveillance requirements for the certification of polio eradication (see Poliomyelitis eradication, p.163).

Papua New Guinea was declared polio free in October 2000. There have been no confirmed cases of endemic wild polio for several years. Sabin vaccine associated poliomyelitis has occurred but is extremely rare. The paralysis is usually asymmetrical and there may be associated features such as symptoms of an aseptic meningitis.

The most likely diagnosis of acute flaccid paralysis is Guillain-Barre syndrome (acute postinfective polyneuritis). The classical presentation is with ascending symmetrical flaccid paralysis with hyporeflexia and downgoing or absent plantar responses and little, if any, objective sensory loss.

Less common causes of acute flaccid paralysis are hypokalaemia, botulism (cases in Papua New Guinea associated with eating turtle meat have been described), myopathy, neuropathy, diphtheria (no recent cases described but something to consider in an unimmunised child) and tick bite paralysis (the tick vector is not established in Papua New Guinea but the diagnosis should at least be considered, see p.333). Hypokalaemia may occur in people used to a diet of sweet potato (high in potassium) who suddenly change to eating rice (low in potassium). The paralysis is rapidly reversed by intravenous infusion of potassium 0.3 mEq/kg/hour MAXIMUM, followed by oral potassium.

Transverse myelitis may also present in the early stages with a flaccid paralysis which, over time, changes to a spastic paralysis.

As noted above, it should also always be remembered that patients with spinal cord compression, which is usually associated with spastic paralysis, may, in the early stages (spinal shock), present with flaccidity and hyporeflexia.

In assessing the child with acute flaccid paralysis the clinician should, therefore, seek a history of trauma to the spine (although vague histories of trauma are sometimes produced by parents as their explanation for what is happening to their child), signs of toxicity (including elevated neutrophil counts, that may suggest abscess formation), evidence of weight loss suggestive of tuberculosis or malignancy (Burkitt's lymphoma) or more subtle clues such as skin pigmentation suggestive of neurofibromatosis. For most clinicians practicing in Papua New Guinea, the only way to exclude a cord compression syndrome (spinal shock) requiring urgent surgical decompression is by performing a myelogram.

IF IN DOUBT, A MYELOGRAM SHOULD BE DONE.

Features suggesting Guillain-Barre syndrome (polyneuritis) rather than cord compression (a myelogram is not necessary):

1. Weakness of the face is never due to cord compression, but may occur in polio or polyneuritis.
2. Optic neuritis or papilloedema occasionally occur in transverse myelitis or polyneuritis, but not in cord compression.
3. Flaccid paralysis for longer than 6 weeks suggests poliomyelitis or polyneuritis rather than cord compression. Spinal shock rarely lasts longer than 6 weeks, so hyperreflexia with upgoing plantars has almost always begun to develop by then in cord compression.
4. Pain and tenderness of the calf muscles (often with slight fasciculation) and CSF lymphocytosis (which may have gone by 14 days after the onset of symptoms) suggest poliomyelitis.
5. A prodromal febrile illness, a latent period of up to 2 weeks, progressive symmetric flaccid paralysis from the legs up over hours to days, downgoing plantars, no sensory loss on testing (although subjective parasthesiae may be present) and normal bowel and bladder function: if ALL these features are present in a child old enough to co-operate with testing of sensation, the diagnosis is polyneuritis and myelography need not be done. Polyneuritis may occur without a prodromal illness, the latent period may be longer than 2 weeks, the paralysis may not be ascending and it may be somewhat asymmetric, and there may be retention of urine, but in such cases a myelogram should be done.

Features suggesting cord compression (a myelogram is indicated):

1. Localised back pain or tenderness.
2. Diminished pinprick sensation in the feet (often with no definite upper level).
3. Loss of position and vibration sense in the feet.
4. Decreased sweating below the level of the lesion.

Late signs of cord compression include the development of hyperreflexia and upgoing plantars, a sensory level to pinprick and/or vibration, and loss of anal tone with absent abdominal reflexes.

INVESTIGATIONS

1. If cord compression is a possibility, x-ray the chest and spine. If the arms and legs are paralysed, x-ray the cervical spine. If only the legs are paralysed, x-ray the thoracic and lumbar spine.
2. Examine the abdomen and look at the chest x-ray for evidence of a primary tumour. Examine the skin carefully for evidence of neurofibromatosis.
3. Do a full blood examination.
4. Do a serum potassium.
5. Do a myelogram if it is indicated.
6. Do NOT do a lumbar puncture until the time of myelography if cord compression is a possibility. If you do a lumbar puncture and viscous fluid is obtained (suggesting a high protein due to cord compression, Froin's syndrome), do NOT remove the needle until you have injected Myodil (p.233).

CSF findings:

1. Cord compression - the CSF is usually normal, but with total obstruction, there is xanthochromia with a high protein (over 1 gram/l).
2. Transverse myelopathy - normal CSF, or a mild increase in protein and cells.
3. Guillain-Barre syndrome - acute idiopathic polyneuritis - the CSF has a normal or slightly raised cell count with a disproportionately high protein after the first few days of illness.
4. Polymyelitis - for the first few days there are many polymorphs, with normal protein and sugar. After 2-3 weeks, the cell count is normal, but the protein increases to up to 3 gram/l.

MANAGEMENT

Care of the paraplegic patient is very demanding. Attention should be paid to the 5 “B”s:

1. Bowels - make sure the patient is not constipated. Give laxative if necessary.
2. Bladder - make sure there is no bladder retention. Expression of the bladder or catheterisation may be required.
3. Bed sores - make sure the patient is turned regularly.
4. Breathing - make sure the airway is kept clear. The best way to do this is by correct positioning. If there is respiratory paralysis, a decision concerning ventilation needs to be made.
5. Behaviour - remember that a paralysed patient may well be very wide awake. It is important to talk with him/her and to show “respect” at all times.

It is also very important to maintain adequate fluid and nutritional intake. This may mean inserting a nasogastric tube.

Regular physiotherapy is required (this can be done by the parents) to prevent contractures.

Guillain-Barre syndrome - postinfective polyneuritis

This is the commonest cause of acute flaccid paralysis in Papua New Guinean children. Steroids should NOT be given. The main threat to survival is respiratory paralysis. Unlike poliomyelitis, recovery from polyneuritis is usually rapid. Tracheostomy (p.364) and ventilation with a Bird respirator (p.46) is indicated if respiratory paralysis develops. Plasma exchange and intravenous human immunoglobulin have been shown to be equally effective treatments. In plasma exchange, whole blood is removed from the patient one unit at a time, the red cells are allowed to settle, and then returned to the patient with plasma from a normal blood donor. This is very labour intensive and time consuming. On the other hand, human immunoglobulin is very expensive. These treatments should probably only be used in very rapidly progressive disease. Recovery should be timed from the day that the paralysis ceases to progress - children (but not adults) who do not start to improve within 2 weeks of that day are unlikely to make a full recovery. Complete recovery usually occurs within 2 months, but may take up to 18 months.

Poliomyelitis

Give paracetamol for relief of pain. Prophylactic antimalarials should be given, but antibiotics should only be given to treat established infection. Steroids should not be given. No intramuscular injections should be given, since they may precipitate paralysis in that limb. Great care should be taken to prevent the development of contractures. A child with bulbar paralysis may die from inhalation of food or secretions. Nurse the child in the semi-prone position, turning him from side to side every two hours. The foot of the bed should be raised 15°. Give nasogastric tube feeds.

Most of the improvement will occur in the first year, but there may be some improvement in the second year. If the child has not been immunised, give three doses of Sabin vaccine to prevent a recurrence. The immediate prognosis depends on whether the patient develops respiratory or bulbar paralysis. Ventilation should not normally be attempted for a child with respiratory paralysis due to poliomyelitis, because prolonged or even indefinite ventilation may be required.

Transverse or ascending myelitis

Myelitis limited to a few segments of the cord is called transverse myelitis; when it spreads progressively upwards it is called ascending myelitis. It may follow a viral infection or any vaccination, or there may be no obvious cause. Occasionally, myelitis is caused by syphilis, pyogenic infection or tuberculosis.

If the patient survives the acute attack, there is usually considerable functional recovery. 60% have a good return to function and only 15% have no significant improvement. Steroids have no effect on the outcome unless swelling is so severe that it causes spinal block.

Spinal shock (cord compression)

Laminectomy should be performed urgently, before the ischaemic necrosis of the cord is irreversible. Some cases of acute paralysis with a block shown on myelogram will be found at laminectomy to be due to myelitis (non-specific inflammation of the spinal cord) - the block is due to gross oedema and swelling of the cord, which usually extends over too many segments for laminectomy to be done to decompress all the swollen cord. Such patients with extensive oedema of the cord causing compression should be given dexamethasone 0.25 mg/kg (maximum 10 mg) daily for one week, then reducing over a further week.

PARALYSIS - CHRONIC - FLACCID

Flaccid (hypotonic) paralysis of gradual onset is usually due to peripheral nerve disease, but it may be caused by a lesion of the anterior horn cells, motor roots, peripheral nerves, motor end plates or muscle. The reflexes are usually preserved until late in the course of muscular and the neuro-muscular junction diseases.

CEREBRAL LESIONS

Focal cerebral lesions usually cause unilateral weakness with increased tone, increased reflexes, upgoing plantars and no sensory loss. However, there may be initial hypotonia with an acute cerebral lesion.

Generalised chronic cerebral lesions may cause hypotonic weakness (which is unexpected, because upper motor neurone lesions usually cause hypertonia), but there are brisk reflexes (as expected in upper motor neurone lesions):

- encephalopathy (damage to normally formed brain): atonic cerebral palsy (atonic diplegia) is the commonest cause of the “floppy infant syndrome”; the plantars are usually upgoing
- dysgenesis (abnormally formed brain): eg Down’s syndrome, Prader-Willi syndrome
- degenerative (regression of normal CNS): storage diseases of lipid, mucopolysaccharide etc. There is a period of normal development, followed by loss of milestones.

SPINAL CORD LESIONS

Occult spina bifida may cause flaccid paraplegia. Spinal cord compression in the lumbar region may cause flaccid paralysis due to a cauda equina lesion. Acute compression of the thoracic or cervical cord may cause flaccid paralysis for a time (spinal shock), but compression of gradual onset causes spastic paralysis.

ANTERIOR HORN CELL DISEASE

Werdnig-Hoffmann disease is a rare progressive form of motor neurone disease that causes progressive hypotonia with absent reflexes and fasciculations. It usually starts in infancy.

PERIPHERAL NERVE LESIONS

Distal weakness with early impairment of reflexes, hypotonia, paraesthesiae and sensory loss. The diagnosis may be confused by the initial stages of a spinal lesion (spinal shock) or acute hemiplegia before hyperreflexia occurs, and by advanced muscle disease. If a child has no reflexes despite reinforcement (biting hard, pulling hands apart), he usually has neuropathy.

There are many causes of neuropathy. These include toxic substances (tick bite, heavy metals), drugs (such as isoniazid, streptomycin, phenytoin, chloroquine, vincristine, DDT), deficiency diseases (beriberi, malabsorption of B12 and folic acid), infections (leprosy, diphtheria, tetanus, dysentery, malaria, TB, typhoid), post-infective (Guillain-Barre), trauma, systemic disease (SLE, PAN, rheumatoid, sarcoid), genetic (peroneal muscular atrophy, Refsum’s disease) and idiopathic.

MYONEURAL JUNCTION DISORDERS

The classic cause is myasthenia gravis. Symptoms include ptosis, ophthalmoplegia, generalised weakness, dysphagia and occasionally respiratory paralysis. The reflexes are normal. There is a transitory neonatal form in the babies of affected mothers. Aminoglycoside antibiotics and hypermagnesaemia (seen in the babies of mothers given magnesium for eclampsia) may affect the myoneural junction.

MUSCLE DISEASE

There is usually gradual onset of proximal muscle weakness. The child “climbs up” his legs with his arms when going from lying to standing because of thigh weakness (Gower’s sign). Neck flexion is weaker than neck extension. Reflexes are normal until late in the disease. Sensation is normal. There may be wasting, but there are no fasciculations. The CSF is normal.

Check for facial involvement (facioscapulohumeral dystrophy), rapid onset of fatigue (myasthenia), pelvic and thigh muscles worse than head and shoulders (limb-girdle dystrophy), myotonia, hypertrophy (Duchenne dystrophy).

Serum CPK, CSF examination and muscle biopsy may help in arriving at a correct diagnosis.

BENIGN CONGENITAL HYPOTONIA

This is a diagnosis of exclusion.

PARALYSIS - SPASTIC (HYPERTONIC)

Spastic paralysis implies disease of the brain or spinal cord (upper motor neurone lesion), with hyperreflexia and upgoing plantars. Active reflexes in a child with downgoing plantars are usually normal. Upgoing plantars are normal in children up to 12 months old.

A child with recent onset of focal spastic paralysis (suggesting a cerebral space occupying lesion) should have a trial of TB therapy, unless neoplasm has been proven histologically.

- Unilateral:
- previous injury - birth, meningitis, subdural haematoma, trauma
 - recent onset - investigation needed to exclude cord compression (Brown-Sequard) or cerebral tumour, tuberculoma, toruloma, haematoma or subdural effusion.
- Bilateral:
- cerebral palsy (spastic diplegia)
 - spinal cord compression (local back pain, sensory level) - familial spastic paraplegia (?family history)
 - spastic tropical paraplegia
 - subacute sclerosing panencephalopathy (with ataxia) (p.358)
- In arms and legs:
- lesion in cervical cord or higher
- In legs only:
- the lesion is below the cervical cord EXCEPT FOR:
 - cerebral palsy (leg fibres selectively damaged by hypoxia) giving spastic diplegia
 - hydrocephalus (parasagittal leg fibres stretched most by dilated ventricles)
 - parasagittal intracranial mass, eg meningioma (presses on area of cortex controlling legs); look for headache, personality change, convulsions.

CEREBRAL SPACE-OCCUPYING LESION

A lesion of the brain usually causes hyperreflexic spastic paralysis (with an upgoing plantar) on the opposite side of the body (though an acute lesion may cause flaccid paralysis initially). There may be raised intracranial pressure (see p.77). Lesions of the cerebral cortex and internal capsule cause paralysis of the face, arm, and/or leg (depending on the extent of the lesion) on the opposite side of the body, but only very large lesions in the anterior fossa cause raised intracranial pressure (with small ventricles). Lesions in the posterior fossa may cause weakness of the face on the same side as the lesion (or on the opposite side) and weakness of the arm and leg on the opposite side, and quite small lesions may cause raised intracranial pressure due to obstruction of the flow of CSF (with enlarged ventricles). Neck stiffness in a patient with focal neurological signs of gradual onset suggests a posterior fossa lesion, or meningitis due to TB or cryptococcus. Subarachnoid haemorrhage and purulent meningitis cause neck stiffness of acute onset.

Do NOT do a lumbar puncture if there is any evidence of raised intracranial pressure.

Do a chest x-ray and Mantoux.

The most likely space-occupying lesion in Papua New Guinea children is a tuberculoma. It is therefore always justified to give a trial of antituberculous treatment.

If there is no improvement after four weeks of anti-TB therapy, or if there is deterioration prior to this, a search for other lesions may be considered - although it should be remembered that the prognosis for intracranial neoplasms in children is poor. Before embarking on invasive and expensive investigations, it is important to discuss the implications with the family.

The availability of a CT scan facility in Port Moresby has made the diagnosis of intracranial neoplasms in all age groups possible by non-invasive means. In young children with "sprung" sutures it may be possible to perform ultrasound scan of the brain through the sutures.

Should these investigations be impracticable, invasive investigation such as burrhole and needle exploration, and air ventriculogram might be considered.

Administration of steroids is likely to produce temporary improvement in symptoms and signs related to intracranial neoplasm, as a result of reduction of inflammatory oedema.

In situations where the prognosis is bad, parents may well wish to take their child home to die rather than stay in hospital.

Pain palliation is something which doctors can help with - even in “hopeless” situations.

TUBERCULOUS SPINAL OSTEITIS (POTT’S DISEASE)

Affected children may present with local pain and tenderness of the spine (usually in the thoracic region), or they may present with difficulty in walking.

NEVER IGNORE BACK PAIN OR LOWER LIMB WEAKNESS OR LIMP IN CHILDREN.

There is often hyperreflexia and sensory loss in the legs, a visible deformity of the spine (gibbus) and a positive Mantoux. Chest x-ray may be suggestive of tuberculosis, and x-ray of the spine almost always shows erosion and collapse of one or more adjacent vertebral bodies. Treatment is with antituberculous drugs.

The place of surgery in spinal cord compression secondary to Potts disease has been controversial, but current opinion is that it is indicated only if the signs have been rapidly progressive and the disability is severe.

SPINAL ADHESIVE ARACHNOIDITIS

This is a disease of older children and adults (usually male). Patients usually give a history of backache followed by weakness and sensory loss which develops over some weeks. In most cases, the lesion is in the thoracic or cervical spine, and there is spastic paraplegia. In some patients, the lesion is in the lumbar spine, and there is flaccid paralysis due to a cauda equina lesion. Myelogram usually shows an irregular block and the diagnosis is confirmed at laminectomy. The prognosis for recovery is very poor. Steroids should not be given (see Wagner F. PNG Med J 22:57-61,1977. Spinal adhesive arachnoiditis).

SPASTIC TROPICAL PARAPLEGIA

This is a progressive myelopathy probably caused by the HTLV-1 virus. HTLV-1 virus is widespread in Papua New Guinea.

PERICARDIAL EFFUSION AND PERICARDIOCENTESIS

The widespread availability and use of ultrasound has confirmed clinical impressions that pericardial effusions are relatively common. Many are small, contribute little to the clinical presentation and resolve on treatment of the underlying condition with no sequelae. Some, however, are large and cause compression of the heart with impairment of function - TAMPONADE. Some are purulent and require drainage of pus. Others, if not drained, may, in the long term, result in fibrosis and constrictive pericarditis.

AETIOLOGY

Causes of pericarditis include:

- viral infection
- staphylococcus*
- uraemia
- SLE
- tuberculosis
- rheumatic fever
- rheumatoid arthritis
- leukaemia

* spread from pneumonia or septicaemia

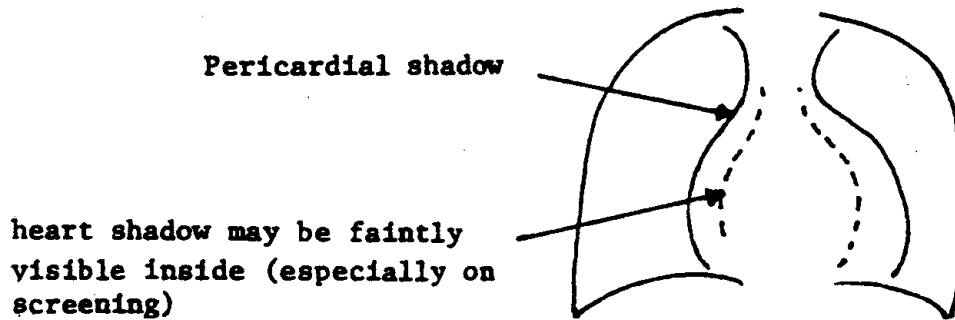
DIAGNOSIS

Ultrasound should be performed whenever there is a suspicion of pericardial effusion. It is vital to diagnose tamponade and purulent pericardial effusion and to drain the pericardium. Tamponade may be mistaken for myocarditis or heart failure due to pneumonia. Tamponade is suggested by Beck's triad (quiet heart, rising JVP, falling BP) in an anxious, ill child. However, many children with tamponade do not have muffled heart sounds.

In cardiac tamponade:

1. there is a rapid pulse, usually with pulsus paradoxus - a small volume pulse that is even weaker on inspiration. The systolic blood pressure usually falls during inspiration, but a fall of over 10 mmHg is abnormal (but not really "paradoxical" at all). It occurs in tamponade, airway obstruction (such as in severe asthma) and SVC obstruction
2. the JVP is ALWAYS raised, though it may be so high that it is hard to see. It may rise further on inspiration
3. the apical impulse is usually markedly reduced (unlike cardiac failure), but this may not be so
4. the heart sounds may be quiet. The presence of a third heart sound or pericardial rub suggest cardiac failure rather than tamponade (but may be present in tamponade)
5. tender hepatomegaly may develop, but is absent initially. Peripheral oedema is rare
6. ECG often shows reduced voltages with flattened T waves
7. chest x-ray usually shows cardiomegaly, but this may not be gross in acute tamponade, which can be caused by quite a small effusion. Pulmonary oedema is very rare
8. ultrasound screening shows a collection of fluid (dark area) around the heart. If it is a serous effusion, it will be black. If it is pyogenic, it may have a speckled greyish appearance and if it is a tuberculous fibrous exudate, there may be readily visible fibrous strands and clots. There is likely to be reduced movement of the heart, though this is more difficult to appreciate for the non expert.

Chest x-ray with pericardial effusion



ALL PRACTICING CLINICIANS SHOULD BE ABLE TO DIAGNOSE A PERICARDIAL EFFUSION USING ULTRASOUND.

Other investigations may be indicated:

- FBE (elevated WCC may suggest pyogenic effusion in a toxic child)
- blood culture
- serum urea
- LE cells and ANA
- Mantoux.

TREATMENT

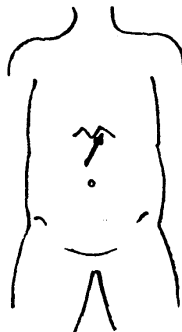
Do NOT give digoxin or diuretics.

PERICARDIOCENTESIS is indicated if:

- there is tamponade
- there is the possibility of pus in the pericardium.

The procedure may be performed under ultrasound guidance - but this is not absolutely necessary.

1. Unless the patient is very sick, give IM pethidine half an hour before the procedure.
2. Prop the patient up in bed at 60 degrees.
3. An assistant should hold the child's arms and legs.
4. Scrub your hands, and put on sterile gloves.
5. Swab an area of 10 cm radius from the xiphisternum with iodine.
6. Inject 2 ml of 1% plain lignocaine into the skin and subcutaneous tissue at the left xiphicostal angle.
7. Aspiration is performed using the biggest Dwellcath available (14-19). The needle is inserted at the left xiphicostal angle and pushed slowly towards the left shoulder, keeping close to the chest wall.



8. Advance the needle slowly until fluid enters the syringe - this is usually at least 2 cm in. Slide the Dwellcath cannula gently further in at the same time as removing the needle and quickly connect a 20 ml syringe and 3-way tap.
9. SLOWLY aspirate the fluid into the syringe. If this is done rapidly, it may cause shock. When the syringe is full, turn the 3-way tap so that the side arm is open. Put 10 ml of fluid in a sterile bottle for bacterial culture, ZN stain for AFB, and protein. Discard the rest of the fluid into a container through a giving-set with the end cut off.
10. Turn the 3-way tap back again, and aspirate again until no more fluid is aspirated.
11. Remove the Dwellcath and clean off the iodine.
12. The dangers of pericardiocentesis include sudden death (from ventricular fibrillation), puncture of the heart, coronary artery or internal mammary artery, and pericardial shock if fluid is removed too rapidly.

Notes

1. If thick pus is obtained it will be necessary to drain the pericardium. This can be done surgically or through a lavage technique. Antibiotic therapy with cloxacillin should be continued for a minimum of 4 weeks.
2. Tuberculous effusions may be serous or fibrous and haemorrhagic. If the latter, surgical drainage through a large pericardial window is required to prevent the subsequent development of constrictive pericarditis. Tuberculosis treatment should be started immediately.
3. In a patient with a history and CXR suggestive of TB and with a small or moderate pericardial effusion which is completely black on ultrasound (serous effusion) and with no signs of tamponade, antituberculous therapy and steroids may result in rapid resolution of the effusion.

REFERENCES

- Hugo-Hamman CT et al. *Pediatr Infect Dis J* 13(1):13-18,1994. Tuberculous pericarditis in children: a review of 44 cases.
- Smith F. *Trop Doctor* 19(3):105-108,1989. Acute pericarditis with effusion and the general surgeon.

PERITONEAL DIALYSIS

This should only be used for acute, reversible renal failure.

1. Empty the bladder with a No.5 or No.8 feeding tube, unless it is certain that the bladder has just been emptied.
2. A sterile technique MUST be used.
3. Add 1000 u heparin to each litre of standard (1.6% dextrose) dialysis fluid.
4. Add potassium chloride 4 mEq (1.25 ml of 2 g in 8 ml solution) to each litre of dialysis fluid if the serum potassium is less than 4 mEq/l.
5. Swab the skin with iodine. Put on a cap, mask, sterile gown and gloves. Using a sterile technique, anaesthetise the skin and subcutaneous tissue with 2% plain xylocaine in the midline 2 cm below the umbilicus (ensure that the liver and spleen are not enlarged).
6. Before inserting the catheter, infuse 25 ml/kg of warmed dialysis solution into the peritoneal cavity over 15 minutes via a Dwellcath or Medicut.
7. Insert the peritoneal dialysis trocar and catheter through the anaesthetised area. After removing the trocar, direct the catheter downward into the pelvic cavity (for small children it may be necessary to cut off part of the catheter).
8. Establish that fluid flows freely from the catheter, then begin the dialysis. Use 50 ml/kg (maximum 500 ml) for each cycle. Run it in over 10 minutes, equilibrate for 30 minutes, and drain for 20 minutes. The peritoneal cavity must be drained completely with each cycle; this may require positioning the patient. The dialysis fluid must be warm.
9. An ACCURATE cumulative record of the volume of fluid infused and withdrawn MUST be kept. If a progressive positive balance develops, hyper tonic (7% dextrose) dialysis fluid can be used. If an excessive negative balance develops, the abdominal cavity should still be drained completely each cycle, but replacement can be made by intravenous fluids.
10. Record pulse and BP 4 hourly, and weigh daily.
11. If the blood glucose exceeds 10 mmol/l, give 0.1 to 0.2 u/kg of plain insulin IV.
12. Dialysis usually lasts about 48 hours. The main danger is from infection (causing peritonitis). In some patients the dialysis catheter blocks and has to be resited or replaced.

REFERENCE

Kempe CH, Silver HK, O'Brien D. Current pediatric diagnosis and treatment, Lange, 1978, p.508-9.

PERITONEAL TAP - DIAGNOSTIC

This may be used to detect the presence of bowel contents, pus or blood in the peritoneal cavity without resorting to formal laparotomy.

The usefulness of the procedure is limited by the fact that a negative tap does not absolutely exclude the presence of these fluids in the peritoneal cavity.

NEVER attempt diagnostic peritoneal tap in a patient who has had a laparotomy, because of the possibility of adhesions and penetration of the bowel with the needle.

1. The site of aspiration is usually to the left or right of the rectus muscle at the level of the umbilicus. Check that there is no hepatosplenomegaly or other abdominal mass in the area and that the bladder is empty.
2. Have the patient supine, but rolled slightly toward the side of the aspiration.
3. Clean the skin over a 10 cm radius with iodine.
4. Draw up 3 ml of sterile saline in a 10 ml syringe with a 21 gauge disposable needle on it.
5. Push the needle through the skin. Then start injecting the saline and push the needle into the peritoneal cavity. Then attempt to aspirate peritoneal fluid. Three attempts are usually made before the tap is said to be negative.
6. Alternatively, a nick is made in anaesthetised skin and a sterile trocar and cannula inserted into the peritoneal cavity. A small plastic catheter (or an infant feeding tube) can then be passed through the cannula into the upper and then the lower abdomen.
7. If any blood, bile, faeces or urine is aspirated, the result of the tap is positive and laparotomy will be required.
8. If the tap is negative, 10 ml/kg of normal saline can be instilled into the peritoneal cavity and aspiration attempted again.

REFERENCE

Radford A. PNG Med J 10:104,1967. Peritoneal tap.

PERTUSSIS (WHOOPIING COUGH)

Pertussis is caused by *Bordella pertussis* growing in the bronchi. Transmission is by droplet spread with coughing.

In many developing countries, pertussis occurs in epidemics every 2 or 3 years. Most cases of clinical 'whooping cough' are in children under 5 years old, and the highest mortality is in children under 1 year old. Epidemics usually spread outward from one region, getting better in the original area as cases increase in the adjacent areas. Separate outbreaks may start due to spread by air or car travel. A single immunisation with triple antigen gives no protection, but 3 injections give about 80% protection and, if it does occur in a fully immunised child, the disease is much milder. Pertussis vaccine is very heat labile, and is rapidly inactivated if the triple antigen is not kept cool at all times. It is therefore MOST IMPORTANT to ensure that all health centre fridges in your area are kept working at all times.

The classical clinical picture is caused by production of very thick sputum, which causes the child to cough out many times so that he becomes cyanosed, then he breathes in so strongly that he produces a loud stridor or whoop. He may vomit the sticky mucus, and he may become so hypoxic that he convulses.

The illness starts with rhinorrhoea, fever and a cough that comes in spasms for 10 days until the whoop starts. The lymphocyte count is often over 20,000 cells/cmm. The cough may last for up to 3 months (100-day cough). The harsh cough may recur with any upper respiratory tract infection in the next year, but this does not mean that the child has got whooping cough again. Adults often get pertussis too, but it is usually a mild infection and they do not whoop. Unimmunised adults form a reservoir of infection.

In babies under one year old the fatality rate is high, and it is harder to diagnose because there is often no whoop. Suspect pertussis if a baby has episodes (paroxysms) of coughing with apnoea and cyanosis, and then vomits, particularly if older children in the area have whooping cough. Chloramphenicol or erythromycin may modify the illness if they are given as soon as fever and rhinorrhoea begin, BEFORE the cough starts.

COMPLICATIONS

1. Pneumonia from secondary infection
2. Dehydration and malnutrition from the vomiting
3. Exacerbation of TB. Suspect this if the cough and weight loss persist for more than 3 months
4. Convulsions, epistaxis, sublingual ulcers, subconjunctival haemorrhage, cerebral haemorrhage resulting from coughing paroxysms
5. Encephalopathy: in one series, a third of children admitted to hospital had convulsions or cerebral depression (PNG Med J 16:36-41,1973)
6. Rectal prolapse
7. Heart failure.

TREATMENT

Outpatient treatment for older children with mild disease

Inpatient treatment for children under 6 months old with complications.

Outpatient treatment

1. Advice to mother:
 - a. the illness may last 6 to 8 weeks
 - b. feed the child every time he vomits
 - c. return if there is shortness of breath, convulsions, weight loss or difficulty feeding

- d. bring any other young child as soon as he gets a runny nose or cough.
2. Triple antigen to unimmunised siblings, and to the patient at the end of his illness.

Note: Cough suppressants should not be given routinely. They often contain substances which are designed to dry secretions, thus making it harder for the sticky mucus to be coughed out. Children who are not sleeping at night because of the cough can be given a small dose of benadryl.

Inpatient treatment

1. Give oral chloramphenicol or erythromycin every 6 hours for 5 days to prevent infection of other patients. Although chloramphenicol makes the child non-infectious, it does NOT alter the course of the disease unless given very early in the illness.
2. For severe paroxysms of coughing with cyanosis, give OXYGEN and GENTLE BRIEF SUCTION.
3. Treat pneumonia and heart failure, if present.
4. If convulsions occur, give IM paraldehyde stat, and start phenobarbitone to help prevent further convulsions.
5. Treat malnutrition, and get the mother to feed the child immediately after every vomit.
6. There is no good evidence for the use of salbutamol or of steroids in children with whooping cough.
7. There is no good evidence to support the use of sedatives or antitussives in the treatment of pertussis (Lancet 2:981,1977; Br Med J 2:619,1970).

REMEMBER - PREVENTION IS BEST. PERTUSSIS IS PREVENTED BY TRIPLE ANTIGEN.

PIGBEL (ENTERITIS NECROTICANS)

Until vaccination began in the early 1980s, enteritis necroticans was the commonest cause of death in children older than 12 months in the highlands of Papua New Guinea. There is concern that with the cessation of the pigbel vaccination programme, there may be a resurgence of the condition, although it is also possible that some of the contributory environmental factors may have changed.

AETIOLOGY

Malnourished children have low trypsin levels and are unable to digest the beta toxin made by *Clostridium welchii* type C, which contaminates protein food (particularly pig meat). This situation is exacerbated by eating partly cooked sweet potato, which contains a trypsin inhibitor, at the same time. The beta toxin causes necrosis of the small bowel (see Lancet 1:125-126, 1976).

DIAGNOSIS

Children with pigbel have:

ALWAYS: SEVERE ABDOMINAL PAIN starting up to 5 days after eating a PROTEIN MEAL (often pig meat)

OFTEN: ABDOMINAL SWELLING
BLACK-FLECKED VOMIT
MILD DIARRHOEA with blood (but sometimes constipation).

The diagnosis of early severe pigbel or of mild pigbel can be difficult:

- gastroenteritis has a lot of diarrhoea and not much pain (pigbel has a lot of pain, but not as much diarrhoea)
- some children with abdominal pain improve after a dose of albendazole and passing ascaris.

Except in very severe cases, children with pigbel are hungry, but eating causes abdominal pain. They therefore lie in bed crying from hunger, but cry out with pain if their parents relent and give them food.

The diagnosis of pigbel rests primarily on the history, but some features in the examination may be helpful:

- the abdominal distension is typically more marked in the upper abdomen, above the umbilicus. Pigbel results in areas of necrosis along the small bowel, which obstruct the flow of bowel contents. The normal bowel between areas of necrosis distends and has forceful peristalsis giving colicky pain, distension, and often easily visible peristalsis
- later on in the course of the disease, the presence of palpable loops of tender thickened small bowel is diagnostic (but do not be misled by the transverse colon full of faeces in the epigastrium).

SEVERITY

Pigbel may present late, sometimes months after a mild attack, with small bowel obstruction or symptoms of malabsorption such as anaemia or oedema. The original episode of pigbel may even have been forgotten by the time of presentation. The management of late pigbel is surgical.

Acute pigbel can be mild, severe or fulminating. Children with fulminating pigbel may die in a matter of hours; indeed, the diagnosis may be made at postmortem in a child who collapses and dies suddenly. Most acute cases can be classified as mild or severe on the basis of distension, vomiting and toxic state:

	Mild	Severe
Abdominal swelling	Some	A lot
Toxic (looks sick, fast pulse)	No	Yes
Black flecked vomit	No	Yes

Mild cases can be treated at health centres. Severe cases should be sent to hospital.

TREATMENT OF MILD PIGBEL

1. Intravenous fluid (half-strength Darrow's):

3 - 5 kg:	25 ml/hour (7 drops/min)
6 - 9 kg:	50 ml/hour (13 drops/min)
10 - 14 kg:	75 ml/hour (20 drops/min)
>15 kg:	100 ml/hour (27 drops/min)
2. Albendazole oral once, then nothing to eat or drink.
3. Pass a large nasogastric tube. Aspirate, then leave on free drainage.
4. Benzyl (crystalline) penicillin IV or IM 6 hourly.
5. If the child gets sicker, or if there is no improvement within 2 days, consider surgery
 - a. No improvement:
 - i. still has abdominal swelling and pain, black-flecked vomit or NG aspirate, fast pulse, looks sick
 - b. Improvement:
 - i. reduced abdominal swelling and pain, no vomiting. Feels hungry and has bowel motions
 - ii. after 24 hours of improvement, stop IV fluids and give sugar-water or oral rehydration solution (ORS)
 - iii. after another 24 hours of improvement, give full strength milk (eg Sunshine, see p.231) for 24 hours
 - iv. then give solid food.

