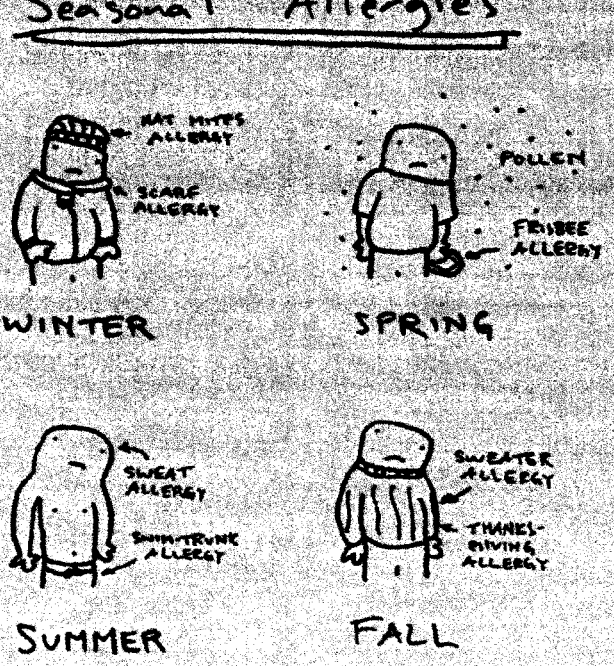
ENDOCRINE & ALLERGIC CONDITIONS

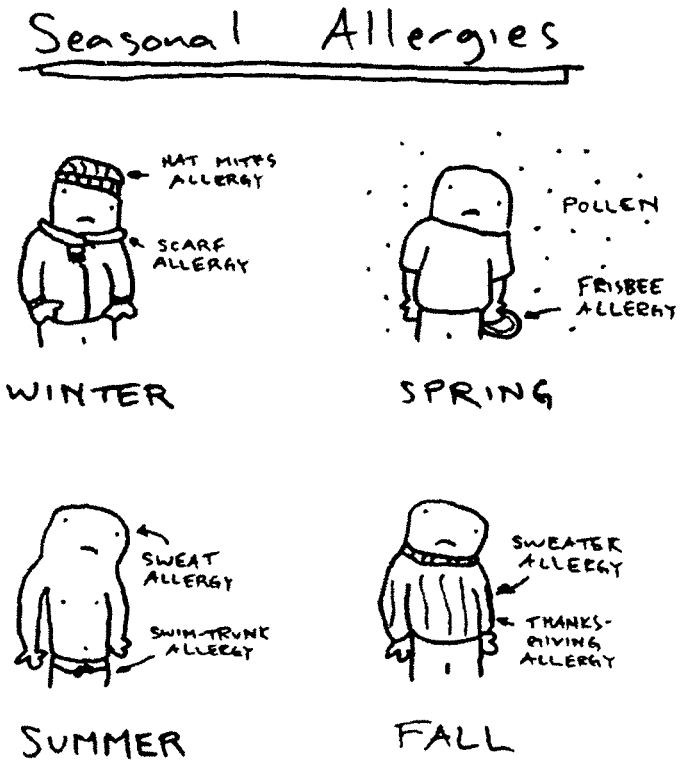
CHRIS - WINDHOEK

GROWTH DISORDERS
SEXUAL DEVELOPMENTAL DISORDERS
WATER BALANCE DISORDERS
ADRENAL CORTEX DISORDERS
PARATHYROID DISORDERS
ALLERGIC RHINITIS
FOOD ALLERGIES



ENDOCRINE & ALLERGIC CONDITIONS

- GROWTH DISORDERS
- SEXUAL DEVELOPMENT DISORDERS
- WATER BALANCE DISORDERS
- THYROID DISORDERS
- ADRENAL CORTECX DISORDERS
- PARATHYRPOID DISORDERS
- ALLERGIC RHINITIS
- FOOD ALLERGIES



ENDOCRINE DISORDERS:

1.) Growth Disorders:

-*In utero*: Maternal nutrition + intact placenta

-Infancy: Nutrition

-Post-infancy: Interaction of endocrine + skeletal systems (in the absence of chronic disease)

- **Growth velocity:** Rate of growth in cm/year

-**Radiological bone age:** Comparative X-rays of left hand + wrist with the norm (see fig.18.1 p362 of Witteberg)

*Familial Short Stature	*Constitutional Growth Delay	*Pathological Short Stature	*Endocrine Short Stature
-family hx of short stature -normal birth weight (>2.5kg) -growth below but parallel to 3rd centile for height -Normal corresponding bone age to chronological age -NO INVESTIGATIONS OR F/U NEEDED!	-More common in boys -Normal nutritional status -Height at or below 3rd centile by age 5 – 7 but normal annual growth rate -Retarded bone age by 2 – 4 years in relation to norm -Delayed pubertal development (may need anabolic steroids or testosterone treatment to aid in puberty) -EXCLUDE HYPOTHYROIDISM!	-e.g. malnutrition, chronic disease, psychosocial deprivation -Very short, slow growth, delayed bone maturation, bony deformities -Catch-up growth can occur when situation corrected -IUGR may fail to catch up	-typically high weight/height ratio -appear obese -Congenital or Acquired Hypothyroidism (test TSH) -Isolated GH deficiency and anterior pituitary def. (may present with hypoglycaemia, typical immature appearance)

-Basic data required for evaluation of short stature

Hx	Examination	Investigations
-Birth weight, GA, Fam hx + fam heights, Feeding hx, Growth and development hx	-Height, weight, span measurements -Plotting on Growth Chart -Exam of nutritional state, development, Organ systems	-Urine + stool -Hb -Serum albumin +blood urea -T4 + TSH -Radiolog. Bone age

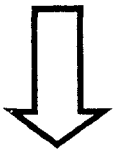
-Short stature: Clinical approach:

ACCURATE MEASUREMENT

Proportionate	**Disproportionate** -Short trunk : Spondylo-epiphyseal Dysplasias Mucopolysaccharidoses -Short limbs : Achondroplasia Metaphyseal Dysplasias

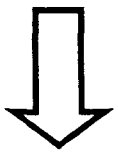
ACCURATE MEASUREMENT

Normal	**Reduced** -Clinically Abn : Growth disorder Syndrome -Clinically norm: Small for dates



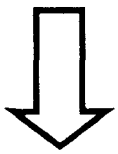
Hx: FEEDING + NUTRITION

Normal	**Abnormal** -Malnutrition



CLINICAL EXAMINATION

Normal	**Abnormal** -Disease: i.e. Cardiac, GIT, Metabolic, Endocrine



GROWTH VELOCITY

Normal	**Reduced** -Emotional, genetic, endocrine

2.) Sexual Development Disorders

***Intersex**

-Ambiguity of external genetalia

-Causes:

Abn development of gonads (chromosome abn)	**Abn genetalia with XY karyotype + testes (male pseudo-hermaphroditism)**	**Abn genetalia with XX karyotype + ovaries (female pseudohermaphroditism)**
-True hermaphroditism -Mixed gonadal dysgenesis	-Dysf of testosterone synthesis -androgen dependant areas not functional -1)complete androgen resistance -2)incomplete androgen resistance	-1)Cong adrenal hyperplasia -with salt-losing syndrome -without salt-losing syndrome -2)Exogenous androgens during pregnancy

Approach:

-Good clinical evaluation of genetalia

-No palpable gonads = EXCLUDE CONGENITAL ADRENAL HYPERPLASIA(CAH)!!

-Ultrasound/exploratory lap may be needed

-Child should be assessed by experts and parents should carefully consider what sex child should be raised as

***Premature thelarche**

-Isolated breast development without other signs of puberty due to excess oestrogen

-usually <2 years of age

-if no hair development (pubic/axillary) = NO TREATMENT, REASSURANCE

***Premature adrenarche**

-Pubic/axillary hair + body odour between age 4 – 7

-Exclude exogenous androgens, late onset CAH, adrenal tumours

-Once excluded = NO TREATMNET, REASSURANCE

***True precocious puberty**

-Prem activation of hypothalamo-pituitary-gonadal axis

-may be due to tumours near hypothalamus (boys), TB meningitis, encephalitis, hydrocephalus

-Frequently in year 1 of life, more common in girls

-Increase in total body growth + sexual development

-Immediate referral + suppressive therapy : *1.)cyproterone acetate 100mg bd*

2.)long acting GnRH analogues

***Precocious pseudopuberty**

-2nd ary sexual characteristics due increased sex steroid activity without axis being activated

-Exclude exogenous administration, gonadal/adrenal tumour, CAH

***Delayed puberty**:

-Consider if no breast development by age 13 – 14 or testicular growth by age 14 – 15

-Exclude undernutrition/long standing chronic disease

-In girls, look for organic causes eg Turners syndrome

-In boys, simple physiological delay more common

-Treat with long acting testosterone 50 – 100 mg IM monthly for 4 months

3.) Water balance disorders

***SIADH**

-Increased water reabsorption by kidneys, = dilutional hyponatremia

-Nausea, vomiting, muscle weakness, oedema, convulsions, coma

-most commonly seen in TB meningitis, head injury

-restrict intake in these pt's, look for weight loss + steady rise in sodium levels

-Emergency mx: *3% saline IV (5ml/kg) combined with furosemide (1mg/kg)*

***Diabetes Insipidus**

-def of ADH or due to lack of effect of ADH

-Polyuria, Polydipsia, dehydration, large volumes of diluted urine

-Administer Vasopressin:

If pt responds :Central	**No response: Nephrogenic**
-organic lesions of hypothalamo-pituitary area Eg trauma, post surgery, tumour	-Genetic X-linked condition, mostly in boys
-Administer Vasopressin	-High water intake -Frequent feeding -HCTZ + Indomethacin

***Diabetes Mellitus type I**

-Often present in DKA (see clinical aspects)

-test for ketones + glucose in all pt's with rapid breathing, abd pain, vomiting, dehydration, decreased LOC

Mx:
-0.9% Saline IVI 1L stat -1L over next hour, next 1L over 2 hours, next 1L over 4 hours, next 1L over 6 hours -4 – 8 units of soluble insulin if plasma glucose >20 -add 2ml of a 15% KCL in every 200 ml saline post insulin administration -use 5%dextrose when glucose <15 -Insulin sliding scale then followed: -cont fluid + K replacement -carefull monitoring

4.)Thyroid disorders

Hypotyhroidism	Hyperthyroidism
-infants of mothers with severe iodine def + goitres -open post fontanelle (>1cm), umbilical hernia, poor sucking, coarse facial features	-infants from mothers with Graves disease -behavioural disturbances, nervousness, sweating, nocturnal enuresis
-Treat with Thyroxine	-Treat with anti-thyroid drugs, subtotal thyroidectomy

5.) Adrenal Cortex disorders

***CAH**

-Autosomal recessive def in cortisol synthesis

-increase in ACTH resulting in adrenal hypertrophy + virilisation of external genetalia

-most common = 21 – hydroxylase deficiency

-suspect in newborns with ambiguous genetalis + impalpable gonads

-electrolyte disturbances present

-Acute Mx: 1.)Bloods for electrolytes, acid-base, urea, cortisol assay

2.)0.9 % NaCl in 5% dextrose @ 20ml/kg in 1st hour, then 60ml/kg over next 24 hours

3.)Solucortef as IV bolus (50mg for small children 100mg for larger children) followed by either 50mg or 100 mg IVI/24hrs added to the maintenance

4.)9-alpha fluorohydrocortisone 0.05 – 0.1mg/ day orally

-when pt is stable, should be referred to tertiary institute for life long maintenance on oral hydrocortisone

***Acute adrenal insufficiency**

- Inadequate secretion of glucocorticoids and mineralocorticoids result in salt loss, hypoglycaemia and circulatory collapse.