Volume of ORS or milk (if improving)	
3 - 5 kg:	75 ml 3 hourly
6 - 9 kg:	100 ml 3 hourly
10 - 14 kg:	150 ml 3 hourly
15 - 19 kg:	200 ml 3 hourly
>20 kg:	250 ml 3 hourly

TREATMENT OF SEVERE PIGBEL

1. Large nasogastric tube. Leave on free drainage, record amount accurately.
2. Albendazole orally once, then nil by mouth.
3. Chloramphenicol IV 6 hourly and metronidazole 500 mg suppository 6 hourly.
4. Treatment B for severe malaria (see p.197).
5. Whole blood transfusion whenever Hb less than 10 g/dl:

5-9 kg:	200 ml ($\frac{1}{2}$ unit)
10-14 kg:	300 ml ($\frac{3}{4}$ unit)
15 kg or more:	400 ml (1 unit)
6. IV fluid
 - a. Rehydrate with normal saline or Haemacel 20 ml/kg over 1 hour, repeated if necessary.
 - b. Replace nasogastric tube losses with an equal volume of IV normal saline with 1 g (4 ml) of KCl per litre.
 - c. Maintenance 4.3% dextrose in 0.18% saline with 4 g (16 ml) KCl per litre:

Under 10 kg:	25 ml/hour	(7 drops/min)
10-14 kg:	50 ml/hour	(13 drops/min)
15-19 kg:	75 ml/hour	(20 drops/min)
20-30 kg:	100 ml/hour	(27 drops/min)

7. IV fluid for severe pigbel in severe cases:
 - with weight under 70%
 - or in a child that will need prolonged IV fluid, or that has been on IV fluid for 5 days with no improvement.

The IV fluid used is very hypertonic, and extravasation into subcutaneous tissues causes necrosis. Administration should be through an intracath or a cutdown cannula with the tip in a large vein. Scalp vein needles should not be used.

- a. Rehydrate with normal saline or Haemacel 20 ml/kg, repeated if necessary.
- b. Replace nasogastric tube losses with an equal volume of IV normal saline with 1 g (4 ml) of KCl per litre.
- c. Maintenance IV fluid of:
 - i. aminofusin 1,000 and 10% dextrose, or
 - ii. aminofusin 600 or Aminosol with 20% dextrose (use 10% dextrose if 20% not available).

	Aminosol or Aminofusin	10-20% dextrose + 5 ml 50% MgSO₄ + 4 g (16 ml) KCl/l
Under 10 kg:	2 ml/kg/hour	6 ml/kg/hour
10-19 kg:	2 ml/kg/hour	4 ml/kg/hour
20-29 kg:	1½ ml/kg/hour	3 ml/kg/hour
30-39 kg:	1 ml/kg/hour	2 ml/kg/hour

- d. Give plasma (same blood group as patient) or SPPS 20 ml/kg IV twice a week.

While still on IV aminofusin/dextrose:

Daily: Review NG losses and adjust saline rate
 Test urine for sugar (reduce IV dextrose concentration if positive)
 Intravite 2 ml IV daily

Monday: Hb, Na, K albumin
 Folic acid 15 mg (1 ml) IV (if available)
 Phytomenadione (Vit K) 1 mg (0.5 ml) IM
 Plasma (same group as patient) 20 ml/kg IV

Thursday: Hb, Na, K
 Plasma (same group as patient) 20 ml/kg IV.

Test the urine for sugar EVERY day. If it is positive (more than trace), change 10% to 5% dextrose. If it is negative for 3 days, change 10% to 20% dextrose (if available) as long as the urine remains negative for sugar.

Transfuse if the haemoglobin is under 10 g/dl.

If the serum sodium falls below 130, substitute 0.9% sodium chloride for aminofusin (at double the rate) for 24 hours. Continue dextrose.

8. If the child gets sicker, or there is no improvement within 2 days, consider surgery. Some surgeons operate on children with severe pigbel as soon as rapid IV rehydration has been achieved.
 - a. No improvement:
 - i. still has large amounts of NG aspirate, no bowel motions, abdominal pain, fast pulse, looks sick.
 - b. Improvement:
 - i. reduced amounts of clear NG aspirate, no pain, develops appetite and has bowel motions
 - ii. after 24 to 48 hours of improvement, stop IV fluids and give sugar-water or Oral Rehydration Solution (see p.305)
 - iii. after another 24 to 48 hours of improvement, give full strength milk for 48 hours
 - iv. then gradually introduce solid food.

PREVENTION

The pigbel vaccine, *Clostridium welchii* type C beta toxoid (Lancet 1:227-230,1979) has dramatically reduced the incidence of, and the mortality and morbidity from pigbel. At least two injections are required for immunity. Reactions to the vaccine are rare. Pigbel vaccine was part of the routine immunisation programme in highlands provinces. It is currently under review.

REFERENCES

- Lawrence G. Lancet 1:125-126,1976. Pathogenesis of enteritis necroticans.
PNG Med J 22(2):March 1979. Review of pigbel.
Shepherd AR. In: Diseases and Health Services of PNG, PHD, 1973, p.264-270.
Smith F. Aust NZ J Surg 38:199-205,1969. Surgery of enteritis necroticans.

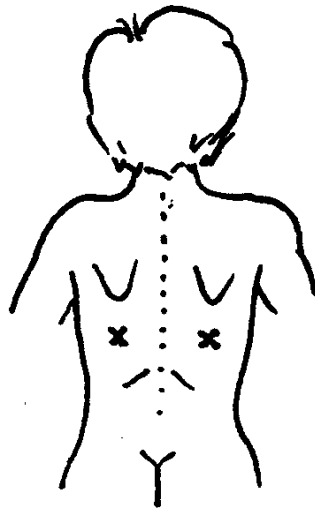
PLEURAL EFFUSION

The classical signs are mediastinal shift, dull percussion and reduced breath sounds and vocal fremitus. These signs are often absent in infants. If a small effusion is suspected on chest x-ray, it can often be confirmed by lying the child on the side (affected side down) and taking an AP view.

PLEURAL ASPIRATION

This should be performed as a diagnostic procedure whenever a pleural effusion of uncertain aetiology is present. With a large effusion, aspiration may be needed to relieve respiratory distress.

1. Have an assistant hold the child in a sitting position. Have the child on intranasal oxygen 2 litre/min.
2. Scrub your hands and put on sterile gloves.
3. Clean the skin with iodine over a 10 cm radius from the bottom of the scapula on the affected side.
4. Aspiration is performed with a sterile disposable 21 gauge needle or a Dwellcath or Medicut attached to a 3-way tap and then a 20 ml syringe. The needle is inserted in the intercostal space below the lower border of the scapula midway between the spine and posterior axillary line. Put the needle in immediately above the rib (to avoid the intercostal vessels which are immediately below each rib).



5. Advance the needle slowly until fluid enters the syringe. This is usually 1-2 cm in, but will depend on how fat the child is. Clamp a pair of artery forceps on the needle at the point it enters the skin to prevent it going in any further, or remove the metal needle from the Dwellcath or Medicut.
6. SLOWLY aspirate the fluid into the syringe. When the syringe is full, turn the 3-way tap so that the side arm is open. Put 5 ml of fluid in a sterile bottle:
 - a. CLEAR FLUID:
 - i. common causes: TB, pneumococcus, neoplasm
 - ii. send the aspirate for cell count, gram stain, AFB, culture
 - iii. discard the rest of the fluid into a container. Turn the 3-way tap back again and continue to aspirate until all the fluid has been removed
 - iv. treat the underlying disease. Repeat aspiration will only be necessary if enough fluid re-accumulates to cause respiratory distress.

- b. PUS (EMPYEMA):
- i. common causes: staphylococcus, pneumococcus, H influenzae
 - ii. send the fluid for gram stain and culture
 - iii. if the pus is thin, aspirate all of the effusion. If the pus is too thick to aspirate properly, remove the needle and arrange for closed underwater drainage of the empyema as soon as possible. Use the same technique as for draining a pneumothorax (see p.315) but use the LARGEST drain tube possible (eg 16 or 18 gauge)
 - iv. treat the child with IV benzyl penicillin (septicaemia doses), cloxacillin, gentamicin and probenecid for AT LEAST 4 WEEKS. Change the antibiotics when culture results are available
 - v. rib resection and drainage may be required.

PNEUMONIA

(PNEUMONIA AND BRONCHIOLITIS - LOWER RESPIRATORY TRACT - LRTI)

See also Neonates - Respiratory Distress (p.271) and WCC over 40,000 per cmm (p.386).

As in other parts of the world, epidemics of bronchiolitis caused by respiratory syncytial virus (RSV) occur in PNG. Very high carriage rates of respiratory pathogens in PNG children appear to contribute to the high incidence of secondary bacterial pneumonia in children with bronchiolitis.

Symptoms and signs of bronchiolitis and pneumonia are similar and the two illnesses are classified together as lower respiratory tract infections (LRTI) and in most cases are treated the same.

SUMMARY

Mild pneumonia:	<ul style="list-style-type: none">• fast breathing (over 40/min) with no chest indrawing• outpatient amoxycillin 3 times daily for 5 days
Moderate pneumonia:	<ul style="list-style-type: none">• chest indrawing• inpatient benzyl penicillin IM 6 hourly
Severe pneumonia:	<ul style="list-style-type: none">• chest indrawing, with cyanosis or not able to drink• inpatient chloramphenicol 6 hourly for 10-14 days, oxygen if cyanosed, digoxin for heart failure
Staphylococcal pneumonia:	<ul style="list-style-type: none">• IV cloxacillin, then oral cloxacillin 4-6 weeks
Persistent pneumonia:	<ul style="list-style-type: none">• trial of cotrimoxazole (trimethoprim 5 mg/kg QID) for 1-2 weeks (give for 3 weeks if responds)• trial of erythromycin for 1-2 weeks (give for 3 weeks if responds)• trial of TB therapy.

Pneumonia is the commonest cause of death and the commonest cause of admission of children in Papua New Guinea. Most deaths from pneumonia occur in infants less than 12 months old. Worldwide, about 4 million children die from pneumonia every year.

Most cases of pneumonia in children are due to *Haemophilus influenzae* or *Streptococcus pneumoniae* or both. *H influenzae* is only moderately sensitive to benzyl penicillin, and pneumococci are becoming increasingly resistant to penicillin in many countries. However, amoxycillin and penicillin in large enough doses will usually cure children with mild or moderate pneumonia. Children with severe pneumonia should be treated with chloramphenicol, which has been shown to be just as effective as giving chloramphenicol and penicillin together. Chloramphenicol is active against haemophilus and pneumococcus. Ampicillin, tetracycline, aminoglycosides and cephalosporins should NOT be used routinely as the initial treatment of pneumonia. However, doctors should be aware that chloramphenicol-resistant *Haemophilus influenzae* has now been clearly documented in Papua New Guinea.

DIAGNOSIS

In young children, pneumonia is diagnosed by WATCHING THE CHILD BREATHE. This is usually far more informative than auscultation and percussion. There are three important rules:

1. COUGH AND NORMAL BREATHING: NO ANTIBIOTIC
2. COUGH AND FAST BREATHING (OVER 40/MIN): GIVE AN ANTIBIOTIC
3. COUGH AND CHEST INDRAWING: ADMIT TO HOSPITAL.

PNEUMONIA OR BRONCHIOLITIS - GUIDELINES

For children **1 month to 5 years** of age

Neonates (0-1 month) with fast breathing or chest indrawing: see Neonates - Infection (Neonatal Sepsis), p.251 and Neonates - Respiratory Distress, p.271.

Feature	Pneumonia		
	Mild	Moderate	Severe
Fast breathing (more than 40/min)	Yes	Yes	Yes
Chest indrawing present	No	Yes	Yes
1. Pulse more than 160/min with large liver, OR 2. Too sick to suck, OR 3. Cyanosis or restless	No	No	Yes
Admit or not?	No	Yes	Yes
Initial antibiotic	Amoxycillin	Crystapen IM 6/24	Chloramphenicol IM 6/24
Antibiotic when improved	Amoxycillin	Amoxycillin 6/24	Chloramphenicol oral
Total course duration	5 days	10 days	14 days
Other treatment	Nil	Suction PRN	<ul style="list-style-type: none"> • Lanoxin oral • nasal oxygen • suction • ?N/G feeds
Malaria treatment	Treatment A	Treatment A	Treatment B
Review frequency	Daily	6 hourly	Every hour
Check on review for	<ul style="list-style-type: none"> • chest indrawing 	<ul style="list-style-type: none"> • pulse rate • liver size • cyanosis • able to suck 	<ul style="list-style-type: none"> • cyanosis • clear airway • fever resolution

**CHECK THE CHILD WITH MILD PNEUMONIA EVERY DAY
- IF CHEST INDRAWING - ADMIT.**

BEWARE OF THE CHILD WHO HAS MENINGITIS AS WELL AS PNEUMONIA. The two diseases often go together. Do a lumbar puncture on any child with pneumonia who is drowsy, not feeding well, or has a stiff neck or bulging fontanelle, unless they are extremely ill (see p.223).

The definition of fast breathing

There has been much controversy over the definition of fast breathing in young children. Current WHO guidelines indicate that below 2 months of age, a respiratory rate of more than 60/min is abnormal, from 2 months to 11 months above 50, and above 12 months above 40 is abnormal. If 50/min is taken, some infants with pneumonia will be excluded from treatment. If 40 is taken some children without pneumonia will be treated. In Papua New Guinea, it has been decided to stick to the long used cut off of 40/min. Electronic timers have been provided to health workers to enable them to count respiratory rate. The main benefit of these is that they do remind the health worker that looking at the child and assessing his respiratory rate is the key to diagnosing pneumonia. Interestingly, it has been shown that if the mother perceives that her child is breathing fast, the child is highly likely to have pneumonia.

TREATMENT

Mild pneumonia

Mild pneumonia is defined as cough and fast breathing (over breaths 40/min), with no chest indrawing. Give outpatient treatment with amoxycillin 3 times daily for 5 days.

Moderate pneumonia

Moderate pneumonia is defined as cough and chest indrawing (intercostal or subcostal retraction), with no cyanosis and drinking well. Admit the child, and give benzyl penicillin IM 6 hourly until improvement occurs. Then give amoxycillin 3 times daily for 5-10 days.

Severe pneumonia

This is defined as cough and chest indrawing with cyanosis or restlessness, inability to drink, or signs of heart failure. Admit the child.

Antibiotic

Give chloramphenicol IM or IV 6 hourly until improvement occurs, then chloramphenicol orally 6 hourly. A total of 10-14 days' chloramphenicol is usually needed.

Oxygen

Give oxygen if the child is cyanosed or very restless. Oxygen can be administered satisfactorily either by nasopharyngeal catheter or by nasal prongs. For nasopharyngeal administration put an 8 FG catheter into the nose to a depth equal to the distance from the side of the nose (ala nasi) to the front of the ear (tragus), and give 1 litre/min of oxygen. Do not push the catheter in too far, and do not give more than 1 litre/min of oxygen. The oxygen should be humidified using a bubble humidifier. The catheter should be removed and cleaned every day, and the water in the humidifier should be changed twice a week.

Nasal prongs are more difficult to obtain, but are effective and more convenient than nasopharyngeal catheters. The availability of pulse oximeters in some hospitals may facilitate rational use of oxygen, but their limitations should be understood. Recommendations concerning the oxygen saturations at which therapy should be instituted vary, and decisions will, to some extent, depend on the availability of oxygen - but a saturation of less than 90% in air would generally be an indication for oxygen therapy.

Digoxin

Give digoxin if the child has tachycardia at rest (a pulse over 160/min) and hepatomegaly (if liver is more than 2 cm below the costal margin).

Other

Give a transfusion of packed cells (and a dose of frusemide) if the Hb is 6 g/dl or less.
Give antimalarials if you are in a malarious area.

Very severe pneumonia

If there is very marked indrawing and cyanosis particularly if the child has recently had measles, consider staphylococcal pneumonia. In malnourished children, consider the possibility of gram negative pneumonia.

Staphylococcal pneumonia is suggested by finding pneumotocoeles, empyema or pyopneumothorax on chest x-ray (but both *Haemophilus* and *pneumococcus* can cause this). Take blood cultures if available, and if there is an effusion do a diagnostic tap (after explaining that the child is extremely ill and why you are doing the tap). Give IV benzyl penicillin, cloxacillin and gentamicin initially and then change to oral cloxacillin, to be continued for 4-6 WEEKS, when the child has improved. Oral probenecid increases serum levels of cloxacillin.

Children with gram negative pneumonia should be treated with ampicillin/amoxycillin and gentamicin for a minimum of 2 weeks.

Sudden deterioration may be due to a pneumothorax.

Children with severe pneumonia and a WCC over 40,000/cmm have a very high mortality (PNG Med J 22:55-8,1979). Consider giving IV benzyl penicillin in high doses plus gentamicin.

REFERENCES

- Berman S. *Rev Infect Dis* 7:674-691,1985. Control of ARI (review).
- Brewster DR, Pyakalya T, Hiawalyer G, O'Connell DL. *PNG Med J* 36(4):285-296,1993. Evaluation of the ARI program: a health facility survey in Simbu, Papua New Guinea.
- Campbell H. *Lancet* 2:742-743,1988. Clinical signs of pneumonia.
- Datta N. *Bull WHO* 65:77-82,1987. Control of acute respiratory infections.
- Duke T, Blanchke AJ, Sialis S et al. *Arch Dis Child* 86(2):108-112,2002. Hypoxaemia in acute respiratory and non respiratory illness in neonates and children in a developing country.
- Duke T, Mgone J, Frank D. *Int J Tuberc Lung Dis* 96:511-519,2001. Hypoxaemia in children with severe pneumonia in Papua New Guinea.
- Dyke T, Lewis D, Heergaard W et al. *J Trop Paediatr* 41:196-201,1995. Predicting hypoxia in children with acute lower respiratory tract infection: a study in the Highlands of Papua New Guinea.
- Ikeogu MO. *Arch Dis Child* 63:1266-7,1988. Pneumonia in Zimbabwe children.
- Moyle JBT. *Arch Dis Child* 74:77-80,1996. Uses and abuses of pulse oximetry.
- Muhe L, Degefu H, Worbu B et al. *Annals Trop Paediatr* (17):273-281,1997. Oxygen administration to hypoxic children: a randomised controlled trial comparing complications in the use of nasal prongs with nasopharyngeal catheters.
- PNG Med J* 34(3):165-224,1991. Focus Issue on ARI.
- Mulholland K. *Lancet* 354:590-592,1999. Magnitude of the problem of childhood pneumonia.
- Savage F. *Trop Doctor* 17:49-51,1987. Acute respiratory infection (review).
- Shann F. Pneumonia in children in PNG. PAHO, 525 23rd St, Washington, 1985.
- Shann F. *New Engl J Med* 313:410-414,1985. Absorption of IM chloramphenicol.
- Shann F. *Arch Dis Child* 60:963-966,1985. Hyponatraemia in pneumonia.
- Shann F. *Lancet* 2:684-686,1985. IM chloramphenicol for pneumonia.
- Shann F. *Br Med J* 292:314-317,1986. Aetiology of pneumonia (serology).
- Shann F. *Pediatr Infect Dis* 5:247-252,1986. Aetiology of pneumonia (review).
- Shann F. *J Pediatr* 110:299-302,1987. Penicillin levels after IM injection.
- Shann F. *Lancet* 2:1238-1240,1988. Nasopharyngeal oxygen in children.
- Shann F, Macgregor D, Richens J, Coakley J. *Pediatr Infect Dis J* 17:1141-1143,1998. Cardiac failure in children with pneumonia in Papua New Guinea.
- Smyth A, Carty H, Hart CA. *Ann Trop Paediatr* 18:31-40,1998. Clinical predictors of hypoxaemia in children with pneumonia.
- Stansfield SK. *Pediatr Infect Dis J* 6:622-629,1987. Control of pneumonia.
- Thilo EH, Park-Moore B, Berman ER, Carson BM. *Am J Dis Child* 145:1137-1140,1991. Oxygen saturation by pulse oximetry in healthy infants at an altitude of 1610m (5280ft).
- Tupasi TE. *J Infect Dis* 157:615-23,1988. Respiratory infections in Manila.
- Usen S, Weber M, Mulholland K et al. *Br Med J* 318:86-91,1999. Clinical predictors of hypoxaemia in Gambian children with acute lower respiratory tract infection: prospective cohort study.
- Weber MW et al. *J Pediatr* 127:378-383,1995. Comparison of nasal prongs and nasopharyngeal catheters for oxygen delivery.
- WHO. Oxygen delivery systems: oxygen therapy for acute respiratory infections in young children in developing countries. Geneva: WHO,1993.
- WHO. Respiratory infections in children. ISBN 924-1542268, Geneva, 1988.

PNEUMONIA - PROLONGED

This is defined as pneumonia persisting despite 2 weeks of antibiotic in adequate doses. Exclude bronchiolitis and asthma (wheeze). Check that the treatment prescribed has actually been given.

SUSPECT

1. tuberculosis if there is a family history, failure to thrive or a pleural effusion
2. foreign body if the child is aged 1 to 5 years and there is a history of aspiration, wheeze, stridor or haemoptysis
3. primary cardiac failure if there is a murmur or a large heart (exclude pericardial effusion) with a high JVP, hepatomegaly or tachycardia
4. staphylococcal pneumonia if there are pneumatoceles, a lung abscess or an empyema
5. chlamydia trachomatis, chlamydia pneumoniae, mycoplasma, ureaplasma and pneumocystis have all been shown to cause pneumonia in children. Chlamydia, mycoplasma, and ureaplasma are sensitive to erythromycin. Pneumocystis and chlamydia are sensitive to cotrimoxazole, although treatment of pneumocystis pneumonia needs high doses of cotrimoxazole. All these organisms cause a similar non-specific clinical picture with tachypnoea, crepitations, hyperinflation and diffuse patchy chest x-ray change. Fever and wheeze are uncommon. With the increasing prevalence of HIV infection, doctors can expect to see an increasing prevalence of pneumocystis pneumonia.

MANAGEMENT

Do progress chest x-rays, a Mantoux (5u PPD), and three gastric aspirates. Take blood and pharyngeal aspirate for culture.

RECORD THE RESTING RESPIRATORY RATE EACH DAY.

If a foreign body, heart failure and staphylococcal pneumonia are unlikely:

1. Give erythromycin 0.5 ml/kg (maximum 10 ml) orally 6 hourly. If there has been improvement after 1 week of erythromycin, continue it for another 2 weeks (a total of 3 weeks).
2. If there has been no improvement with erythromycin, give cotrimoxazole in high dose (trimethoprim 20 mg/kg/day in 3-4 divided doses). If there has been improvement after 1 week of cotrimoxazole, continue it for another 2 weeks (a total of 3 weeks). High doses of cotrimoxazole may cause megaloblastic anaemia or cyanosis due to methaemoglobinaemia; both resolve if the drug is stopped.
3. If there is still no improvement after erythromycin and cotrimoxazole, start TB treatment (In some instances, eg strong contact history, it would be reasonable to start TB treatment earlier).

PNEUMOTHORAX

In neonates, this is often due to meconium aspiration. In older children, it is often due to staphylococcal pneumonia, foreign body or tracheostomy.

1. The classical signs of pneumothorax are sudden deterioration of clinical state with tachypnoea and cyanosis. Examination may show mediastinal shift, resonant percussion, reduced breath sounds, and amphoric breathing if there is a fistula into the pleural cavity. However, these signs are often absent in neonates and infants.
2. In staphylococcal pneumonia, pneumothorax should be differentiated from lung cyst. Do NOT insert a chest drain into a lung cyst.
3. A small pneumothorax without respiratory embarrassment does NOT need to be drained. Observe the child closely, and try to prevent excessive crying.
4. If there is a large pneumothorax causing respiratory embarrassment, give oxygen by nasopharyngeal catheter at 2 litre/min (oxygen helps reabsorption of the pneumothorax) and try to confirm the diagnosis by chest x-ray. Then insert an underwater seal pleural drain.
5. If the child is too sick to wait for an x-ray, a needle thoracocentesis should be performed. A large “over the needle” plastic cannula is fitted onto a 10 or 20 ml syringe. It is then inserted into the chest just above the rib into the third to fifth intercostal space in the mid-axillary line on the affected side, with gentle aspiration applied. If a pneumothorax is present, air will be easily aspirated as the needle enters the pleural cavity. The needle is removed and the cannula secured in place. The cannula can be left open until an underwater seal pleural is inserted (Note: the cannula may become occluded and a tension pneumothorax may reaccumulate).

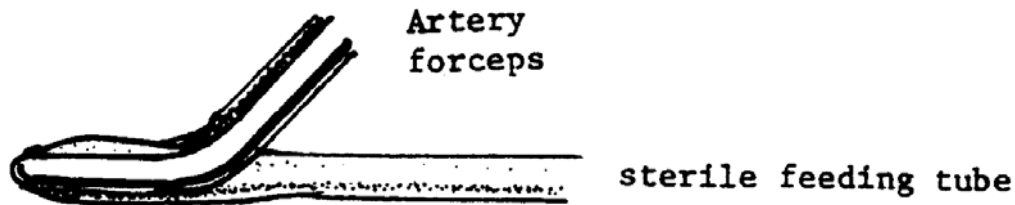
TEMPORARY UNDERWATER SEAL DRAIN

In some situations (eg a neonate with a tension pneumothorax), a temporary underwater seal pleural drain can be easily set up using a large bore plastic cannula and an inverted intravenous giving set with the venous end attached to the plastic cannula and the flask end in a container of water below the patient

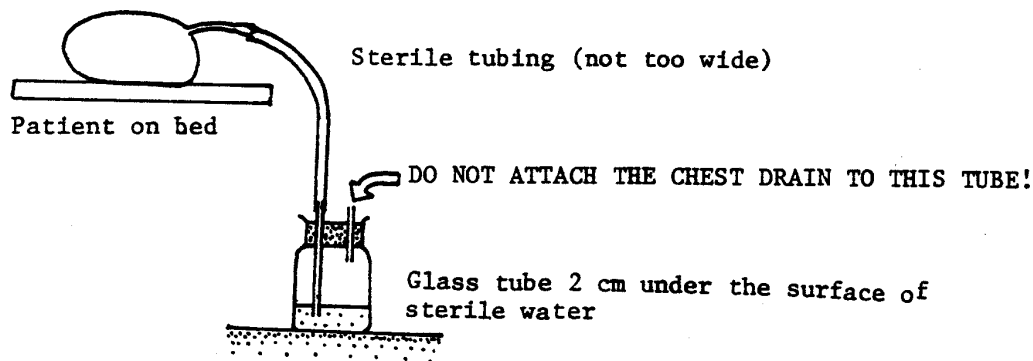
INSERTION OF AN UNDERWATER PLEURAL DRAIN

1. Have the child on nasopharyngeal oxygen at 1 litre per minute and firmly held sitting up with hands above the head.
2. Scrub your hands and put on sterile gloves.
3. The tube should be inserted in the third to the fifth intercostal space in the mid axillary line. Clean the skin with iodine over a 10 cm radius and drape the area. Inject 1% plain lignocaine through a 23 gauge needle.
4. Make an incision in the skin 1 cm long with a sterile scalpel blade.
5. Take a pair of closed artery forceps and grasp them near the end you are going to push into the chest (so you cannot push them in too far). With the forceps held at right angles to the chest wall, push the point through the chest wall and into the pleural cavity. You will have to push quite hard, and they will go through the pleura with a pop. Open the forceps to make a slightly bigger hole into the pleural cavity, then close them and remove them.

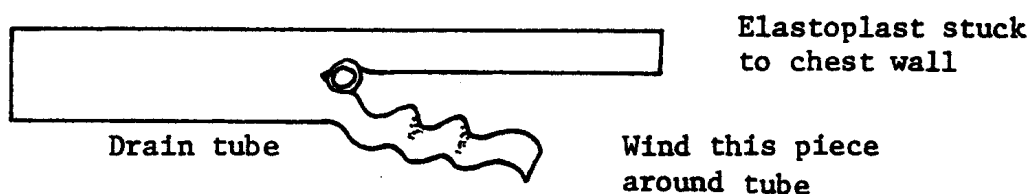
6. Take a sterile 12 gauge infant feeding tube and make a mark on the tube 5 cm from the tip. Grasp the tip with a pair of artery forceps. Push it through the chest wall along the hole you have just made. Angle the tube anteriorly towards the xiphisternum (J Pediatr 99:629,1981).



7. Remove the forceps, but leave the tube inside the chest.
8. Push the tube in until 4 cm is inside the chest. Do NOT push it in too far because this will cause it to kink or block. Clamp the tube until you have connected it to an underwater seal.
9. Connect the tube with sterile tubing to an underwater seal. MAKE SURE THAT ALL JOINS IN THE TUBING ARE AIRTIGHT. It is best if the tubing is of clear plastic. You may have to adjust the position of the tip of the intercostal tube so that either air bubbles out through the underwater drain, or so that the fluid level in the glass tube swings freely.



10. Firmly suture the wound closed - so that it is airtight. Suture the tube firmly to the skin. Put Elastoplast across the wound and around the tube.



11. The tube is only patent if air bubbles out or the fluid level in the tube swings as the child breathes. If the bubbles stop and the fluid does not swing with respiration, examine the child carefully and get another chest x-ray:
 - a. the tube may be blocked and the pneumothorax reaccumulating. You will have to replace the tube
 - b. the tube may not be correctly connected to the underwater drain bottle (it must be connected to the tube going under the water)
 - c. the air may have all drained out and the lung re-expanded. Clamp the tube for 4 to 6 hours, then take a chest x-ray. Undo the clamp on the tube; if there is still no swing or bubbles of air and you are sure the pneumothorax has not reaccumulated, remove the drain tube.

POISONING

See also Poisoning - Bites and Stings (p.333), Poisoning - Seafood Ingestion (p.336) and Snakebite (p.351).

ANTIMALARIAL OVERDOSE IS PARTICULARLY DANGEROUS.

Doses as low as 5 mg/kg IM or 20 mg/kg orally of chloroquine or amodiaquine may be fatal. Sudden death may occur in a seemingly well child. There is no specific antidote - make the child vomit IMMEDIATELY and give activated charcoal 1 g/kg every 4 hours (with a laxative). Death is from quinidine-like cardiac toxicity: treat arrhythmias with phenytoin 10 mg/kg IV over 30 min, and convulsions with diazepam. Hypotension should be treated with 8.4% sodium bicarbonate IV and dopamine infusion. Correct hypokalaemia and hypovolaemia. Convulsions should be treated with diazepam. Adrenaline has been used for chloroquine overdosage.

Useful references about poisoning are:

1. Dreisbach RH. Handbook of poisoning. Lange. This book should be kept at every hospital.
2. Poisoning by Pesticides, Division of Occupational Health and Radiation Control, Health Commission of NSW, Australia.
3. A poisons information service is available at the New Children's Hospital, Sydney (tel 0561298450000) (international access from PNG).

IF IN DOUBT, ASK FOR HELP.

MANAGEMENT

Assess the situation

Identify the suspected poison. Find out the probable dose taken, and the time of ingestion.

Dilute the poison

Give the child a cupful of milk to drink.

Remove the poison

Induce vomiting by rubbing the back of the child's throat with a spatula. If he does not vomit, give 15 ml syrup of Ipecac (NOT the concentrated Liquid Extract of Ipecac, which is itself a poison) OR a few mouthfuls of soapy water. Rub the back of the throat again.

DO NOT MAKE THE CHILD VOMIT IF THE POISON IS:

1. a corrosive acid or alkali
2. kerosene or other petroleum distillate
3. strychnine.

DO NOT MAKE THE CHILD VOMIT IF HE OR SHE IS UNCONSCIOUS.

If attempts to induce vomiting are unsuccessful, gastric lavage can be performed through a nasogastric tube. In the case of an unconscious child, gastric lavage should be performed after endotracheal intubation to protect the airway from aspiration.

Delay absorption

Use activated charcoal.

Increase excretion

Use activated charcoal.

Activated charcoal not only inhibits absorption of chemicals from the gastrointestinal tract, but also increases the clearance of many chemicals after they have been absorbed (New Engl J Med 307:676-

678,1982). To remove a poison from the body, give 0.25 g/kg of activated charcoal by NG tube every hour. Also give a laxative such as diphylac to increase bowel emptying.

DO NOT GIVE CHARCOAL OR LAXATIVE UNLESS THERE ARE ACTIVE BOWEL SOUNDS (CHECK THIS EVERY HOUR).

Inactivate the poison

Give the pharmacological antidote, if any, that is recommended in the Handbook of Poisoning.

“Antidote” management of specific poisons

Anticholinergics, tricyclic antidepressants, antihistamines and Angel’s Trumpet flowers:

Correct hypokalaemia and hypovolaemia. Arrhythmias should be treated with 1 ml/kg of 8.4% sodium bicarbonate IV (Anaesth Inten Care 1:203, 1973) and phenytoin 10 mg/kg IV over 30 min (Ann Emerg Med 10:270, 1981). Coma and convulsions can be treated with physostigmine 0.02 mg/kg IV every 5 minutes until there is a response (maximum 0.1 mg/kg), then 0.1-0.2 mg/kg/hour by IV infusion (JAMA 230:1405,1974; Lancet 2:368-9,589-90,1980). Do NOT give physostigmine if there are conduction abnormalities or arrhythmias.

Cyanide poisoning from cassava (tapioca) or lima beans:

Give oxygen, amyl nitrite by inhalation (one 0.2 ml ampoule every 5 minutes), sodium nitrite 3% 0.25 ml/kg IV over 5 minutes, and sodium thiosulphate 25% 1.5 ml/kg IV over 5 minutes.

Diazepam or opiate overdose:

Give naloxone (Narcan) 0.1 mg/kg IV, then 0.01 mg/kg/hour IV by infusion.

Metal poisoning:

1. Iron: give desferrioxamine by IV infusion at 10 mg/kg/hour for 2 hours, then 1 mg/kg/hour
2. Lead: give calcium disodium edetate 7.5 mg/kg by infusion over 1 hour every 4 hours for 5 days and dimercaprol (BAL) 4 mg/kg IM 4 hourly for 5 days
3. Other (arsenic, mercury, copper, cobalt, nickel, antimony, gold): give dimercaprol (BAL) 4 mg/kg IM 4 or 6 hourly for 3 days, then daily for 10 days (or until total recovery occurs).

Methaemoglobinaemia from anti-rust tablets, aniline dyes or chlorates (gargles, mouth washes, weed-killers):

Give methylene blue 2 mg/kg/dose IV over 3 minutes.

Paraquat (weed-killer):

Induce vomiting. Give 3 ml/kg 6 hourly of a 30% suspension of Fuller’s earth (or ordinary clay soil) in water. Do NOT give oxygen or steroids.

Preserved wood poisoning (contains arsenic pentoxide and sodium dichromate with either copper sulphate or boric acid and sodium fluoride):

Give dimercaprol (BAL) 2.5 mg/kg IM 6 hourly for 3 days, then daily for 10 days (or until total recovery).

Pesticides organophosphates such as maldion (Malathion), primiphos ethyl (Solgar), primiphos methyl (Actetic) or trithlorphon (Klorfon):

Give atropine 0.05 mg/kg (maximum 2.5 mg) IV, repeated every 5 minutes until the pulse rate rises to normal. Huge doses may be needed (much higher than the doses in textbooks). Give pyridine-2-aldoxime methochloride (PAM, pralidoxime) 25-50 mg/kg (maximum 1g) slowly IV, and repeat this dose hourly if required.

Carbamates such as mancozeb (Dithrane):

Give atropine as above, but no PAM.

Chlorinated hydrocarbons such as gamma benzene hexachloride (scabies lotion), Gammaphex 7 oil, dieldron (Dieldrex), DDT or Lindane (Septane 80, Resistox):

Do NOT give milk or oil. Give diazepam, paraldehyde or phenobarbitone for convulsions. Ventilation may be needed.

Supportive treatment

Control convulsions with IV diazepam (Valium) or IM paraldehyde. Support respiration and the circulation.

Prevention

Educate parents to keep kerosene and other poisons LOCKED UP.

POISONS ANTIDOTE CUPBOARD

Every hospital should have a poisons antidote cupboard. This should contain:

- | | |
|-------------|---|
| Parenteral: | <ul style="list-style-type: none">• adrenaline, amyl nitrite, atropine• benztropine (Congentin)• calcium disodium edetate, calcium gluconate 10%• desferrioxamine, dextrose 50%, diazepam, dimercaprol (BAL) 10% in oil• isoprenaline• methylene blue• naloxone (Narcan)• paraldehyde, phenobarbitone, physostigmine, promethazine, pyridine-2-aldoxime-methochloride (PAM, pralidoxime)• sodium bicarbonate, sodium nitrite 3%, sodium thiosulphate 25%• vitamin K1 |
| Oral: | <ul style="list-style-type: none">• activated charcoal, acetylcysteine• duphalac• glycerin• magnesium hydroxide mixture (Milk of Magnesia)• peanut oil• starch. |

Ipecacuana syrup is best kept refrigerated.

The 'Handbook of Poisoning' must also be readily available.

TABLE OF POISONS

Substance	TR* (see p.332)	Comments	Treatment
ACIDS	Depends on the concentration	Corrosive	Do not induce vomiting. Give very large amounts of milk or water followed by milk of magnesia or aluminium hydroxide mixture and olive oil or beaten egg white. Do not give sodium carbonate or bicarbonate.
ADHESIVES General purpose glue	2-3	eg "Stephens" liquid glue	Milk
Pastes	1-2	eg "Perkins" paste	Milk
Epoxy resin and polystyrene cements	3	May cause local irritation and skin sensitisation. The vehicle is usually trichlorethylene ("Trilene") or xylene	Milk
ALCOHOLS Ethanol	4		Emesis followed by activated charcoal.
Methanol (see also METHYLATED SPIRITS)	5	A latent period of 8 or more hours may precede gastrointestinal symptoms.	Gastric lavage using sodium bicarbonate and give large doses of ethanol
ALKALIS	Depends on the concentration	Corrosive	Do not induce vomiting. Dilute with large amounts of milk or water and neutralise with lemon juice (50%), vinegar (25%), olive oil or beaten egg whites.
AMMONIA AND CLOUDY AMMONIA		Corrosive if in a strong concentration. Most household cleaners contain ½% which is no problem, but cloudy ammonia contains 8% ammonia and may cause burning.	See ALKALIS.
ANTI-CHOLINERGICS	5	Toxic dose for atropine and other natural alkaloids in children is 5 mg and in adults 50 mg. No fatalities have been reported from synthetic anti-cholinergics. Symptoms include hallucinations, thirst and dry flushed skin.	Syrup of Ipecac for small overdoses. Physostigmine (p.318).
ANTIDEPRESSANTS (tricyclics, eg imipramine, amitriptyline)	5	More than 10 mg/kg can cause serious poisoning. Symptoms include lethargy proceeding to coma and cardiac irregularities.	Physostigmine and bicarbonate, see p.318.

Substance	TR* (see p.332)	Comments	Treatment
ANTI-FREEZE	3	Toxic ingredients are methanol, isopropyl alcohol or ethylene glycol in varying concentrations. Symptoms are of gastric irritation.	Immediate emesis and ethyl alcohol (which lowers toxicity of methyl alcohol). Activated charcoal will absorb the remaining alcohols after emesis.
ANTI-HISTAMINES	4	Toxic dose is 10-50 mg/kg. Children become hyper-excitable, ataxic and may convulse if the dose is high enough, while in adults CNS depression dominates.	Emesis for ingestion of small quantities. Physostigmine (p.318).
ANTI-RUST	3-4	Most anti-rust agents contain phosphoric acid or chromates (1%). Common agents containing phosphoric acid include "Ferropro rust converter" (15%) or "Rustrinse" (56%). Corrosive damage is not likely except for "Rustrinse". "Rustiban" contains hydrofluoric acid 40% and is very corrosive.	See ACIDS. Methylene blue for cyanosis (p.318). Give oral calcium gluconate and full procedure for corrosive burns. See ACIDS.
ANTISEPTICS	3	These contain chlorinated phenols ("Dettol", 1.3%), or volatile oils ("Solyptol" eucalyptus oil, 50%). Most cause vomiting if more than a few millilitres are swallowed. This is due both to the active ingredient and the soap vehicle. Bronchial aspiration is the main danger. Organic mercurials ("Mercurochrome") are poorly absorbed and so cause no problem.	Milk. Emesis or lavage if more than 25 ml is swallowed.
ANT KILLERS	5	The toxic ingredient is either tartar emetic 3% (antimony) or arsenic trioxide 0.5% (old formulations 3%). "Dedant" is an example of the former, while "Rodax" and "Loyalstone Ant Banisher" contain arsenic. Symptoms occur within 1 hour and are proportional to the severity of the poisoning. These include vomiting and diarrhoea.	Emesis as soon as possible. Then give large quantities of olive oil, milk, egg whites and 5 heaped teaspoons of activated charcoal. BAL is the antidote for arsenic and antimony.
ARSENIC	5	Many weedkillers contain up to 60%.	As for ANT KILLERS.
ASPIRIN	4	Toxic dose - children under 1 yr, 500mg; children of 2 yr, about 3 g; adults, 15 g.	Syrup of Ipecac 15 ml. IV bicarbonate. See Handbook of Poisoning.

Substance	TR* (see p.332)	Comments	Treatment
BARBITURATES	4	Ten times the normal hypnotic dose is regarded as a serious overdosage. Moderate intoxication resembles alcohol inebriation. In severe intoxication, the patient is comatose, the level of reflex activity conforming in a general way to the intensity of the central depression.	Emesis if the patient is seen early. Aggressive supportive care. Forced diuresis is useful only for methyl phenobarbitone and phenobarbitone.
BLEACH	2-3	Most liquid bleaches contain hypochlorite 5%. These include "Marvo-Linn", "Sno-White", "Zixo" and "Dynamo". It is quite non-corrosive but a strong gastric irritant. "Action" bleach and other powdered bleaches contain dichloroisocyanurates.	Milk. Emesis often occurs spontaneously.
BORAX AND BORACIC ACID	4	First symptoms are those of gastrointestinal irritation.	For more than 2 g, emesis with syrup of Ipecac. Fluids.
BRAKE FLUID	3	Most PNG brake fluids are the same and contain 100 cellosolves (monoalkylethers of ethylene glycol). They are very irritant to mucous membranes but not corrosive. Main symptoms is CNS depression.	Emesis with syrup of Ipecac for more than 15 ml.
BROMIDES	4	Deaths are relatively rare unless combined with other drugs. Potentiated by alcohol. The toxic dose is in excess of 10 g (adult) and 2 g (child). Symptoms are similar to barbiturate overdose.	Treatment is similar to barbiturate overdose.
CALAMINE LOTION.	2	Phenol content is only 0.5%.	Milk
CAMPBOR, CAMPBORATED OIL AND CAMPBOR BLOCK	4	Camphor is a potent convulsant in doses as small as 1 g or 5 ml of the oil. Glucose-6-phosphate dehydrogenase deficiency will lead to haemolytic anaemia.	Induce vomiting if detected immediately. Otherwise, perform gastric lavage followed by activated charcoal, a cathartic and a large amount of liquid paraffin. Diazepam is used for convulsions.
CARBON TETRACHLORIDE	5	Poisoning occurs after ingestion, inhalation and skin absorption. First symptoms are usually nausea and vomiting, CNS depression and visual disturbances.	Fresh air, immediate emesis and wash affected areas. Avoid all animal and vegetable fats and alcohol.
CARPET SHAMPOO	2	Soaps with pH about 10, ie irritant but not corrosive.	Milk
CAR POLISH	2-3	Aqueous or kerosene based wax emulsions of low toxicity. The risk of bronchial aspiration is low.	Milk

Substance	TR* (see p.332)	Comments	Treatment
CHLORAL	4	Toxicity low. Toxic dose is 3 g for children and 10 g for adults. Potentiated by alcohol. Symptoms are similar to barbiturate overdose.	Treatment is similar to barbiturate overdose.
CIGARETTES		Two cigarettes contain enough nicotine to cause vomiting and diarrhoea.	Activated charcoal.
CLEANERS (see also DRAIN, OVEN & TOILET BOWL CLEANERS)	1-2	Powders (“Vim”, “Ajax”) and pastes (“Gumption”) are soaps in an abrasive base. Liquids (“Sprint”, “Nifti”, “Handy Andy”) are anionic soaps of pH 10. They are irritant but not corrosive. Contents of ammonia, formaldehyde or cellosolves are not important because they are present in small percentages	Milk. Emesis is contraindicated as it may increase the hazard of frothing and inhalation.
CODEINE	5	Children tolerate codeine well. For a 2 year old, the toxic dose is about 120 mg. Symptoms of restlessness, excitability, nausea, ataxia may appear.	Emesis with syrup of Ipecac. Nalorphine is antidotal, if necessary, for respiratory depression.
DEODORANT Blocks Liquids Aerosols	3-4	Active ingredient is p-dichlorobenzene 99% in “Parry’s Fresh Air”, “Fragrasan”, “Mosom”. Toxic dose in a child is 20 g. Symptoms of gastrointestinal irritation and CNS depression are most prominent. Other blocks may contain pyrethrins and piperonyl butoxide but these are non-toxic. “Airwick” is inert in small quantities. Aerosol packs are non-toxic eg “Floriant”.	Emesis or fruit juice for small quantities; milk enhances absorption. Milk As for KEROSENE.
DESSICANT CRYSTALS	1	Silica gel is non-toxic. It is used to dehydrate cameras, tablets and other products, which are subject to deterioration from moisture.	Not necessary.
DETERGENTS	2	Household ingredients are anionic synthetics of low toxicity (“Bushland”, “Kwit”). Main action is gastrointestinal irritation causing diarrhoea.	Milk. With emesis, there is the risk of inhalation of foam.

Substance	TR* (see p.332)	Comments	Treatment
DISINFECTANTS 2 main types: Phenyl disinfectant	4	Contains 3-10% cresylic acid and can be corrosive. Contents and concentration are always on the label.	See PHENOLS.
Volatile oil disinfectant	3	Contains no more than 15% volatile oil with a methylated spirits and soap vehicle ("Pine-O-Clean", "DX", "Bushland", "Dutch").	Milk.
DRAIN CLEANERS	5	Most contain very high concentrations of caustic alkali NaOH 60%, eg "Drano", "Fissolve". Absence of oral burning does not preclude the possibility of whole granules being swallowed and causing severe oesophageal and gastric damage.	See ALKALIS.
DRY CLEANING FLUIDS (except for carbon tetrachloride)	3	Most contain white spirit (turpentine-like petroleum distillate, eg "Murlex"). Some contain trichlorethylene ("Trilene") and perchlorethylene.	Give milk. For white spirit preparation, see KEROSENE.
FELT PEN INK	1	Non-toxic.	
FERTILISERS AND LIQUID MANURES	3	Contain ammonium sulphate, nitrates and urea ("Zest"). Gastric irritation is possible but symptoms are unlikely to occur.	Milk
FLOOR POLISH	3-4	Some have a kerosene vehicle ("Durosil") but because it is emulsified, aspiration is not a likely problem. Most are aqueous emulsions of soaps and waxes and are no real problem.	Milk. Avoid emesis.
FLOOR STRIPPER	4	All are alkaline with pH about 13, so in susceptible cases, minor corrosion of the oesophagus could occur, resulting in possible strictures. Examples include "Bourne Strips" and "Super Kleen".	See ALKALIS.
FLUORIDE TABLETS		Up to 50 tablets (and probably much more) is required before symptoms occur. First symptom is diarrhoea, due to the irritant capacity of the fluoride ion in the gut.	Milk or oral soluble calcium (calcium fluoride is insoluble and therefore inert, so calcium is an antidote).

Substance	TR* (see p.332)	Comments	Treatment
FLYSPRAY Aerosol	1	Ingredients are of low toxicity, eg pyrethrins, piperonyl butoxide and dichlorvos and the propellant is freon.	See KEROSENE.
Kerosene base	3	Toxicity is purely due to the vehicle and so bronchial aspiration is the major hazard.	
“Shelltox pestrip”		Unlikely to cause any symptoms unless there is physical contact with the strip for half an hour or more.	
FURNITURE POLISH	4	Both aerosol and liquid forms contain waxes in white spirit (turpentine-like petroleum distillate). White spirit is the only toxic ingredient. Aerosols include “Pledge” and “Favor” while liquid forms include “Sheraton” and “O-Cedar”.	See KEROSENE for the liquid forms. Aerosols, being emulsified, require no treatment.
HAIR DYES	3-4	The more permanent the dye the more toxic it is likely to be. Phenylene diamines (aromatic nitroamines) are the major toxic ingredients. Irritation of mucosal surfaces and methaemoglobinaemia are the likely problems.	Emesis if more than 10 ml is swallowed. Otherwise, milk. Methylene blue is an antidote for methaemoglobinaemia.
HAIR SPRAYS Aerosols	1	Both the lacquer (polyacrylate, nitro-cellulose) and the vehicle (freon) are inert.	See METHYLATED SPIRITS.
Liquid	2	The vehicle contains a high percentage of methylated spirits.	
HAND AND BODY LOTION	2	Basically aqueous emulsions with small amounts of perfume and colouring. Mild diarrhoea is possible.	Milk
HEXACHLORO-PHENE EMULSIONS	3	Theoretical fatal dose is extrapolated from animal data. For the child, 45 ml of 3% emulsion is considered toxic. For the adult, 6-10 g of hexachlorophene or 200-350 ml of the emulsion is considered toxic. Symptoms of acute toxicity are nausea, abdominal cramps and diarrhoea. In severe cases, lethargy, drop in blood pressure and dehydration may occur.	Emesis within the first hour after a single oral dose is recommended. The principal treatment consists of sedation, fluid and electrolyte correction and, in severe cases, anti-convulsants and isoprenaline. Oxygen may be administered if cyanosis occurs.

Substance	TR* (see p.332)	Comments	Treatment
INKS Writing and stamp pad	2		Milk
Marking ink	4	They mostly have a phenolic type (toluene, xylene 80%) or aniline vehicle.	Emesis if more than a few ml ingested.
INSECT REPELLANT Aerosols	2	Large quantities will cause vomiting due to gastric irritation; may cause CNS depression.	Emesis where necessary. Otherwise, milk.
Liquids	3		
IRON TABLETS	5	More than 2 tablets are very toxic to a 2 year old and may be lethal. Symptoms may be delayed.	Desferrioxamine. See p.318.
KEROSENE AND OTHER PETROLEUM DISTILLATES	5	Symptoms of gastric irritation frequently cause vomiting. CNS depression possible.	No vomiting, give evaporated milk. See Handbook of Poisoning.
LAUNDRY BLUE	2	Both liquid and solid formulations constitute little hazard. Ultra-marine blue is the colouring matter. Borax is usually present in about 0.1%.	Milk
LAXATIVES	3	Phenolphthalein aloes, senna and bisacodyl all have a potent cathartic action but are not very toxic systematically. Symptoms are usually delayed 4-6 hours.	Emesis
LEAD PENCIL AND INDELIBLE PENCIL	1	Non-toxic, contains graphite and waxes.	
LIGHTER FLUID	3	100% petroleum distillate (naphthas).	See KEROSENE.
LUBRICATING OIL	2	Long chain hydrocarbons that are poorly absorbed, if at all. Likely to have similar effect to paraffin oil. No toxic contaminants.	Milk
MATCH HEADS AND MATCHBOXES		In the quantities likely to be eaten, toxic effects are unlikely. Match heads contain potassium chlorate 60% (toxic dose in 100 matches) which causes gastro-intestinal irritation. The striking surface of the matchbox contains red phosphorus which is quite insoluble and therefore inert.	Milk

Substance	TR* (see p.332)	Comments	Treatment
MERCURY From thermometers	1	Insoluble and inert.	Milk
From mercurial antiseptics	2	Organic mercurials are poorly absorbed and have a toxic dose for a child in the vicinity of 3 g. Their concentrations are rarely above 1/200 and therefore poisoning is unlikely. Examples include “Metaphen” and “Mercurochrome”.	
METAL CLEANERS AND POLISHES	3	These vary greatly in content. Ingredients include white spirit and ammonia (“Brasso”), thiourea (“Goddards Silver Dip”) and methylated spirits (“Silvio”). Toxicity is generally low with bronchial aspiration of the white spirit being the major hazard.	Milk. See KEROSENE for white spirit formulations.
METHYLATED SPIRITS	3	Contains 2% methanol in ethanol. Symptoms are those of ethanol poisoning. Methanol is not toxic in this dose.	See ALCOHOL.
METHANOL	4	Delayed onset of symptoms.	See ALCOHOL.
METHYL SALICYLATES			See ASPIRIN.
MINOR TRANQUILLISERS/ MUSCLE RELAXANTS eg chlorodiazepoxide, diazepam		Surprisingly low toxicity. No fatalities are recorded where this was the only drug involved. Symptoms are of drowsiness, ataxia and muscle relaxation. Potentiation with other CNS depressants is very marked.	Emesis
MOSQUITO COIL	1-2	“Tiger” contains less than 1% pyrethrins in an inert base.	Milk
MOTH BALLS (naphthalene)	4	One naphthalene moth ball is enough to make a child quite sick. Symptoms include diarrhoea and vomiting followed by excitement, restlessness and confusion. Glucose-6-phosphate dehydrogenase deficiency will lead to haemolytic anaemia.	Emesis

Substance	TR* (see p.332)	Comments	Treatment
NAIL HARDENERS	3	There are two types - those with weak concentrations of formalin ("Tuff") or those with a strengthened lacquer base ("Hard as Nails"). Formalin is primarily a gastric irritant and no toxicity would be expected from the weak solution. The lacquer based products are irritant.	Emesis if a large quantity swallowed (unlikely because of burning sensation). Otherwise, milk.
NAIL POLISH	2	Contains insoluble pigments and nitrocellulose lacquer in acetone or butyl acetate, which cause local irritation.	Milk
NAIL POLISH REMOVER	3	Contains acetone or butyl acetate. Both cause irritation of mucous membranes and mild CNS depression in quantities greater than 20 ml.	Emesis is indicated if more than 20 ml has been swallowed. Otherwise, give milk.
NAPPY WASHES	3	"Nappee" contains cationic detergent 8% which can be neutralised by anionic detergent (household soap). "Napisan" contain anionic detergent and hypochlorites. Gastric irritation is main effect.	For cationic soaps, give a weak solution of handsoap in water. Otherwise, milk is adequate.
ORAL CONTRACEPTIVES		Toxicity is low in children. One month's supply may cause some nausea but little else.	
ORGANIC PHOSPHATES	5	Toxicity ranges from very high (parathion, fenthion) to low (malathion). First symptoms are usually nausea, vomiting, diarrhoea, blurred vision, headache and abdominal pain. The Poisons Information Centre has all the registered products on file.	Emesis. Atropine and PAM (pyridine-2-aldoxime methochloride) are antidotes. See p.318.
OVEN CLEANERS	5	Some contain sodium hydroxide 10%, others only 3% or less. Corrosion is unlikely to occur in the latter concentrations but 10% will need full procedure for corrosives. All products containing sodium hydroxide will state the exact concentration on the label.	See ALKALIS.

Substance	TR* (see p.332)	Comments	Treatment
PAINTS Artists' oils and tempera	5	Most are not toxic, but possible ingredients include lead, zinc, mercury, barium and arsenical pigments.	Induce vomiting.
House paint and varnish	3	Some are aqueous. Others have a turpentine base but, because the paint is emulsified, the risk of inhalation is minimal. Pigments are insoluble and therefore inert. Lead is mainly a problem in old, peeling paint.	Milk
Water colour	2	Non-toxic in all but large quantities.	
PARACETAMOL AND PHENACETIN	4	Toxic dose in children 3-5 g and adults 20 g. Symptoms are cyanosis (due to methaemoglobinaemia), gastric irritation, hypotension and sweating. Toxic necrosis of the liver occurs in severe cases.	Immediate emesis. Antidote is N-acetyl-cysteine (Mycomyst).
PERFUME	2-3	Toxicity is due to the ethanol present - usually about 60%. The expensive concentrates are almost pure volatile oil and about 15 ml would be necessary to cause any serious symptoms.	Treat as for alcohol or give milk.
PHENOLS	4-5	These include cresylic acid, lysol. They are corrosive to skin and mucous membranes.	Wash the skin very well and rub in olive oil. If ingested, induce vomiting immediately and give milk, egg whites and olive oil. Watch for CNS depression and renal damage.
PHENOTHIAZINES	4	These include "Melleril", "Anatensol", and "Stelazine". In mild overdose, the main symptom would be extreme drowsiness and the possibility of Parkinsonian dystonic reaction.	Emesis. Benztropine (Cogentin) 0.03 mg/kg IV for extrapyramidal symptoms.
PHOLCODINE	5	Toxic dose in a child is about 200 mg and much more in an adult. Symptoms of restlessness, excitement and ataxia.	Emesis for quantities over 100 mg.

Substance	TR* (see p.332)	Comments	Treatment
PHOSPHOROUS	5 for white or yellow phosphorus	Red phosphorus is insoluble and inert. Rat poisons ("Rid-O-Rat") contain 0.6% yellow phosphorus. The first symptoms are gastro-intestinal irritation and thirst. Lethal dose is 1 mg/kg. Matches do not have phosphorus heads.	Emesis if more than 0.25 mg/kg eaten.
PLANTS (see Poisoning-Plants, p.335)	1	Many plants are toxic if eaten in sufficient quantity, the most frequent symptoms being due to oral and gastrointestinal irritation with the risk of dehydration. Only a few could be regarded as poisonous when only a few leaves or berries swallowed. These include castor oil beans, oleander and deadly nightshade, angel's trumpets, cassava and lima beans.	Emesis or lavage where a sufficient quantity had been eaten but this situation is rare. Cassava may cause cyanide poisoning (p.318). Angel's trumpet flowers cause atropine poisoning (p.318).
PUTTY	1-2	For household putties, linseed oil and calcium carbonate are the main ingredients.	Not necessary.
RAT POISON	1-2	Most commonly contain warfarin ("Ratsak"). Only dangerous if there have been repeated ingestions daily for more than 4 days. Some contain phosphorus ("Rid-O-Rat").	Oral vitamin K. See PHOSPHORUS.
ROOM DEODORANT BLOCKS			See DEODORANT BLOCKS.
SALICYLATES			See ASPIRIN.
SHAMPOO	2	Most anti-dandruff preparations contain insignificant quantities of detergents.	See DETERGENTS.
	3-4	However, those which contain selenium sulphide ("Selsun") or cadmium sulphide are potentially toxic.	Emesis if more than 10 ml swallowed.
	3	Some contain hexachlorophene.	See HEXACHLOROPHENE.
SHOE POLISH	4	The pigments are not very toxic.	Emesis if large amount ingested.
SOAP AND SOAP POWDERS	2-3	Vomiting occurs frequently because of gut irritation, but toxicity is low. Diarrhoea will probably occur.	Milk
SOLDERING FLUX AND FLUID	5	Very corrosive if they contain zinc chloride. Many contain high proportions of borax, sodium fluoride or methanol.	All should be treated immediately. Zinc chloride - see ACIDS. Borax - see BORAX. Fluoride - see FLUORIDE and methanol - see METHANOL.

Substance	TR* (see p.332)	Comments	Treatment
SNAIL KILLER	2	All snail killers contain metaldehyde ("Defender" and "Sellys Snailbait"). Toxic dose of metaldehyde in a child is 200 g and most preparations contain about 2%, so there is no problem.	
STERILISING SOLUTIONS	2	These include "Milton" and "Babee". May be mild gastric irritation.	Milk
SWIMMING POOL CHEMICALS	Depends on the concentration	Acids and alkalis are used to adjust the pH of water. The chlorinating agents used are mainly hypochlorites and chloroiso-cyanurates. Preparation with more than 10% would be corrosive.	See ACIDS and ALKALIS. Dilute with large amounts of water or milk. Give antacids; avoid acids. Unreacted hypochlorites in the stomach can be reduced by giving 5-10 g sodium thiosulphate dissolved in 200 ml of water. Start full corrosive precaution. Symptomatic and supportive care.
TOADSTOOLS		PNG has very few poisonous toadstools but an occasional fatal case has occurred. See p.335.	
TOILET BOWL CLEANERS		Vary considerably in formulation. "Harpic" and "Sani Flush" "Sanpic" "Sno-Gen"	See ACIDS See ANTISEPTICS See BLEACHES
TURPENTINE	5	Turpentine is a petroleum distillate with approximately 20% aromatic content. Toxicity is slightly higher than kerosene but it should be treated in the same way.	See KEROSENE.
UPHOLSTERY CLEANERS	3	Contain strong soaps which may cause gastric irritation and vomiting but definitely no corrosion.	Milk
VITAMINS		Acute toxicity dose of Vitamin A to a child is at least 300,000 units. Vitamin B and C are non-toxic. Acute Vitamin D toxic dose is at least 1,000,000 units.	Emesis if necessary.

Substance	TR* (see p.332)	Comments	Treatment
WEEDKILLERS	5	Toxicity varies but some are extremely dangerous ("Paraquat"). The Poisons Information Centre has all registered weedkillers on file. Doctors should refer to a publication such as "Agricultural chemicals - a synopsis of toxicity and a guide to treatment", obtainable free from the Department of Industrial Hygiene, 5 Parliament Place, Melbourne, Vic, Australia.	Paraquat: 3 ml/kg QID of 30% suspension of Fuller's earth (or ordinary soil) in water, no oxygen or steroids.
ZINC CHLORIDE	5	Present in soldering fluxes and is extremely corrosive. "Bakers" soldering fluid and other liquid zinc chloride preparation are very corrosive, one mouthful being sufficient to cause extensive necrosis.	See ACIDS.
ZINC CREAM	2	Non-toxic. If a very large quantity is swallowed, obstruction is a theoretical problem.	

*Toxicity Rating		
1	Non-toxic	Huge quantities needed to cause toxicity
2	Slightly toxic	Adult toxic dose 1 litre or 1 kg Child toxic dose 0.5 litre or 0.5 kg
3	Moderately toxic	Adult toxic dose 0.25 litre or 0.25 kg Child toxic dose 30 ml or 30 g
4	Very toxic	Adult toxic dose 30 ml or 30 g Child toxic dose 5 ml or 5 g
5	Extremely toxic	Adult toxic dose 2.5 ml or 2.5 g Child toxic dose 0.25 ml or 0.25 g

POISONING - BITES AND STINGS

See also Snakebite (p.351).

For a general review of this topic, see Warrell DA, Animal poisons, in Manson's Tropical Diseases, edited by G Cook.

SPIDERS

There are fortunately very few venomous spiders in Papua New Guinea. Large spiders may inflict painful bites - but these can be treated symptomatically.

TICKS

Fortunately the paralysis tick is not established in Papua New Guinea. However, it should always be borne in mind in a person with unexplained paralysis since there exists the possibility that it may enter the country.

HORNET

Massive attacks by a large number may be fatal. The lethal factor appears to be a nephrotoxin.

CENTIPEDE AND BLISTER BEETLE

Bites are common and painful but rarely dangerous.

GIANT MILLIPEDE

Some giant millipedes in the Pacific region and on the North Coast of the PNG mainland can squirt a corrosive fluid two feet or more. Contact with the eye may cause blindness. If the fluid lands on the skin, wash it off immediately with ether, alcohol or water, then treat as for a burn. If the fluid lands in the eye, wash it thoroughly with sterile saline or water, apply local anaesthetic and an eye pad if there is severe pain, put in antibiotic eye ointment and, if fluorescein staining reveals ulceration, apply atropine drops.

HAIRY CATERPILLARS

These very attractive orange/brown caterpillars produce a highly irritant substance which causes an urticarial eruption on the skin and can cause conjunctival oedema. The skin and eyes should be thoroughly washed and the urticaria and swelling is relieved with antihistamines.

STONEFISH (AND OTHER STINGING FISH)

The sting is very painful, but rarely fatal. Severe cases suffer intense pain, paralysis, respiratory depression, peripheral vasodilation, shock and cardiac arrest. The stonefish toxin is heat labile. The easiest and most readily available treatment is to immerse the affected limb in water hot enough to denature the venom - but not hot enough to burn the skin. In practice, this will be water at a temperature that the patient can only just tolerate. Other measures include the injection of 2% plain lignocaine into the area of the sting. This is preferable to the traditional treatment of emetine or hyoscine-N-butyl bromide (Buscopan). In the rare situation where there are severe general symptoms, stonefish

antivenene, if available, can be given. Very rarely, intubation and ventilation may be required for paralysis. Give tetanus prophylaxis.

STINGRAY

The sting causes local pain, bleeding, swelling and necrosis. The systemic effects are nausea, vomiting, diarrhoea, frequency, salivation, tonic paralysis, shock and death. Immersing the limb in water of a temperature that the patient can only just tolerate is highly effective in reducing the pain, since, like that of the stonefish, the toxin is denatured by heat. The spine should be removed. 2% plain lignocaine can be injected into the sting site. The systemic effects may require morphine, IV fluids, and ventilation. Give tetanus prophylaxis. Arterio-venous fistula is a relatively common long-term complication.

BLUE BOTTLE (PORTUGUESE MAN-OF-WAR) AND OTHER JELLYFISH

The sting is very painful, but rarely fatal. It may cause cramps, weakness, nausea, cyanosis and collapse. Apply a weak alkali locally (eg ammonia, soap), analgesics, hydrocortisone. If a tentacle is still on the skin, it is very important not to rub it - this will precipitate the firing of more poison cells.

CONE SHELL

25% of stings cause fatal paralysis. Inject 2% plain lignocaine into the wound if it is very painful. Intubation, ventilation and IV fluids may be required.

REFERENCES

Radford AJ. PNG Med J 18:138-141,1975. Giant millipede burns in PNG.
Warrell DA. Animal poisons. In: Cook G, ed. Manson's tropical diseases. 20th edition, 1996.

POISONING - PLANTS

MUSHROOM OR TOADSTOOL POISONING

The usual symptoms are vomiting and diarrhoea. Death is rare. Some fungi are hallucinogenic.

WILD LIMA BEANS

These are common in parts of Papua New Guinea. The wild varieties of lima bean usually have dark seeds and contain linamarin, which is converted to prussic acid in the gut. Vomiting and convulsions may be followed by death. The mottled bean contains less linamarin, and white beans do not contain any at all. Prolonged cooking with many changes of water is said to render most of the poisonous types safe. The symptoms are due to cyanide poisoning

CASSAVA

Careless preparation, particularly of wild cassava, can lead to cyanide poisoning. See p.318.

TRUMPET FLOWER

Ingestion of any part of this tree is a common cause of atropine poisoning in the South Pacific. There is excitability, formication (agitated picking movements), dilated pupils, dry skin, tachycardia and bladder distension. The treatment is IV physostigmine (see p.318).

DERRIS PLANT

This vine root is used in coastal areas of the Papua New Guinea and other parts of the Pacific as a fish poison and as a means of suicide. The roots can be cooked and eaten safely.

PHYSIC NUT

Ingestion causes vomiting and diarrhoea.

OLEANDER (THEVUTIA PERUVILA)

This plant with dark green long shiny leaves, yellow, white or orange trumpet shape flowers (horizontal, rather than the larger vertical hanging angel trumpets), a green fruit and a sticky white sap is common in coastal areas of Papua New Guinea. All parts contain a cardiac glycoside. Ingestion results in the signs and symptoms of digoxin toxicity - nausea and vomiting, and rhythm abnormalities - particularly heart block. Treatment is primarily supportive.

REFERENCES

Taufa T. PNG Med J;16(1):60-61,1973. Poisoning of children with physic nuts.
Vince J, Salamon B, Tau G, Zilibowicz M. PNG Med J:27:167-169,1984. Digoxin poisoning from Thevetia peruvila.

POISONING - SEAFOOD INGESTION

Seafood causes poisoning in different ways:

1. Bacterial contamination causing gastrointestinal symptoms identical with other forms of food poisoning. It is usually due to poor preparation and storage.
2. Tetradon (Puffer, Toad, Parrot and Box Fish) poisoning. These are toxic unless ALL the skin and ALL the internal organs (especially the gall bladder) are removed and the fish is placed directly on the fire for 30 minutes or more. Poisoning causes abdominal pain, vomiting, dizziness, parasthesiae, paralysis and death.
3. Poisoning from normally edible fish that have ingested toxic algae or plankton. Ciguatera is a blue-green algae that is eaten by small fish, which are eaten by carnivorous fish (which are then toxic to man usually causing paralysis). Scombroid fish poisoning, due to the growth of surface organisms, occurs after the ingestion of scombroid fish - the most common of which is tuna - which have not been adequately prepared. Paralytic shellfish poisoning is caused by ingestion of a dinoflagellate plankton, which cause red or brown patches in the sea known as "red tide". The plankton accumulates in some molluscs, and ingestion of the mollusc causes ataxia, paralysis, convulsions and death. Mannitol 1 g/kg IV over 1 hour is an effective antidote (Med J Aust 151:77-80,1989).

TURTLE MEAT POISONING

There are several species of turtle that are normally edible, but that become poisonous due to ingestion of toxic coral vegetation prior to being caught, or due to bacterial contamination after being killed (eg botulism).

PRIMARY HEALTH CARE

Children in Papua New Guinea die primarily from infectious diseases: pneumonia, malaria, diarrhoea, measles, pertussis, meningitis, tuberculosis and typhoid. Malnutrition often contributes to death from these diseases. Simple and effective means are available to prevent or treat these conditions. Such treatment is immensely worthwhile and can dramatically improve the health of a population if it is widely available.