-can be caused by infection (TB), enzyme deficiency, haemorrhage, auto-immune disease, steroid therapy, CAH

-High K, low to low-normal Na + a poorly compensated metabolic acidosis

-Medical EMERGENCY! (See acute mx of CAH)

***Addisons disease**

-Adrenal cortex atrophy due to auto-immune disease, infection (TB), metabolic disorder

-Pt's are weak, anorectic, vomiting, diarrhoea, dehydration, and hypotension

-poor response to ACTH stimulation test

Life long treatment with oral hydrocortisone

*Hyperadrenocorticism

-Hyperfunctioning cortex may present with Cushing's syndrome or marked virilisation (depending on specific steroid secretion)

-Cushing's syndrome causes:

1.)Excess steroid therapy (common)

2.)Adrenal tumour (associated virilisation)

3.)ACTH-secreting micro-adenoma

4.)Ectopic ACTH-secreting tumours (rare)

-Characteristic moon face, buffalo hump, truncal obesity

-Growth failure + retarded bone age

-muscle weakness, thinning skin, purple skin striae

-hypertension + virilisation may be present

-24 hour urine cortisol secretion is diagnostic

-surgical resection

6.)Parathyroid disorders

Primary hyperparathyroidism	Secondary hyperparathyroidism	Hypoparathyroidism
-very rare in children -clinical sx's of hypercalcaemia -high calcium, low phosphate with normal urea -confirmed by PTH levels -Surgical removal indicated	-more common -due to chronically decreased calcium eg. Vit D def or chronic renal failure -resultant parathyroid hyperplasia due to overstimulation -Treat with vit D	-1.)congenital -presents within a few days - months -2.)idiopathic acquired -presents later

ALLERGIC DISORDERS:

1.)Allergic Rhinitis

Seasonal allergic rhinitis	**Persistent allergic rhinitis**
-seasonal -usually precipitated by pollens, grass, spores -intense sneezing, nasal d/c, itching, itching of palate +auditory canals as well -Dx easily made	-year round -caused by sensitivity to everyday exposure to household allergens (dust, mites) -itching not common, but nasal d/c +congestion -typical pale allergic facies + blue discolouration of lower eyelids -allergic "salute" crease on nose -glue ears
-good hx, skin testing, CAP-RAST	
Treatment: -impossible to avoid seasonal allergens -non-sedative anti-histamines e.g. loratadine + cetrizine -Intranasal steroid sprays e.g. budesonide + fluticasone -desensitisation (very effective)	Treatment: -Environmental control NB! -non-sedative anti-histamines -Intranasal steroid sprays -desensitisation -Oral steroids (only if very severe symptoms)

2.)Food allergies

-very rare

-vomiting, diarrhoea most common symptoms

-other symptoms include skin reactions, nasal obstruction, wheezing

-avoidance is strongly advised

CONNECTIVE TISSUE DISORDERS

LEANIE - VICTORIA

Childhood Rheumatological Disorders

- →JIA
- →CT Disease
  - -SLE
- →Vasculitides
  - -HSP
  - -Takayasu's arteritis
  - -Kawasaki disease

Connective Tissue Disorders

Childhood Rheumatological Disorders

- →JIA
- →CT Disease
  - -SLE
- →Vasculitides
  - -HSP
  - -Takayasu's arteritis
  - -Kawasaki disease

Connective Tissue Disorders

The following Rheumatological problems may be encountered:

1. PUO
2. Eyes-Conjunctivitis, uveitis
3. Skin: Palmar erythema
   Maculopapular rashes or linear rashes
4. Joints: Mono or polyarthritis
5. Reticulo endothelial system activation
   LN's not matted together
   HSM
6. Organ dysfunction

Childhood Rheumatological disorders

- Juvenile idiopathic arthritis
  - Systemic onset JIA
  - Oligoarticular JIA
  - Polyarthritis
  - Psoriatic arthritis
  - Enthesitis related arthritis (enthesitis= local tenderness at insertion of tendon, ligament or joint capsule into bone)

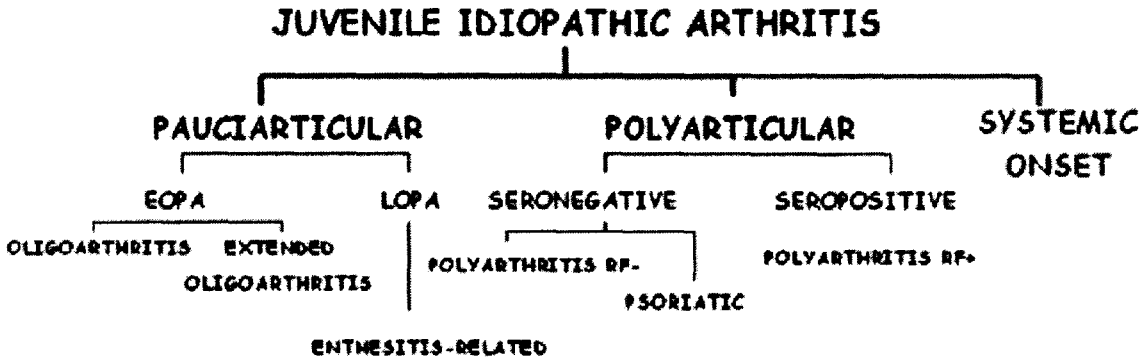
- CT Diseases
  - SLE
  - Juvenile dermatomyositis
  - Scleroderma

- Vasculitides
  - Henoch-Schonlein purpura
  - Kawasaki disease (mucocutaneous LN syndrome)
  - Takayasu's arteritis
  - Poly-arteritis nodosa

- Skeletal dysplasias
  - Osteochondrosis: Perthe's disease

- Infectious and post-Infectious Artheritis

Juvenile idiopathic arteritis



Criteria:

Age of onset <16
Arthritis in one or more joint
Duration of disease > 6 wks
Exclusion of other forms or arthritis
Different onset patterns

Systemic: Persistent high spiking fever and arthritis, HSM or pericarditis and rash
Oligoarticular: up to four joints within the first 6moths
Polyarticular: five or more joints within the 1st 6 months
Enthesitis related: inflammatory spinal pain and enthesitis

Complications:

- Pain malaise, irritability anorexia
- Contractures and deformities
- Joint failure
- Anaemia
- Chronic anterior uveitis
- Growth disturbances

S/I

- WCC
- Platelet count
- CRP
- RF and ANA
- Arthroscopy and synovial biopsy
- X-rays and CT

Mx

Relieve symptoms: Anti-inflam!

- σ Step 1: NSAIDS: Aspirin, ibuprofeb, indomethacin, diclofenac
- σ Step 2: Cytotoxic agents: methotrexate
- σ Step3: Prednisone Intra-articular injection

Monitor for compx
Physio and OT

CT diseases:

SLE:

- Multisystem autoimmune disease, underlying pathology is vasculitis affecting small bld vessels
- Neonatal SLE: placental transfer of maternal auto-antibodies. Mother's SLE may be sub-clinical
- Most NB congenital consequence: heart block.
- Most common compx= renal failure or chronic nephritis

Dx: at least 4 of the following:

1. Malar rash
2. Discoid lupus
3. Photosensitivity
4. Oral or nasopharyngeal ulcers
5. Arthritis
6. Pleuritis or pericarditis
7. Proteinuria or cellular casts
8. Anaemia
9. ANA
10. LE cells, anti-native DNA Ab's, anti-SM Ab's, false +ve WR
11. Neuro: psychosis or convulsion

Mx: High dose CS-
Refer to specialist rheumatologists.

Vasculitides:

Henoch-Schonlein purpura: HSP

- ♣ Most common vasculitis in childhood
- ♣ 2/3 of cases are transient, 1/2 will recur with diminishing severity
- ♣ Symptoms are self-limiting, but renal disease will persist

Symptoms:
- ♣ Abdo pain
- ♣ Palpable purpura over the buttocks and pressure bearing areas
- ♣ Large joint arthrits
- ♣ Rash can be urticarial or maculopapular in atypical cases.

C/S:
- ♣ Nephritis
- ♣ Microscopic haematuria, proteinuria and HT

Mx:
- ♣ Supportive: Pain control, maintenance of hydration and nutrition.
- ♣ GIT and joint involvement: prednisone
- ♣ Refer if renal disease present! Rx: steroids, azathioprine, IV immunoglobulins

Takayasu's arteritis

- Arteritis confined to aorta and large vessels
- Occurs mainly in older girls

Sx and Sg's:
- Acute phase: fever, arthralgia myalgia and fatigue.
- Later: symptoms due to arterial occlusion (stenosis, thrombosis or aneurysmal dilation:
  - Sv HT and visceral ischaemia
  - Cerebral anoxia
  - Absent or reduced pulses

S/I: angiography

Rx: control HT and convulsions
If in active phase **(RAISED ESR AND IgG)**
CS and Cyclophosphamide
Vascular surgery to correct stenosis.

Kawasaki disease

- Acute multisystem disease
- Small to medium sized arteritis
- Sx's last for a few weeks and resolve spontaneously.

Dx: At least 5 of the following:
- Fever≥ 6 days
- Bilat conjunctivitis
- Oropharyngeal changes: Mucosla erythema, dry, strawberry tongue
- Erythema of palms and soles
- Polymorphous exanthem of trunk without vesicles or crusts
- Acute non-purulent cervical lymphadenopathy of >1.5cm

* 20% develop coronary artery aneurysms-can be fatal!

S/I: WCC, ESR and Platelets.

Rx: high dose aspirin for two weeks, low dose for for 2 months.
IV gammaglobulins

Neoplastic Disorders

LEANIE-VICTORIA

- Leukemia
- Lymphomas
  - →Non-Hodgkin's Lymphoma
  - →Hodgkin's Lymphoma
  - →Malignant B-cell Lymphoma
  - →Lymphoblastic Lymphoma
  - →Large cell Lymphoma
- Malignant Solid Tumours
  - →Nephroblastoma
  - →Neuroblastoma
  - →Rhabdomyosarcoma
  - →Retinoblastoma
- Liver Tumours
  - →Hepatoblastoma
  - →Hepatocellular Ca
- Germ Cell tumours
- Osteogenic Sarcoma
- Ewing's Sarcoma
- Kaposi's Sarcoma

Neoplastic Disorders

Neoplastic Disorders Neoplastic Disorders Neoplastic

- **Leukemia**
- **Lymphomas**
  - →**Non-Hodgkin's Lymphoma**
  - →**Hodgkin's Lymphoma**
  - →**Malignant B-cell Lymphoma**
  - →**Lymphoblastic Lymphoma**
  - →**Large cell Lymphoma**
- **Malignant Solid Tumours**
  - →**Nephroblastoma**
  - →**Neuroblastoma**
  - →**Rhabdomyosarcoma**
  - →**Retinoblastoma**
- **Liver Tumours**
  - →**Hepatoblastoma**
  - →**Hepatocellular Ca**
- **Germ Cell tumours**
- **Osteogenic Sarcoma**
- **Ewing's Sarcoma**
- **Kaposi's Sarcoma**

Neoplastic Disorders

Neoplastic Disorders Neoplastic

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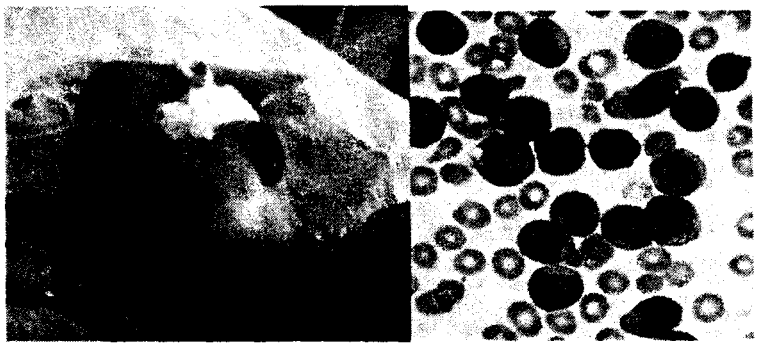
Neoplastic Disorders:

Leukemia



- Def: Uncontrolled proliferation or defective maturation of WBC's
- Most childhood leukemias are of the acute subtype:
  - Acute lymphocytic leukemia (ALL) 1—5 yrs
  - Acute non-lymphocytic leukemia (ANLL) AML
- ALL has the highest incidence btwn 1 and 5 yrs.
- Chronic Myelocytic leukemia is rare and chronic lymphocytic leukemia does not occur in children

Sx & Sg's: (in order of prevelance)

- Fatigue and pallor
- HSM
- Fever
- Bleeding or bruising
- Lymphadenopathy
- Bone pain

Dx:

FBC: Hb < 10/dL, normochromic and normocytic
Thrombocytopaenia <100
WCC: normal, raised, or low
Abnormal WC's (blasts) on smear

Bone marrow aspirate: this is a diagnostic test and stages and classifies the disease.

CXR: mediastinal involvement

LP: assess for blasts in CSF

Mx:

- ChemoRx
- Supportive Rx
- Preventative Rx: Allopurinol (tumour lysis syndrome)
  Cotrimazole (pneumocystis carinii)

severe infxn + Metabolic syndrome (2° to tumor lysis syndr)
- Leukostasis (in 1st week after Dx)
- Recurrence
- Rx

*treat oppurtunistic infections accordingly. Keep a high index of suspicion for TB, H. Zoster, HSV and V. Zoster.

Prognosis

70% of ALL pt's are cured permanently.
Reduction in blast count to <1.0 after a week of prednisone indicates good prognosis

Lymphomas

Histological Classification:

Non-Hodgkin's lymphoma
Malignant B-cell lymphoma
Hodgkin's Lymphoma
Lymphoblastic lymphoma
Large-cell lymphoma

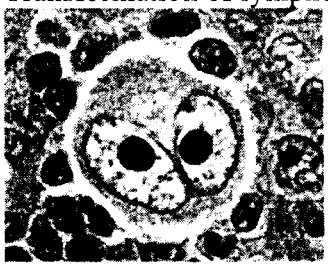
Non-Hodgkin's lymphoma

4 types: mature B cell (Burkitts) / mature T-cell + natural killer cell / precursor pre-B lymphoblastic lymphoma / precursor T cell

Highly malignant
Major sub groups: Undifferentiated (Burkitt's, non-Burkitt's- B-cell origin)
Lymphoblastic (T-cell)
Large-cell histiolcytic
Dx: fine needle aspiration, Biopsy (bibopski :)
Staging: CXR, CT, CSF analysis, U/S, bone marrow examination

Murphy staging system:
Stage I: Single nodal or extranodal site (not in abdo or mediastinum)
Stage II: 1 or more extranodal sites + regional LN OR 2 extranodal sites on the same side of the diaphragm
Stage III: 2 or more site on both sides of the diaphragm including all primary intrathoracic and extensive abdo tumours.
Stage IV: I to III + bone marrow involvement (<25% infiltration) and/or CNS disease

Hodgkin's Lymphoma
Transformation of lymphocytes into *Reed-steinberg multinucleated giant-cell.*



EBV is thought to play a roll
Male predominance. Rare before the 3YOA.

Classification: Nodular lymphocyte predominance
Classical HL (95%)

Dx: Biopsy or FNA

Most common presentation: painless cervical lymphadenopathy. Systemic signs: LOW, night sweats, pruritis, pyrexia

Also: enlarged inguinal or axillary lymphadenopathy, hepato/splenomegaly

Staging:

The principal stage is determined by location of the tumor:

Stage I : single region, usually one lymph node and the surrounding area.

Stage II : two separate LN regions, confined to one side of the diaphragm

Stage III : LN regions both sides of the diaphragm, including one organ or area near the lymph nodes or the spleen.

Stage IV : one or more extralymphatic organs

Modifiers: These letters can be appended to some stages:

A or *B:* the absence of constitutional (B-type) symptoms is denoted by adding an "A" to the stage; the presence is denoted by adding a "B" to the stage.

E: is used if the disease is "extranodal" (not in the lymph nodes) or has spread from lymph nodes to adjacent tissue.

X: is used if the largest deposit is >10 cm large ("bulky disease"), or whether the mediastinum is wider than 1/3 of the chest on a chest X-ray.

S: is used if the disease has spread to the spleen.

NHL

Malignant B-cell lymphoma

Peak age: 7.

Predominantly male

Two types: Endemic Burkitt's lymphoma and Sporadic Burkitt's lymphoma.

Theory: EBV infection at young age stimulate B-cell proliferation.

swollen mandible & maxilla → nasopharynx & orbit

Endemic Burkitt's:

Most common presentations:
1. Swelling of the maxilla or mandible + extension to the nasopharynx nose and orbit.
2. Abdo disease, ascites+ infiltration of retroperitoneal organs.
3. CNS involvement in: paraplegia, CN palsies

Sporadic Burkitt's:

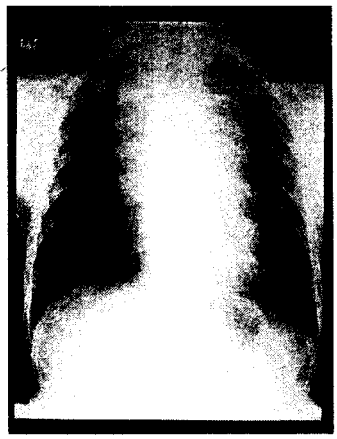
Most common presentation:
1. Abdo mass (ileocaecal region)
2. Primary tumour in head and neck region

Rx (for both)5 months chemo course. Extremely sensitive to chemo!!.
1 year survival rate= >95%

Lymphoblastic Lymphoma:

< precursor B
precursor T - sensitive to steroids clinically)

mediastinal mass



Peak age: 12 T cell = older than B
Male predominance.
Most common presentation:
Mediastinal mass.
Rapidly enlarging painless cervical, supraclaviculare and axillary LN.
Rx: Chemo!

tumours grow and cause obstruction of large vessels → emergency resp distress = dyspnoea dysphagia pain swelling - neck face UL

Rx low dose radiotherapy + steroids

B lymphoma

Large Cell Lymphoma

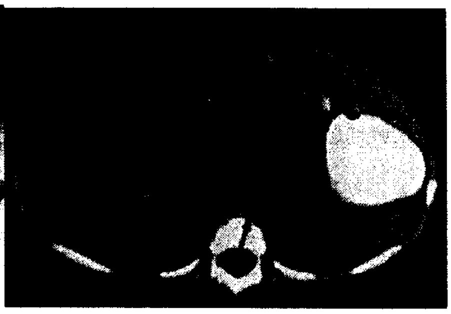
—tumours in GIT / mesentery / abd
└ wandering ring

Most uncommon type.
Occurs in older children, male predominance.
Assoc with immune def and inheritance.
Rx: chemo and radioRx.

Malignant Solid Tumours:

Nephroblastoma (Wilm's tumour)

Diff Dx - neuroblastoma
hydronephrosis
polycystic kidneys
mesoblastic nephroma (benign)
renal cell carcinoma

assoc congen abN
WAGR syndr - Wilms, aniridia, genital, mental retardation
Beckwith Wiedemann synd
Denys Drash

Most common solid tumour in RSA.
Boys and girls equally affected, peak age: 1-5 YOA
Assoc with congenital abN: Aniridia, hemihypertrophy, urogenital abN.

(large, firm, irregular, nodular)

C/Presentation: Abdo mass,usually painless. Fever, abdo pain, HT, haematuria.

S/I: FBC, UKE, LFT, urinalysis, level of catecholamine excretion (exclude neuroblastoma)
U/S, CXR, CT/MRI, IVP (optional) FNA

Staging: USA National Wilm's Tumour Study System (NWTS)
Stage I: tumour limited to kidney and completely resected. 5 yr survival >90%
Stage II: completely resected, but extends beyond kidney.
Stage III: Residual tumour confined to Abdo
Stage IV: Bilateral renal involvement. 5 yr survival: 50%

Rx: shrink tumour with chemo before surgery.
All children need post-op chemo.
RadioRx added in Stage II-IV

Neuroblastoma:

* Ca in 1st yr of life = neural crest cell tumour

in primitive sympathetic cells

Origin: Sympathetic NS. Most common site: adrenals. also along thorax / cervical region
Spinal cord tumour=>"dumb bell tumours" thru neural foramen
extending through neural foramina.
80%: produce catecholamines.
Dx: detect homovanillic acid (HVA) and vanillyl mandelic (VMA) acid in urine.

Mets * - periorbital ecchymosis
- bone pain
- hepatomegaly "blue berry muffin"
skin nodules

C/Sx's:
Depends on site of disease.
Abdo disease: Large irregular tumour, digestive Sx and pain
Thoracic tumour: Resp sx's
Head and Neck; Horner's
Pelivic: Urinary Sx's and disturbed bowel movements.

Staging
I: localised tumour, microscopically excised
IIA: unilateral, incomplete excision
IIB: like IIA with ipsilateral regional LN
IV: Mets to distant LN, bone or bone marrow, liver, skin, or other organs.
IVS: localised primary tumour with spread limited to liver skin and/or bone marrow.

Diagnositc Criteria of Neuroblastoma

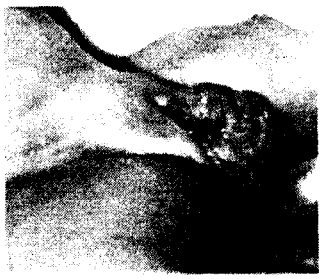
1. Histo Dx OR
2. Marrow infiltrations + catecholamines in urine
* Abdo XR may show calcifications in tumour
* Abdo U/S, CT, MRI used to determine extent
* bone marrow aspirate used for staging.