85% of children in Papua New Guinea live in rural villages. Most of these children are not able to get to centralised urban health services. Within the urban centres themselves, an increasing proportion of the population live in squatter settlements with minimal resources, and in practice, difficult access to central health services. Hospitals in the urban centres are in danger of being overwhelmed with outpatients. For this reason, it is important that doctors do not concentrate solely on curative hospital medicine, but work to support and improve the many health services provided by paramedical workers to rural villages and urban clinics.

MATERNAL AND CHILD HEALTH SERVICES

Maternal and Child Health (MCH) Services hold mobile clinics in rural villages. They are attended by children under 5 years of age with their mothers, and are a major avenue for the delivery of health education (on nutrition, hygiene and family spacing), immunisation, treatment of common diseases and follow up of high-risk children.

Unfortunately, it is often difficult to get MCH sisters to work for long periods in rural areas. Staff at outlying health centres, therefore, tend to be young and inexperienced, with a high turnover. Young sisters may find it hard to gain the respect of village mothers. Transport for clinics may be a problem and, if clinics are not held regularly, attendances will fall. Sisters often do not arrive to start a clinic at an outlying village until 9 or 10 a.m., by which time many mothers have left to work in their gardens. A large proportion of staff time is spent doing paper work (how much of it is really necessary?). It is important that doctors be aware of these potential problems and do what they can to help.

Within the urban centres, MCH services are incorporated into the urban clinic system.

The major functions of MCH services are:

1. Health education: this is a long-term aim and staff should not be discouraged if they do not see immediate results.
2. Immunisation: prevention of pertussis, tetanus and measles is of great importance, but the major causes of death in children are pneumonia malaria, diarrhoea and meningitis. Immunisation will become even more important when effective vaccines against these diseases become available. Giving local health workers responsibility for immunisation in their area and providing feedback about their performance has been shown to increase immunisation rates (Brit Med J 296:1654-6,1988). Great care must be taken to preserve the vaccine cold chain (Lancet 1:1466,1988).
3. The treatment of common diseases: in the rural areas a monthly visit does not provide adequate facilities for treating the acute infectious diseases of children; this must be done by community health workers at the aidposts. Within the urban clinic system, preventative services should go hand in hand with curative services. For example, a sick child with an upper respiratory infection who needs vaccination with measles vaccine should receive the measles vaccine together with advice and symptomatic treatment.

AIDPOSTS OR VILLAGE CLINICS

Aidposts are the only health service permanently available to most rural villagers. Their potential for improving health through prevention and treatment is enormous. They are the most important part of the health service, but they receive the least attention and the least money. Aidposts need close supervision

and support; one of a doctor's main tasks is to persuade health workers to provide this. You should make a point of visiting aidposts regularly to see what goes on there and to identify problems.

The large contribution which aidposts in Papua New Guinea make to health was demonstrated by Lombange (PNG Med J 23:126-31,1980), who showed a dramatic reduction in morbidity in a rural part of Enga Province following the establishment of an aidpost.

VILLAGE HEALTH AIDES (VHA)

A VHA is someone chosen by the village for training in the prevention or treatment of a limited number of diseases eg malaria, pneumonia, diarrhoea or malnutrition. Older members of the community are probably best. Payment is often a major problem but should be by the village (perhaps in kind); a feast twice a year in honour of the VHA has been found satisfactory in the Madang area of Papua New Guinea. Very careful planning with provision for adequate supervision is mandatory before VHA schemes are introduced.

The theoretical advantages of VHA programmes are that the VHAs have local knowledge and are always available. They are seen as "one of us", the community, rather than "one of them", the health professionals. They can give villagers responsibility for their own health care, rather than encouraging dependence on government.

Care has to be taken not to expect too much from such workers initially: perhaps treatment of pneumonia (with oral amoxycillin) and diarrhoea (with sugar-water), and treatment of fever with antimalarials are enough to begin with. Nutrition education (and even MUAC measurement) and family planning activity might be considered later.

VHA schemes require continuous close supervision and support. Keen individuals can establish VHA services in a limited area and maintain them adequately, as long as they remain to supervise them, but such schemes often collapse after the instigator has left. It is by no means certain that such services can be run effectively in Papua New Guinea at present.

SCHOOLS

Primary schools are widely distributed throughout Papua New Guinea and have close contact with a large number of children. Health education of school children is not only a good investment for the future, but it is also a means to get information to villagers now. There is a good health curriculum for Papua New Guinean primary (and secondary) schools, but it is not clear how well the subject is being taught. It is well worthwhile making contact with your local education authorities to find out what is being done already and how you can help.

REFERENCES

Garner P, Heywood P, Baea M et al. PNG Med J 39(1):6-11,1996. Infant mortality in a deprived area of Papua New Guinea: priorities for antenatal services and health education.
Tulloch J. Lancet 354(suppl 11):16-20,1999. Integrated approach to child health in developing countries.

RABIES

Fortunately, rabies is not established in Papua New Guinea. However, there is a large potential reservoir in the mammalian population and it is vitally important that quarantine regulations are enforced.

Rabies is a zoonosis caused by a rhabdovirus. There is usually a history of a bite from a mammal 20-90 days before (although the incubation period may be 4 days to several years). After a few days of fever, headache and irritability, hydrophobia develops (furious rabies): attempts to drink water or a draft of air on the face precipitate violent inspiratory spasms with wild or aggressive behaviour. In contrast to tetanus, muscle tone is normal between spasms. Patients die from a spasm, or progress to flaccid paralysis and coma. Somewhat less than 20% of cases develop flaccid paralysis without spasms (paralytic rabies).

The diagnosis is confirmed by histological examination of the animal or, in the patient, by immunofluorescence of nerves in a skin biopsy in the first week of the illness, and by antibody in serum or CSF after the first week.

Even with intensive care, the prognosis is very poor once clinical rabies has developed. Control is by immunisation:

1. Pre-exposure prophylaxis is given to humans at high risk (eg vets and dog catchers and handlers) in endemic areas. Tissue culture vaccine should be given IM into the deltoid muscle, not into the buttocks.
2. Post-exposure prophylaxis must be started as soon as possible after exposure. Tissue culture vaccine should be given IM into the deltoid muscle, not into the buttocks. Vaccination is commenced immediately in all cases, but can be stopped after 5 days if the animal is under observation and remains well, or if the animal's brain fluorescent antibody test is negative. Antiserum is given IM to all cases (human 20 IU/kg, animal 40 IU/kg), except after minor exposure to a domestic cat or dog that is available for observation (minor exposures are licks of skin, not mucous membrane, or a minor bite to a covered part of the trunk, arm or leg); but if the animal becomes ill or is proven to have rabies, antiserum should be given immediately. All wounds should be scrubbed with soap, iodine or alcohol.

REFERENCES

- Editorial. *Lancet* 1:917-8,1988. Rabies vaccine failures.
Steinhoff MC. *Indian J Pediatr* 51:561-565,1984. Prevention and treatment.
Symposium. *Rev Infect Dis* 10:S573-S815,1988. Research on rabies prevention.

RENAL FAILURE - ACUTE

Acute renal failure (ARF) is a sudden decrease in renal function accompanied by retention of nitrogenous wastes and a disturbance of water and electrolyte balance. ARF may be classified as:

1. Pre-renal - usually due to dehydration or shock
2. Renal - due to damage to the renal parenchyma
3. Post-renal - due to obstruction of urinary flow.

Whilst the most common cause of ARF in children in a Western setting is haemolytic uraemic syndrome, the most common causes in Papua New Guinean children are probably pre-renal failure due to dehydration or septicaemic shock, and renal failure secondary to glomerulonephritis and malaria.

DIAGNOSIS

In the vast majority of children, ARF presents with, or is associated with, oliguria/anuria.

Oliguria in neonate - urine output < 0.6 ml/kg/hour

Oliguria in infant/child - urine output < 300 ml/m²/day, < 0.5 ml/kg/hour

Anuria - urine output < 1 ml/kg/day.

CLINICAL ASSESSMENT/OBSERVATIONS (REGULARLY REVIEWED)

General condition - alertness, lethargy, nausea, appetite, etc

Hydration - skin turgor, dependent oedema, ascites

Circulating blood volume - capillary return, JVP, skin temperature

Blood pressure - 4 hrly.

MONITORING

As soon as the possibility of acute renal failure is suspected, monitoring of the following parameters should be commenced:

Parameter	Frequency
Urine output	Continuous
Other output (diarrhoea, vomit etc)	Continuous
Fluid intake	Continuous
Body weight	Daily - charted on graph
Blood	
• sodium, potassium, urea, creatinine	1 - 2x daily
• total bicarbonate	Daily
Urine (if possible)	
• sodium, potassium, urea, creatinine	Daily
• osmolality	Daily

TREATMENT

Fluid and sodium balance

Maintaining fluid balance is the essence of the management of acute renal failure.

The patient may not be in balance when acute renal failure is first suspected and it is important to correct this.

1. If low circulating volume/severe volume depletion: give normal saline or colloid 20 ml/kg IV
2. If volume overload/hypertension: give frusemide 2 mg/kg IV stat.

The latter treatment is often particularly useful in patients with acute nephritis where hypertension is primarily secondary to volume overload.

Maintenance

To maintain fluid balance in oliguric/anuric patient give:

1. insensible loss (300 - 400 ml/m²/24 hours or one fifth maintenance)
2. plus urine output
3. plus other losses.

Notes

1. This maintenance regimen does not correct fluid imbalance. A low serum sodium is usually indicative of fluid overload.
2. Intake must be re-assessed at least 12 hourly as urine output may change dramatically.
3. Note must be taken of weights, sodium levels and PCV as they will give early warning of over- or under-hydration.
4. In anuric patients (as opposed to oliguric patients), it is most appropriate to give fluids which are free of electrolytes in order to compensate for insensible loss. In patients on IV fluids 5% dextrose would be most appropriate initially, although 0.18% NaCl 4% dextrose may be required later to compensate for sodium loss from sweat.
5. Sodium losses in urine and in other fluids (diarrhoea, gastric aspirate, fistula) should be replaced. In the majority of patients, enough sodium will be taken in the diet, but in those with large fluid losses, IV fluids may be required with careful calculations to balance electrolyte losses.
6. In anuric/oliguric patients, watch carefully for signs of improvement of renal function which may be indicated by increase in urine volume, and a fall in serum creatinine. Such improvement is an indication for a careful increase in fluid intake.
7. When a diuretic phase occurs, it will be necessary to rapidly increase fluid intake, and electrolyte replacement including potassium will be required. When the situation is stable, gently reduce fluid intake to normal.

Correction of acidosis

Metabolic acidosis may develop as a result of an inability to excrete the daily endogenous production of non-volatile acids - 1-3 mmol/kg/day. Oral supplementation of bicarbonate in a similar dose may be given to correct for this, although there is a danger of sodium overload.

Correction of hyperkalaemia (or hypokalaemia)

Hyperkalaemia will lead to cardiac arrest or serious arrhythmias. ECG monitoring is essential if potassium is 6.0 mmol/l or more. Ion exchange resin should be started (Resonium A 1 g/kg po or pr - can repeat 1-2 hours).

Watch for development of prolonged P-R interval and peaked T wave. As toxicity worsens the P wave is lost, QRS widens and S-T depression develops. These changes are indicators for urgent measures to reduce potassium levels and to organise peritoneal dialysis:

1. 10% calcium gluconate 0.5 ml/kg IV over 3-5 minutes (works in seconds)
2. soluble insulin 0.1 unit/kg with concurrent administration 0.2 ml/kg of 50% dextrose IV (works in minutes).

If K levels are >7 mmol/l but there are no ECG abnormalities:

1. give sodium bicarbonate 1-3 mmol/kg IV (risk of tetany) (works within an hour)
2. dextrose 0.5 g/kg/hr (10% dextrose at 5 ml/kg/hr until blood sugar reaches 14 mmol/l) (works within an hour).

Note

Hypokalaemia is also dangerous and although potassium intake should normally be severely restricted in acute renal failure, if the patient becomes potassium depleted from heavy ongoing losses (fistula or diuretic phase), it is most important that replacement is given.

Nutrition

Common sense should prevail. Avoid high potassium-containing food and encourage the child to eat high energy foods.

Indications for dialysis

The indications for dialysis in acute renal failure are as follows:

1. Fluid overload
2. Uncontrolled hypertension
3. Potassium toxicity
4. Metabolic acidosis
5. Convulsions
6. Loss of general wellbeing \pm alteration in conscious level.

See Peritoneal Dialysis (p.300).

RENAL FAILURE - CHRONIC

The aetiology of chronic renal failure (CRF) in Papua New Guinean children is not known. It is likely that chronic glomerulonephritis is the most likely cause, with chronic pyelonephritis and congenital abnormalities of the kidney and renal tract also accounting for a significant proportion of affected children.

It is quite possible for children with chronic renal failure treated conservatively to have a relatively normal quality of life for many years. Therefore, whilst chronic ambulatory peritoneal dialysis and renal transplantation are not realistic options for Papua New Guinean children, clinicians responsible for children with CRF should adopt a positive, rather than a nihilistic approach.

Objectives of management of patients with CRF:

1. preservation of remaining renal function
2. maintenance of health and well being.

MONITORING RENAL FUNCTION

Creatinine measurements

Creatinine measurements are done in most hospitals and provide a basic indicator of renal function. If the child can cooperate, it is possible to measure creatinine clearance by taking a serum creatinine level during the course of a 24 hour urine collection for creatinine (creatinine clearance = UV/P , where U is the urinary creatinine concentration, V is the 24 hr volume and P the plasma concentration).

CAUSES AND PREVENTION OF DETERIORATION OF RENAL FUNCTION

Renal function may deteriorate gradually or acutely in patients with chronic renal failure for a number of reasons:

1. continuing damage from original pathology
2. hyper-perfusion of remaining nephrons
3. uncontrolled hypertension
4. unwanted effect of angiotensin converting enzyme (ACE) inhibitor
5. obstruction
6. dehydration
7. infection
8. super-imposed acute nephritis.

A low protein diet may reduce the rate of deterioration from hyperperfusion. Control of hypertension, awareness of ACE inhibitor side effects and their avoidance if necessary, prevention and treatment of urinary obstruction and infection, and prevention and rapid correction of dehydration are all important ways of preventing deterioration in renal function.

MONITORING HEALTH AND WELLBEING

Anaemia

This is common in children with chronic renal failure and is usually normocytic normochromic.

- Iron should not be given unless iron deficiency is confirmed by low serum ferritin levels
- Folic acid may be given for megaloblastic anaemia.

Acidosis

Chronic acidosis will have an adverse effect on growth and, if severe, may give rise to tachypnoea. A standard bicarbonate of <20 mmol/l is an indication for replacement therapy.

Treatment with 1-3 mmol/kg/day of sodium bicarbonate is usually sufficient. When poorly tolerated, sodium citrate may be substituted in the same molar dosage.

Hypocalcaemia

Patients with CRF are usually hypocalcaemic. Indeed, hypocalcaemic tetany is one of the presentations of CRF commonly seen in Papua New Guinea. The hypocalcaemia of CRF is usually associated with hyperphosphataemia, and it is reasonable to reduce the latter by the use of phosphate-binding agents to prevent absorption from the gut.

Treatment: calcium carbonate is the most appropriate agent, starting at 40 mg/kg/day rising to 1 g/kg/day as required. If this is not available, aluminium hydroxide (5-50 mg/kg/dose 3 or 4 times daily) is a less satisfactory alternative (aluminium toxicity may occur).

Osteodystrophy

This is an indication of very severe CRF. Unfortunately, simply giving large doses of vitamin D is unlikely to be very effective (because the kidneys are unable to respond by manufacturing the active metabolite 1-25 OH cholecalciferol). It is possible to obtain calcitriol or activated AT-10 but this is very expensive, and unlikely to be available to most children with CRF).

Water balance

As renal concentrating mechanisms are impaired these children are very susceptible to dehydration. In hot weather, they must have adequate access to fluids and any episode of diarrhoea and vomiting must be treated promptly with IV fluids if required.

Sodium balance

Sodium retention or wasting may occur. Sodium supplements may be given as sodium bicarbonate or merely added to the food.

Potassium balance

Hyperkalaemia may occur and a low potassium diet may then be indicated. When dietary measures alone do not control the potassium, then frusemide may also be used to increase excretion.

Blood pressure

It is very important to keep the blood pressure below the 90th centile for the age of the child (see Normal Values, p.280). Most patients with renal hypertension will be controlled with a betablocker and thiazide diuretic or a betablocker and alpha methyl dopa. ACE inhibitors are now available in Papua New Guinea but are expensive and not without side effects.

Growth

This is particularly likely to be a problem in children <1 year as growth lost at this stage is rarely recovered.

Attention to correction of acidosis, salt wasting and osteodystrophy (if possible) will help to ameliorate the problem. Adequate energy intake is essential.

RENAL TUBULAR ACIDOSIS

Distal renal tubular acidosis (RTA) appears to occur with a relatively high frequency in children in PNG, especially from the western north and south coasts, and Manus. Brown and Polume (PNG Med J 37:45-9,1994) reported three cases, and since then numerous other cases have been seen. When severe, the disease leads to failure to thrive, tachypnoea due to acidosis, and clinically obvious rickets. Signs of rickets are:

- clinical - flaring of wrists, genu valgum (knock knees), rickety rosary
- radiological - metaphyseal flaring and cupping
- biochemical tests (if available) demonstrate a repeatedly low serum bicarbonate (<16 mmol/l), elevated chloride and alkaline phosphatase, and a high urinary pH. Serum potassium may be low.

There is frequently a mis-diagnosis of recurrent pneumonia or tuberculosis. Affected children do become susceptible to pneumonia and other infections due to their malnutrition and chest wall weakness, and frequently in severe cases die in infancy or early childhood if not treated (there may be a history of a previously affected sibling who has perished).

Effective therapy can be given by administration of Shohl's (alkali) solution TID or QID. The usual starting dose is 10-15 ml TID. Sometimes, potassium supplementation is required as well. Growth and general health dramatically improve, and the child can lead a normal life. Some children develop severe genu valgum, and may have episodes of hypokalaemic paralysis precipitated by gastroenteritis. Nephrocalcinosis may also occur.

REFERENCE

Brown N, Polume H. PNG Med J 37(1):45-49,1994. Renal tubular acidosis in Papua New Guinea.

SEPTIC SHOCK

Septic shock is common in babies and children in Papua New Guinea. It is caused by release of a lipopolysaccharide (endotoxin) from the cell wall of gram negative (eg E coli, pseudomonas, Bacteroides) bacilli. Endotoxin damages cell walls, causes release of lysosomal enzymes from leucocytes and activates complement. In late septic shock, there is intense alpha sympathetic activity with constriction of arterioles, which impairs blood supply to the tissues. Tissue perfusion is further reduced by hypovolaemia due to pooling of blood in dilated capillaries and increased capillary permeability with leakage of plasma proteins into the interstitial fluid.

The main features of septic shock are hypotension and oliguria. Early in the disease, some patients have an increased cardiac output with warm hands and feet, but in advanced septic shock there is a reduced cardiac output with cold hands and feet (but normal or high rectal temperature). There is usually prostration, pallor, confusion, tachycardia, hyperventilation, acidosis, hypocalcaemia and hypoalbuminaemia. Many patients have hyponatraemia, anaemia, thrombocytopaenia and clotting abnormalities. Patients with thrombocytopaenia have a very high mortality. Confusion, acidosis and cold hands and feet are also associated with a poor outcome.

MANAGEMENT

1. Give intranasal oxygen.
2. Establish intravenous access - a central line is best but usually impractical. An intraosseous line should be used if venous access is not achieved within 2 or 3 minutes (p.171).
3. Take blood for dextrostix, haemoglobin, white cell count and platelets, urea and electrolytes, and malaria parasites and culture.
4. Give a bolus of 20 ml/kg normal saline or Haemacel. Repeat this until there is clinical improvement (very large amounts - over 80 ml/kg - may be required).
5. If hypoglycaemia is present (dextrostix < 2.2 mmol/litre), give 5 ml/kg of 10% dextrose. Repeat as necessary (use 50% dextrose 1 ml/kg if no 10%).
6. Commence antibiotics. Ampicillin and gentamicin IV. If staphylococcal infection is likely, give cloxacillin as well.
7. Insert a urinary catheter and monitor hourly urine output (there should be an output of 1-2 ml/kg/hr).
8. If perfusion is still poor after 60 ml/kg of fluid has been given, consider the use of inotropes. Ideally, dobutamine (1-20 microgram/kg/min) and low dose dopamine (1-5 microgram/kg/min) are used. If dobutamine is not available, dopamine can be used on its own, but higher doses may be required (up to 10-20 microgram/kg/min) with the likelihood that renal perfusion may decrease rather than increase. If neither is available, or there is no response, isoprenaline (0.05-0.5 microgram/kg/min) or adrenaline (0.05-1.0 microgram/kg/min) can be used.
9. Once adequate perfusion is established, give IV 4.3% dextrose in 0.18% N saline at maintenance rates. Add 1 g KCl and 10 ml 10% calcium gluconate to a litre.
10. If acidosis persists in spite of adequate fluid replacement, give 2 ml/kg 8.4% sodium bicarbonate over 30 min.
11. If there is inadequate urinary output in spite of adequate fluid replacement, give a bolus of 5 ml/kg of 20% mannitol with 1 mg/kg of frusemide. This can be repeated after 6 hours.
12. If there is any obvious focus of pus, this should be drained early.

REFERENCES

The child in shock. Chap 10, p.99-116. In: Advanced paediatric life support. The practical approach. 3rd ed. London: BMJ Books, 2001.
Schierhout G, Roberts I. Br Med J 316:961-964,1998. Fluid resuscitation with colloid or crystalloid solutions in critically ill patients: a systematic review of randomised trials.

SEXUALLY TRANSMITTED DISEASE

Children can contract STD through sexual abuse (a form of child abuse that is now known to be quite common), when the disease is the same as in adults. STD infections may also be contracted in utero or during delivery; the clinical effects of congenitally acquired disease are not the same as when the diseases are contracted sexually.

GONOCOCCAL CONJUNCTIVITIS IN BABIES

Pregnant women may have gonococci growing in their vagina, which may or may not cause symptoms. The baby's eyes may be infected as he or she is being born. This is a serious infection which can make the baby blind. Within about 48 hours of birth, the baby's eyes become red and inflamed, the eyelids swell, and pus oozes from between them.

The prevention of gonococcal conjunctivitis is of great importance. As soon as possible after the baby is born, wipe the eyes with clean gauze and put in oxytetracycline eye ointment once only.

If a newborn baby presents with severe conjunctivitis:

1. take a smear of the pus from the eye
2. treat immediately. Do NOT wait for the result of the smear
3. rinse the pus out of the eye with saline
4. give crystalline penicillin IM 3 times a day for 5 days
5. put oxytetracycline (or compound antibiotic or chloramphenicol) eye ointment into both eyes 4 times a day for 5 days
6. if there is not a rapid response, consider adding gentamicin to the treatment (penicillin resistant gonococcus is unfortunately now common).

Make sure you treat both the mother and father with the standard treatment for gonorrhoea if the diagnosis is confirmed.

CONGENITAL SYPHILIS

If a woman has primary or secondary syphilis during pregnancy, her child may be born with congenital syphilis. The diagnosis may be difficult. The baby may seem normal for the first few weeks or months after birth, then fever, anaemia and poor weight gain occur. But there are often signs earlier than this:

1. In the mother, there may be a history of syphilis or a positive VDRL.
2. In a newborn baby, there may be a big liver and spleen, anaemia, skin rash, jaundice, oedema, or periostitis on x-ray.
3. In infants 3-12 weeks old, there may be a blocked nose, a skin rash (especially of the hands and feet), sores around the mouth and anus, or painful swelling of the limbs (from periostitis).
4. In children, there may be a saddle-nose, deformed teeth, poor vision, deformed legs (sabre shins), or a gumma (lump) of the nose or palate.

The diagnosis can be confirmed by doing a VDRL test on the baby each month for 3 months. A stable or rising titre confirms congenital syphilis. A falling titre suggests that the antibodies came from the mother and that the baby is not infected.

Prevention is important. In areas where syphilis is common, all pregnant women should have a VDRL blood test for syphilis done at their first ante-natal visit. Women with a positive VDRL should be given benzathine penicillin 2.4 million units IM (half into each buttock) each week for 3 weeks (if the woman has signs and symptoms of primary or secondary syphilis), or benzathine penicillin 2.4 million units IM (half into each buttock) once (if the woman has no signs and symptoms of syphilis).

All babies born to VDRL positive mothers should receive intramuscular benzathine penicillin. If no signs of congenital syphilis are present, this is all that is required for the baby. If signs are present, the treatment of congenital syphilis in a baby is:

- benzathine penicillin: 240,000u if >2.5 kg
120,000u if <2.5 kg IM once
- AND crystalline penicillin twice daily for 10 days.

GONOCOCCAL VULVO-VAGINITIS, PROCTITIS OR URETHRITIS

Treat this with a single doses of Augmentin (ampicillin and clavulinic acid), amoxycillin, and probenecid:

Weight	Amoxycillin	Augmentin	Probenecid
<10 kg	1 gram (4 x 250 mg)	Half tablet	Half tablet
>10 kg	1½ gram (6 x 250 mg)	1 tablet	1 tablet

HIV INFECTION

See section on HIV (p.151).

SKIN DISEASES

BOILS OR ABSCESSES

1. If pus is present, incise the lesion and drain it. Antibiotics are not usually required.
2. If pus is not yet present:
 - a. If the child is not sick:
 - i. check the abscess every day
 - ii. when pus has formed, incise and drain
 - b. If the child has a high fever or looks sick or has multiple abscesses:
 - i. give chloramphenicol oral or IM every 6 hours
 - ii. when pus has formed, incise and drain.
3. Remember that a hot and swollen limb may be caused by osteomyelitis or septic arthritis.

BURNS (MINOR)

1. If the burn is clean, wash it with an antiseptic solution eg chlorhexidine (Savlon) or normal saline. Cover it with gauze soaked in an antiseptic solution and squeezed dry. Change the dressing every 3 days.
2. If the burn is infected or more than 6 hours old, do daily dressings.
3. If there is dead tissue in the burn, remove it and clean the burn with chlorhexidine or normal saline.
4. Give tetanus toxoid 0.5 ml IM.
5. All patients with burns need increased fluids.
6. For the treatment of serious burns, see p.60.

ECZEMA

This causes an itchy rash that usually begins on the face after 2-3 months of age. The elbow and knee flexures are often involved in older children (In contrast, seborrhoeic dermatitis is not itchy, usually starts in the first two weeks of life, and usually clears by 3 months of age).

1. If the eczema is infected, give amoxycillin orally for 5 days.
2. Apply 1% hydrocortisone cream sparingly 3 times a day for 1-2 weeks.
3. Avoid the use of ordinary soap (this dries the skin and makes eczema worse). Use a moisturising soap if available.
4. Use a moisturising cream such as emulsifying ointment or aqueous cream if available, both as a soap and as a skin application. If not available, application of coconut oil may help.

IMPETIGO

This causes multiple crusting sores, usually on the face.

1. Clean the scabs away with an antiseptic solution eg chlorhexidine (Savlon) or normal saline.
2. Apply crystal violet (gentian violet).
3. If the infection is spreading or there is a fever, give amoxycillin 3 times daily for 5 days.

RINGWORM, TINEA (GRILLE)

1. Clean the skin with soap and water.
2. Apply benzoic acid (Whitfield's) ointment or salicylic acid paint (Grille lotion) once a day for 4 weeks. Apply to no more than a quarter of the body on any one day. Do not apply to the face.
3. Tofnilate (Tinaderm or Grille Ointment) is very effective. It can be purchased relatively cheaply at private pharmacies.

SCABIES

1. Take off all the child's clothes.
2. Wash the child and scrub him or her with a brush. This is important to remove the tops of the burrows so that the lotion can penetrate to the scabies mite to kill it.
3. Apply scabies lotion to ALL the body except the face. Leave the skin unwashed for 24 hours.
4. Explain to the mother that she must wash all the family's clothes and blankets and dry them in the sun. After 4 days, she must scrub the child and apply scabies lotion again. The rest of the family must be treated at the same time if they have scabies too.
5. If the lesions are infected, give amoxycillin 3 times daily for 5 days.

SEBORRHOEIC DERMATITIS

This is a greasy, scaling rash that is not itchy. It usually starts in the first two weeks of life on the scalp (as cradle cap). Other sites are the forehead, eyelids, retro-auricular folds, neck, axillae, groins, gluteal cleft and trunk. The rash usually clears by 3 months of age (In contrast, eczema is an itchy rash that usually begins on the face after 2-3 months of age).

1. If the seborrhoea is infected, give amoxycillin 3 times daily for 5 days.
2. Apply 1% hydrocortisone cream sparingly three times a day for 1-2 weeks.
3. Avoid the use of ordinary soap (this dries the skin and makes eczema worse). Use a moisturising soap if available.
4. Use a moisturising cream such as emulsifying ointment or aqueous cream if available, both as a soap and as a skin application. If not available, application of coconut oil may help.

SORES OR ULCERS

1. Clean any dirt and dead tissue away with antiseptic lotion, eg chlorhexidine (Savlon) or normal saline.
2. If the lesion is infected, painful, swollen and red, put a little acriflavine emulsion on a gauze dressing and put it on the sore. Do this daily until the wound or ulcer is clean.
3. Dress clean and moist ulcers, sores or cuts with gauze soaked in antiseptic solution and squeezed dry.
4. If the sore is larger than 2.5 cm in diameter, if there are multiple sores, or if there is surrounding cellulitis, give amoxycillin 3 times daily for 5 days.
5. If there is no improvement, give cotrimoxazole orally twice daily for 5 days and tinidazole orally once daily for 5 days.
6. Large ulcers should be skin grafted when they are clean.

SNAKEBITE

There have been a number of changes to the understanding of snakebite and its treatment in Papua New Guinea over the last decade. It was previously widely held that the Death Adder was responsible for the majority of venomous snakebites in the highlands and mainland northcoast region, whilst on the Papuan south coast, there were three common venomous snakes - the Papuan Taipan, the Papuan Black and the Death Adder. A recent study (Lalloo et al 1995) indicated that the Papuan Taipan accounted for by far the largest majority of snakebites on the Papuan coastal region. The Papuan Black snake would appear to be very rare. Bites by the Papuan Whip snake (a “thin” snake as opposed to the rather “thick” Taipan) occur but are rarely fatal. Bites by other much less common snakes such as the Small Eyed snake, the Common Brown snake (not at all common!) and some varieties of aquatic snake have been reported but are rarely fatal.

The antivenom available from Australia is made against Australian snakes. Polyvalent antivenom is effective against Death Adder, Taipan and Papuan Black snake (a close relative of the Australian Eastern Brown snake). Death Adder monovalent antivenom is available. The cost for a single ampoule of antivenom is in the region of 7,000 Kina.

The venoms of the Death Adder, the Taipan and the Papuan Black are all very potent neurotoxins (the Taipan venom is said to be the most potent of any snake in the world!). Death is usually the result of respiratory paralysis. The venoms also have complex effects on blood coagulation and clot lysis and haemorrhagic complications are common. Renal failure may also occur - possibly as a result of myotoxicity. Local necrosis does not occur.

The available antivenoms reverse coagulopathy, and the Death Adder antivenom has some beneficial effect on the neuropathy, but Taipan neuropathy is not significantly reversed by Taipan antivenom. It is therefore important to give the antivenom - if available - before respiratory paralysis has occurred. A high level of care of the airways, artificial ventilation and good nursing care are essential in the management of envenomated patients. With such care, outcome is relatively good, even when antivenom is not available.

Should antivenom not be available, fresh frozen plasma should be given to reverse the coagulopathy.

WHICH ANTIVENOM TO GIVE?

Highlands and North Coast:	Death Adder antivenom, unless snake otherwise identified, in which case give polyvalent
Papuan Region:	Polyvalent if identity of snake unclear Taipan or polyvalent if “black” snake Death Adder if positive identification

FIRST AID

1. Immediately apply a firm bandage to the length of the bitten limb, starting distal to the bite. Splint the limb as well as bandaging it. These measures prevent dissemination of the venom by obstructing the lymphatics. Do not remove the splint and bandage until full facilities are available.
2. Reassure the patient and transport him or her safely and without panic to the nearest hospital or health facility.
3. Alert the hospital to the patient’s arrival, if possible. Deterioration may occur following removal of the bandage.
4. DO NOT use tourniquets
 cut the skin around the bite
 suck the bite.

Admit:

1. All children who have definitely been bitten by a poisonous snake.
2. All children who may have been bitten by a poisonous snake.

Look for symptoms and signs of envenomation:

- Nausea and vomiting
- Painful lymph glands
- Abdominal pain
- Weakness of limbs
- Difficulty seeing properly
- Drooping eyelids
- Difficulty with breathing
- Dribbling of saliva

Take a blood sample for clotting time:

A clotting time of >15 min is highly suggestive of envenomation.

TREATMENT

No signs of envenomation or clotting time less than 15 min

1. Keep on bed rest
2. Hourly observations for at least 12 hours after admission. If signs of envenomation appear, treat as below:
3. Do not touch the site of the bite
4. Give amoxycillin for 5 days
5. Give tetanus toxoid.

Signs of envenomation or clotting time more than 15 min

1. Keep child on bed rest. Nurse on the side and keep the airway clear.
2. Put up a drip of 1/5 N saline in 4.3% dextrose.
3. Make sure you have resuscitation equipment, adrenaline and hydrocortisone ready in case of anaphylactic shock.
4. Give a dose of promethazine 1 mg/kg slowly IV over 1 minute, followed by hydrocortisone 100 mg IV over 1 minute.
5. Give ½ ml of antivenom through the running drip over 1 minute.
6. Wait for 5 minutes
 - a. If no reaction, give the remainder of the antivenom IV over 20 minutes
 - i. then run drip at maintenance rate
 - ii. give benzyl penicillin IV 6 hourly
 - iii. give tetanus toxoid
 - iv. do not touch the site of the bite
 - b. If there is a reaction to the antivenom
 - i. Mild reaction (fever, skin rash)
 - give another dose of IV hydrocortisone
 - continue the antivenom slowly over 1 hour. STOP if there is any deterioration.
 - ii. Severe reaction (wheezing, shock)
 - immediately STOP giving the antivenom
 - give another dose of IV hydrocortisone
 - give diluted adrenaline IV over 1 minute (see Anaphylaxis, p.29)
 - give oxygen
 - start artificial ventilation if necessary.

IF PARALYSIS IS PROGRESSING IN SPITE OF ANTIVENOM, INTUBATION AND VENTILATION SHOULD BE PERFORMED EARLIER AND ELECTIVELY RATHER THAN LATER AND IN AN EMERGENCY.

THE COMMONEST CAUSE OF DEATH IN INTUBATED SNAKEBITE CHILD VICTIMS IS A TUBE ACCIDENT. RIGOROUS ATTENTION TO TUBE MANAGEMENT IS VITAL.