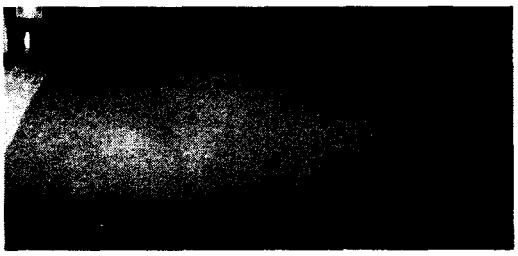
Treatment:
Stage I and IIA: surgery
Stage IIB and III: chemo followed by resection
Stage IV: bone marrow transplant. If not possible: palliative Rx.

Rhabdomyosarcoma


Orbital


½ in Head + Neck


Genital ¼


Left Arm

Most common soft tissue sarcoma of childhood - striated mm/precursor cells of this mm.
Some fams carry an autosomal dominant gene.
Tumour presents as soft tissue mass, depending on site, can be confused with:
Orbit: Retinoblastoma, neuroblastoma, Burkitt's lymphoma. Extremities: acute abscess.
Peak Age: 5yoa.
50%: Head & Neck region
25%: Genitourinary system

Dx: FNA/biopsy
Rx: primary total surgical resection, chemo. RadioRx for inoperable tumours.

Retinoblastoma

Cause: Loss of both of a pair of anti-oncogenes one gene is long arm of chromosome 13 in developing retinal cell.
Usually unlilat, peak age of presentation: 2years.
Local spread: into orbit or along optic nerve and intracranial extension.
Later: haematogenous spread to bone marrow.

C/S: white spot in the pupil (leukocoria or "cat's eye reflex"), a squint, proptosis, orbital mass.

DDx: neuroblastoma, rhabdomyosarcoma, Burkitt's lymphoma, visceral larva migrans.

S/I:
Opthalmological EUA, local XR and skeletal survey, CT or MRI, bone marrow and CSF exam.

Rx:
Small lesions: photocoagulation or localised radioRx (brachioRx)
Large lesions, still confined to eyeball: enucleation
Optic nerve infiltration: Add chemo and radioRx
Advanced and mets: palliative.

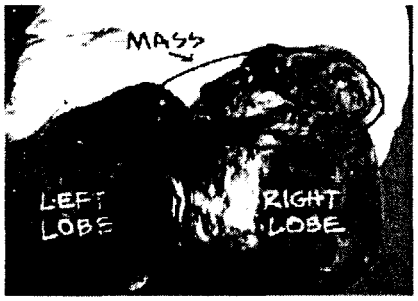
F/U: regular opthalmological check-ups until 5yoa.

Liver Tumours

1. Hepatoblastoma
2. Hepatocellular carcinoma

Presentation: abdo swelling, enlarged, irregular, firm liver +/- tenderness.
S/I: increased serum alphafetoprotein
Prevention: routine vaccination of infants against Hep B
Common mets: lungs

Hepatoblastoma: male predominance, peak presentation before 3yoa. Usually R lobe.
Other anomalies: virilisation and hemihypertrophy.
Hepatocellular carcinoma: rarely occurs before 6yoa. Worse prognosis.



Dx: Abdo mass on sonar + raised s.alfphafetoprotein.+ child <4yoa
CXR: Exlude lung mets

Rx: Shrink with chemo ---> resect ---> post-op chemo
Liver transplant in unresectable disease

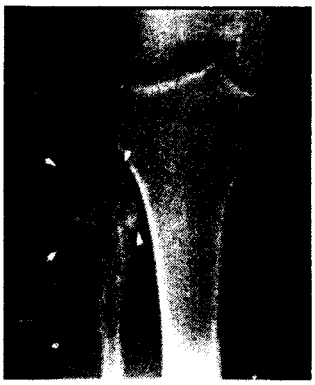
Germ Cell Tumour

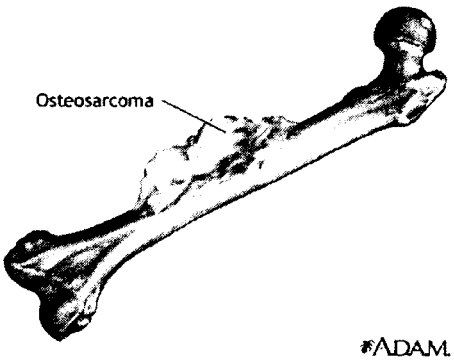
Tumour develops from embryonal germ cells.
Presents as ovarian or testicular tumour. Most common extragonadal site: sacrococcygeal region.
Tumours secrete alphafetoprotein and HCG.

WHO classification:
*Mature teratome
*Embryonal carcinoma
*Immature teratoma
*Yolk-sac tumour
*Germinoma (testes: serminoma. Ovary: dysgerminoma)

Rx: combo of chemo and primary or delayed resection cures most children.

Osteogenic sarcoma



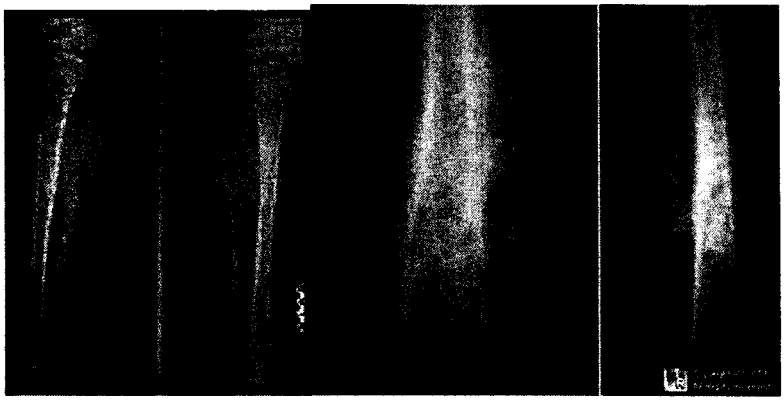


Osteolytic lesions, margins poorly defined. Cortex is breeched:
"sunray" appearance
Codman's triangle: new bone between perios and cortex.
Mataphysis!!

Commonest primary bone tumour in kids
Median onset: 12yoa
Presentation: localised swelling and pain.
Most commonly affected: femur, tibia and humerus
DDx: Osteomyelytis, traumatic #, lymphoma adn eosinophilic granuloma.
Dx: Biopsy, CXR, CT or MRI

Rx: 50% can be cured: Chemo and resection/amputation.

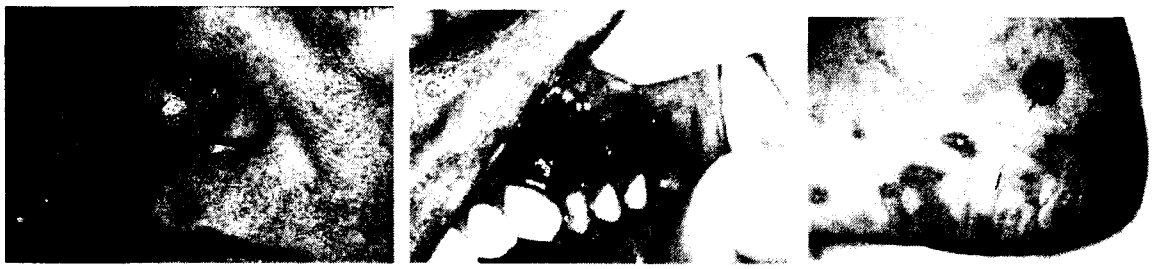
Ewing's Sarcoma



Bone destruction: Mid diaphysis
Onion-peel effect.

Arrise from endothelial cells in the bone marrow., most commonly involve pelvis, femur, humerus and ribs
Presentation: Local swelling and pain.
Rx: as above.

Kaposi's Sarcoma:



Most important cause of gen lymphadenopathy.
Assoc with wasting anaemia and HIV+ve.
Clinical course is rapidly progressive and die within weeks.
Dx: skin biopsy. CXR and HIV test for counseling purposes.
Rx: palliative

POISONING

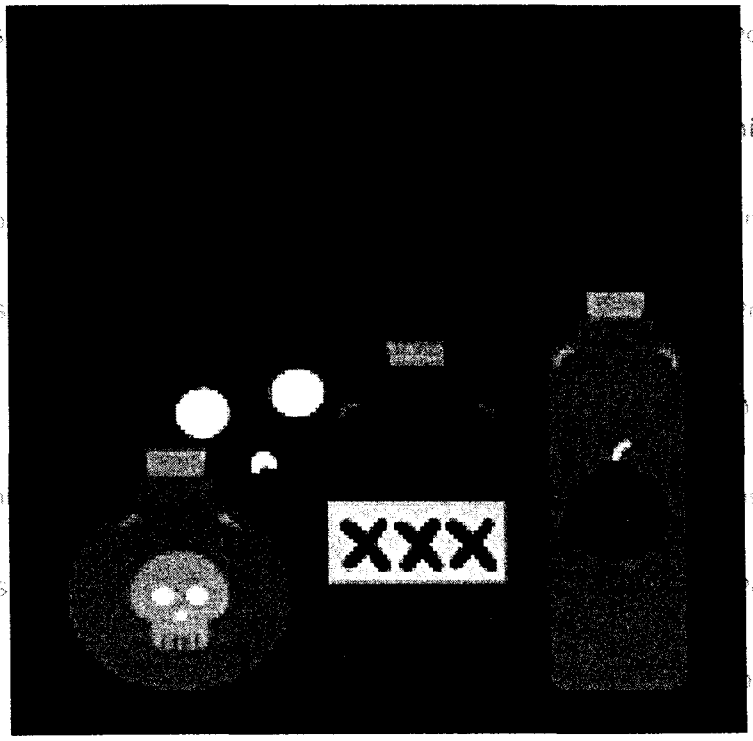
WYNAND - PIETERMARITZBURG

ROUTES OF EXPOSURE
SPECIFIC POISONS:
→PARACETAMOL
→ALKALIS & ACIDS
→ALCOHOL
→INSECTIDES
→CARBON MONOXIDE
→BELLADONNA & ATROPINE
ANTIDOTES

Poisoning

Routes of exposure
Specific poisons
- **→Paracetamol**
- **→Alkalis & Acids**
- **→Alcohol**
- **→Insectides**
- **→Carbon Monoxide**
- **→Belladonna & Atropine**

Antidotes



Poisoning

Routes of exposure	+	Method of preventing absorption or enhancing elimination
Direct eye contact	-	Remove contact lenses + **irrigate** with N saline for @ least 15min. Examine for corneal damage
Inhalation	-	O2 & bronchodilators (B2 agonist of wheeze)
Ingestion	-	If at home, a glass of H2O or milk can be given **Emesis** – Ipecacuanha 10 – 15ml syrup, followed by a glass of water. Within 6h of ingestion (12h for salicylates and TCA) Contra-indications: ↓ LOC (any ↓ coughing/swallowing reflexes. Convulsions and coma. Poisoning with petroleum products, paraffin, hydrocarbons (benzene/turpentine), corrosive products, acids and alkalis **Gastric lavage** – Life threatening, within 1h, not with acids, alkalis or petroleum distillates **Activated charcoal** - 30g with 150ml water, either po or through NGT. Used for Aspirin, phenobarbitone, carbamazapine, phenothiazines, TAD, phenytoin, dapsone, quinine, digoxin, theophylline/aminiohylline **Whole body irrigation** – Polyethylene glucol po, in iron, lithium and theophyllene
Blood	-	**Urinary alkalinisation** with 1,26% NaHCO3 for salicylates and phenobarbitone (watch for ↓ K^+) **Haemodialysis** – Salicylates, theophylline, ethylene glycol, methanol, carbamazepine
Direct Skin contact	-	**Remove clothing, wash** skin thoroughly with water