REFERENCES

- Sutherland SK. Aust Fam Phy 19(1):21-41,1990. Treatment of snake bite.
- Lalloo DG et al. Am J Trop Med Hyg 52(6):525-531,1995. Snake bites by the Papuan Taipan.
- Lalloo DG et al. Trans Roy Soc Trop Med Hyg 89:178-182,1995. The epidemiology of snake bite in the Central Province and national capital district, Papua New Guinea.
- Lalloo DG et al. . QJM 89(1):25-35,1996. .Neurotoxicity, anticoagulant activity, and evidence of rhabdomyolysis in patients bitten by death adders in Southern Papua New Guinea.
- Warrell DA et al. QJM 89(7):523-530,1996. The emerging syndrome of envenoming by the New Guinea small eyed snake.
- Brian MJ, Vince JD. Trans Roy Soc Trop Med Hyg 1:850-852,1987. Treatment and outcome of venomous snake bite in children in Port Moresby General Hospital.
- Kiromat P. Snake bite in children admitted to Port Moresby General Hospital. MMed Part 2 Project 1997 (Dept Clinical Sciences).

STRIDOR

Always suspect foreign body. Take a chest x-ray. Give all patients with acute stridor and fever chloramphenicol for 5 days.

IT IS DANGEROUS TO EXAMINE THE THROAT OF A CHILD WITH CROUP OR EPIGLOTTITIS - COMPLETE RESPIRATORY OBSTRUCTION MAY BE PRECIPITATED.

FOREIGN BODY

A foreign body is suggested by:

1. age 1 to 5 years
2. a history of inhalation: there is a sudden onset of stridor or cough while eating or when there was an object in the child's mouth (eg a coffee bean)
3. wheeze as well as stridor (this is very strong evidence)
4. a chest x-ray taken in expiration may show air trapping (a darker lung) on the affected side.

IF YOU SUSPECT FOREIGN BODY, REFER THE PATIENT TO A BASE HOSPITAL FOR BRONCHOSCOPY.

CROUP (LARYNGO-TRACHEO-BRONCHITIS)

A need for tracheostomy is suggested by:

1. severe rib recession
2. poor air entry
3. pallor, restlessness and tachycardia
4. cyanosis (tracheostomy is URGENT).

For ill understood reasons, croup is less common in Papua New Guinea than in "Western" countries; when it does occur, it is often caused by measles or by pyogenic bacteria (see Jones R, JAMA 242:721-6, 1979, for a discussion of bacterial tracheitis). It is often severe, and many affected children require tracheostomy.

EPIGLOTTITIS

A rare, fulminant illness with septicaemia due to *Haemophilus influenzae*. Give IV or IM chloramphenicol. Patients with epiglottitis are pale, toxic, sitting up and drooling; they have an expiratory snore, high fever and a very sore throat. Unlike children with croup, they do not have a harsh cough, and they are not hoarse. Tracheostomy is often necessary.

TB

If stridor persists, the chest x-ray is suspicious (especially if there is a widened mediastinum), there is a history of fever and weight loss prior to stridor, or a family history of TB, do a Mantoux, three gastric aspirates, and review the chest x-ray carefully.

DIPHTHERIA

This is now extremely rare in Papua New Guinea, but could still occur. The diagnosis is suggested by a membrane on the tonsils (The three conditions which cause membranous tonsillitis are diphtheria, streptococcal tonsillitis and infectious mononucleosis).

STREPTOCOCCAL TONSILLITIS

Although the classic presentation appears to be rare in PNG children, a case of severe disease causing respiratory obstruction and “bull neck” has been reported (Brown N, Lagani W. PNG Med J 36(3):246-248,1993).

IT IS DANGEROUS TO EXAMINE THE TONSILS OF A CHILD WITH STRIDOR.

Examine the throat GENTLY with the child sitting up and facilities for intubation available if you suspect diphtheria.

TEMPORARY RELIEF OF STRIDOR

Temporary relief of stridor can be obtained with the use of nebulised adrenaline (put 0.5 ml of adrenaline 1 in 1000 and 1.5 ml normal saline in the nebuliser). This procedure “buys time” - about 10-15 minutes and can be repeated - to enable organisation of planned tracheostomy or intubation.

STRONGYLOIDIASIS

There are two species of *Strongyloides* that cause significant problems in Papua New Guinean children.

STRONGYLOIDES STERCORALIS

Previously thought to be rather uncommon, this parasite has been shown to be relatively common, particularly in urban areas. It is a gut nematode, with a life cycle very similar to that of the hookworm (*Necator Americanus* and *Ankylostoma duodenale*). The adult worm burrows under the mucosa of the small intestine and causes a protein losing enteropathy. The classical presentation is of hypoproteinaemic oedema.

Occasionally a “hyperinfection” syndrome may occur with severe diarrhoea, pulmonary signs and symptoms and profound hypoproteinaemia.

DIAGNOSIS of *strongyloides stercoralis* infection should always be considered in a child presenting with oedema, and is easily made by examination of fresh stool, which will reveal larvae (*Strongyloides stercoralis* is the only human parasite whose eggs may hatch before excretion).

TREATMENT is with albendazole (chewed or crushed) daily for three days.

STRONGYLOIDES FULLEBORNI V KELLYI

Strongyloides fulleborni is a parasite primarily of non-human primates, but which successfully adapts to human transmission, and is common in many periurban areas of Africa.

Overwhelming infection with what was originally thought to be *Strongyloides fulleborni* was discovered to be the cause of an unusual and almost invariably fatal illness in infants in the Kamea area of the Gulf province (Ashford et al 1979, Vince et al 1979).

Since Papua New Guinea has no non-human primates, the discovery of this parasite was a cause of considerable interest. It has been given its own classification - *v Kellyi* - after the parasitologist who first described the ova.

Epidemiological studies have revealed that infection is widespread by the age of 6 months, and declines in later childhood and is at a low level in adults.

The mode of transmission remains a mystery. The parasite has also been found in other areas geographically similar to the Kamea area.

Affected children are usually less than 6 months old. They develop marked abdominal distension due to ascites, cough and shortness of breath (suggesting pneumonia) and diarrhoea. Other features often present are peripheral oedema with pleural or pericardial effusion, hepatomegaly, staring eyes and eosinophilia. Anaemia is not usually present.

Unless the correct diagnosis is made, these children are often thought to have septicaemia and pneumonia. But cultures are sterile and antibiotics are ineffective. The faeces contain large numbers of embryonated ova - which hatch within a short time of excretion.

TREATMENT

1. Give thiabendazole (Mintezol) 25 mg/kg BD for 3 days. If thiabendazole is not available, give albendazole 1 tab daily for 3 days if <10 kg, 2 tab daily for 3 days if >10 kg. The albendazole should be crushed.
2. If oedema is severe, give IM or IV frusemide and plasma (of the same blood group as the patient) or SPPS 40 ml/kg IV over 4 hours.
3. Give chloramphenicol IV.

4. Give antimalarials.
5. Give nasopharyngeal oxygen at 1 litre/min if the child is cyanosed.

REFERENCES

- Ashford RW, Vince JD, Gratten M, Banakiori JBK. PNG Med J 22:128-135,1979. Strongyloides infection in a mid mountain Papua New Guinea community: results of an epidemiological survey.
- Ashford RW, Vince JD, Gratten MJ, Miles WF. Trans Roy Soc Trop Med Hyg 72(5):554,1998 (letter). Strongyloides infection associated with acute infantile disease in Papua New Guinea.
- Barnish G, Harari M. PNG Med J 32(1):51-54,1989. Possible effects of strongyloides fulleborni-like infection on children in the Kramui area of Simbu province.
- Vince JD, Ashford RW, Gratten M, Banakiori JBK. PNG Med J 22:120-127,1979. Strongyloides infection in young infants in Papua New Guinea. Association with generalised oedema.

SUBACUTE SCLEROSING PANENCEPHALITIS (SSPE)

INTRODUCTION AND EPIDEMIOLOGY

Subacute sclerosing panencephalitis (SSPE) is an invariably fatal degenerative condition of the central nervous system which predominantly affects children and young adolescents, which is classed as a “slow virus” disease, and which is associated with measles virus infection. It is generally agreed that the incidence of SSPE is related to the age of measles infection. The younger the age at infection, the higher the incidence of SSPE. Other host and environmental factors may well play a role. SSPE has been reported to be more common in males than in females, in rural than in urban areas, and in some racial groups than in others. Both temporal and geographical clustering occurs. Recent confirmation of considerable genomic variation in wild measles virus has strengthened the long held suspicion that SSPE may be linked to particular strains of virus. Papua New Guinea has the dubious distinction of having had the highest reported incidence of SSPE anywhere in the world. The IMR reported an incidence of 56 cases per million population under the age of 20 years in 1991- some 100 times that in the USA. The incidence may be even higher than this. The Paediatric Surveillance Unit received 58 reports of SSPE - the majority from the Eastern Highlands Province - in an 18 month period between July 96-Dec 97.

SIGNS AND SYMPTOMS

The earliest indications of pathology are mild and perhaps unrecognised changes in the child’s behaviour and in social and academic performance. These “mental” changes deteriorate to a stage of dementia. Characteristic “neurological” features are the onset of myoclonic jerks which are generally symmetric and the development of hypertonicity, which may progress to a state of decerebrate rigidity. Cerebellar ataxia and hypotonic state may also occur.

The neurologic and mental impairment gradually impair the child’s ability to feed, and in the absence of nutritional support, a state of severe malnutrition eventuates.

Staging systems for SSPE have been proposed, eg:

Stage 1- cerebral signs (mental and behavioural)

Stage 2 - convulsive motor signs

Stage 3 - mutism, loss of cerebral cortex functions, myoclonus

Stage 4 - coma, opisthotonus.

A second system proposes 3 instead of 4 stages. This may be helpful in discussing prognosis in relation to the speed of progression, though in practice there is considerable overlap between the stages which merge into each other.

The symptoms and signs of SSPE begin from months to years after measles infection. In a Port Moresby study of 19 patients, the time interval from measles infection to diagnosis of SSPE was 4.4 years - somewhat shorter than that reported from the USA. There is some variation in the progression of the disease. Death may occur within 6 months of the onset of symptoms in a few patients but the majority survive for at least a year with a mean of around 18 months. Survival beyond 3 years is extremely uncommon. The course of the disease in the majority of patients is of insidious onset and inexorable deterioration. A few patients may have clinical remissions of varying duration.

DIAGNOSIS

The clinical features are themselves highly characteristic. Diagnosis is confirmed by finding markedly elevated levels of antibody against measles virus in both serum and CSF (the CSF antibodies being synthesised within the CNS).

If available, an EEG may demonstrate characteristic but not specific changes of “suppressive-burst episodes”. CT scan and MRI may be normal in the early stages but with progression of the disease may demonstrate cortical atrophy with ventricular enlargement and other nonspecific changes.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes chronic meningitis (tuberculosis and cryptococcosis), other forms of neurodegenerative disease, and neoplastic conditions.

TREATMENT

No treatment has cured SSPE. Claims have been made for benefit in terms of slowing disease progression from the use of inosiplex, interferon alpha and cimetidine.

The mainstays of treatment are:

1. control of convulsions as far as possible (benzodiazepines such as clonazepam or nitrazepam are probably likely to be the most helpful)
2. nutritional support
3. nursing care of the bedridden and eventually incontinent patient
4. support of the family as far as is possible.

In a person presenting in the early stages of the disease in areas where TB is common, it is not unreasonable to commence treatment for CNS TB pending exclusion of this diagnosis and confirmation of SSPE.

PREVENTION

There is incontrovertible evidence that SSPE is very largely (but not completely) preventable by vaccination against measles. Relative risks for SSPE after measles compared with vaccination were 29 - and were 100 for children acquiring measles before the age of 1 year. The introduction of measles vaccination in the USA led to a 100-fold reduction of SSPE.

Unfortunately, vaccination coverage rates have been too low and problems of cold chain maintenance too large in Papua New Guinea for the measles vaccination policy to have had a major preventative effect as yet. Hopefully, though, the incidence of SSPE will, with improvement in these factors, fall within the next 5-10 years.

REFERENCES

- Jabour JT et al. JAMA 220:959-961,1972. Epidemiology of subacute sclerosing panencephalitis.
Dyken PR. Neurol Clin 3:179,1985. Subacute sclerosing panencephalitis. Current status.
Lucas K. PNG Med J 36(3):185-186,1993. Subacute sclerosing panencephalitis in Papua New Guinea.
Lucas KM et al. Epidemiol Infect 108:547-553,1992. Subacute sclerosing panencephalitis in PNG. A high incidence in young children.
Tovilu M. A four year review of subacute sclerosing panencephalitis at the Port Moresby General Hospital. MMed (Child Health) Dissertation 1993. Dept of Clinical Sciences UPNG.

TETANUS

See also Neonates - Tetanus (p.275). Tetanus is a serious disease with a high mortality.

1. AVOID ALL UNNECESSARY HANDLING AND NOISE.
2. If the spasms are severe, give nasopharyngeal oxygen until sedation is achieved.
3. Give paraldehyde 0.2 ml/kg IM stat for initial sedation.
4. So that diazepam and tetanus immunoglobulin can be given IV, insert an IV drip of 0.9% sodium chloride (normal saline):

Under 10 kg:	run the IV at 10 ml/hour
10 kg or more:	run the IV at 20 ml/hour

The IV can usually be removed after good sedation has been achieved in about 24 hours (then give all drugs NG, oral or IM). If the IV stays in for more than 48 hours, change from 0.9% saline to 4.3% dextrose in 0.18% sodium chloride with 1 g KCl per litre.

5. Sedation is a most important part of the management of tetanus. Use diazepam and chlorpromazine. Give chlorpromazine (Largactil) 5 mg/kg IM stat, then 5 mg/kg 12 hourly by NG tube.
6. The maintenance dose of diazepam (Valium) is adjusted according to the response. It is important to understand the pharmacology of diazepam:
 - a. IV diazepam has a marked initial effect due to the high serum levels. IT MUST BE GIVEN SLOWLY
 - b. this effect rapidly wears off because the drug is redistributed into fat, so that extra doses have to be given frequently at first
 - c. diazepam has a long half life (24 to 48 hours), so that once levels of diazepam in fat have risen, 12 hourly doses are adequate for maintenance: more frequent doses will cause accumulation of the drug and may cause toxicity
 - d. IM diazepam is very poorly and irregularly absorbed, and the drug is much better given IV or orally
 - e. IV diazepam is incompatible with dextrose. Intravenous injection of undiluted diazepam causes severe thrombophlebitis. Diazepam is relatively insoluble in water or saline, and will precipitate if it is diluted at less than 1 in 20
 - f. diazepam cannot be given by slow IV infusion, because it is bound to the plastic of IV tubing. Diazepam should therefore be given orally or by NG tube, or the undiluted preparation injected slowly into a freely flowing IV drip of 0.9% sodium chloride.

The loading dose: initially, give diazepam 0.5 mg/kg (0.1 ml/kg of 10 mg/2 ml amp) SLOWLY every 15 to 30 minutes until severe spasms have been controlled, usually after 2 to 4 doses.

Maintenance doses: give the same dose (0.5 mg/kg) NG or oral every 12 hours, changing to half this dose every 12 hours as the child improves. If severe spasms recur on the maintenance dose, give 0.5 mg/kg (0.1 ml/kg of a 10 mg/2 ml ampoule) IV every 15 to 30 minutes until control is achieved again.

7. Give benzyl (crystalline) penicillin IV 6 hourly. When the IV is removed, change to oral amoxycillin. Change to chloramphenicol if secondary infection occurs.
8. Give tetanus immunoglobulin on admission after the initial sedation.
 - a. If intravenous human immunoglobulin is available:
 - use a 2,000 unit ampoule of intravenous human immunoglobulin. Perform a careful lumbar puncture, and remove 2 ml of CSF for microscopy, culture, protein and glucose. Then SLOWLY inject 3 ml (about 200 units) of immunoglobulin intrathecally through the LP needle. Give the remainder of the 2,000 unit ampoule (about 25 ml) and the whole of another 2,000 unit ampoule IV over one hour.
 - b. If the IV preparation is not available:
 - use 250 unit IM ampoules of human immunoglobulin. DO NOT GIVE THIS INTRATHECALLY - give 750 unit (3 ampoules) IM stat. Give another 500 unit (2 ampoules) IM on the following 2 days.

- c. If horse antitoxin has to be used, give 750 unit IM daily for 3 days
9. Pass a nasogastric tube and give NG milk feeds: see Oral Fluid Requirements (p.397). Mild cases can take fluids by mouth. Very severe cases may need IV fluids, but NG feeding is preferable.
10. Clean, turn and suction the child 3 hourly. Keep a careful record of all spasms, and adjust the sedation accordingly.
11. Tetanus often causes autonomic dysfunction. Monitor the pulse rate and blood pressure carefully, and give morphine 0.1-0.5 mg/kg IV over 30 min every 2-8 hours for hypertension or unexplained tachycardia (Crit Care Med 17:371-5,1989). Propranolol has also been used, but may cause sudden death.

	Keep the pulse rate below	Normal BP
3 years or less	120	90/60
4 years or more	100	95/70
The pulse rises by 18 beats for every 1 °C rise in temperature.		

12. Tracheostomy should be performed routinely in all but the mildest cases of tetanus, so that hypostatic pneumonia can be prevented by careful suction.
13. If the tetanus spasms are not controlled with the above treatment, consider paralysing and ventilating the child.
14. Start active immunisation with tetanus toxoid 0.5 ml IM during convalescence and give a total of 3 doses at 2 month intervals.

REFERENCES

- Editorial. Lancet 2:464,1980. Intrathecal TIG (human).
 Gupta PS. Indian J Pediatr 49:15-17,1982. Intrathecal immunoglobulin.
 Sanders RKM. Lancet 1:974-7,1977. Intrathecal ATS (horse) in tetanus.
 Sanders RKM. Tropical Doctor 7:99-104,1977. Treatment in rural areas.
 Shann F. Med J Aust 2:604-605,1983. Intrathecal tetanus antiserum.
 Wright DK. Crit Care Med 17:371-375,1989. Autonomic dysfunction in tetanus.

THALASSAEMIA

See also Anaemia - Persistent or Recurrent (p.20).

INTRODUCTION

Both alpha- and beta-thalassaemia genes are common in Papua New Guinea, and there is good evidence that they are protective against malaria. There is very interesting recent evidence that the alpha + homozygous state is protective not only against malaria, but also against other infections.

The situation of having no alpha globin genes is incompatible with survival, and alpha thalassaemia is not a clinical problem. By contrast, beta thalassaemia is a major problem in the coastal areas of the country (and, interestingly, in the southern highlands, representing spread of the gene from the Gulf coast).

Beta thalassaemia is the most important haemoglobinopathy in Papua New Guinea.

Normal Hb electrophoresis and foetal Hb

The Hb A2 is normally less than 3% (it is high in thalassaemia minor). Hb F after the age of 6 months is normally less than 2% (it may be up to 15% in thalassaemia minor, and over 20% in thalassaemia major). Blood transfusion invalidates these values for 3 months. If Hb electrophoresis is unavailable, simpler tests for the presence of cells containing foetal Hb can be used.

THALASSAEMIA MINOR (HETEROZYGOUS)

This causes a mild hypochromic anaemia that is unresponsive to iron. In uncomplicated cases, the haemoglobin is rarely less than 10 g/dl. The diagnosis is confirmed by finding Hb A2 over 3% on Hb electrophoresis. No treatment is indicated. Iron should not be given.

THALASSAEMIA MAJOR (HOMOZYGOUS)

Both parents have thalassaemia minor. Mild anaemia and splenomegaly develop by 6 months of age, severe anaemia by 1 year and hepatomegaly and massive splenomegaly by 2 years. Growth is impaired and there is widening of the flat bones of the skull and face (frontal bossing, with prominent cheeks and upper lip). Infections are common.

There is a severe hypochromic microcytic anaemia with target cells, nucleated red cells and a reticulocytosis. The serum bilirubin is high. Haemoglobin electrophoresis shows elevated Hb F and Hb A2. Skull x-ray shows a "hair on-end" appearance.

Ideally, treatment consists of regular blood transfusions to maintain Hb above 10 g/dl (this prevents the development of splenomegaly and hypersplenism and greatly improves the quality of life) combined with regular iron chelation by means of subcutaneous infusions of desferrioxamine. The cost of desferrioxamine is prohibitive, and it is not used in Papua New Guinea. There is considerable interest in the development of an oral chelating agent. The only one which has been trialled to any extent, L1 or Diferriprone, has unfortunately been associated with a relatively high incidence of side effects, and efforts to obtain it for trial use have been unsuccessful.

In Papua New Guinea, it should be possible (but is by no means easy) to maintain children with a Hb >8.5 g/dl. Provided that the patients attend regularly and blood transfusion is given as soon as the Hb drops below 9 g/dl, the amount of blood used is actually no more - and may be less than the amount required when the Hb is allowed to drop to low levels, and then brought up to above 10 with large infusions. Weekly folic acid and antimalarials should be given.

Splenectomy is only indicated in an older child with hypersplenism that causes greatly increased transfusion requirements. It is dangerous in malarious areas. Immunisation against pneumococcus and

Haemophilus influenzae type B should be given before splenectomy. After splenectomy, give phenoxymethylpenicillin 1 tablet daily and antimalarials for life.

Unfortunately, thalassaemic children in Papua New Guinea die between the ages of 8-20 years from haemosiderosis resulting from multiple transfusions.

If the parents have further children, each child has a one in four chance of being affected. The parents should be strongly advised to accept family planning.

REFERENCES

- Olivieri NF. Lancet 341(2):99-107,1999. The B thalassaemias.
Olivieri NF, Brittenham GM, McLaren CE, Templeton DM et al. New Engl J Med 339:417-423,1998.
Long term safety and effectiveness of iron chelation therapy with deferiprone for thalassaemia major.
(Editorial commentary by Kowdley KV and Kaplan MM, p.468-469).

TRACHEOSTOMY

This is a difficult procedure in a small child. Except in an emergency, you should not attempt this operation yourself unless you have had considerable surgical experience. Transfer patients to an experienced surgeon BEFORE severe symptoms develop if you can.

Cannulating the cricothyroid membrane with a large bore needle does NOT provide an adequate airway for a patient with severe obstruction (Br Med J 1:854,1978). In a hospital, peroral endotracheal intubation is almost always possible, and then can be followed by a leisurely formal tracheostomy.

ANAESTHESIA

No anaesthesia is required for an emergency procedure in a moribund child. In a hospital without good anaesthetic facilities or if there is no halothane, an urgent tracheostomy can be performed under local anaesthesia (1% plain lignocaine).

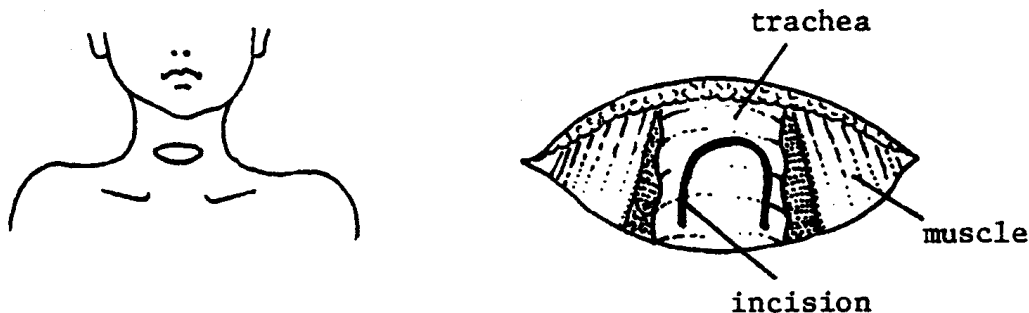
In croup, ideal conditions are provided by the induction of general anaesthesia with halothane, followed by peroral endotracheal intubation. Do NOT attempt this unless you are experienced at intubating children. Give IV or IM atropine. Have the child breathe 100% oxygen by mask, then gradually introduce halothane up to 4% and keep giving this until automatic respiration has been established and no other spontaneous movement is occurring. In a child with severe airways obstruction, induction often takes 10 to 15 minutes, because the tidal volume is very small and little halothane enters the lungs. Intubate the child with an uncuffed peroral tube, it often helps to use a stilette. A smaller tube than usual should be used, the size that just provides an adequate airway.

Weight of child	Size of ETT (mm)
Neonate	2.5
Less than 8 kg	3.0
8-12 kg	3.5
13-18 kg	4.0
19-30 kg	4.5

Have a sterile size 8 FG catheter connected to a sucker ready at the time of intubation. In children with croup, there is usually a large amount of sputum in the trachea; send a swab for culture and sensitivity if these tests are available. Tape the endotracheal tube in securely; do not push it in too far (you can usually feel the tip of the tube in the suprasternal notch if you move the tube up and down a small distance). Tape a stethoscope over the child's heart. Maintain general anaesthesia with oxygen and 1-2% halothane.

TRACHEOSTOMY

1. Put a pad under the child's shoulders to push the trachea forward. Ensure that the child is lying PERFECTLY straight and keep your dissection in the mid-line. Define the position of the thyroid and cricoid cartilages. Make a transverse incision in the skin midway between the cricoid cartilage and the sternal notch using a number 15 blade. From now on, use only blunt dissection with forceps until you reach the trachea. KEEP IN THE MIDLINE. Ligate or diathermy all bleeding vessels. Have an assistant hold the wound open gently on each side with retractors. The thyroid isthmus is a potential hazard, but it is rarely seen in a small child and retracts upwards easily. Once the trachea has been reached, you may have to carefully snip off the fascia in front of it. Feel carefully for the tracheal rings. Make an inverted U-shaped incision through the third and fourth tracheal rings (well below the cricoid cartilage), just big enough to admit the appropriate sized uncuffed tracheostomy tube.



2. Insert the tracheostomy tube. Choose the largest size tube that fits LOOSELY into the trachea. NEVER use a tube that is too large, because it may cause tracheal stenosis, and it will be hard to reinsert when it is removed for cleaning. Some tracheostomy tubes of the correct diameter are too long and have to be cut short so that they do not reach the carina.

Portex tracheostomy tube size	
Weight	(internal diameter in mm)
Neonate	3.0
5 kg	3.5
10 kg	4.0
12 kg	4.5
16 kg	5.0
20 kg	5.5

3. Hold the tube with tapes tied loosely but securely around the neck. Tie the tapes in a bow so that they can be undone quickly if the tube blocks. Loosely close the wound with a skin stitch on each side.

TRACHEOSTOMY CARE

The main risk is obstruction of the tube by mucus:

1. Put 1 ml of sterile saline down the tube every hour. DO NOT USE DEXTROSE OR WATER.
2. Suck out the trachea every hour (after instilling saline). Use at least an 8 FG catheter (5 FG is too small). It is most important that suction be regular and effective. Soak the catheter in aqueous chlorhexidine 1 in 30 (fresh daily) between suctions and rinse it in clean water before use.
3. After 5 days, remove the tracheostomy tube and clean it. The tube should be removed and cleaned every week thereafter. Reinsertion may be difficult the first two or three times until a tract has formed into the trachea. When reinserting the tube, keep the child's head in the midline and the neck extended. Be careful that the tube goes into the trachea, do not force it into the neck or you may make a false passage into the soft tissues.
4. Make sure that all staff understand that if the child suddenly deteriorates, develops chest indrawing and goes cyanosed, they should immediately remove the tube and call a doctor. These symptoms suggest obstruction of the tube.
5. If the tracheostomy has been performed for croup, you can test the amount of laryngeal obstruction by briefly covering the tracheostomy stoma with a gauze swab while the tube has been removed for cleaning: note the amount of stridor and chest indrawing. When this is minimal, leave the tube out and cover the wound with a clean, dry gauze dressing. Do not suture the wound, but allow it to close by itself.

TROPICAL SPLENOMEGALY SYNDROME (TSS)

(HYPERIMMUNE MALARIAL SPLENOMEGALY - HMS)

This syndrome is common in older children and adults in several parts of the developing world. It is due to an abnormal immune response to chronic malaria infection, with circulating complexes of IgM antibody, malaria antibody, malaria antigen and complement. There is probably a genetic predisposition.

TSS is characterised by persistent gross splenomegaly (the spleen often reaches below the umbilicus) in a malarious area, hepatic sinusoidal lymphocytosis, high serum IgM levels and high malarial antibody levels. There may be mild hepatomegaly, anaemia (mainly due to expansion of the plasma volume), mild leucopaenia and mild thrombocytopaenia. Episodes of severe acute haemolysis or overwhelming sepsis may occur.

TREATMENT

The treatment of choice is long-term weekly suppressive doses of antimalarials. Treatment must be regular and prolonged. Response is rare in less than 6 months, and complete regression of splenomegaly may take more than 3 years. Despite the difficulties involved, strenuous efforts should be made to give weekly antimalarials indefinitely to every patient with TSS. Relapse occurs rapidly if antimalarials are stopped, even if splenomegaly has completely resolved.

Splenectomy reduces morbidity, but it is hazardous and does not increase life expectancy. Acute haemolysis responds dramatically to steroids.

REFERENCE

Crane GG. PNG Med J 20:6-14,1977. Pathogenesis of TSS.

TUBERCULOSIS

Tuberculosis is now the commonest infectious disease in the world. It infects one third of the world's population, and in 1995 there were about 9 million new cases with 3 million deaths.

In Papua New Guinea, the incidence of tuberculosis is about 135/100,000 population. In National Capital District, the incidence is about 750/100,000. There are currently about 8,000 newly diagnosed patients in Papua New Guinea per year, one third of whom are children. Tuberculosis is now the second or third commonest cause of hospital admission in coastal areas and ranks in the top 5 causes of death in both children and adults. Improved transport and increased mobilisation of the population has resulted in a rapidly increasing incidence of TB in areas of the country such as the highlands region where previously it was uncommon.

There is very great concern that unless urgent steps are taken, with the Tuberculosis programme of the 80s and 90s having failed, the HIV epidemic will greatly accelerate the spread of TB within the country. Multidrug resistance, currently at a relatively low level, could well become a very serious problem in the near future. The Health Department is following WHO recommendations in introducing DOTS (Directly Observed Treatment Shortcourse). It is imperative that a major effort by all concerned in the prevention and treatment of tuberculosis is supported by adequate financial and manpower resources.

The virtual eradication of TB in Europe between 1945 and 1975 was achieved by the interplay of three factors:

1. Prevention
 - a. The treatment of open cases
 - b. BCG vaccination
 - c. Improved nutrition, improved housing conditions and reduction of other diseases
 - d. The tuberculin testing of cattle and pasteurisation of milk
2. Treatment
 - a. The identification of cases
 - b. Drug treatment, with effective drugs freely available
 - c. Nonspecific treatment (good nutrition and treatment of concomitant diseases)
3. Education
 - a. That TB is an infection: it does not occur by chance, and it is not inevitable
 - b. That TB can be cured by treatment
 - c. That TB can be prevented by BCG vaccination
 - d. That prevention or treatment need not be expensive nor disruptive to a person's life.

THE NATURAL HISTORY OF UNTREATED PRIMARY TB

Initial infection is followed by a period of incubation during which the primary focus forms, the regional nodes enlarge, and organisms escape into the blood stream. At this stage, the body has not developed tuberculin sensitivity. This stage is symptomless and it is not until sensitivity develops 6-8 weeks after infection that fever, malaise and symptoms may occur. The infection in most children, however, is never clinically manifest. At this time, sensitivity phenomena are seen in a small proportion of children. Erythema nodosum is very rare in young children, but phlyctenular conjunctivitis is seen occasionally, and should always alert the clinician to the probability of tuberculosis.

After this initial phase, the clinical syndromes can be divided into those arising from the primary focus, the regional lymph glands, and dissemination. Complications from each of these three situations can occur simultaneously, especially in young children. The risk of serious dissemination is related inversely to age from birth to 10 years, with a rise again at puberty.

The tuberculin test, once positive, will usually remain so except for temporary reversion after measles, streptococcal infection or malnutrition, and permanent reversion with HIV infection.

Progressive enlargement of the primary focus leading to cavitation sometimes occurs, especially in young children (a progressive primary lesion). Pleural effusion, a manifestation of a high degree of

tuberculin sensitivity, is uncommon in children under 5 years. If it occurs, it will do so within 3 months of infection in 25% of cases and within 6 months of infection in 75% of cases.

The regional lymph nodes always enlarge in primary TB, and are bigger in young children. Symptoms and signs are related to lymph glands pressing on a bronchus and producing obstructive emphysema, or collapse with consolidation (a segmental lesion). Bronchial erosion is commoner in early childhood; it usually occurs between 3 and 9 months after infection.

Disseminated disease is more frequent in young children. The liver and spleen are often enlarged, and 20% of children under 1 year old, and 4% of children infected under 5 years old will develop tuberculous meningitis or miliary TB. In 90% of cases, this will develop within 12 months of infection. Bone and joint lesions occur mainly within 3 years of infection. Renal and skin tuberculosis are late complications, mostly occurring five or more years after infection.

Adult or secondary tuberculosis has been considered to be mainly a reactivation of a focus in the lung which originated during the state of dissemination of the primary infection and which remained dormant until the body's resistance became weakened - by malnutrition, corticosteroids, puberty, or intercurrent infection.

Tuberculosis meningitis presents either in an acute or slowly progressive form. It nearly always develops after the rupture, slow or sudden, of a pre-existing meningeal or subcortical caseous lesion occurring either alone or as part of a generalised miliary spread. Many patients have multiple caseous lesions. It sometimes presents as a space occupying lesion - an intracranial tuberculoma. In the young child, the onset of meningitis is usually insidious; there is often neck stiffness, and Kernig's sign is usually absent.

Tuberculosis of the CNS may present in 3 different ways:

1. as meningitis, with fever, headache, vomiting, and neck stiffness. CSF examination shows mainly lymphocytes, the sugar is reduced and the protein raised
2. as encephalitis, where the pathology is essentially massive caseation, with progressive changes in one or both cerebral hemispheres. Little reaction is evident in the meninges. There is fever, headache and vomiting, but no meningeal signs. The CSF contains only a few lymphocytes, but the protein is raised
3. as a space occupying lesion with headache, vomiting, papilloedema with or without localising neurological signs.

Note: The commonest cause of space occupying lesions in children in most tropical countries is tuberculoma (in up to 50% of cases). The tuberculin test is often negative, and x-rays of the chest are usually normal. A skull X-ray may show evidence of raised intracranial pressure.

The clinical picture of tuberculosis depends on the degree of tuberculin sensitivity. When the sensitivity is low, as in young infants and malnourished children, the acute inflammatory component of the pathological reaction is absent or reduced. When it is high, as in older children, the tuberculin test is strongly positive and there is often a pleural effusion or erythema nodosum (though erythema nodosum seems to be uncommon in Papua New Guinean children).

Tuberculosis should always be considered in the differential diagnosis of a pyrexia of unknown aetiology (or PUO). The diagnosis may be helped by doing a tuberculin test, a chest x-ray, fasting gastric aspirates and laryngeal swabs for AFB, and tuberculin testing and chest x-rays of household contacts. Sometimes, a therapeutic trial of antituberculous drugs is necessary.

Other conditions that may cause PUO and malaise without obvious physical signs, apart from wasting, include typhoid, malaria, urinary tract infections, bacterial endocarditis, HIV infection and malignant disease.

In children with a PUO, typhoid should always be suspected. Abdominal signs (loss of appetite, diarrhoea or constipation, and tender tumid abdomen), meningeal signs and chest signs are common. Diagnosis is made by blood culture early in the disease, and later by culture of the stools. The Widal agglutination test becomes positive after the 10th day.

CLINICAL FEATURES SUGGESTIVE OF TUBERCULOSIS IN CHILDREN

Tuberculosis is often difficult to diagnose in children. There are relatively few TB bacilli present and sputum cannot be obtained for examination. The following features suggest the diagnosis:

1. a family history of TB
2. the child comes from an area where TB is common
3. failure to gain weight
4. failure to recover after illness (eg measles or whooping cough)
5. a chronic cough
6. cough with unilateral wheeze
7. cervical lymphadenopathy (consider biopsy)
8. persistent fever (a 4 hourly temperature chart is often helpful in a child suspected of having TB)
9. absence of BCG scar
10. pneumonia that does not respond to antibiotics
11. ascites without other oedema
12. raised intracranial pressure with focal neurological signs
13. signs of meningitis, with CSF lymphocytosis and a high protein
14. signs of spinal cord compression
15. sterile pyuria while not on antibiotics, or painless haematuria
16. a positive 5u PPD Mantoux test.

Often children with features of TB have to be treated without bacteriological proof of the diagnosis. However, the decision to put a patient on potentially dangerous medication for 6 months should never be taken lightly and the clinician should always be able to justify this course of action. The use of the TB score chart (below) often helps in decision making.

MANTOUX TESTING

Use 5u PPD (not 1u) by intradermal injection. The reaction to measure is the area of induration, not the area of erythema. A positive reaction is over 5 mm induration if the child has not had a BCG, and over 15 mm if the child has had a BCG.

Malnourished children often have a negative Mantoux even if they have TB. A person given his or her first BCG vaccination who does not have TB usually starts to react (erythema, swelling, ulceration) at about 5-7 days. A person given a BCG vaccination who has TB (or who has had a previous BCG) has an "accelerated reaction" starting at 2-3 days - even if he or she is malnourished. Providing a BCG has not been given before, BCG vaccination can be used instead of a Mantoux test in malnourished children who are suspected of having TB: a reaction at 2-3 days demonstrates past or present TB (or previous BCG), and a reaction at 5-7 days suggests no TB (past or present) or overwhelming TB.

In "developed" countries, there is usually a bimodal distribution of tuberculin reactions into small reactions caused by non-specific sensitivity (or sensitivity to atypical mycobacteria), and large reactions caused by infection with pathogenic human tubercle bacilli. In "developing" countries, this bimodal pattern is obscured by a high proportion of intermediate reactions. In areas where tuberculosis is prevalent, and where BCG vaccination is practiced, the finding of a positive Mantoux reaction in apparently healthy individuals makes the interpretation of the test very difficult.

THE PAEDIATRIC TB SCORE

Because it is often difficult to diagnose childhood TB with certainty (by positive bacteriology or histology), a TB score has been devised. If a child scores 7 and no other disease is more likely, TB treatment should be started.

Paediatric Tuberculosis Score Chart				
<i>Basic score chart - for each feature decide on score and write in box:</i>				
Feature	0	1	3	Score
Length of illness	Less than 2 weeks	2 to 4 weeks	More than 4 weeks	
Nutritional status	More than 80% line	Between 60-80% line	Less than 60% line	
Family history of TB	No family history	Verbal family history	Sputum +ve family history	
<i>Give score for any other features (if present) as below:</i>				
Significant Mantoux (mm)			Score 3	
Enlarged, painless rubbery neck glands			Score 3	
Night sweats or unexplained fever			Score 2	
Angle deformity of spine			Score 4	
Malnutrition not improved after 1 month treatment			Score 3	
Firm, non-fluid, non-traumatic swelling of joint			Score 3	
Unexplained abdominal swelling (ascites)			Score 3	
Coma for more than 48 hours (with or without convulsions). Send to hospital if possible.			Score 3	
			TOTAL	
If total score is 7 or more and the child has NO OTHER DISEASE MORE LIKELY TO EXPLAIN THE ILLNESS, then commence TREATMENT FOR TUBERCULOSIS according to the child's weight (see STB, p.106).				
Notes:				
1. Beware of OVER SCORING a child, as each item may be wrongly assessed if care is not taken. If you have not used the score chart before, then you must refer to the notes in the Standard Treatment Book.				
2. Always keep a record of the score chart result in the child's notes so that it can be checked later.				

TREATMENT OF TUBERCULOSIS BY SHORT COURSE CHEMOTHERAPY - RATIONALE

At the start of chemotherapy, there is a large number of actively dividing tuberculosis bacilli in the body. These bacteria are killed by the bactericidal action of rifampicin and INAH. At the end of 2 months, all these organism should have been killed. There is also, however, another population of bacteria which multiplies slowly either inside macrophages or inside solid caseous lesions - the so-called "Persisters".

PZA during the intensive phase deals with the bacilli in the macrophages, whilst rifampicin penetrates caseous lesions. Not all of the persisters will have been destroyed by the end of 8 weeks - hence the need for continuation treatment to kill the organisms. The longer the continuation phase, the lower the relapse rate after cessation of treatment. Six months is the shortest duration which gives acceptable relapse rates.

TREATMENT OF TUBERCULOSIS IN PAPUA NEW GUINEA

There are basically two regimens in Papua New Guinea.

1. "Standard" treatment for pulmonary TB, glandular TB, and TB pleural effusion, consisting of:
Intensive phase 2 months daily (rifampicin, INAH, PZA). "A" Treatment
Continuation phase 4 months twice weekly (rifampicin, INAH). "B" Treatment*
ie total of 6 months: 2HRZ + 4H2R2
2. Treatment for "severe" extrapulmonary TB ie TB of CNS, bones, joints, pericardium, abdomen, kidneys:
Intensive phase 2 months daily (rifampicin, INAH, PZA). "A" Treatment
Continuation phase 7 months daily (rifampicin, INAH). "CDT" (Continuous Daily Treatment)
ie total of 9 months: 2HRZ + 7HR.

IT IS VERY IMPORTANT TO NOTE THAT THE DRUG DOSES OF INAH USED IN "B" (twice weekly) and "CDT" (continuous daily treatment) ARE DIFFERENT. BE ABSOLUTELY SURE THAT

YOU AND YOUR STAFF KNOW EXACTLY WHAT REGIMEN THE PATIENT IS FOLLOWING AND ALWAYS CHECK THEY ARE TAKING THE CORRECT DOSES OF DRUGS.

*It is possible that during the life of this book, “B” Continuation Treatment will consist of drugs given three times per week instead of twice.

The use of a fourth drug in the intensive phase of treatment of children with TB

The risk of the spontaneous development of resistance to the standard antituberculous drugs is proportional to the number of organisms present. Most children with primary and progressive primary tuberculosis have relatively few organisms - “paucibacillary”. Treatment with three drugs during the intensive phase is adequate. A few have large numbers of organisms - “multibacillary”. These are children who are sputum or gastric aspirate positive, or who have CXR changes of very severe tuberculous bronchopneumonia. These children are treated with a fourth drug. Those below the age of 7 years are treated with streptomycin, and those 7 years or older are treated with ethambutol for the duration of the intensive phase of treatment (ethambutol can cause a retrobulbar neuritis and optic atrophy. Children less than 7 years will probably not report early changes in vision - so it is not used. Older children taking ethambutol should be checked regularly for visual disturbance).

ANTITUBERCULOUS DRUG DOSES

Rifampicin

10 (8-12) mg/kg daily in “A” and “CDT” regimens, twice weekly in “B” regimen

INAH

5 (4-6) mg/kg daily in “A” and “CDT” regimens (maximum of 300 mg)

15 mg/kg twice weekly in “B” regimen (pyridoxine added if dose >300 mg)

PZA (Pyrazinamide)

25 (20-30) mg/kg daily in “A” regimen

Streptomycin, the fourth drug for children less than 7 years of age with multibacillary disease

15 (12-18) mg/kg daily in “A” regimen

Ethambutol, the fourth drug for children 7 years or older with multibacillary disease

15 mg/kg (maximum) daily in “A” regimen.

INAH PROPHYLAXIS

Untreated, 20% of children under the age of 1 year and 4% of those under 5 years who have a primary infection will develop miliary TB or TB meningitis. Ninety percent of those children who develop miliary TB or TB meningitis do so within 1 year of their initial infection. It is therefore very important to protect young children with a primary infection from these severe forms of tuberculosis. INAH is given in a dose of 5 mg/kg daily for 6 months to children under the age of five years who are household contacts of known sputum positive patients.

Anyone who is asymptomatic but has recently converted from Mantoux negative to Mantoux positive (without the conversion being the result of a recent BCG vaccination) should be given INAH prophylaxis for 6 months (in practice, this situation does not commonly present).

MANAGEMENT OF A NEWBORN BABY WHOSE MOTHER HAS TUBERCULOSIS

This depends on the likely infectivity of the mother.

1. If the mother
 - a. is newly diagnosed
 - b. has been on treatment for less than 2 months
 - c. has been on treatment for more than 2 months but is still sputum positive, or

- d. if there is another sputum positive household contact
 - i. DO NOT give the baby BCG in the newborn period
 - ii. DO give the baby prophylactic INAH for 6 months
 - iii. DO give BCG after prophylaxis is complete
 - iv. DO encourage the mother to look after her child
 - v. DO encourage the mother to breastfeed
2. If the mother
 - a. has been on treatment for more than 2 months
 - b. and is sputum negative
 - i. GIVE BCG.
 - ii. Encourage the mother to breastfeed.

PREVENTION

1. BCG to all newborn. This has been a controversial subject but the combined evidence from around the world and from PNG indicates some protective effect even though this is far from complete.
2. FIND AND TREAT INFECTIOUS CASES. Do sputum smears for AFB on all patients with cough of more than one month's duration.

INDICATIONS FOR STEROIDS IN PAEDIATRIC TB

There are 3 situations in which steroids are probably of benefit:

1. CNS TB (tuberclous meningitis or tuberculoma).
2. Mediastinal compression from tuberculous lymphadenopathy.
3. Pericardial effusion.

In these situations give prednisolone 1-2 mg/kg daily for 4 weeks and then taper the dose over the next 2 weeks.

REFERENCES

- Biddulph J. J Trop Pediatr 34:20-23,1988. Short course therapy in children.
 Biddulph RJ. Pediatr Infect Dis J 9:794-801,1990. Short course chemotherapy.
 Crofton J, Horne N, Miller F. Clinical tuberculosis. The Macmillan Press Limited, 1992. (Essential reading for an understanding of TB).
 Donald PR. Pediatr Infect Dis J 7:469-71,1988. Pyrazinamide for TB meningitis.
 Dooley et al. Clin Infect Dis 25(4):872-887,1997. Steroid therapy in TB.
 Fox W. Bull IUAT 60:40-49,1985. Short course chemotherapy.
 Jacobs RF. Pediatr Infect Dis 4:513-517,1985. Treatment TB in children.
 McMaster P, Ezilio N, Vince J. Ann Trop Paediatr 20(3):223-226,2000. Retrospective study of lymph node tuberculosis in Port Moresby.
 Malavi A. Med Clin N Am 69:315-331,1985. TB meningitis.
 Miller FJW. Trop Doctor 3:66-71,1973. Regional differences in TB.
 Morley D. Paediatric priorities in the developing world. 1973: Chap 15.
 Murtagh K. Arch Dis Child 55:795-799,1980. Mantoux 1 unreliable.
 Shennan DH, Kibel MA. Tuberculosis. In: Stanfield P, Brueton M, Chan M, Parkin M, Waterston T. Diseases of children in the subtropics and tropics. Chap 5:519-551. 4th ed, Edward Arnold.
 WHO. Treatment of tuberculosis: guidelines for national programmes. 2nd edition. Geneva: WHO, 1997.
 WHO. TB: a clinical manual for South East Asia. Geneva: WHO, 1997.

TYPHOID

INTRODUCTION

Typhoid has been endemic in Papua New Guinea for many years, but it is only during the 1980s and 1990s that it has become a major health problem. There have been a couple of major water borne epidemics in educational institutions - but by far the most common means of spread is by the faecal-oral route, the result of poor personal and food hygiene. A typhoid control programme has not been particularly effective, and there are about 7,000 new cases reported each year with a case fatality of about 3%. Typhoid ranks in the top 6 causes of hospital admission in most parts of the country.

CLINICAL PRESENTATIONS

Typhoid may present in a very similar fashion to malaria. Certainly, it should be considered in any febrile child not responding to antimalarial therapy. Common features are:

- headache
- abdominal pain
- abdominal tenderness
- abdominal distension
- constipation
- diarrhoea, with or without blood
- confusion
- talking nonsense
- dehydration
- looking/feeling very sick.

Experienced practitioners are often able to make an accurate diagnosis based on the “typhoid facies” but children with prolonged malaria and with TB also sometimes have a similar appearance. Diarrhoea is as common as constipation in children with typhoid, relative bradycardia is not so prominent a feature as it is in adults and the classic leucopaenia may be absent in children.

Indications of severity include:

1. distended tender abdomen
2. rectal bleeding
3. severe abdominal pain
4. semi/unconsciousness or confusion.

DIAGNOSIS

Do blood, stool and urine cultures and a Widal test, if possible. Positive H and O titres of $>1/160$ or rising titres are virtually diagnostic. Leucopenia is supportive evidence - but is by no means invariable in children - who may have leucocytosis.

TREATMENT

Antibiotic - chloramphenicol

Treatment with chloramphenicol should be continued for three weeks to ensure a low relapse rate. It is usually given parenterally initially and then changed to orally as soon as the child will tolerate it. The quinolone antibiotics such as ciprofloxacin are currently used in many parts of the world - but are very expensive - and no more efficient in their cure rates than chloramphenicol (though the temperature may come down more quickly).

It is important to understand that the patient may remain febrile for up to a week and sometimes longer in spite of antibiotics. Patients with typhoid only rarely make a rapid recovery.

Antimalarials

Patients should be treated as for severe malaria even if the blood slide is negative unless the evidence for typhoid is unequivocal.

Fluids

Attention to fluid balance is important.

If the child is dehydrated:

- rehydrate with ORS or with IV half strength Darrow's solution

If the child is not dehydrated but is vomiting a lot, and the abdomen is distended or tender:

- give intravenous maintenance fluids

If the child is not dehydrated, not vomiting and has a soft abdomen:

- give milk and other fluid orally.

Nutrition

Ensure that the children receive plenty of food and vitamin supplements.

Education

It is important that the parents gain understanding of the fact that typhoid is spread from faeces onto hands and food, and that they appreciate the need for hand washing and food hygiene. Studies from Goroka have indicated that in PNG, it is common for patients having completed treatment for typhoid to continue to excrete the organism for 7 months or longer.

TYPHOID VACCINE

A number of typhoid vaccines are available - but none has been shown to be more than moderately effective. The main educational message and the main preventative strategy must be hygiene.

ULTRASONOGRAPHY

Ultrasonography is a good example of medical high technology which has found an important place in the management of patients of all ages and with a wide variety of medical and surgical disorders. Basic ultrasonography machines are by no means cheap - but they are relatively robust, and if handled with care, should provide years of service. The great advantage of ultrasonography is that it is non-invasive.

There is no doubt that ultrasonography is best done by those with training and experience. However, there can similarly be no doubt that a basic understanding of ultrasound and some hands-on experience of examination of patients with a limited number of paediatric problems is of considerable value to the clinician practicing in a setting without a trained ultrasonographer. Ultrasonography can save the patient invasive investigations, and can rapidly facilitate diagnosis.

Those medical officers responsible for the care of children should try to have some experience in the ultrasonography of:

1. the brain in neonates and infants - diagnosis of hydrocephalus
2. the liver and spleen - to detect abscesses and deviations from normal appearance
3. the peritoneal cavity - to detect the presence of ascites
4. the kidneys - to assess abnormalities in structure (eg cysts - hydronephrosis) and in appearance and size (eg chronic glomerulonephritis)
5. the mediastinum - to detect pericardial effusion.

CLINICIANS ARE STRONGLY ADVISED TO TAKE ANY OPPORTUNITY AFFORDED TO BECOME FAMILIAR WITH THE ABOVE BASIC EXAMINATIONS.

It should be possible for a reasonably experienced clinician to examine the heart with a simple ultrasound machine, looking for VSD and ASD. However, this is not always easy and artefacts are common.

UPPER RESPIRATORY TRACT INFECTION (URTI)

Give outpatient treatment, but tell the parents to return if the child becomes short of breath.

If afebrile

Explain to the parents that the cough is useful to the child in getting rid of the rubbish from the nose which is dripping into the chest. This is the reason that infant cough mixture is no longer in the Standard Treatment Book.

Give routine immunisation if the child is due for it - a child who is well enough to be sent home is well enough to be immunised.

If febrile

Give antimalarials and search for a cause of the fever. There is a high incidence of bacteraemia in children less than 6 months old with a temperature over 39.5 °C per axilla. If the child has otitis media, give amoxycillin orally for 5 days.

It is reasonable to give paracetamol to febrile children over the age of three months.

Cool sponging is not as effective as paracetamol - but it does have some effect and has the advantage of the parents being involved in the child's care.

REFERENCE

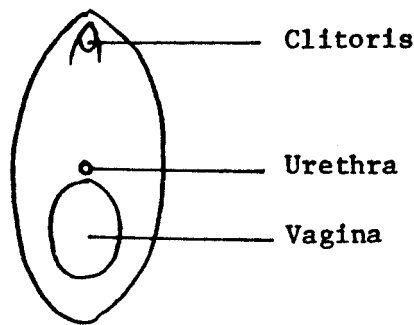
Agbolosu NB, Cuevas LE, Milligan P et al. Ann Trop Paediatr 17(3):283-288,1997. Efficacy of cool sponging versus paracetamol in reducing temperature in febrile children.

URETHRAL CATHETER

This procedure must always be done very carefully because of the risk of causing infection. Prolonged catheterization always results in infection.

1. Clean the genitalia as if you were taking a mid-stream urine sample.
2. Put on sterile gloves and drape the area. Clean the skin with aqueous chlorhexidine.
3. Lubricate the catheter tip with xylocaine jelly:

Under 3 kg:	use a 5 gauge feeding tube
3-8 kg:	use an 8 gauge feeding tube
9-12 kg:	use a 12 gauge catheter.
4. In males: hold the penis vertical. Hold the catheter with sterile forceps about 2 cm from its tip and gently push it along the urethra. After a short distance, resistance may be met at the external sphincter. If this cannot be overcome by gentle steady pressure, use a smaller catheter.
5. In females: in small children, identification of the urethra can be difficult. Hold the labia apart. The urethral orifice lies just anterior to the vagina. Hold the catheter 2 cm from its tip and angle it slightly backwards. Only a few centimetres are required to reach the bladder. Be careful that the catheter does not go into the vagina.



6. For a discussion of catheter care see Lancet 1:89-90,1982.

URINARY TRACT INFECTION AND URINE COLLECTION

INTRODUCTION

Urinary tract infections (UTI) are relatively common. In some series, UTI have been found in around 5% of febrile children. UTI has been found in more than 10% of hospitalised malnourished children. UTI in early childhood, when associated with ureteric reflux, can lead to renal scarring and eventually to hypertension and chronic renal failure in adult life.

Collection of urine is frequently omitted in the workup of a child who is febrile, malnourished or failing to thrive. Blood is collected but urine is not. Part of the reason for this is the mistaken belief that urine collection is a time consuming business and cannot be done on the spot. It is important that this misunderstanding is corrected. It is possible to collect samples of urine from babies and young children easily and relatively quickly.

DIAGNOSIS

Urinary tract infection should be suspected and urine cultures performed in patients with the following symptoms:

- | | |
|-----------------|--|
| Neonates: | <ul style="list-style-type: none">• “septic baby”• prolonged or severe jaundice• failure to thrive• recurrent vomiting |
| Infants: | <ul style="list-style-type: none">• septic or febrile child• changes in voiding pattern• screaming on passing urine• malodorous urine (fishy rather than ammoniacal) |
| Pre-school: | <ul style="list-style-type: none">• febrile illness• abdominal or flank pain• frequency, urgency, dribbling, wetting, dysuria• secondary enuresis• malodorous urine• haematuria |
| Older children: | <ul style="list-style-type: none">• loin or lower abdominal pain• frequency, urgency, dysuria• secondary enuresis• haematuria. |

Urinary tract infection should always be considered in malnourished children.

URINE COLLECTION

Midstream (older child)

1. Clean the genitalia with cotton balls soaked in aqueous chlorhexidine (Savlon):
 - a. in males: retract the foreskin, and swab from the tip of the penis to the base
 - b. in females: swab the vulva and perineum from anterior to posterior
2. Use each swab once, then discard it.
3. Dry the area thoroughly after it has been cleaned.
4. The patient then voids and, after a few seconds of micturition, a sterile specimen bottle is inserted into the stream for a short time.

Clean catch technique

This technique is simple, can be used in neonates, infants and young children, is non-invasive, takes only a few minutes and is almost as sensitive and specific as suprapubic urine collection.

1. Clean the genitalia and peritoneum (have a sterile urine collecting bottle handy - sometimes the cleaning procedure itself will precipitate micturition).
2. Ask the parent or assistant to hold the baby or child upright (in a "standing" position).
3. Either gently percuss suprapubically or gently "bounce" your finger or thumb suprapubically. Continue this for up to a minute. Very often, you will be able to feel the bladder contracting.
4. Collect the midstream part of the urine in the sterile container.

Urine bag (young child)

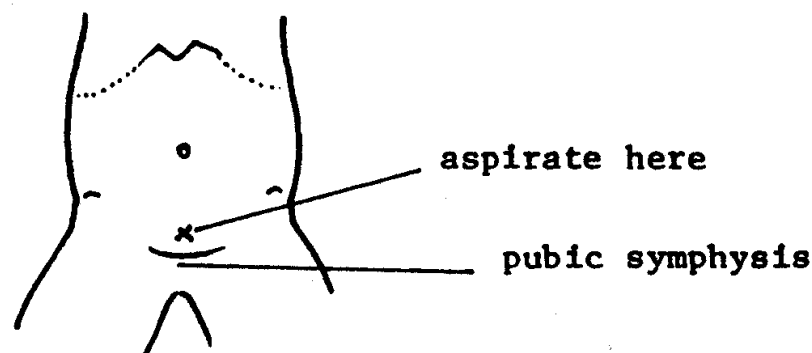
This method often yields a contaminated specimen. It is really not worth the effort in females. In males, it is only useful if care is taken with cleaning the penis and the bag is removed and the urine cultured immediately as soon as voiding has occurred.

1. Clean and then dry the genitalia as above.
2. Remove the paper to expose the adhesive on a plastic urine collection bag, insert the penis through the opening in the bag and press the adhesive onto the skin. As soon as urine has been passed, remove the bag and seal it by folding the two adhesive surfaces together.
3. A contaminated specimen is suggested by:
 - a. the absence of white cells (but the presence of white cells does not prove infection)
 - b. the presence of epithelial cells
 - c. scanty growth (less than 100,000 organisms per ml) or mixed growth.
4. The presence of pus cells with no growth suggests prior antibiotic treatment or TB.

Suprapubic aspiration (bladder tap)

This is the most reliable means of obtaining uncontaminated urine for micro and culture. ONLY attempt it when the bladder is palpable, or when the child has not voided for at least one hour.

1. Have an assistant FIRMLY hold the child supine with legs together and extended.
2. Prepare the skin over the lower abdomen with 70% alcohol. Do not use local anaesthetic.
3. Insert a 23 gauge needle on a 5 ml syringe perpendicularly to the skin 1 cm above the pubic symphysis in the midline (this is usually just above the skinfold).



4. The depth of insertion varies with the size of the child and how fat he or she is. Do not insert the needle too far.

Under 2 kg:	about 1 cm
2 - 4 kg:	about 1.5 cm
4 - 6 kg:	about 2 cm
6 - 10 kg:	about 3 cm

5. Aspirate with the syringe. If you do not get urine at first, SLOWLY withdraw the needle, aspirating as you withdraw.
6. If you do not get urine on the first attempt, you can try ONCE more. Check that the needle is patent first. Insert it 1 cm further than the first time. If you are still unsuccessful, give the child a big drink and wait another hour before trying again.
7. If the specimen cannot be examined immediately, store it in the bottom of the refrigerator (do not freeze it).

URINARY TRACT INFECTION - TREATMENT

NEONATES

Neonatal urinary tract infection is usually associated with pyelonephritis. Treat with parenteral antibiotics - ampicillin and gentamicin for 10 days would be a reasonable treatment.

INFANTS AND OLDER CHILDREN

This depends on how sick the child is:

1. If the child is toxic and there is a possibility of septicaemia:
 - a. treat with parenteral antibiotics at least until the child improves - and then change to oral treatment, depending on the culture and sensitivity results. Treat for a minimum of 10 days.
2. If the child is not toxic:
 - a. treat with oral antibiotics. Either amoxycillin or cotrimoxazole would be reasonable. Treat for one week.

Note: It is important that children under 5 years of age are continued on low dose prophylactic antibiotic until ureteric reflux has been either found or excluded by a micturating cystourethrogram (see below).

Children with established vesicoureteric reflux should be treated with long-term prophylactic antibiotics (eg cotrimoxazole, amoxycillin, or nitrofurantoin, given at night) and regular urinary cultures, until the reflux - which is likely to improve over time - no longer occurs.

Repeat a urine culture one week after stopping treatment.

FURTHER INVESTIGATION

Because about one in 3 children with UTI have ureteric reflux, and because reflux nephropathy is a significant cause of chronic renal failure and hypertension, and because UTI may be associated with other abnormalities of the urinary tract (obstruction, calculi, anatomical features such as bladder diverticulae or duplex ureters), it is very important that a child with confirmed UTI is investigated appropriately. There have been a number of suggestions as to which children should receive which investigations - but in Papua New Guinea, it is reasonable to adopt the following approach:

1. All children with confirmed UTI should have ultrasonography of the upper and lower renal system. This can be done at any time during the infection or soon afterwards.
2. If any abnormalities in the kidneys are detected at any age, an IVP should be performed to assess renal function.
3. Children under the age of 5 yrs should have a micturating cystourethrogram to evaluate the possibility of ureteric reflux. This is usually done 4-6 weeks after the infection has subsided (since UTI itself may cause reflux).

REFERENCE

Hoberman A, Wald ER. *Pediatr Infect Dis J* 16:11-17,1997. Urinary tract infections in young febrile infants.

URINE TESTS

All hospitals should have a supply of Multistix (or equivalent) dipstick urine testing strips. The test strip should have tests for glucose, albumen, blood, bilirubin and urobilinogen as the minimum requirement. Some strips contain tests for urinary pH, specific gravity, nitrite and leucocyte esterase (these latter two tests are helpful in the diagnosis of urinary tract infection). Always check the expiry date on the Multistix (or other test strips).

In the absence of test strips, the older laboratory tests can be used. All doctors should be able to examine a urine sample microscopically and to interpret the findings.

LABORATORY TESTS

Albumin

Boil the urine in a test tube, and if it turns cloudy, add 3 drops of dilute acetic acid. Albumin is present if it stays cloudy. Allow the test tube to stand. Over 1/8th solid is significant albuminuria.

Bilirubin

Dark urine may be due to dehydration (concentrated urine), bilirubin or haemoglobin. Shake the urine vigorously in a closed test tube. If the froth on top is yellow, bile is present. If the froth is white, bile is not present.

A jaundiced patient with no bile in the urine has haemolysis (p.177), and should be admitted for investigation. A patient with bile in the urine who does not look sick probably has hepatitis (p.178), and can be sent home.

Blood

Many patients say that their urine contains blood when it is merely dark. Always check the urine yourself by microscopic examination for red blood cells. Up to two RBC per HPF is normal.

Haemoglobin

Test strips do not distinguish between the presence of blood and haemoglobin.

If the urine is red but red cells are not present on microscopy, and there is a positive stix test for blood, haemoglobinuria is highly likely.

If test strips are not available, add 3 drops of dilute acetic acid to the urine and allow it to stand. Haemoglobinuria is probably present if there is no precipitate and the urine still looks red after one hour. Haemoglobinuria is caused by blackwater fever (p.200), G6PD deficiency (p.140), incompatible blood transfusion, severe burns, snakebite, septicaemia, and severe exertion.

Microscopy

Put a drop of well-mixed uncentrifuged urine on a clean counting chamber and examine for white cells, red cells, epithelial cells, casts, crystals and candida albicans (monilia). Use the 40x objective to count white cells.

1. Fuchs-Rosenthal Chamber: count the white cells in 5 of the 16 large squares.
2. Neubaur Chamber: count the white cells in 5 of the 9 large squares AND MULTIPLY BY TWO (as for CSF, p.70). This gives the number of white cells per cmm:

Less than 10 WBC/cmm:	normal
10 - 30 WBC/cmm:	moderate
Over 30 WBC/cmm:	heavy

Sugar

Add 8 drops of urine to 5 ml of Benedict's solution in a test tube and boil. Alternatively, put 5 drops of urine and 10 drops of water in a test tube and add one Clinitest tablet. Blue means no sugar. Green is +, yellow ++ and brown or orange +++. Benedict's solution or Clinitest tablets detect any reducing sugar (glucose, galactose, lactose, fructose, maltose or pentose). False positives may be caused by aspirin, massive doses of penicillin, chloral hydrate, streptomycin, isoniazid, probenecid, creatinine or uric acid. Clinistix and Boehringer glucose test strips detect ONLY glucose.

A positive test with Benedict's or Clinitest, but a negative test strip suggests the presence of galactose, fructose, maltose, pentose or a drug.

A negative test with a Clinitest tablet but a positive Benedict's test or test strip suggests that only a small amount of sugar is present.

The same tests (Benedicts and Clinitest) can be used for testing liquid stool for reducing substances.

VAGINAL DISCHARGE (PREPUBERTAL)

See also Child Abuse, p.72.

Vaginal bleeding is common in the first few days of life (neonatal “period” due to oestrogen withdrawal). A mucoid vaginal discharge is normal in neonates. Prepubertal girls are susceptible to vulvo-vaginitis because of the proximity of the vagina to the anus, the high pH and atrophic epithelium of the vagina, and poor hygiene (wiping the perineum from back to front, and insertion of contaminated fingers or foreign bodies into the introitus). Sexual abuse of young girls is common in Papua New Guinea (as common as it is in other countries) and you should always be suspicious of this.

1. Perform a careful clinical examination for a foreign body.
2. Take a swab of the discharge for culture.
3. Give a dose of albendazole (for pinworm).
4. Educate the child to clean the perineum from front to back once with each sheet of paper, and to remove all remnants of toilet paper.

If the culture grows gonococcus:

1. Take blood for a VDRL.
2. Give a single dose of augmentin, ampicillin and probenecid (p.348).
3. Attempt to find the person who is abusing the child.
4. Repeat the culture after one week.

Gonococcal infection usually causes a profuse purulent discharge with pruritis, swelling and dysuria. There is inflammation and excoriation of the vulva and vagina.

A non-sanguinous discharge that does not grow gonococcus:

This is usually due to coliform infection, a low-grade chronic infection, often with a foul-smelling discharge. If the discharge persists despite improved hygiene:

1. Examine the child under anaesthesia for a foreign body.
2. Using a small-orifice 2 ml syringe, inject 1 ml of oestrogen cream into the vagina BD for 10 days.
3. Give appropriate oral antibiotic therapy.
4. Instruct mother to bath the child’s genitalia with salt water.

A blood-stained discharge:

This is usually due to the presence of a foreign body or to Shigella or group A beta haemolytic Streptococcus infection.

Profuse, purulent, bloody, foul-smelling discharge is pathognomonic of a foreign body. Small objects can often be removed by irrigation using a soft rubber catheter. If this fails, instrumental removal under anaesthesia will be required.

Shigella infection should be treated with cotrimoxazole for 7 days (check the sensitivity, if possible). Beta haemolytic streptococcus infection should be treated with amoxycillin for 10 days.

WHEEZING

See also Asthma (p.41).

All that wheezes is not asthma! The common causes of wheezing are:

Under 12 months of age	Over 12 months of age
Acute viral bronchiolitis (RSV infection)	Asthma
Chlamydia trachomatis pneumonia (p.314)	Foreign body (DO NOT FORGET THIS)
Cardiac failure	Tuberculosis
Inhalation bronchitis and bronchiolitis	Cardiac failure
	Tropical eosinophilia

Occasionally, wheezing may occur as part of a sensitivity reaction.

If the wheeze is severe:

1. admit, give oxygen, and treat cardiac failure or pneumonia, if present
2. obtain a CXR if you are in doubt as to the cause of the wheeze
3. it may help to stop milk feeds and give IV fluid
4. make a decision about a trial of nebulised salbutamol. This sounds easy, but most children less than 18 months old do not respond to salbutamol - though a few do. In addition, in severe bronchiolitis, the use of salbutamol may cause desaturation, probably due to shunting. If you decide to use salbutamol, use either a nebuliser or a Metered Dose Inhaler and spacer. If there is no response (or if there is a deterioration), do not persist.

WHITE CELL COUNT OVER 40,000 PER CMM

A high white cell count (over 40,000 per cmm) not due to leukaemia is known as a leukaemoid reaction.

POLYMORPHS

1. A high white cell count with predominant neutrophilia is a well recognised entity. Many children with very high WCC have severe pneumonia, and many of them die. In a study from Goroka Hospital:

- a. WCC 20,000 - 40,000: one quarter of the children died
- b. WCC over 40,000: three quarters of the children died.

If there is no improvement in the child's condition after 24 hours of standard treatment with chloramphenicol, it may be best to treat the patient with intravenous benzyl penicillin 3 hourly and intravenous gentamicin. If the child has meningitis, continue with chloramphenicol or, if it is available, give a third generation cephalosporin.

2. Other causes of a very high WCC are TB, sepsis, post splenectomy, acute haemolysis and lymphoma.
3. Myeloid leukaemia is suggested by a high proportion of myeloblasts, anaemia and thrombocytopaenia. Do a bone marrow aspiration (p.57).

LYMPHOCYTES

1. Pertussis.
2. Other causes are TB, infectious lymphocytosis (self-limiting disease with fever, rash, vomiting, diarrhoea and meningo-encephalitis), infectious mononucleosis (rarely diagnosed in Papua New Guinea) and carcinomatosis.
3. Lymphatic leukaemia is suggested by a high proportion of lymphoblasts, anaemia and thrombocytopaenia. Do a bone marrow aspiration (p.57).

REFERENCE

Shann F. PNG Med J 22:55-8,1979. Leukaemoid reactions in Goroka children.

YAWS

INTRODUCTION

Yaws eradication campaigns in the 1960s were thought to have all but eliminated this disease. It has, however, made a resurgence and is quite commonly seen in several areas of the country.