Common Specific Poisons

Paracetamol

Poisoning either accidental (usually small children) or suicide attempt (adolescents)
Causes centrilobular hepatic necrosis. Renal tubular necrosis possible.
Hepatotoxic dose: 150mg/kg
Clinical features: Usually delayed for 48 – 72hours
Initially GIT: Nausea, vomiting, abdominal pain
Later Liver: Jaundice + signs of liver failure
Management: → fatty liver

- ABCs
- Activated charcoal po, unless antidote has to be given po.
- Measure plasma paracetamol levels and plot on curve (plasma levels on a time scale)
- Do not take blood in first 4h as plasma levels have not peaked yet. If substantial OD, start Acetylcystine ASAP. Rather discontinue later if necessary.
- If levels above risk line on graph, start acetylecystine treatment following a 48h regime:
  First 15min: 150mg/kg
  Next 4h: 50mg/kg
  Next 16h: 100mg/kg
  Next 24h: 150mg/kg
  (Work out in how much fluid it needs to be dissolved according to the child's weight)
- Monitor blood glucose
- Limit fluid (paracetamol – fluid retention)

Acelylcystine
- Gluthatione donor
- Use with caution in asthmatics
- Monitor K^+ and BCG
- Relatively safe in pregnancy

Salicilates

Mostly accidental, some preparations taste pleasant.
Mild: >150mg/kg
Direct stimulation of respiratory system → Respiratory alkalosis
Sx's: Nausea, vomiting, tinnitus, deafness, tachypnea, fever
Moderate: > 250mg/kg
Sx's: Profuse sweating, peripheral vasodilatation, bounding pulses
Petechia, subconjunctival haemorrhages, haemorrhage
Hypoglycaemia
Dehydration
Severe: >500mg/kg
Sx's: Metabolic acidosis, due to uncoupling oxidative phosphorilation
Sg's: Convulsions, coma, renal or liver failure

Management

- ABCs
- Induce emesis or gastric lavage
- Administer activated charcoal
- Rehydration (beware of pulmonary oedema)
- Test: Serum salicylate level, U&E, arterial blood gas
- Correct acid-base imbalance
- Forced alkaline diuresis
- Dialysis in severe cases

Tricyclic antidepressants

Children can become very sick from a small amount of TAD due to the narrow therapeutic/toxic ratio.

Acts by blocking acetylcholine, nor-adrenaline, α-adrenergic, serotonin, and 5-hydroxitryptamine and dopamine reuptake → anticholinergic syndrome.

Sg and Sx: Drowsiness, dry mouth, papillary dilatation, excitability, hallucinations, convulsions and coma.

Cardiac arrhythmias → Tachycardia/tachyarrhythmia
Cardiac arrhythmias → Bradcardia/bradyarrhythmia
Cardiac arrhythmias → Direct suppression of myocardial contraction

Management

- ABCs
- Induce emesis (unless c/i)
- Gastric lavage
- Administer activated charcoal (repeated doses of 15-30g q4h)
- NaHCO3 titrated to blood pH of 7,45-7,55
- Anti-arrhythmic drug depending on type of arrhythmia

Antimalarials

Quinine: Fatal dose - 900mg in children
Stimulates pancreatic β cells → ↑insulin → hypoglycaemia
Causes retinal vasoconstriction
Quinine, mefloquine and halofantrine can cause arrhythmias
Chloroquine can cause hypocalaemia

Sx: Blurred vision, constriction of visual fields, blindness, dilated pupils,
Nausea, vomiting
Tinnitus, deafness
Headache, tremor, ataxia, drowsiness, coma, respiratory depression
Hypotension and arrhythmias

Management

- ABCs
- Induce vomiting
- Administer activated charcoal
- Cardiac monitoring and treated as indicated
- Diazepam has protective effect with choloquine poisoning

Alkalis and acids

Found in many domestic cleaning products. Children may accidentally ingest these.
Alkalis tend to cause more damage than acids by binding with fats and oils in tissue and causing necrosis.
Alkalis cause damage to the oesophagus whereas acids cause damage to the stomach.
The mouth and sometimes the eyes are also involved.

Clinical: Acute inflammation and ulceration of the mouth and oesophagus and eyes.
Tissue necrosis may lead to stricture formation.

Management:

- Milk/water po to dilute
- Opiods for pain
- CXR to exclude perforation
- Oesophagoscopy may be performed after 48h
- Surgery as indicated

DO NOT

- Try to neutralize the chemical
- Induce emesis
- Insert a NG tube
- Perform a gastric lavage

Tranquilizers, sedatives and hypnotics

All cause a depression of CNS ranging from drowsiness to coma. In addition the specific classes cause the following:

Benzodiazepines: ↓ CNS + Respiratory depression
Phenothiazines: ↓ CNS + extrapyramidal Sg (might mimic psychotic episode)
Barbiturates: Initially confusion, ataxia, hypotension and miosis.↓ CNS + Respiratory depression
Antihistamines: Might initially have hyperexitability. ↓ CNS + anticholinergic effect (dry mouth, fever and dilated pupils).

Management

- ABCs (ventilate if respiratory depression)
- Gastric lavage
- Activated charcoal
- Physostigmine (0.5 – 2mg to reverse anticholinergic effects of antihistamines)

Alcohol

Ethyl alcohol found in alcoholic beverages, comparing with methanol (methylated spirits) and ethylene glycol (found in antifreeze, industrial solvents and cleaners).

Clinical features: Depresses CNS → sedation, ataxia, slurred speech followed by progressive incoordination, stupor, coma and respiratory failure. Cerebral oedema is also possible in severe cases.
Interferes with glucose metabolism → Hypoglycaemia (sweating, tachycardia, convulsions)

Management:

- ABCs (protect airway!)
- DO not induce emesis if ↓ consciousness
- Administer activated charcoal
- Check blood glucose and alcohol levels
- Hydrate with glucose replacement if necessary
- In case of methanol or ethylene glycol poisoning, give ethanol → competitively inhibits damage to brain and liver

Insecticides

Organophosphates and cabamates

Inhibits cholinesterase → ↑acetycholine

Clinical picture (Sequential)

1. Acute cholinergic syndrome

Within minutes of exposure

Muscarinic effects: ↑Secretion – lacrimation, urination, diarrhoea, miosis, bradycardia, bronchorrhoea, bronchospasm

Nicotinic effects: ↑Depolarization – Muscle fasciculation, hyper-reflexia, flaccid muscles

CNS effects: Headache, dizziness, confusion, drowsiness, coma, fits, central respiratory depression

Pt also presents with garlic breath

2. Intermediate syndrome

±48h after exposure in ±20% of patients

Presents with muscle weakness, respiratory distress + paralysis of muscles progressing over 24h. May develop over or continue for 2 – 18 days.

3. Organophosphate induced delayed polyneuropathy

Presents 1-3 weeks post-exposure

Sg & Sx:

Cramping muscles pain. Numbness in distal upper & lower limbs

Weakness follows & spreads to hands. Shuffling gait, wrist drop

Muscle wasting, sensory loss, ↓tendon reflexes

Management

- ABCs

Cholinergic syndrome

- Remove clothing and wash child (take precautions for yourself)
- Induce emesis if conscious or gastric lavage
- Activated charcoal via NG tube
- Atropine 0.05mg/kg initially, followed by 0.02mg/kg every 15 min until salivation stops and pupils begin to dilate
- If convulsing, administer diazepam
- Oximes (pralidoxime or obidoxime) must also be given if available (reactivates phosphorylated AChE)
- Monitor child's ECG, ABG, temperature, UCE, amylase and glucose

Intermediate syndrome

- Ventilatory support before the child in respiratory failure
- Sedate using diazepam/midazolam

Organophosphate induced delayed polyneuropathy

- Physiotherapy to reduce deformity caused by muscle wasting

Hydrocarbons and Petroleum products

Local: Leaking of oils and fat from skin → local irritation

Aspiration/inhalation: Severe necrotizing and haemorrhagic pneumonitis. Causes coughing, tachypnoea and dyspnoea

Systemic: CNS depression, sensitization of heart muscle, liver- and kidney damage

Management

- ABCs
- Supportive – hydration and ventilation where necessary.
- NO EMESIS OR GASTRIC LAVAGE

Iron

Children might ingest their mother's antenatal supplements as it resembles sweets.

Clinical features:

Early: (0-12h) Causes local irritation and necrosis of intestinal mucosa → vasodilatation, inflammation and bleeding → Vomiting, abdominal pain, haematemesis

Intermediate: (12-24h) Absorbed iron accumulates in mitochondria, interfering with electron transfer across membrane → organ damage (especially liver) → Sx of shock + metabolic acidosis, fever, hypotension. Bleeding tendencies and hypoglycaemia may also develop.

Late: Pyloric stenosis due to fibrosis after local corrosive effect.

Management

- ABCs
- Induce emesis
- Gastric lavage
- Instillation of desferrioxamine (1g diluted in 1l of water with NaHCO3)
- IV desferrioxamine (slow infusion of 15mg/kg/h)
- Peritonial or haemodialysis in severe cases

Carbon Monoxide

CO binds to Hb → carboxyhaemoglobin.

t½: 200min in room air vs 40min in 100% oxygen

Leads to hypoxia → cerebral oedema and damage to cardiac muscles and organs.

Clinical:

Initial: Headache, dizziness + progressive ↓LOC

Intermediate: Tachypnoea, tachycardia (as hypoxia sets in)

Ultimately: Convulsions, coma, respiratory and circulatory failure

NB: Do ABG, do not believe saturation monitor!!

Management:

- ABC
- 100% oxygen, hyperbaric if available
- Mannitol IV + dexamethasone 1mg/kg q6h to prevent cerebral oedema
- Monitor for 1 week for pulmonary oedema, cardiac failure and myoglobinuria

Botanical poisons

Mushrooms

Many of the toxins are inactivated by cooking, but not all.