Yaws is caused by infection with *Treponema pertenue*, a spirochaete morphologically identical to *T. pallidum* (which causes syphilis). Spread is by direct contact (non venereal). Skin, subcutaneous tissues and bone are affected. Only the primary and secondary lesions are infectious: they contain large numbers of organisms. The incubation period is 2-8 weeks.

PRIMARY YAWS (ULCERATIVE)

This is a small erythematous macule which develops into one or more papules surrounded by erythema. It may ulcerate. It is usually painless, but it may itch, and scratching can cause secondary infection. It heals over weeks or months, and may persist into the secondary period. The commonest site is on the legs or buttocks, but there may be no history of a primary lesion.

SECONDARY YAWS (GRANULOMATOUS)

Secondary lesions come on a few weeks to months after the primary one appears, and are similar but smaller. They are single or multiple, grouped around the region of the primary yaw or scattered over the body. There may be successive crops. Common sites are the face, axillae, vulval cleft, anus or buttocks, but not the scalp. Lesions on the soles or palms cause painful ulcers. Less commonly, there are macules, papules, areas of desquamation, or circinate “fungal” lesions.

Secondary bone lesions cause painful, often oedematous, deformities of the shafts of bones, particularly the tibia, fibula, ulna or fingers. There is anterior bowing of the legs or swelling of the fingers. X-ray shows focal cortical rarefactions and periosteal changes with deposition of new bone. Later, there is dense bone formation.

TERTIARY YAWS (GUMMATOUS)

Tertiary lesions appear some years later in untreated cases; they are gummatous granulomata which cause ulceration of the skin and deeper tissues, including bone. There is initially a subcutaneous nodule, which may form an indolent ulcer with secondary infection and severe scarring. Tertiary nodules may occur in the neighbourhood of large joints, particularly the knees.

Tertiary bone lesions may cause considerable destruction and deformity, particularly with involvement of the nasal process of the maxillary bones and hard palate (“gangosa”). The result resembles severe cleft lip and palate. Skull or long bones may be affected. X-ray shows well-defined oval cortical rarefactions that may contain spicules of dead bone.

DIAGNOSIS

Clinical diagnosis is usually easy, particularly if there are other cases in the area. Confusion may occur with syphilis, mycobacterium ulcerans, leprosy and tropical ulcers. The VDRL is positive. *Treponema* are seen by dark-ground illumination of fluid from primary or secondary lesions. Remove the crust, and clean the lesion with saline if there is secondary infection. Lightly scrape the lesion, and gently express the fluid for examination. The fluid can be transported by running it into a capillary tube, which is then strapped to a tongue depressor with tape after the ends of the tube have been sealed with putty.

TREATMENT

1. Give a single injection of IM benzathine penicillin:

Less than 20 kg:	0.6 million units
20 kg or more:	1.2 million units
2. If you do not have benzathine penicillin, give amoxycillin TID for 10 days.
3. Survey the area to determine the prevalence of yaws:

Under 5% prevalence:	treat the immediate family and close contacts
5 - 19% prevalence:	treat all the children in the village, and any adults with clinical disease
20% or more:	treat the entire community.

REFERENCES

Garner MF. PNG Med J 16:189-93,1973. Yaws in New Guinea.
Hackett C. An international nomenclature of yaws lesions, Geneva: WHO, 1957 (an excellent colour atlas of yaws).

DRUG DOSES - HOSPITAL

See also Anaesthetics - Drug Doses p.27, Neonates - Drug Doses p.244.

1. **WEIGH EACH CHILD ACCURATELY, OTHERWISE YOU CANNOT USE THE TABLE**
 - a. **ZERO THE SCALE FIRST**
 - b. **WRITE THE WEIGHT IN THE SCALE BOOK (AND HISTORY, IF ADMITTED)**
 - c. **PLOT THE WEIGHT ON THE ROAD TO HEALTH CHART - TREAT IF UNDERWEIGHT**
2. **CHECK THAT THE DRUG THAT YOU ARE GOING TO USE IS THE SAME TYPE AND STRENGTH AS THE ONE LISTED ON THE TABLE. ALSO CHECK THE CONCENTRATION AND VOLUMES AFTER ADDITION OF WATER FOR INJECTION.**
3. **FOR ACCURATE MG/KG DOSES, REFER TO “DRUG DOSES” BY FRANK SHANN (AVAILABLE FROM PAEDIATRIC SOCIETY OF PNG).**

*DENOTES CATEGORY C (SPECIALIST ONLY) DRUG, () DENOTES DRUG IS DILUTED

Drug Doses - Hospital	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
ADRENALINE. Amp 1/1000 in 1 ml, 0.01 ml/kg SC	ml	-	-	-	0.2	0.25	0.3	0.4	0.5
ALBENDAZOLE. Tab 200 mg (must be crushed or chewed)	tab	1	2	2	2	2	2	2	2
ALDOMET - see METHYLDOPA									
AMINOPHYLLINE (toxicity: tachycardia, vomiting, headache)									
• Amp 250 mg/10 ml, ¼ ml/kg (maximum 10 ml) IV over 1 hour (put in burette) every 6 hours	ml	-	1.5	2	3	4	6	8	10
• Elixir 25 mg/5 ml. 6 hourly maintenance dose. Start with 5 mg/kg, increase to 7.5 mg/kg if poor control and no toxicity	ml	-	6	10	15	-	-	-	-
• Tab 100 mg. 6 hourly maintenance dose. Start with low dose, increase if poor control and no toxicity	tab	-	¼	½	¾	1	1	1	1
AMODIAQUINE (INFANT CAMOQUIN). Tab 100 mg						Use chloroquine Use chloroquine			
• Treatment: 10 mg/kg daily for 3 days oral	tab	½	1	1½	1½				
• Prophylaxis: 5 mg/kg weekly oral	tab	¼	½	1	1				
AMOXYCILLIN 25-50 mg/kg/dose									
• Vial 250 mg (add 2 ml sterile water) IM or IV 6 hourly.....	ml	2	3	4	5	6	7	8	8
• Cap/tab 250 mg TID oral	tab	½	½	¾	1	1½	2	2	2
• Susp 125 mg/5 ml TID oral	ml	2.5	5	7.5	10	-	-	-	-

Drug Doses - Hospital	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
AMPICILLIN 25-50 mg/kg 6 hourly									
➤ Vial 250 mg (add 1 ml sterile water), or 500 mg (add 2 ml sterile water)									
• meningitis, osteomyelitis, septicaemia (give oral probenecid as well): 50 mg/kg 6 hourly	ml	1	2	3	3	4	4	4	4
• pneumonia and other infections: 25-50 mg/kg 6 hourly IM or IV.....	ml	1	1.5	2	2	2	2	2	2
➤ *Syrup 125 mg/5 ml, 1 ml/kg 6 hourly oral	ml	4	8	12	-	-	-	-	-
➤ *Cap 250 mg, 6 hourly oral	cap	-	-	1	1	2	2	2	2
ARTEMETHER	See Treatment for Severe Malaria (B1), p.198								
ARTESUNATE	See Treatment for Severe Malaria (B1), p.198 See Treatment for Treatment Failure Malaria (C1), p.199								
ASPIRIN									
• Tab 300 mg, antipyretic	tab	-	-	-	-	-	1½	2	2
• For arthritis and rheumatic fever ONLY, 30 mg/kg 6 hourly oral	tab	-	½	1	1½	1½	2½	3	4
ATROPINE. Amp 0.6 mg/ml									
• Add 3 ml sterile water	ml	(0.1 ml/kg)		-	-	-	-	-	-
• Undiluted 0.02 mg/kg	ml	-		0.25	0.25	0.5	0.75	0.75	1
BACTRIM - see COTRIMOXAZOLE									
BENADRYL - see DIPHENHYDRAMINE									
CAMOQUIN - see AMODIAQUINE									
CAPTOPRIL. Tab 25 mg 0.1-1 mg/kg/dose TID	tab	-	-	¼	¼	½	½	1	1
*CARBAMAZEPINE (TEGRETOL). Tab 200 mg oral. 10 mg/kg BD	tab	-	¼	½	1	1	1½	2	2
CEFOTAXIME. 50 mg/kg IV or IM 6 hourly	ml	Dilute as per manufacturer's instructions							
CEFTAZIDINE. 50 mg/kg IV or IM 6 hourly	ml								
CEFTRIAZONE. 50 mg/kg IV or IM 12 hourly	ml								
CHLORAL HYDRATE 150 mg/5 ml, 25-50 mg/kg/dose	ml	5	10	15	20	20	20	20	20
CHLORAMPHENICOL. 25 mg/kg (maximum 500 mg in children) 6 hourly:									
• Vial 1g (add 4 ml sterile water) IM or IV 6 hourly	ml	0.5	6-7kg: 0.75 8-9kg: 1	1.5	2	2.5	3	3	4
• Susp 125 mg/5 ml, 6 hourly oral	ml	1 ml/kg	1 ml/kg	1 ml/kg	1 ml/kg	-	-	-	-
• Cap 250 mg, 6 hourly oral	cap	-	-	1	1	2	2	3	4

Drug Doses - Hospital	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
CHLOROQUINE (NIVAQUINE)									
➤ Treatment									
• tab 150 mg base, 10 mg/kg oral daily for 3 days	tab	¼	½	1	1	1½	2	3	3-4
➤ Prophylaxis									
• tab 150 mg base, 5 mg/kg oral weekly	tab	¼	¼	½	½	1	1	2	2
CHLORPROMAZINE (LARGACTIL). Half dose if dehydrated									
➤ Amp 50 mg/2 ml, IM BD									
• sedation, vomiting: 1 mg/kg	ml	-	0.25	0.5	0.5	0.75	1	1.5	2
• tetanus: 5 mg/kg (0.2 ml/kg)	ml	0.75	1.5	2	3	4	6	7	8
➤ Tab 25 mg, oral BD									
• sedation, vomiting: 1 mg/kg	tab	-	¼	½	½	1	1	1½	2
• tetanus: 5 mg/kg	tab	1	1½	2	3	4	6	7	8
CIMETIDINE.									
• Amp 100 mg/ml, IV 10 mg/kg 6 hourly	ml	0.5	0.75	1	1.5	2	2	2	2
• Tab 200 mg, oral 6-8 mg/kg 6 hourly	tab	¼	¼	½	½	1	1	1½	2
*CLOFAZIMINE (LAMPRENE). Tab 100 mg daily	tab	See p.186		1	1	1	1	1	1
*CLOXACILLIN. 25-50 mg/kg 6 hourly. Give oral probenecid as well.									
• Vial 250 mg (add 1 ml sterile water), IV (in burette) or IM 6 hrly	ml	0.5	1	1.5	2	3	4	4	4
• Cap 250 mg, 6 hourly oral	cap	-	-	1	1	1	1	2	2
COTRIMOXAZOLE (SEPTRIN, BACTRIM). (Trimethoprim 80 mg + sulphamethoxazole 400 mg). High dose for chronic pneumonia is trimethoprim 5 mg/kg QID (see p.314).·									
• Normal dose (trimethoprim 4 mg/kg BD), BD oral	tab	¼	½	½	1	1½	2	2	2
• Susp 40 mg/200/5 mg	ml	2.5	5	7.5	10	-	-	-	-
DAPSONE (DDS - see also MALOPRIM). Tab 50 mg									
➤ Daily for 12 weeks	tab	½	½	½	½	1	1½	1½	2
➤ Then, either:									
• twice a week (supervised)	tab	1½	1½	1½	1½	3	5	5	6
• or daily at home	tab	½	½	½	½	1	1½	1½	2
*DEXAMETHASONE (DECADRON). Amp 4 mg/ml									
For cerebral oedema, IV, 6 hourly	ml	0.25	0.25	0.5	0.5	1	1	1.5	2
DIAZEPAM (VALIUM) 10 mg/2 ml, 0.2 mg/kg (0.04 ml/kg)·									
• Slow IV	ml	0.25	0.5	0.5	0.75	1	2	2	2
• Rectal	ml	0.25	0.5	1	1.5	2	2	2	2

Drug Doses - Hospital	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
DIETHYLCARBAMAZINE (HETRAZAN). Tab 50 mg									
• Filariasis: 2 mg/kg TID for 3 weeks	tab	¼	¼	½	½	1	1½	2	2
• Tropical pulmonary eosinophilia: 4 mg/kg TID for 5 days	tab	¼	½	1	1½	2	3	4	4
DIGOXIN (LANOXIN) 6 hourly for 3 doses:									
• Elixir 50 microgram/ml oral	ml	2	3	5	-	-	-	-	-
• Tab 0.25 mg oral	tab	-	-	-	1	1	1	1	1
Then maintenance if needed daily:									
• Elixir 50 microgram/ml oral	ml	1	1.5	2.5	3	-	-	-	-
• Tab 0.25 mg oral	tab	-	-	-	-	1	1	1	1
DILANTIN - see PHENYTOIN									
DIPHENHYDRAMINE (BENADRYL). Elixir 10 mg/5 ml, 8 hourly oral	ml	2	4	6	8	10	Use promethazine		
DOPAMINE		See Septic Shock, p.346							
ENALOPRIL 0.2-1 mg/kg daily. Tab 10 mg	tab	-	-	¼	¼	½	½	1	1
*ERYTHROMYCIN, 6-12.5 mg/kg 6 hourly									
• Susp 125 mg/5 ml, 6 hourly oral (0.5 ml/kg)	ml	2	3	5	7.5	10	-	-	-
• Tab/Cap 250 mg, 6 hourly oral	tab	-	-	-	1	1	1	2	2
ETHAMBUTOL. Tab 400 mg, 15 mg/kg daily oral	tab	-	-	-	1	1	1½	1½	2
*FANSIDAR (Pyrimethamine 25 mg + sulfadoxine 500 mg). Tab, single dose	tab	-	½	1	1	1½	2	2	3
FERRIC MIXTURE, FERROUS TABS - see IRON									
FLAGYL - see METRONIDAZOLE									
FOLIC ACID. Tab 5 mg									
• Malnutrition, daily oral	tab	1	1	1	1	1	1	1	1
• Anaemia, weekly oral	tab	1	1	1	1	1	1	1	1
FRUSEMIDE (LASIX). Amp 20 mg/2 ml, 1-2 mg/kg IM or IV	ml	0.5	0.75	1	1.5	2	2	2	2
FURADANTIN - see NITROFURANTOIN									
*GENTAMICIN. 5-7.5 mg/kg IM or IV daily	ml	5-7.5 mg/kg daily dose							
HETRAZAN - see DIETHYLCARBAMAZINE									
HYDROCHLOROTHIAZIDE (ESIDREX). Tab 50 mg									
• Oedema: 1-2 mg/kg daily oral	tab	-	-	¼	¼	½	½	¾	1
• Hypertension: 0.5-1 mg/kg daily oral	tab	-	-	¼	¼	¼	¼	½	½
HYDROCORTISONE (SOLU-CORTEF). Amp 100 mg/2 ml, 4 mg/kg IV 4 or 6 hourly	ml	0.5	1	1	1.5	2	2	3	4

Drug Doses - Hospital	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
IMFERON - see IRON									
IPECACUANA SYRUP, give once, oral	ml	-	15	15	15	15	15	15	15
IRON									
• Amp 2 ml or 5 ml (Imferon). Do not give more than 5 ml IM per day. Total dose, IM	ml	3	5	7	10	15	20	20	20
• Tab (Ferrous sulphate or Fefol) 200 mg oral	tab	-	¼ daily	½ daily	½ daily	1 daily	1 BD	1 BD	1 BD
• (If iron suspension - Sulphate Fumarate or Gluconate - is available, the dose is based on 6 mg/kg of elemental iron.)									
ISONIAZID (INAH)									
• Tab 100 mg, 10-20 mg/kg daily for 2 months	tab	½	1	1½	2	3	3	3	3
• THEN 15-20 mg/kg twice a week, oral	tab	1	1½	2½	3	4½	6	7½	9
ISOPRENALINE		See Septic Shock, p.346							
KETAMINE (KETALAR) 500 mg/10 ml									
• IV 2 mg/kg (0.04 ml/kg) SLOWLY	ml	0.04 ml/kg		0.5	0.75	1	1.5	2	3
• IM: first dose 10 mg/kg (0.2 ml/kg)	ml	0.75	1.5	2.5	3.5	5	7.5	7.5	10
• further doses 5 mg/kg (0.1 ml/kg)	ml	0.5	0.75	1.5	2	2.5	4	4	5
LAMPRENE - see CLOFAZIMINE									
LANOXIN - see DIGOXIN									
LARGACTIL - see CHLORPROMAZINE									
LASIX - see FRUSEMIDE									
LYTIC COCKTAIL. Pethidine 50 mg (1 ml) + promethazine 25 mg (1 ml) + sterile water 8 ml. MAXIMUM dose 0.3 ml/kg IV	ml	0.3 ml/kg			5	7	10	10	10
MAGNESIUM HYDROXIDE MIXTURE (MILK OF MAGNESIA), daily	ml	-	2	5	10	10	15	15	20
MALOPRIM (pyrimethamine 12.5 mg + dapsone 100 mg). Tab, weekly oral	tab	-	-	-	-	½	1	1	1
MANNITOL 20%. 1.25-2.5 ml/kg (0.25-0.5 g/kg) IV over 30 minutes. May be repeated every 6 hours	ml	10	20	30	40	60	80	110	125
MEBENDAZOLE. Tab 100 mg BD for 3 days	tab	½	½	1	1	1	1	1	1
METHYLDOPA (ALDOMET). Tab 250 mg, 5-10 mg/kg	tab	-	¼	¼	½	½	1	1½	2
METRONIDAZOLE (FLAGYL)									
• Tab 200 mg or 250 mg, 15-20 mg/kg TID for 5 days, oral	tab	½	1	1½	2	2	3	3	4
• Suppos 500 mg (for half dose, split lengthways), TID into rectum	suppos	½	½	1	1	2	2	2	2
MOGADON - see NITRAZEPAM									

Drug Doses - Hospital	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
MORPHINE. Amp 10 mg/ml (NOT 15 mg/ml), 0.2 mg/kg IM 6 hourly	ml	-	-	0.25	0.5	0.5	0.75	1	1.5
NITRAZEPAM (MOGADON). Tab 5 mg. For epilepsy 0.2-0.4 mg/kg. Start daily, slowly increase to TID oral	tab	¼	½	½	1	1	1	1	1
*NITROFURANTOIN (FURADANTIN) 1-2 mg/kg 6 hourly									
• Susp 25 mg/5 ml, 6 hourly oral	ml	1.5	3	5	7	10	-	-	-
• Tab 100 mg, 6 hourly oral	tab	-	-	-	-	½	¾	1	1
NIVAQUINE - see CHLOROQUINE									
NYSTATIN. Susp 100,000 u/ml, 6 hourly oral	ml	1	1	1	1	1	1	1	1
OMNOPON and SCOPOLAMINE - see PAPAVERETUM and HYOSCINE									
(OXY)TETRACYCLINE. Do not give tetracycline to children weighing less than 20 mg (except for cholera). Cap 250 mg oxytetracycline, 5-10 mg/kg QID oral	cap	-	-	-	-	1	1	1-2	2
PAPAVERETUM 20 mg and HYOSCINE 0.4 mg (OMNOPON and SCOPOLAMINE). Amp 1 ml, 0.02 ml/kg IM	ml	-	-	0.25	0.5	0.5	0.75	1	1
PARACETAMOL. Susp 10-15 mg/kg/dose 6 hourly	ml	-	2.5	5	5	7.5	10	-	-
PARALDEHYDE. Amp 5 ml, 0.2 ml/kg IM with glass syringe or NG	ml	1	1.5	2.5	3	4	5	7.5	10
PENICILLIN									
➤ Benzathine. Vial 2,400,000 u (add 5 ml sterile water). Yaws. IM stat	ml	1	1	2	2	3	4	5	5
➤ Benzyl (Crystalline). Vial 1,000,000 u (add 2 ml sterile water):									
• septicaemia, osteomyelitis, very severe pneumonia: IV or IM 3 hourly	ml	1	1	2	2	3	4	4	4
• pneumonia and other infections: IM or IV 6 hourly	ml	0.5	0.5	1	1	1.5	2	2	2
➤ Phenoxyethyl (Pen V):									
• rheumatic fever prophylaxis: tab 250 mg oral, daily or BD	tab	-	-	-	1	1	1	1	1
➤ Procaine, aqueous. Vial 3,000,000 u (add 10 ml sterile water), IM daily	ml	1	1.5	2	2	3	3	3	4
PETHIDINE. Amp 50 mg/ml or 100 mg/2 ml									
• Usual dose: 1 mg/kg IM 6 hourly	ml	-	-	0.25	0.5	0.5	0.75	1	1
• For tetanus or for very severe pain: 2 mg/kg IV or IM	ml	-	0.25	0.5	0.75	1	1.5	2	2
PHENERGAN - see PROMETHAZINE									

Drug Doses - Hospital		Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
PHENOBARBITONE										
➤ Stat: 15-20 mg/kg as:										
• amp 200 mg/ml IM	ml	0.25	0.5	0.75	1	1	1	1	1.5	
• or tab 30 mg oral	tab	2	3	5	6	7	7	7	8	
➤ Then: 5 mg/kg daily as:										
• tab 30 mg daily oral	tab	½	1	2	3	4	5	5	6	
• or amp 200 mg/ml IM daily										
○ add 3 ml sterile water	ml	(0.5)	(0.75)	-	-	-	-	-	-	-
○ undiluted	ml	-	-	0.25	0.5	0.5	1	1	1	
PHENYTOIN (DILANTIN). Toxicity: ataxia, nystagmus, drowsiness										
• Vial 250 mg/5 ml SLOW IV - Stat 15 mg/kg (max 250 mg)	ml	1	2	3	4	5	5	5	5	
- Then 4 mg/kg BD	ml	0.25	0.5	1	1.25	1.75	2.75	3.5	4	
• Susp 30 mg/5 ml, 5-8 mg/kg daily oral	ml	2.5	5	8	11	-	-	-	-	
• Tab 30 mg, 4 mg/kg daily oral, NOT 100 mg tab/cap	tab/cap	1	2	2	3	4	5	6	8	
• Tab 100 mg, 4 mg/kg oral	tab	-	-	½	¾	1	1½	2	2-3	
POTASSIUM CHLORIDE SLOW RELEASE Tab 600 mg. 40-80 mg/kg BD (½-1 mmol/kg BD), oral		tab	½	½	1	1	1	2	2 BD	2 TID
PRIMAQUINE. Tab 7.5 mg-0.4 mg/kg daily for 14 days (eradication course)		tab	¼	¼	½	½	1	1½	2	2
PROBENECID. Tab 500 mg, oral										
• 20 mg/kg BD	tab	¼	-	-	-	-	-	-	-	-
• 10 mg/kg QID	tab	-	¼	¼	½	½	¾	1	1	
PROMETHAZINE (PHENERGAN)·										
• Amp 50 mg/2 ml, 0.5 mg/kg IM or slow IV	ml	-	0.25	0.5	0.75	1	1.5	2	2	
• Tab 25 mg, 0.5 mg/kg BD or TID oral	tab	Use diphenhydramine					1	1	1	
PYRAZINAMIDE. Tab 500 mg, 20-30 mg/kg/day oral		tab	¼	½	¾	1	1½	2	2	3
QUININE										
• Amp 120 mg/2 ml or 600 mg/10 ml. Give 10-15 mg/kg 12 hourly IM, or IV in 10 ml/kg of dextrose saline via burette over 4 hours	ml	3kg: 0.5 4-5kg: 1	1½	2	3	20-24kg: 4 25-29kg: 5	6	8	10	
• Tab 300 mg, 10 mg/kg TID for 3-5 days	tab	¼	¼	½	½	1	1½	1½	2	

Drug Doses - Hospital	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
*RIFAMPICIN. Cap 150 mg, susp 100 mg/5 ml (10 mg/kg daily (before breakfast))									
• Susp	ml	2.5	5	7.5	10	-	-	-	-
• Cap	cap	-	-	-	-	2	3	3	4
*SALBUTAMOL (VENTOLIN)·									
• Tab 4 mg, 4 times a day oral	tab	-	¼	¼	½	½	1	1	1
• Respirator solution through nebuliser 0.5 ml + 1.5 ml normal saline	ml	Give 1-3 hourly (see Asthma, p.41)							
SEPTRIN - see COTRIMOXAZOLE									
STREPTOMYCIN. Vial 1 g (add 1½ ml sterile water). For children <7 years old with sputum positive for TB. IM daily for 2 months	ml	0.25	6-10.9kg: 0.5	11-15.9kg: 0.75	16-20.9kg: 1	21-30.9kg: 1	-	-	-
SULFADOXINE - see FANSIDAR									
SULPHADIMIDINE. Tab 500 mg (0.5 g), 25-50 mg/kg QID oral	tab	¼	½	1	1	1½	2	2	2
SULPHAMETHOXAZOLE - see COTRIMOXAZOLE									
TETRACYCLINE - see OXYTETRACYCLINE									
*THIABENDAZOLE (MINTEZOL). Tab 500 mg, 25 mg/kg BD for 3 days, oral	tab	¼	½	½	1	1	1½	2	3
TINIDAZOLE. Tab 500 mg. Stat dose for persistent diarrhoea, daily for 3 days for dysentery	tab	¼	½	1	1 ½	2	3	4	4
TRIMEPRAZINE (VALERGAN). Syrup. 30 mg/5 ml. Premedication, 2-4 mg/kg oral	ml	2	4	6	8	10	-	-	-
TRIMETHOPRIM CO - see COTRIMOXAZOLE									
TRIPLOPEN - see PENICILLIN									
VALERGAN - see TRIMEPRAZINE									
VALPROATE. 250 mg tab, 5-25 mg/kg BD	tab	¼	¼	½	½	1	1	1½	2
VALIUM - see DIAZEPAM									
VENTOLIN - see SALBUTAMOL									

IV AND ORAL FLUIDS

IV fluid requirements	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
NORMAL MAINTENANCE	ml/hr	20	25	50	75	75	75	100	100
4.3% dextrose in 0.18% normal saline	drops/min	5	7	13	20	20	20	25	25
HEART FAILURE, COMA									
Do not give oral fluid as well	ml/hr	10	15	20	25	25	50	50	50
4.3% dextrose in 0.18% normal saline	drops/min	3	4	5	7	7	13	13	13
DIARRHOEA (½ strength Darrows)									
quickly if dehydrated	ml	100	150	250	350	500	700	900	1000
THEN	ml/hr	25	50	75	100	100	150	150	150
	drops/min	7	13	20	25	25	40	40	40
BURNS (0.9% sodium chloride)									
quickly if more than 10% burn	ml	100	150	250	350	500	700	900	1000
THEN	ml/hr	25	50	75	100	100	150	150	150
	drops/min	7	13	20	25	25	40	40	40
Note drops/min using standard burette. If using a paediatric burette, drops/min = ml/hr.									

Oral fluid requirements	Weight (kg):	3-5.9	6-9.9	10-14.9	15-19.9	20-29.9	30-39.9	40-49.9	Adult
MAINTENANCE									
Give every 3 hours (6 times a day) oral or NG tube	ml	120	240	300	350	400	450	450	500
DIARRHOEA WITH DEHYDRATION: ORS, oral or NG tube									
FAST,	ml	100	150	250	350	500	500	500	500
THEN every hour for 4 hours	ml	50	100	150	200	300	400	500	600
MENINGITIS, HEART FAILURE, COMA									
If patient not drinking and unable to insert IV, give NG fluid 4 times a day, oral	ml	100	150	200	250	300	350	350	350

INDEX

- 10-step checklist, 11
- 5-fluorocytosine, 227
- Abbreviations, 13
- Abdomen
 - distension, 15, 304, 305
 - mass, 39, 205
 - pain, 304
 - paracentesis, 39
 - tap, 301
- Abnormalities
 - congenital, 85–87
- ABO incompatibility, 95–96, 247, 255
- Abscesses
 - brain, 77, 83
 - liver, 16, 178
 - lung, 314
 - superficial, 349
- Acetazolamide
 - for hydrocephalus, 158, 159
- Acid ingestion, 320
- Acidosis
 - causes, 160
 - treatment, 135
- Acriflavine, 32
- Acute flaccid paralysis, 289–92
- Acute nephritis, 283
- Adhesive arachnoiditis
 - spinal, 296
- Admission to hospital, 156
- Adrenaline, 389
 - for anaphylaxis, 29
 - for cardiac arrest, 64
- Age
 - from teeth, 210, 281
- Aidposts, 143, 337
- AIDS, 151, 153, 163, 217
- Albendazole, 389
 - for anaemia, 18
 - for ascites, 40
 - for eosinophilia, 115
 - for malnutrition, 213, 217
 - for oedema, 283
 - for pigbel, 304
 - for strongyloidiasis, 356
- Albuminuria, 382
- Alcohol
 - as disinfectant, 33
 - ingestion of, 320
- Algid malaria, 200
- Alkali ingestion, 320
- Amino acids
 - intravenous, 218, 306
- Aminofusin, 218, 306
- Aminophylline, 243, 244, 389
 - for apnoea, 237
 - for asthma, 42
 - poisoning, 83
- Amodiaquine, 197, 201, 244, 389
- Amoebiasis, 16
 - and hepatitis, 178
- Amoxycillin, 389
- Amphotericin B, 227
- Ampicillin, 243, 244, 390
 - for gonorrhoea, 348
 - for neonatal meningitis, 262
- Anaemia, 17–21
 - and apnoea, 236
 - bone marrow aspiration, 57–58
 - in burns, 60
 - in malaria, 195, 200
 - teaching about, 145
 - transfusion, 55
- Anaesthetics, 22–28
 - for tracheostomy, 364
- Anaphylaxis, 29–30
- Ancylostomiasis. *See* Hookworm
- Angel's trumpet flower poisoning. *See* Trumpet flower poisoning
- Angiogram, carotid, 67
- Anorectal malformations, 85
- Anoxia, neonatal, 273–74
- Anterior horn cell lesions, 289–94
- Anthropometry and nutrition, 210–13
- Antibiotics, symbols, 31
- Anticholinergic poisoning, 318
- Anticonvulsant therapy, 82, 92–93, 224
- Antihistamine poisoning, 321
 - antidote, 318
 - coma in, 83
- Antisepsis, 32–33
 - in cholera, 76
- Antiseptic ingestion, 321
- Anuria, 283
- Aortic stenosis, 147–49
- Apgar score, 238, 273
- Apnoea
 - Alarm, 117
 - neonatal, 236–37, 273–74
- Arachnoiditis
 - spinal adhesive, 296
- Arnold-Chiari malformation, 80
- Arterial puncture, 34
- Arthritis, 35–38, 189
 - acute, 35–37
 - chronic, 38
 - juvenile chronic, 38
 - non-specific, 37
 - pyogenic, 35, 37
 - reactive, 38
 - septic, 284
- Ascites, 39–40
- Asphyxia
 - neonatal, 238–39, 273–74
- Aspiration
 - lung, 193
 - pericardium, 297
 - peritoneal cavity, 301
 - pleural effusion, 308–9
 - subdural collection, 226
- Aspirin, 390
 - causing haemolysis, 140
 - in breast milk, 181
 - in urine, 383
 - poisoning by, 83, 321

- Asthma, 41–44, 385
 - metered dose inhaler (MDI) for, 41, 42
 - nebuliser for, 41, 43, 119
 - spacer for, 41, 42, 43
- Astrocytoma, 79
 - ataxia, 45
- Atlanto-axial dislocation, 35
- Atracurium (Tracurium), 27
- Atrial septal defect, 147–49
- Atropine, 27, 390
 - Angel's trumpet flower poisoning, 335
 - for cardiac arrest, 65
- Auriscopes, 117
- Babies. *See* Neonates
- Baby Friendly Hospital Initiative, 9
- Bacterial infection. *See* Sepsis
- Barbiturate poisoning, 322
- BCG vaccine, 164, 367–72
- Beans, poisonous, 335
- Beck's triad, 297
- Benedict's test, 383
- Benztropine, for dystonic reactions, 88
- Bicarbonate. *See* Sodium bicarbonate
- Biliary atresia, 255
- Bilious vomiting, 15, 85
- Bilirubin. *See also* Exchange Transfusion, Jaundice,
 - Phototherapy
 - in urine, 178, 382
 - serum level, 280
- Bilirubinometer, 117
- Biopsy forceps, rectal, 120
- Bird respirator, 46–49
- Birth
 - asphyxia, 273–74
 - interval, 126
- Bites, poisonous, 333–34, 351–53
- Blackwater fever, 200
- Bladder tap, 380
- Bleach ingestion, 322
- Bleeding, abnormal, 50–52
- Blood
 - anaemia, 17–19
 - crossmatch, 95–96
 - culture, 53
 - in urine, 382
 - pressure, 54, 281
 - taking, 34, 129
 - transfusion, 18, 55–56, 60, 280
 - warmer, 117
- Body fluids. *See* Fluid therapy
- Boils, 349
- Bone marrow
 - bone marrow aspiration, 57–58
 - fluid into, 171, 346
- Books, 12, 59
- Bordetella pertussis. *See* Pertussis
- Born before arrival, 240
- Bottle feeding, 167–70
- Botulism, 289
- Bowel obstruction, 15
- Box Fish poisoning, 336
- Boyle's machine, 23
- Breastfeeding. *See* Feeding, Lactation
- Bronchiolitis, 385
- Burkitt's lymphoma, 205
- Burns, 60–61, 349
 - to eyes, 122
- Calcium, 135
 - for convulsions, 91
 - for neonatal convulsions, 242
- Calcium gluconate, 244
- Camoquin. *See* Amodiaquine
- Camphor poisoning, 140, 322
- Cancrum oris, 62
- Capillary disorders, 50
- Captopril, 390
- Carbamazepine, 93, 390
- Cardiac
 - arrest, 63–65
 - congenital malformation, 86, 147–49
 - disease in neonates, 271
 - failure, 55, 69, 282–83, 310–14, 385
 - massage, neonates, 274
 - murmur, functional, 150
 - tamponade, 66, 297–99
- Cardiomegaly, 66
- Cardiomyopathy, 66
- Carotid angiogram, 67
- Cassava poisoning, 83, 318, 335
- Cataracts, 86, 122
- Catheters. *See also* Intravenous catheters
 - urethral, 377
- Cefotaxime, 243, 244, 390
- Ceftazidime, 244
- Ceftazidone, 243, 390
- Ceftriaxone, 243, 244
- Ceftriazone, 390
- Centipede bites, 333
- Central venous
 - catheters, 68–69, 346
 - pressure, 69
- Cerebral
 - abscess, 77–79
 - angiogram, 67
 - malaria, 82, 88–91, 195, 197
 - oedema, 224
 - palsy, 293–96
 - tumour, 77–79, 83, 141, 209, 295
- Cerebrospinal fluid. *See also* Lumbar puncture
 - examination, 70
 - normal values, 71
- Child abuse, 72–74
- Child health nursing services, 7, 337
- Children, number of, 5
- Chlamydia pneumonia, 314, 385
- Chloral hydrate, 390
 - in urine, 383
 - overdose, 323
- Chloramphenicol, 243, 245, 390
 - abdominal distension due to, 15
 - and haemolysis, 140
 - drug interactions, 223, 224
 - for diarrhoea, 105
 - for meningitis, 223–24, 262–63
 - for osteomyelitis, 284
 - for pertussis, 302–3
 - for pigbel, 305
 - for pneumonia, 310–13
 - oedema due to, 282
- Chlorhexidine, 32
- Chloroquine, 194–203, 391
 - for leprosy reaction, 187
 - in anaemia, 17
 - in haemolysis, 140