Clinical:

Vomiting, diarrhoea and abdominal cramps

Inocybe: Contains muscarine → cholinergic crisis (lacrimation, salivation, bronchospasm, miosis, urinary and faecal incontinence. (Reversed by 0.05mg/kg atropine)

Amanita and Galerina: Most poisonous, causes cell necrosis of liver, kidneys and gut
Treatment is supportive

Belladonna and atropine poisoning
Deadly nightshade (belladonna) and other plans containing belladonna alkaloids (stramonium, Jimsonweed, green and sprouting potatoes) cause atropine poisoning.
Sg & Sx: Dry mouth, dilated pupils, fever, decreased sweating and tachycardia.
Only last for 4-6h

Management
- ABCs
- Induce emesis
- Activated charcoal
- Physostigmine 0.5-2mg

Impala (Callilepsis laureola)
Ingredient in some herbal medicines.
Clinical:
Short hx of ↓LOC, convulsions and GIT Sx
CNS Sx: Hypotonic, hyporeflexic, convulsions & ↓LOC (no focal Sx or meningeal irritation)
Resp: Tachypnoea with acidotic-type breathing
Biochemical: Hypoglycaemia, renal impairment with hyperkalaemia, uraemia and acidosis. Raised liver enzymes and prolonged prothrombin time and raised ammonia.
None of the following: Jaundice, hepatic foetor

Management
- ABCs
- Glucose for hypoglycaemia
- Manage hepatic and renal failure

Specific Antidotes

B-blockers	-	Glucagon, adrenaline
Warfarin	-	Vit K, FFPs
Digixin	-	Specific antibodies (Digibind)
Methanol	-	Ethanol
Iron	-	Desferrioxamine
Paracetamol	-	N-acetylcystein, Methionine
Opoids	-	Nalaxone
TCA	-	NaHCO3
Cyanide	-	Thiosulphate, O2, nitrites
Heavy Metals	-	EDTA
CCB	-	CaCl, Ca gluconate, glucagon
Lead	-	DMSA, DMPS, Disodium, Calcium edentate
Mercury	-	DMPS
Organophosphates	-	Atropine, Oxime (pralidoxime)
Carbon Monoxide	-	O2
Metoclopromide	-	Akineton
Atropine & Belladona	-	Physiostigmine
Ethelyne Glycol (Antifreeze)	-	Ethanol
Sulphonylurea	-	Glucose – glucagon if resistant
Fluoride		
Black widow bite	-	Ca Gluconate
Copper	-	Penicillamine

INFECTIONS

TIM - PIETERMARITZBURG

HIV
TB
CHILDHOOD INFECTIONS

"Now, don't panic, but I'd like you to take off all your clothes so we can burn them."

INFECTIONS

HIV
TB
CHILDHOOD INFECTIONS

"Now, don't panic, but I'd like you to take off all your clothes so we can burn them."

PREVENTION OF 1° INFECTION► 2010 PMTCT REGIMEN

(women not on HAART)

- ❑ **MOTHER**
- ❑ Antenatal: AZT 300mg bd po dly from 14w GA
- ❑ Intrapartum:
- ❑ Single dose NVP 200mg po
- ❑ Single dose Truvada (TDF 300mg + FTC 200mg)
- ❑ AZT 300mg po 3hrly during labour

- ❑ **NEONATE/INFANT**
- ❑ Birth – 6 weeks
  - o ≤ 2.5kg 10mg/day po
  - o ≥ 2.5kg 15mg/day po
- ❑ Stop NVP at 6w if
  - o exclusively formula fed infant, mother is on HAART,
  - o infant confirmed HIV +
- ❑ Otherwise:
- ❑ 6w-9/12: 20mg/day
- ❑ 6/12-9/12: 30mg/day
- ❑ 9/12 to end of breastfeeding 40mg/day

WHICH KIDS TO TEST?

- ❑ All HIV exposed infants (so all whose mums were on the PMTCT program)
- ❑ Clinical features of HIV
- ❑ Severe acute illness
- ❑ Kids fitting IMCI criteria for suspected HIV
- ❑ All kids with current or previous TB
- ❑ Suspicious family/social history
- ❑ Breastfed by woman of unknown status
- ❑ ?sexual assault
- ❑ If in best interest of the child

WHEN TO TEST? (notes on HIV exposure too!)

- ❑ In kids < 18 months old, HIV ELISA & rapid antibody tests cannot distinguish between maternal and infant HIV antibodies
- ❑ BUT, HIV PCR (sensitivity 98.8% and specificity 99.4%) can confirm HIV status► typically done at 6 weeks
- ❑ Must perform a confirmatory viral detection assay ► a viral load of > 10 000 copies/mL (>4 log) = confirmation of HIV infection
- ❑ Kids >18/12► HIV ELISA as for adults
- ❑ **PRACTICAL Tip: use 6w & 10w immunization visits to perform PCR and report on results**

HIV basic Approach ...

NB NB NB !Cotrimoxazole prophylaxis for all HIV-**exposed** infants must also begin at 6 weeks of age!

WHO staging (know this & DO it)

also read over CDC staging – kalafong Docs likes to ask about it!

STAGE 1:
- ❑ Asymptomatic
- ❑ Persistent gen. Lymphadenopathy

STAGE 2:
- ❑ Unexplained persistent HSM
- ❑ Popular pruritic eruptions
- ❑ Extensive warts
- ❑ Extensive molluscum contagiosum
- ❑ Fungal nail infections
- ❑ Recurrent oral ulceration
- ❑ Persistent parotic enlargement
- ❑ Herpes zoster
- ❑ Lineal gingival hyperplasia

STAGE 3:

Unexplained moderate malnutrition
Unexplained diarrhoea for >14days
Unexplained fever for longer than 1/12
Persistent oral candidiasis (only after 1st 6-8weeks of life)
Oral hairy leukoplakia
Pulmonary TB
Severe bacterial pneumonia
Symptomatic LIP
Chronic HIV-assoc lung disease
Unexplained anaemia

NB Growth faltering may be the 1st sign of treatment failure!

STAGE 4:
- ❑ Unexplained severe wasting, stunting, malnutrition
- ❑ PJP
- ❑ Recurrent severe bacterial infections
- ❑ Chronic herpes simplex infections
- ❑ Extrapulmonary TB
- ❑ Kaposi sarcoma
- ❑ Oesophageal candidiasis
- ❑ CNS toxoplasmosis
- ❑ HIV encephalopathy
- ❑ CMV infection..

Child tests POSITIVE ► Now what??

- ❑ STAGE child Clinically (WHO)
- ❑ Baseline bloods (CD4 count, % & VL)

THEN, assess eligibility for ARVS:

Age	Eligibility for treatment
Child< 1 year	all these kids qualify
1-5 years	Symptomatic (stage III or IV) or CD4 ≤ 25% or absolute count ≤ 750 cells/mm3
≥ 5 years	Symptomatic (stage III or IV) or CD4 < 350 cells/mm3

Also ENSURE ROUTINE MANAGEMENT is performed on all these kids:
- ❑ Growth monitoring and promotion
- ❑ Immunisation (see notes)
- ❑ Vitamin A supplementation
  - o 6 -11 months: 100 000 IU stat
  - o 1-5years stat at 12/12, then q6/12 until 5years
- ❑ Routine deworming
  - o 12-24/12 or <10kg: mebendazole 100mg bd for 3 days q6 months
  - o >24/12 or >10 kg: 500mg single dose q6 months
- ❑ Cotrimoxazole prophylaxis for PJP (see below)
- ❑ **DEVELOPMENTAL ASSESSMENT** must be done (screens for encephalopathy & used for treatment progress monitoring!)

Approach to HIV exposed children & BACTRIM prophylaxis

▶ the HIV dept. at Kalafong is *passionate* about prophylaxis

HIV-EXPOSED INFANTS

An HIV-exposed child is defined as a child born to a mother living with HIV until HIV exposure stops (6 weeks after the complete cessation of breast feeding) and HIV infection can be excluded. HIV-exposure status should be determined before birth as part of the PMTCT programme – where the mother's status is not known, this should be determined after birth.

Table 3: HIV testing at six week visit

Positive maternal HIV status	All infants born to HIV-infected women require a PCR.
Negative maternal HIV status	Rapid test should be offered to mother to ensure she has remained HIV-uninfected.
Unknown maternal HIV status	Offer a rapid test to the mother. If she tests positive then her infant should have a PCR at the same visit. Provide the mother with the care she requires.
Unknown maternal HIV status and mother refuses testing	Offer an HIV rapid test (on the infant) to assess HIV-exposure. If the infant's rapid test is positive, perform a PCR test on the infant during the visit and counsel the mother to seek further HIV testing and care. NOTE in the RTHC so that mother receives continued support during infant follow-up.

All HIV-infected and HIV-exposed infants must receive cotrimoxazole prophylaxis from six weeks of age as outlined in Table 7.Dapsone should be used in cotrimoxazole intolerant patients. The recommended dose is 2 mg/kg/day or 4mg/kg/week. The maximum daily dose is 100 mg (1 tablet).

Table 7: Pneumocystis Jiroveci Pneumonia (PCP) Prophylaxis

All HIV-exposed newborns	Start from 4-6 weeks after birth	Stop when PCR negative ≥ 6 weeks after full cessation of breastfeeding AND infant is clinically HIV negative.
All HIV-exposed Exclusive formula feeding children (EFF)	Start from 4-6 weeks after birth	Stop when PCR negative AND infant is clinically HIV negative AND EFF is expected to continue
All HIV-exposed breastfeeding children	Start from 4-6 weeks after birth	Stop when PCR negative ≥ 6 weeks after full cessation of breastfeeding AND infant is clinically HIV negative.
HIV-infected infants < 12 months old	Start from 4-6 weeks after birth or as soon as possible after HIV diagnosis even if on ART.	All infants < 12 months should remain on prophylaxis.
For HIV-infected children 1-5 years with or without ART	All symptomatic children (WHO clinical stage 2, 3 or 4) or CD4 < 15% or < 500 cells/mm^3.	Stop once ART-associated immune reconstitution has occurred for ≥ 6 months i.e. CD4 percentage ≥ 15% or CD4 count ≥ 500 cells/mm^3 on ≥ 2 occasions, 3-6 months apart.
HIV-infected children ≥ 6 years of age with or without ART	Start if CD4 count < 200 cells/mm^3 or <15% OR WHO clinical stage 3 or 4 disease (including TB).	Stop once ART-associated immune reconstitution has occurred for ≥ 6 months in children over 1 year of age: CD4 ≥ 15% or ≥ 200 cells/mm^3 on ≥ 2 occasions, 3 – 6 months apart.
Any HIV-infected child with high risk for bacterial infections or at risk of malaria	Start cotrimoxazole prophylaxis even with ART immune-reconstitution.	Do not stop until risk has been eliminated and all CD4 cell percentage or CD4 cell count criteria listed above have been met
HIV –infected child with previous PCP infection	Start as soon as first PCP episode has been treated	Stop at age 5 years

* NOTE: Any one of the criteria could be used for starting therapy

Table 8: Recommended doses of cotrimoxazole for prophylaxis

Age/weight of child	[illegible] 100mg SMX/20 mg TMP/5 mL			
< 6 months or < 5 kg	100mg SMX/ 20 mg TMP	2.5 mL	¼ tablet	–
6 months – 5 years or 5–15kg	200mg SMX/ 40 mg TMP	5 mL	½ tablet	–
6-14 years or 15–30 kg	400mg SMX/ 80 mg TMP	10 mL	1 tablet	½ tablet
> 14 yrs or > 30 kg	800mg SMX /160 mg TMP	–	2 tablets	1 tablet

HIV Continued:

- ❑ Pre ARV requirements
- ❑ 1st line Regimens
- ❑ Routine monitoring
- ❑ 2nd line regimens

BEFORE INTITATING ARVs→

BASELINE CLINICAL DATA & LAB Info:

- ❑ Child's weight & height (as well as relevant calculations)
- ❑ WHO clinical staging
- ❑ Presence of TB symptoms
- ❑ Developmental Level
- ❑ CD4 count & percentage
- ❑ Viral Load
- ❑ Recent FBC
- ❑ ALT if starting NVP regimen

2010 1st line ARV REGIMENS

<3yrs or <10kg	>3yrs or >10kg
❑ **Abacavir** ❑ **Lamivudine** ❑ **Lopinavir/ritonavir**	❑ Abacavir ❑ Lamivudine ❑ Efavirenz

For all children on stavudine with no side effects, stavudine may be continued. Abacavir should be substituted once any lipodystrophy is suspected

Routine Monitoring Tests in kids on ARVS:

Test	Timing
CD4 count/ %	At initiation, then 6/12, 1yr then annually
VL	At initiation, 6/12, 1yr, annually
FBC	Baseline; then if child on AZT: 1/12, 3/12, 6/12 & then annually
LDL cholesterol & Triglycerides	Child on lopinavir/ritonavir
ALT	NVP → baseline & repeat if kid develops rash or jaundice

NB: manage intecurrent illness, monitor respone to ARVS (weight gain, developmental assessment, staging, bloods); assess adherence; routine car e/ immunizations; psychosocial support

Switching to 2nd Line ARV REGIMENS

- ❑ Only change if treatment failure →defined as VL > 1000 copies/ml (despite good adherence)
- ❑ First do the following:
  - Allow ≥ 24w trial therapy before condluding failure has occurred
  - Always optimize/ensure adherence 1st
  - Treat intecurrent opportunistic infections
  - Exclude IRIS
  - Ensure adequate nutrition

Viral load monitoring & recommended action:

VIRAL LOAD	RESPONSE
<400 copies/ml	6/12 VL monitoring & routine adherence support
400-1000 copies/ml	Repeat VL in 6/12; begin step up adherence package if VL still < 400-1000
>1000 copies/ml	Begin step up adherence package Repeat VL in 3/12

- If < 400, return to routing 6/212 monitoring
- If 400-1000, continue step up adherence & repeat VL after 6/12
- If > 1000 & child on NNRTI based regimen, switch to 2nd line Rx only if adherence <80%
- If > 1000 & on PI based regimen, reinforce adherence
- Switch to 2nd line Rx if VL 5000 only if adherence >80%

Criteria for virological failure:

- ❑ Sustained ↑ in VL> 5000 copies/ml
- ❑ A ↓ in VL < 1 $\log_{10}$ (tenfold) 6-8w after starting ARVS (1° virological failure)

2nd line ARV REGIMENS

Regimen which Failed	ACTION
[illegible]	[illegible]
❑ **Abacavir** ❑ **Lamivudine** ❑ **Efavirenz**	Change to ❑ Zidovudine ❑ Didanosine ❑ Lopinavir/ritonivir
[illegible]	[illegible]

The goal of ART is to increase survival and decrease HIV related morbidity and mortality. On ART:

- ❑ The child's CD4 count should rise and remain above the baseline count
- ❑ The child's viral load should become undetectable (< 400 copies/mL) by 24w after starting Rx, and remain undetectable

It wasn't possible to summarise everything about ARV sided effects for this book – just too much info. BUT I've got copies of the 2010 guidelines for the management of HIV in children for whoever is interested. Below are just 2 tables of useful things to know...

Table 19: Adverse effects of ARVs

NRTI	Zidovidine	Anaemia, granulocytopenia Myopathy, Lactic acidosis
	Didanosine ddI	Common: abdominal pain, nausea and vomiting Uncommon: pancreatitis, peripheral neuropathy, lactic acidosis
	Stavudine	Common: abdominal pain, nausea and vomiting Uncommon: lipoatrophy, lipodystophy, peripheral neuropathy, lactic acidosis
	Abacavir	Hypersensitivity reaction (with or without rash) – may be fatal in adults and children
	Lamivudine	Common: headache, fatigue and abdominal pain, Uncommon: pancreatitis and peripheral neuropathy, lactic acidosis
NNRTI	Nevirapine	Skin rash, sedative effect and diarrhoea, LIVER TOXICITY
	Efavirenz	Skin rash CNS – Sleep disturbance, confusion, abnormal thinking. Teratogenic in primates
PI	Ritonavir	Nausea, vomiting, diarrhoea Hypercholesterolaemia and hypertriglyceridaemia
	Lopinavir /Ritonavir	Nausea, vomiting, diarrhoea Hypercholesterolaemia and hypertriglyceridaemia

5. Standardized national monitoring for infants and children with HIV

At initial Diagnosis of HIV	Purpose
Check HIV result	Ensure that national testing algorithm including HIV DNA PCR and HIV viral load (RNA) for infants and children less than 18 months has been followed
Document weight and height	To monitor growth and development + identify eligibility for ART
Screen for TB symptoms	To identify TB/HIV co-infected
Do the CD4 count	To identify eligibility for ART or ARVs
Hb or FBC is available	To detect anaemia or neutropenia

At Routine Follow-Up Visits	Purpose
Document weight and height	To monitor growth and development and to see if they have become eligible for ART
Check that CD4 has been done in the last 6 months	To see if they have become eligible for ART
WHO clinical staging	To see if they have become eligible for ART
Screen for TB symptoms	To identify TB/HIV co-infection

If eligible for ART	Purpose
ALT if starting on a NVP-based regimen	If ALT raised, do HepBSAg and avoid NVP
Hb or FBC if available if starting on an AZT-based regimen	If less than 8g/dl refer

On ART	Purpose
Height + weight + development	To monitor response to ART
Clinical stage	To monitor response to ART
CD4 at month 6, 1 year into ART, and then every 12 months	To monitor response to ART
VL at month 6, 1 year into ART, then every 12 months	To monitor response to ART To identify problems with adherence
ALT if on NVP an develops rash or jaundice	to identify NVP toxicity
FBC at month 1, 2,and 3 if on AZT	To identify AZT toxicity

TB (+) interrupt ART

APPROACH TO PATIENTS WITH CONCOMITANT TB

- ❑ Starting HAART in patients on TB Rx may be complicated by IRIS in 8-45% of cases

CD4 Count	Action
>200	Start ARVs after completing Tb Rx
<200	Delay ARVs until after intensive phase of TB rx (2/12)
<50	Stabilise on TB Rx for 2 weeks then start ARVs ▶ less chance of IRIS according to Prof Avenant

- ❑ NB: rifampicin significantly ↓ NVP levels (also shared toxicity ▶ rash & hepatitis) thus avoid this combo if possible since NVP dosages must be ↑ with ↑ risk of adverse effects. ALT should be monitored in these kids & those who develop signs or symptoms of hepatitis should be referred to a treatment expert immediately.
- ❑ NB: A rash in a child on nevirapine with mucosal involvement OR associated with fever/systemic symptoms /derangement in liver functions should be treated as a Grade 4 toxicity. All ARVs should be stopped immediately. Patients should be referred to a specialist for advice regarding restarting ARVs. **The patient should never be rechallenged with nevirapine.**
- ❑ If the child is on an EFV containing regimen, there should be no change to the ARVs and standard dose TB Treatment should be added to the regimen.
- ❑ If the child is on a Lopinavir/ritonavir solution containing regimen, added ritonavir should be added at a dose of 0.75x the volume of the Lopinavir/ritonavir dose. TB treatment should be dosed at standard doses.
- ❑ In older children (taking Lopinavir/ritonavir tablets) the dose should be doubled to roughly 600 mg/m2 of Lopinavir (this is similar to the adult guidelines).
- ❑ If the child is unable to tolerate the large number of drugs, ART may have to be interrupted until TB therapy has been completed – however this should only be done if the child is stable and has a good CD4 count, and in consultation with a treatment expert.

IMMUNE RECONSTITUTION INFLAMMATORY SYNDROME (IRIS)

occurs in 6 wks of starting HAART

This paradoxical clinical deterioration after starting ART is also known as Immune Reconstitution Inflammatory Syndrome (IRIS). It is due to the improving immune system interacting with organisms that have colonized the body during the early stages of HIV infection.

CAUSES

A wide range of pathogens may induce IRIS including Mycobacterium tuberculosis (MTB), BCG, *Mycobacterium avium complex, Mycobacterium leprae, Cryptococcus neoformans, Aspergillus fumigatus, Aspergillus terreus, Candida albicans, Pneumocystis jiroveci*, CMV, Human Herpes viruses, Human Papilloma virus and Hepatitis B and C viruses.

PRESENTATION

IRIS usually presents during the first 6 weeks after starting ART. Clinical presentations vary and depend on the causative organism and the organ-system that is colonized. For example IRIS caused by MTB may present with high fever, lymphadenopathy, worsening of the original tuberculous lesion, and/or deteriorating chest X-ray features including the development of a miliary pattern or pleural effusion.

MANAGEMENT

– specialist on case RR

Includes specific antimicrobial therapy e.g. TB treatment for IRIS caused by TB. In severe reactions glucocorticosteroids and/or temporary discontinuation of ART may help.

SINGLE DRUG SUBSTITUTION OF STAVUDINE WITH ABACAVIR

According to the new guidelines, kids are no longer initiated on Stavudine (d4T). Kids currently stable on d4T regimens should continue Rx BUT maintain a high index of suspicion for lipodystrophy. Kids who develop lipodystrophy or other toxicity to d4T & are virologically suppressed should have a single drug substitution to Abacavir.

Toxicity warranting a switch:

- ❑ Lactic acidosis
- ❑ Peripheral neuropathy
- ❑ Metabolic syndrome
- ❑ Lipodystrophy - Lipoatrophy/Lipohypertrophy

HIV-associated lipodystrophy can present with:

- ❑ Lipoatrophy : facial fat loss ±involvement of the buttocks and limbs
- ❑ Lipohypertrophy (fat accumulation): including abdomen, buffalo hump &breast hypertrophy
- ❑ Metabolic syndrome▶. These kids are at risk of T1DM & CAD
- ❑ Lipodystrophy occurs in 18-33% of patients on ART and is associated with a longer duration of therapy (> 1year) and the use of Stavudine, Didanosine and protease inhibitors

Early substitution of d4T to Abacavir will prevent clinical progression of lipodystrophy.

PCP

RESPIRATORY COMPLICATIONS OF HIV in KIDS

- ❑ Pneumocyctis Jiroveci Pneumonia
- ❑ Severe/recurrent Bacterial Pneumonia
- ❑ Lymphocytic Interstitial Pneumonia
- ❑ Tuberculosis
- ❑ CMV

Sorry guys I ran out of time but LIP and CMV are both very NB topics worth reading!

PNEUMOCYSTIS JIROVECI (FORMALLY CARINII) PNEUMONIA (PCP)

PCP, which is an AIDS defining condition, accounts for a high proportion of mortality in HIV-infected infants. The majority of cases can be prevented with cotrimoxazole prophylaxis, whilst early and appropriate treatment significantly improves the outcome. PCP is characterized by the following features:

- ❑ Tachypnoea
- ❑ Hypoxaemia - characterized by disorientation, confusion and with cyanosis if the child is not anaemic
- ❑ Absent or low-grade pyrexia – however acute onset of fever may be a feature

Clinical findings on chest auscultation may be negligible and thus not in keeping with the degree of respiratory distress. On chest X-ray one might see a diffuse interstitial infiltrate. Early and appropriate treatment, significantly improves the prognosis.