- peripheral neuritis due to, 293
- poisoning, 317
- resistance to, 194, 199
- Chlorpromazine, 245, 391
 - for tetanus, 275, 360
 - hepatitis due to, 178
 - lactation induction, 183
- Cholera, 75–76
- Chorea, 45
- Ciguatera poisoning, 336
- Cimetidine, 391
- Cirrhosis, 40
- Cisternal puncture, 80
- Cleft lip, 86
- Cleft palate, 86
- Clinitest, 383
- Clofazimine, 186–87, 391
- Clostridia. *See* Pigbel, Tetanus
- Clotting disorders, 50–52
- Cloxacillin, 243, 245, 391
 - for pneumonia, 310
 - in septic shock, 346
- CNS tumours, 209
- Coagulation disorders, 50–52
- Coarctation of aorta, 147–49
- Colostomy, 81
- Coma, 82–84
- Community health, 5–10, 337–38
- Cone shell sting, 334
- Congenital malformations, 85–87
- Continuous positive airways pressure
 - in apnoea, 237
- Contraception, 127
- Contractures, in burns, 60, 86
- Convulsions, 88–93
 - apnoea due to, 236
 - in pertussis, 302, 303
 - neonatal, 241–42
- Corneal ulcer, 123, 137
- Corticosteroids. *See* Dexamethasone, Hydrocortisone, Prednisolone
- Cotrimoxazole, 391
 - for pneumonia, 310, 314
- Cough
 - pneumonia, 311
 - tuberculosis, 369
 - URTI, 376
- Craniopharyngioma, 79
- Creatinine, 280
 - in urine, 383
- Cretinism, 94
- Crossmatch of blood, 95–96
- Croup, 354, 364, 365
- Cryptococcus
 - cerebral, 77, 295
 - meningitis, 227
- Crystal violet, 32
- Culture and health, 5, 8, 156–57
- Cut down, venous, 97–98
- Cyanosis
 - and antirust tablets, 318, 321
 - in pneumonia, 310–14
 - on respirator, 47
- Cyclophosphamide
 - for Burkitt's lymphoma, 205
 - for nephrotic syndrome, 279
- Dapsone, 186–88, 391
 - and haemolysis, 140
 - and hepatitis, 178
- Darrow's solution, 75, 133
- Deafness, 85
- Death Adder, 351
- Dehydration. *See* Diarrhoea
 - hyperventilation, 160
 - in malaria, 200
 - in pertussis, 302
- Demographic data, 3
- Derris plant poisoning, 335
- Development, milestones, 99–100
- Dexamethasone, 391
 - for raised intracranial pressure, 78
 - for tetanus, 275
- Dextrose, 245. *See* Glucose
- Dextrostix, 117
- Diabetes
 - and coma, 83
 - and hyperventilation, 160
 - mellitus, 101–2
- Dialysis, peritoneal, 300
- Diaphragmatic hernia, 271
- Diarrhoea, 103–7
 - and hyperventilation, 160
 - in amoebiasis, 16
 - in cholera, 75
 - in measles, 221
 - in neonates, 264
 - teaching about, 145
- Diazepam, 243, 245, 360, 391
 - for convulsions, 90, 242
 - in breast milk, 181
 - in meningitis, 224, 262
 - in tetanus, 275, 360
 - overdose of, 318
- Diethylcarbamazine, 392
 - for eosinophilia, 116
- Digestelact, 106, 168, 231, 264
- Digoxin, 148, 245, 392
 - in severe pneumonia, 312
- Dinoflagellate poisoning, 336
- Diphenhydramine, 392
- Diphtheria
 - and paralysis, 289, 293
 - and stridor, 354
- Diplegia, 293
- Directly Observed Treatment Shortcourse (DOTS), 367
- Discharge from hospital, 156
- Disinfection. *See* Antisepsis
- Distension. *See* Abdominal distension
- Dithrane poisoning, 318
- Dobutamine, 29, 346
- Doctors
 - number of, 5
 - role of, 1–2
- Dopamine, 29, 64, 196, 346
- Drowning, 108
- Ear
 - foreign body, 137
 - otitis media, 285
- ECG, 118
 - and serum potassium, 341
 - interpretation, 109–12
- Eczema, 349
- Effusion

- pericardial, 66, 297–99
- pleural, 308–9
- subdural, 141, 225–26, 295
- Electric blanket, 119
- Electrocardiogram. *See* ECG
- Electrolyte mixture, 135, 218, 230
- Electrolytes
 - administration, 133–36
 - normal values, 280
- Emergency trolley, 118
- EMO, 25
- Emotional abuse, 73
- Empyema, 309, 312
- Enalapril, 392
- Encephalitis, 83
 - and hyperventilation, 160
 - CSF findings, 71
 - in measles, 221
 - in pertussis, 302
- Encephalocele, nasal, 86
- Endocarditis
 - subacute bacterial, 148
- Endotracheal intubation, 113–14
 - in neonates, 274
- Entamoeba histolytica, 16
- Enteritis necroticans. *See* Pigbel
- Eosinophilia, 115–16, 385
- Ependymoma, 79
- Epiglottitis, 354
- Epilepsy. *See* Convulsions
- Equipment, 117–21
 - in health centres, 144
- Erythema
 - nodosum leprosum, 187
- Erythromycin, 392
 - in pertussis, 303
 - in pneumonia, 310, 314
- Ethambutol, 371, 392
- Ether, 25
- Ethinylestradiol
 - lactation induction, 182
- Eusol, 32
- Exchange transfusion, 246–49
 - blood for, 280
 - tray, 118
- Exomphalos, 85
- Extrinsic clotting pathway, 51
- Eye
 - disease, 122–25
 - foreign body, 124, 137
- Fallot's tetralogy, 147–49
- Family planning, 126–28, 212, 218
- Fansidar, 202, 194–203, 392
- Febrile convulsions, 88, 200
- Feeding. *See also* Lactation
 - bottles, 167–70
 - breast, 8, 104, 106, 167
 - in meningitis, 224–25
 - infant, 167–70
 - milk mixtures, 167–70
 - of low birth weight, 259
 - of neonates, 250, 264
- Femoral puncture, 129
- fetoprotein
 - alpha, 280
- Fever, 130–32
- Fibrinogen, 51
- Filariasis
 - arthritis, 38
- Fish hook, 137
- Fish, poisoning, 336
- Fluid therapy, 133–36
 - for diarrhoea, 103–7, 397
 - for maintenance, 397
 - in anuria, 283
 - in burns, 60, 397
 - in cardiac failure, 397
 - in coma, 397
 - in meningitis, 224–25, 262
 - intravenous, 397
 - oral, 397
 - via bone marrow, 171
 - with phototherapy, 254, 270
- Fluothane. *See* Halothane
- Folic acid, 392
 - for anaemia, 18
 - for malnutrition, 217
- Foreign body, 137–38, 354
 - and pneumonia, 314
 - ear, 137
 - eye, 124, 137
 - gastrointestinal, 138
 - nose, 137
 - pharynx, 137
 - trachea, 138, 354, 385
 - urethra, 138
 - vaginal, 384
- Frog breathing, 139
- Fröin's syndrome, 71
- Furosemide, 245, 392
- Functional murmurs, 150
- Funnel chest, 85
- Galactosaemia, 255
- Gamma benzene hexachloride
 - poisoning, 318
- Gangosa, 387
- Gastroenteritis. *See* Diarrhoea
- Gentamicin, 243, 245, 392
 - for neonatal meningitis, 262
 - for septic shock, 346
- Gentian violet, 32
- Germ cell tumours, 209
- Gestational age, 257–58
- Glasgow Children's Coma Scale, 84
- Glasgow Coma Scale, 84
- Glaucoma, 122, 124
- Glioma, 78, 79
- Glomerulonephritis, acute, 283
- Glucose
 - blood for, 280
 - for apnoea, 236
 - for neonatal convulsions, 242
 - in cardiac arrest, 64
 - in CSF, 71
 - in urine, 383
- Glucose 6 phosphate dehydrogenase
 - anaemia in, 20, 21, 177
 - blood for, 280
 - deficiency, 140
- Glycosuria, 383
- GOBI-FFF, 9
- Goitre, 94
- Gonococcus, 347–48, 384
- Grille, 350

- Growth. *See* Nutrition
- Guillain-Barre disease, 291
- Gummata in yaws, 387
- Haemarthrosis, 50
- Haemoglobin
 - in urine, 382
- Haemoglobin electrophoresis, 280
- Haemoglobinuria, 382
- Haemolysis, 19, 20, 21, 176, 177
 - and leukaemoid reaction, 386
 - and neonatal jaundice, 254–55
 - transfusion in, 55
- Haemostasis, 50
- Halothane, 22, 24, 25
 - for tracheostomy, 364
- Hand dryers, 118
- Hansen's disease. *See* Leprosy
- Hartmann's solution, 75, 134
- Head. *See also* Cerebral
 - circumference, 141–42
 - injury, 83
 - tumour, 77–79, 83
- Health care
 - health centres, 143–46
 - hospitalisation, 156–57
 - primary, 337–38
 - provision of, 7–10
- Health centres
 - visiting, 143–46
- Health indices, 3
- Health, attitudes to, 5–10, 156–57
- Heart disease. *See* Cardiac disease
- Heart rate, 281
- Heating, 118
- Hemiplegia, 295–96
- Hepatitis, 176, 178–79
 - neonatal, 255
- Hepatomegaly
 - and abdominal distension, 15
 - in amoebiasis, 16, 178
 - in cardiac failure, 282
- Hernia
 - diaphragmatic, 271
 - inguinal, 86
- Hess test, 50
- Hetrazan. *See* Diethylcarbamazine
- Hexachlorophane, 32
- Hip disease, 189
- HIV
 - and breastfeeding, 154
 - case definition, 151, 153
 - clinical features, 151, 152
 - laboratory diagnosis, 153
 - management, 153
 - prevention of perinatal transmission, 154
 - prognosis, 154
 - staging in children, 154
 - transmission, 151
- Hookworm
 - anaemia, 18
 - oedema, 283
- Hornet bites, 333
- Hospital
 - absconsions, 156–57
 - admission, 156–57
 - discharge, 156–57
- Hospital and mothers, 8
- Humidicribs, 258
- Hyaline membrane disease, 272
- Hydrocephalus, 77, 141, 158–59, 295
 - medical treatment, 158
 - surgery, 159
- Hydrochlorothiazide, 392
 - and hepatitis, 178
 - for ascites, 40
- Hydrocoele, 86
- Hydrocortisone, 392
 - for anaphylaxis, 29
 - for asthma, 42
- Hyperglycaemia, 83
- Hyperimmune malarial splenomegaly, 366
- Hyperkalaemia, 344
 - renal failure, 341
- Hypertension, 82, 283
 - renal, 344
- Hyperventilation, 160
- Hypocalcaemia, 91
- Hypoglycaemia, 83, 91, 161, 198, 200, 259
 - and apnoea, 236
 - and coma, 83
 - and convulsions, 91
- Hypokalaemia
 - in abdominal distension, 15
 - paralysis, 289
 - renal failure, 342
- Hypoplastic left heart, 147–49
- Hypothermia, 234, 258
- Hypothyroidism, 162, 255
 - cretinism, 94
- Hypovolaemia
 - central venous pressure, 69
- Ileostomy, 81
- Illness
 - beliefs about, 5–10, 156–57
 - causes of, 6
- IMCI, 9
- Immunisation, 163–66, 337
 - for pertussis, 302
 - for pigbel, 307
 - for tetanus, 361
- Immunoglobulin
 - for Guillain-Barre, 291
 - for tetanus, 275, 360
- Impetigo, 349
- Incubators, 258
- Indomethacin
 - for juvenile chronic arthritis, 38
 - for non-specific arthritis, 37
 - for patent ductus, 267
 - for reactive arthritis, 38
- Infant mortality, 5
- Infection. *See* Sepsis
- Infectious lymphocytosis, 386
- Infectious mononucleosis, 282, 386
- Ingestion. *See* Poisoning
- Injection palsy, 189
- Intracaths
 - insertion, 172–74
 - purchase of, 119
- Intracranial pressure, raised
 - acute, 82–84
 - chronic, 77–79, 92–93
- Intraosseous fluid, 171, 346
- Intrauterine infection, 141

- Intravenous catheters
 - central, 69
 - cut down, 97–98
 - insertion of, 172–74
 - purchase of, 119
- Intravenous fluids. *See* Fluid therapy
- Intravenous nutrition. *See* Parenteral nutrition
- Intravenous pyelogram, 175
- Intrinsic clotting pathway, 51
- Intubation, endotracheal, 113–14
- Iodine
 - as antiseptic, 32
 - in cretinism, 94
- Ipecacuana, 393
- Iritis, 122, 124
- Iron, 393
 - and infection, 17
 - for anaemia, 17–18
 - for neonates, 260
 - in feeds, 168
 - poisoning, 318, 326
- Isoniazid, 245, 370–72, 393
 - and hepatitis, 178
 - and peripheral neuritis, 293
 - in breast milk, 181
 - in urine, 383
- Isoprenaline infusion, 29, 64, 346
- Jaundice, 177–79
 - exchange transfusion, 246–49
 - neonatal, 254–55
- Jellyfish stings, 334
- Jones Criteria, 36
- Journals, medical, 180
- Jugular vein, catheter, 69
- Kerosene poisoning, 317, 326
- Ketamine, 22, 23, 27, 393
- Knee, disease of, 189
- Kwashiorkor
 - malnutrition, 215–19
 - oedema, 282
- Lactation
 - drugs in breast milk, 181
 - induction, 183
- Lactogen, 168
- Lactose, 106
- Lactose intolerance, 230
- Laryngo-tracheo-bronchitis, 354, 364, 365
- Lead poisoning, 318
 - anaemia in, 20, 21
 - and peripheral neuritis, 293
 - coma in, 83
 - macrocephaly, 141
- Leprosy, 184–88
 - and peripheral neuritis, 293
- Leucocytosis, 386
- Leukaemia, 206
 - and anaemia, 21
- Leukaemoid reaction, 386
- Library, 59, 180
- Lima bean poisoning, 318, 335
- Limp, 189
- Literacy rate, 5
- Liver. *See also* Hepatitis
 - abscess, 16
 - biopsy, 190–91
 - chronic disease, 283
- Liver tumours, 209
- Lumbar puncture, 82, 192
 - contraindications, 77, 290, 295
- Lung. *See also* Pneumonia
 - aspiration, 193
 - foreign body, 138
 - hyperventilation, 160
- Lymphoma, 208
 - and leukaemoid reaction, 386
 - Burkitt's, 205
- Lytic cocktail, 393
- Macrocephaly, 141
- Magnesium, 135
 - for neonatal convulsions, 242
 - in diarrhoea, 105
- Magnesium hydroxide, 393
- Magnesium sulphate, 245
- Maintenance fluids. *See* Fluid therapy
- Malaria, 194–203
 - and haemolysis, 177
 - and hepatitis, 178
 - and hyperventilation, 160
 - and neonatal jaundice, 255
 - and nephrotic syndrome, 279
 - and oedema, 282
 - and peripheral neuritis, 293
 - in anaemia, 17, 20
 - in fever, 131
- Malathion poisoning, 318
- Malformations, congenital, 85–87
- Malignant disease, 204–9
- Malnutrition, 210–20. *See also* Nutrition
- Maloprim, 201, 202, 393
- Mannitol, 393
 - for raised intracranial pressure, 78
 - for Reye's syndrome, 83
 - in burns, 60
 - in septic shock, 346
- Mantoux, 369
- Marasmus, 210
- Marrow
 - aspiration, 58
 - fluid into, 171
- Mastoiditis, 85
- Maternal and child health services, 7, 337
- Measles, 221–22
 - immunisation, 165
- Mebendazole, 393
 - in malnutrition, 217
- Meconium aspiration, 271, 273
 - neonatal, 261
- Medical
 - books, 59
 - journals, 180
- Medroxyprogesterone
 - contraception, 128, 218
 - lactation induction, 182
- Medulloblastoma, 79
- Menghini biopsy, 190
- Meningioma, 295
- Meningitis, 223–26, 262–63
 - and apnoea, 236
 - and hyperventilation, 160
 - cryptococcal, 227–28
 - CSF findings, 71
 - teaching about, 145
 - tuberculous, 368
- Meningocele, nasal, 86

- Methaemoglobinaemia, 318
- Methanol poisoning, 320
- Methotrexate
 - for Burkitt's lymphoma, 205
 - for juvenile chronic arthritis, 38
 - for leukaemia, 206
- Methylated spirit poisoning, 327
- Methyldopa, 393
- Metoclopramide
 - lactation induction, 182
- Metronidazole, 245, 393
 - for amoebiasis, 16
 - for cancrum oris, 262
 - for diarrhoea, 218
 - for mastoiditis, 85
 - for necrotising enterocolitis, 265
 - for pigbel, 305
 - in breast milk, 181
- Microcephaly, 141
- Micturating cystourethrogram, 229
- Mid upper arm circumference, 210, 214
- Milestones, developmental, 99–100
- Milk. *See* Feeding
- Milk oil formula, 168, 216, 231
- Millipede attack, 333
- Mitsuda test, 186
- Moles, 135
- Morbidity, causes of, 6
- Morphine, 394
- Mortality
 - 1–4 years, 3
 - infant, 5, 126
 - maternal, 3
 - under 5, 5
- Motor neurone disease, 293
- Mucopolysaccharidosis, 141, 293
- Mushroom poisoning, 335
- Myasthenia gravis, 293
- Mycobacterium ulcerans, 232
- Myelitis, 289–92
- Myelogram, 233
 - indications for, 289, 290, 296
- Myocarditis, 66
- Myodil, 233
- Myopathy, 289
- Myxoedema, 94
- Naloxone, 246, 274, 318
- Nasal encephalocele, 86
- Nasal meningocoele, 86
- Nebulisers
 - in asthma, 43
 - purchase of, 119
- Neck pain, 35
- Neglect, 73
- Neonates, 234–78
 - admission, 234–35
 - apnoea, 236–37
 - asphyxia, 238–39
 - born before arrival, 240
 - convulsions, 241–42
 - drug dilutions, 243
 - drug doses, 243–46
 - exchange transfusion, 246–49
 - fluids and feeds, 250
 - infection, 251–53
 - jaundice, 254–55
 - low birth weight, 260
 - meconium aspiration, 261
 - meningitis, 262–63
 - milk feeds, 264
 - necrotising enterocolitis, 265–66
 - patent ductus arteriosus, 267–68
 - phototherapy, 269–70
 - respiratory distress, 271–72
 - resuscitation, 273–74
 - tetanus, 275–76
 - umbilical vein catheterisation, 277–78
- Neostigmine, 27
- Nephritis, acute, 283
- Nephroblastoma, 207
- Nephrotic syndrome, 279, 282
- Neuroblastoma, 45, 207
- Neutrophilia, 386
- Newborn. *See* Neonates
- Nitrazepam, 394
- Nitrofurantoin, 394
- Non-accidental injury, 72
- Normal values, 280–81
- Nose
 - foreign body, 137
- Nursery, heating, 118, 258
- Nursing services, 7, 337
- Nutramigen, 106, 168, 264
- Nutrition, 210–20
 - and anaemia, 18
 - and pigbel, 304
 - classification, 210
 - in burns, 61
 - in measles, 221
 - in renal failure, 342
 - rules, 145
 - teaching about, 145, 211–12
- Nuts, poisonous, 335
- Nystatin, 394
 - in diarrhoea, 106, 218
 - in malnutrition, 217
- Oedema, 282–83
- Oesophageal atresia, 271
- Oleander poisoning, 335
- Omnopon. *See* Papaveretum
- Oral rehydration, 75, 103, 305
- Organophosphate poisoning, 318, 328
- Osteomyelitis, 189, 284
- Otitis media, 285
- Oxygen, 236, 238
 - causing abdominal distension, 15
 - for neonates, 259
 - for pneumothorax, 315
 - for severe pneumonia, 312
- Oxytetracycline, 394
 - for cholera, 76
- Packed cells, 19, 55
- Paediatric Rules, 286–87
- Paediatrician
 - role of, 1–2
- Papaveretum, 394
- Papilloedema, 77, 82
- Papuan black snake, 351–53
- Paracentesis. *See* Aspiration
- Paracetamol, 394
- Paracetamol poisoning, 329
- Paradoxical pulse, 297
- Paraldehyde, 246, 394
 - for convulsions, 90

- for neonatal convulsions, 242
- in meningitis, 224, 262
- in tetanus, 275, 360
- Paralysis, 289–96
 - acute flaccid, 289–92
 - chronic flaccid, 293–94
 - post-infectious, 189
 - spastic, 295–96
- Paramedical staff, 1, 6
 - in health centres, 143–46
 - teaching, 145–46
- Paraplegia, 289–96
- Paraquat poisoning, 318, 332
- Parasite density, 203
- Parasites, intestinal, 211
- Parenteral nutrition
 - for diarrhoea, 218
 - in pigbel, 304–7
- Partial thromboplastin time, 51
- Patent ductus arteriosus, 147–49, 267–68
- Penicillin, 243, 246, 394
 - and eosinophilia, 115
 - for cancrum oris, 62
 - for gonorrhoea, 347
 - for necrotising enterocolitis, 265
 - for neonatal conjunctivitis, 123
 - for neonatal meningitis, 262
 - for nephrotic syndrome, 279
 - for pigbel, 305
 - for pneumonia, 310
 - for rheumatic fever, 36
 - for syphilis, 347
 - for tetanus, 360
 - for yaws, 388
 - in urine, 383
- Pentavite, 168
- Pentothal, 28
- Pericardial effusion, 66, 297–99
- Pericardiocentesis, 297–99
- Pericarditis, constrictive, 40
- Peripheral neuropathy, 293
- Peritoneal
 - dialysis, 300
 - fluid, 171
 - tap, 301
- Perthes' disease, 189
- Pertussis, 302–3
 - and leukamoid reaction, 386
 - and oedema, 282
 - immunisation, 165
- Pesticides poisoning, 318
- Pethidine, 28, 243, 394
 - and neonatal depression, 274
- Pharynx
 - foreign body, 137
- Phenobarbitone, 90, 92, 243, 246, 395
 - and chloramphenicol, 224
 - in breast milk, 181
 - in convulsions, 242
- Phenytoin, 90, 93, 246, 395
 - and chloramphenicol, 224
 - and hepatitis, 178
 - and peripheral neuritis, 293
 - in breast milk, 181
 - in convulsions, 242
- Phototherapy, 120, 269–70
 - indications, 254
- Physic nut poisoning, 335
- Physostigmine, 318
- Pigbel, 304–7
 - abdominal distension, 15
 - blood transfusion in, 55
 - immunisation, 165
- Pigeon chest, 85
- Pineal tumour, 79
- Plants, poisoning by, 335
- Platelet disorders, 50
- Pleural
 - aspiration, 308–9
 - drain, 315–16
 - effusion, 308–9
- Pneumocystis pneumonia, 314
- Pneumonia, 310–14
 - and apnoea, 236
 - and foreign body, 314
 - hepatitis in, 178
 - hyperventilation in, 160
 - in measles, 221
 - in pertussis, 302
 - leukaemoid reaction in, 386
 - lung aspiration, 193
 - neonatal, 271
 - teaching about, 145
 - with meningitis, 311
- Pneumothorax, 315–16
 - in neonates, 271
 - in pneumonia, 312
- Poisoning, 317–36
 - and acidosis, 160
 - and coma, 83
- Poliomyelitis, 290, 291
 - and limp, 189
 - immunisation, 165
- Polydactyly, 86
- Polyneuritis, 289–92
- Potassium, 135, 395
 - and ECG, 341
 - and paralysis, 289
 - in abdominal distension, 15
 - in burns, 60
 - normal values, 280
- Pott's disease, 296
- Prednisolone
 - for asthma, 42
 - for juvenile chronic arthritis, 38
 - for nephrotic syndrome, 279
 - for rheumatic fever, 36
 - for tuberculosis meningitis, 372
- Pregestimil, 120, 218, 230, 264
- Prematurity, 260
- Prevention of illness, 6
- Primaquine, 194, 195, 202, 395
- Primary health care, 5–10, 143–46, 337–38
- Priorities for child health services, 7–8
- Probenecid, 395
 - for gonorrhoea, 348
 - in urine, 383
- Proguanil, 194, 201
- Prolactin, 183
- Promethazine, 56, 395
 - for anaphylaxis, 29
 - for envenomation, 352
- Propranolol, in tetanus, 361
- Protein

- in burns, 60
- in oedema, 282–83
- in urine, 282, 382
- Prothrombin time, 51
- Pseudotumour cerebri, 77
- Puffer fish poisoning, 336
- Pulmonary eosinophilia, 115–16
- Pulmonary stenosis, 147–49
- Pulse rate, 281
- Pulsus paradoxus, 297
- Purpura, 251
- Pyloric stenosis, 134
- Pyomyositis, 284
- Pyrazinamide, 370–72, 395
- Pyrexia, 130–32
- Pyrimethamine, 194, 201. *See also* Fansidar, Maloprim
- Quinine, 194, 195, 198, 199, 202, 246, 395
 - and haemolysis, 140
- Rabies, 339
- Radiators, 118
- Rectal atresia, 85
- Rectal biopsy forceps, 120
- Red tide poisoning, 336
- Refrigerators, 121
 - in health centres, 144
- Renal disease
 - and hyperventilation, 160
 - and oedema, 282–83
 - coma, 83
 - fluid therapy in, 283
 - IVP, 175
 - MCU, 229
 - nephritis, 283
 - nephrotic syndrome, 279
- Renal failure
 - acute, 340–42
 - chronic, 283, 343–44
- Renal tubular acidosis, 345
- Reserpine
 - in breast milk, 181
- Respiration rate, 281
- Respirator, Bird, 46–49
- Respiratory distress
 - in neonates, 259, 271–72
- Respiratory tract infection
 - otitis media, 285
 - pneumonia, 310–14
 - upper, 376
- Resuscitation
 - cardiac arrest, 63–65
 - of neonates, 273–74
- Reticulocytes, 20, 280
- Retinoblastoma, 208
- Reye's syndrome, 83
 - and hyperventilation, 160
- Rheumatic fever, 36
 - and limp, 189
- Rheumatoid arthritis. *See* Still's disease
- Rifampicin, 396
 - and hepatitis, 178
 - for leprosy, 186, 188
 - for tuberculosis, 370–72
- Ringworm, 350
- Room heaters, 118
- Rural health, 7–10, 143–46, 337–38
- Rust tablets, poisoning, 318, 321
- Sabin vaccine, 165
- Salbutamol, 396
 - for anaphylaxis, 29
 - for asthma, 41–44, 385
 - for pertussis, 303
- Sanitation, 5
- Sarcomas, 209
- Scabies, 350
 - lotion, poisoning, 318
- Scalp vein needle, 172
- Schools, 338
- Sea snake, 351–53
- Seafood, poisonous, 336
- Seborrhoea, 350
- Semimembranous bursa, 87
- Sepsis
 - abdominal distension, 15
 - and apnoea, 236
 - and haemolysis, 177
 - and hepatitis, 178
 - and neonatal jaundice, 255
 - arthritis, 284
 - blood transfusion in, 55
 - central venous pressure, 69
 - cerebral abscess, 77–79
 - in burns, 60–61
 - leukaemoid reaction in, 386
 - neonatal, 251–53
 - shock in, 346
- Septic shock, 346
- Septicaemia, 346
- Sexual abuse, 73, 151
- Sexually transmitted disease, 347–48
- Shock
 - anaphylactic, 29
 - and hyperventilation, 160
 - in burns, 60–61
 - in cholera, 76
 - in malaria, 200
 - intraosseous fluid in, 171
 - venous pressure in, 69
- Sitting, age of, 99
- Skin, 349–50
 - cleaning, 32–33
 - sores, 32–33, 350
 - ulcers, 350
- Small for gestational age, 260
- Snakebite, 351–53
- Sodium, 135
 - in convulsions, 91
 - in parenteral nutrition, 306
 - normal values, 280
- Sodium bicarbonate, 135, 244
 - for septic shock, 346
 - neonates, 274
- Sodium cromoglycate, 42
- Solid food, introduction, 211
- Sores, 32–33, 350
- Spastic tropical paraplegia, 296
- Spasticity, 295–96
- Speech, development, 99
- Spider bites, 333
- Spina bifida, 293
- Spinal
 - adhesive arachnoiditis, 296
 - anaesthesia, 26
 - cord compression, 289–92, 293, 295–96

- shock, 292
- tuberculosis, 296
- Splenectomy
 - and leukaemoid reaction, 386
 - for thalassaemia, 362
 - for tropical splenomegaly, 366
- Splenomegaly
 - thalassaemia, 362
 - tropical, 366
- Squint, 86
- Standard Treatments, 1
- Staphylococcus
 - meningitis, 262–63
 - pneumonia, 310–14
- Steroids. *See* Dexamethasone, Hydrocortisone, Prednisolone
- Stethoscopes, 121
- Still's disease
 - anaemia in, 20, 21
- Stingray, 334
- Stings, poisonous, 333–34
- Stonefish, 333
- Strabismus, 86, 124
- Streptomycin, 243, 246, 371, 396
 - and peripheral neuritis, 293
 - in urine, 383
- Stridor, 354–55
- Strongyloides, 356–57
 - and oedema, 283
- Strongyloidiasis
 - Strongyloides Fulleborni V Kellyi, 356
 - Strongyloides Stercoralis, 356
- Stunting, 211
- Subacute bacterial endocarditis, 148
 - anaemia in, 20
- Subacute sclerosing panencephalitis, 45, 358
- Subdural collection, 141, 295
 - in meningitis, 225–26
- Subgaleal inclusion cyst, 86
- Suckling, lactation induction, 182
- Suction, 121
 - with tracheostomy, 365
- Sugar water, 103
- Sugar-milk, 168, 231, 259, 264
- Sugar-water, 264
- Sulphadimidine, 396
- Suprapubic aspiration of urine, 380
- Suxamethonium, 23, 24, 28
- Syphilis, 347
- Syringes, plastic, 120
- Taipan snake, 351–53
- Talipes, 87
- Tamponade, cardiac, 297–99
- Tear duct, blocked, 86, 125
- Teeth, and age, 210, 281
- Temperature
 - and apnoea, 236
 - fever, 130–32
 - malaria, 200
 - neonates, 258
 - room heaters, 118
 - with phototherapy, 270
- Testes, undescended, 86
- Tetanus, 360–61
 - and convulsions, 88
 - immunisation, 165
 - immunoglobulin, 246
 - neonatal, 275–76
- Tetracycline. *See* Oxytetracycline
- Tetradon poisoning, 336
- Thalassaemia, 362–63
 - anaemia in, 21
- Thiabendazole, 396
 - for eosinophilia, 116
 - for oedema, 283
 - for strongyloidiasis, 356
- Thiacetazone, 186
- Thiopentone, 28
- Thoracocentesis, needle, 315
- Thrombasthaenia, 50
- Thrombocytopaenia, 346
- Thromboplastin time, 51
- Thyroid gland. *See* Cretinism, Hypothyroidism
- Tick paralysis, 289, 333
- Tinea, 350
- Tinidazole, 396
- Toad fish poisoning, 336
- Toadstool poisoning, 335
- TORCHES, 141, 255
- Torula. *See* Cryptococcus
- Trachea
 - foreign body, 138, 354, 385
- Tracheo-oesophageal fistula, 271
- Tracheostomy, 364–65
 - in croup, 354
 - in tetanus, 361
- Trachoma, 125
- Traditional practices, 8
- Transfusion. *See* Blood transfusion
- Transillumination, of head, 141
- Transpositions of great vessels, 147–49
- Transverse myelitis, 289–92
- Tricyclic antidepressant poisoning, 318, 320
- Triglyde, 120, 264
- Trimeprazine, 396
- Trimethoprim. *See* Cotrimoxazole
- Tropical
 - eosinophilia, 115–16, 385
 - splenomegaly, 366
- Trucut biopsy, 191
- Trumpet flower poisoning, 83, 318, 335
- Tuberculoma, 77, 141, 295
- Tuberculosis, 367–72
 - anaemia, 17
 - and limp, 189
 - and peripheral neuritis, 293
 - and pertussis, 302
 - and prolonged pneumonia, 314
 - and stridor, 354
 - and wheeze, 385
 - ascites, 40
 - ataxia, 45
 - fever in, 131
 - leukaemoid reaction in, 386
 - spinal, 296
- Tubing, oxygen and suction, 120
- Turtle meat poisoning, 336
- Typhoid, 373–74
 - anaemia, 17
 - and peripheral neuritis, 293
- Ulcers, 350
- Ultrasonography, 375
- Umbilicus
 - catheter, 277–78

- granuloma, 85
- hernia, 85
- Unconscious child. *See* Coma
- Urea, 280
- Ureaplasma pneumonia, 314
- Urethra
 - catheter, 377
 - foreign body, 138
- Urinary tract infection, 131, 378–81
- Urine
 - bilirubin in, 382
 - blood in, 283, 382
 - clean catch technique, 379
 - collection of, 380
 - haemoglobin in, 382
 - in burns, 60
 - in septicaemia, 346
 - microscopy, 382
 - midstream, 378
 - protein in, 282–83, 382
 - sugar in, 383
 - suprapubic aspiration, 380
- Vaccination. *See* Immunisation
- Vaginal discharge, 384
- Valergan. *See* Trimeprazine
- Valium. *See* Diazepam
- Valproate, 396
- VDRL, 253, 347
- Venepuncture. *See* Blood taking
- Venereal disease. *See* Sexually transmitted disease
- Ventilation
 - frog breathing, 139
 - mechanical, 46–49
- Ventolin. *See* Salbutamol
- Ventricular septal defect, 147–49
- Vertigo, 45
- Village health aides, 2, 338
- Vim-Silverman biopsy, 191
- Visiting health centres, 143–46
- Vitamin
 - keratomalacia, 215
- Vitamin A supplementation, 9
- Vitamins, 168, 216
 - in diarrhoea, 105
 - in malnutrition, 216
 - phytomenadione, 246
 - poisoning, 331
- Walking, 99
- Wasting, 211
- Weaning, 211
- Weight-for-age, 210
- Wheeze, 385
- White cell count, 386
- Whooping cough. *See* Pertussis
- Widal, 280
- Wilm's tumour, 207
- Wood preservative poisoning, 318
- Xerophthalmia, 125
- Yaws, 387–88
- Zinc
 - in electrolyte mixture, 135, 230