Suspect PCP if the child:

- ❑ Is less than 12 months old and
- ❑ Has tachypnoea > 50 or more breaths/minute in infants 2 to 12 months, 40 or more breaths/minute in children 12 months up to 5 years
- ❑ Is dyspnoeic
- ❑ Has few crackles relative to the degree of dyspnoea, and decreased breath sound intensity on auscultation
- ❑ Is hypoxaemic – many children who are anaemic may be profoundly hypoxaemic without appearing
- ❑ cyanosed

Begin treating for PCP immediately on suspicion (in addition to usual treatment of pneumonia) even if the HIV status of the child has not yet been established. All infants and children with suspected PCP should be treated in hospital.

INPATIENT MANAGEMENT OF SUSPECTED PCP (PNEUMOCYSTIS PNEUMONIA)

Treat aggressively. Palliative care principles with an emphasis on relieving respiratory distress should also be applied.

INVESTIGATIONS

- Check oxygen saturation: If PCP is present, oxygen saturation is usually less than 90% on room air
- Chest X-ray: Diffuse bilateral alveolar or interstitial infiltrate/ ground class appearance (findings can vary)

TREATMENT

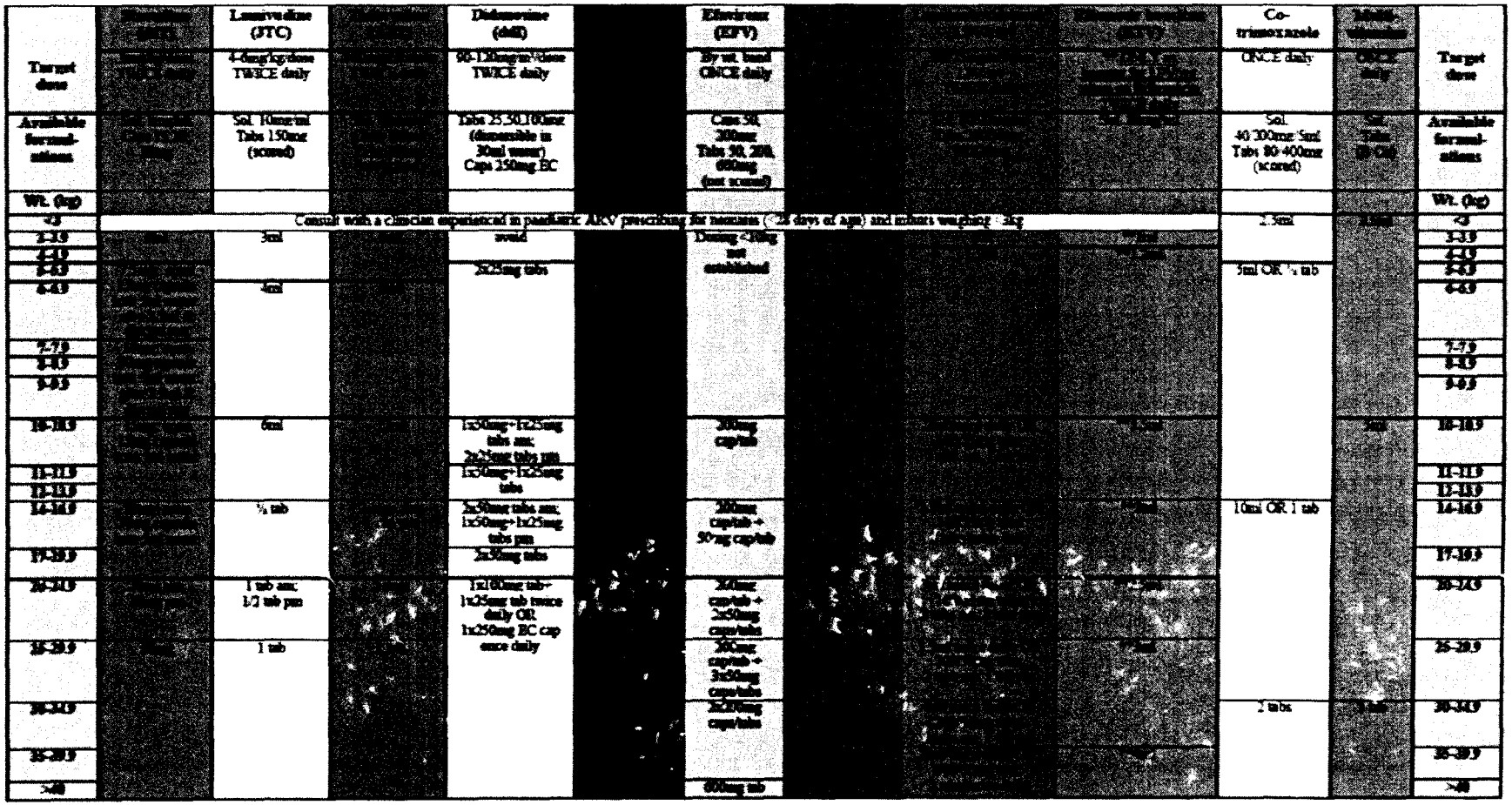
- ❑ Oxygen (nasal prongs)
- ❑ Cotrimoxazole. Load with 250mg/m2 of the trimethoprim component, then give 20mg/kg/day of trimethoprim component 6 hourly IV for 5 days changing to orally for 3 weeks if response adequate. NOTE: this is a higher dose than that used for prophylaxis.
- ❑ If PCP is confirmed or if child is hypoxaemic, give Prednisone (1-2mg/kg) daily for two weeks
- ❑ Consider adding clindamycin 30 – 40mg/kg/day for severe disease
- ❑ Paracetamol 10-15mg/kg 6 hourly for pain or fever > 37.5OC
- ❑ Morphine must be given if severe respiratory distress is not responding to other medical management, and admission to an intensive care unit is not an option

  Morphine oral starting doses:
  - o < 1 year: 0.2- 0.4 mg 4 hourly
  - o 1 – 5 years: 0.5- 5 mg 4 hourly
  - o 6 - 12 years: 5-7.5 mg 4 hourly
- ❑ PCP prophylaxis should continue after discharge as per guidelines

PREVENTION OF PCP

Most cases of PCP can be prevented through administration of routine prophylactic cotrimoxazole.

Antiretroviral Drug Dosing Chart for Children (2009)



* A lead-in dose of nevirapine is given for the first 14 days of treatment equivalent to half of maintenance dose i.e. usual maintenance dose but given once-daily. Increase to full maintenance dose after 14 days if no rash develops.

Compiled by J. Nuttall & S. Eaton for the Paediatric HIV/TB Policy Reference Group, Western Cape. Adapted from World Health Organization guidelines, 2006 & 2008.

NEED HELP?
CALL NATIONAL HIV HCW HOTLINE
0800 212 506/ 021 406 6782
OR
send an sms or "please call me" message to
071 840 1572

0800 212 506

Body Surface Area (BSA) $m^2 = \sqrt{\frac{\text{Weight (kg)} \times \text{Height (cm)}}{3600}}$

Dx * standard = Mantaux Test

Tuberculosis = infection with Mycobacterium Tuberculosis, a slow growing aerobe (doubling time of 18 hours) capable of intracellular survival, even replicating in macrophages. Clinical manifestations depend on the interplay between microbial and host factors- hence an immunopathological disease. Classic lesions are granulomas with central caseous necrosis. CD4+ cells release cytokines 2° to antigen presentation (esp INF-γ), resulting in the recruitment of monocytes to form granulomas which limit replication and spread of the organism. Cells derived from monocytes in granulomas are termed epitheliod histiocytes.

1. EPIDEMIOLOGY:
- 1/3 of world population has TB
- incidence rate of ± 9 million pa
- of these, ± 11% occur in kids

CONTENTS:

2.1 TRANSMISSION:
- Inhalation of droplet nuclei <10 μm diameter into alveoli.
- 65% of children living with a smear + adult become infected

2.2 DEVELOPMENT OF DISEASE

Steps in the development of disease -
1. Bacilli inhaled to lung periphery→ primary lesion formed = "ghon focus"→ uncontrolled muktiplication with spread to regional lymph nodes → Ghon Complex (focus plus nodes)
2. Silent bacteraemia→ minute mets to lung apices, 1 vertebrae, 2 long bones, 3 meninges, 4 lymph nodes & 5 kidneys
3. Delayed type hypersensitivity→ ± 2/52 later sensitized T cells activate macrophages→ become histiocytes & eventually form granulomas
4. 2 options then exist, either:
   a. Controlled 1° infection
   b. Progressive 1° infection

1° focus + LN involvement

Complications of focus	Complications of nodes
• Effusion	Extension into bronchus
• Cavitation	Consolidation
• Coin shadow	Hyperinflation

3.1 CLINICAL MANIFESTATIONS: Extra Thoracic disease

Miliary TB	• due to uncontrolled 1° infection • develops 6/12 – 1 yr after infection • fever, wasting, HSM, lymphadenopathy • NB reticulonodular patter on both lung fields on CXR very suggestive • High mortality!! • NB lung fields may be clear
TB meningitis (see full section)	Close assoc. with military TB Most dangerous form of TB Cortical/meningeal TB focus = "Rich focus" →caseation →D/C into CSF→ typically takes 3/12 to occur Thick protenacious exudate→ cover base of brain, cranial nerve, obstructs CSF Thus communication HC in 80% of kids at time of Dx ± cranial nerve fallout ± vasculits/infarction
Lymph nodes	(typically matted due to periadenitis) Exclude: haematological malignancy, acute pyogenic infction & chronic fungal infection
Skin (see pg 634 of coovadia)	Lupus vulgaris is most common Papulonecrotic tuberculitides Lichen scrofulosorum Nodular tunerculitides
Bone & joint → search for a cold abscess!	Potts disease- beware the gibbus! Hip = 2nd most common TB dactiliits- indurated, red fingers with thick phalanges!
Liver & spleen	HSM occurs with or without military spread Enlargement
Adrenal	Addisons disease many years after bilateral infection → typically only in adults
Blood	If BM infected: anaemia, pancytopaenia, or may mimic malignancy
Renal	Multiple manifestations, but with incidental discovery of sterile pyuria – rule out TB
Genital	Epididimytis, orchitis, vaginal, salpingitis
ENTTB	Asymmetrically enlarged tonsils with ulcers Chronic painless ear D/C, chronic OM
Abdomen (Low grade sticky peritonitis)	• Ascites • Palpable lymph nodes • GIT disease
Pericarditis	↑JVP, hepatomegally, peripheral oedema
Eyes	Phlyctenular conjunctivitis with pre-auricular lymphadenopathy

3.2 CLINICAL MANIFESTATIONS: Intra Thoracic disease

Infection without disease	• ☑mantoux • Normal CXR • May need chemoprophylaxis
Aymptomatic mediastinal lymph-adenopathy	• ☑mantoux • CXR shows hilar or paratracheal nodes • No other signs • Qualify for treatment
Progressive 1° focus	• Unusual • Primary focus enlarges • May fill segment or lobe • Cavitation
Lympho Bronchial TB	• Thoracic nodes adjacent to bronchi may either: • Airway collapse • Ball valve effect →hyperinflation • Ulceration + D/C of a node → collapse/consolidation or expansile pneumonia • Cx: permanent collapse, bronchiectasis
TB broncho-pneumonia	• D/C of caseous material from a node/granuloma → diseemination of bacilli • CXR shows bronchopneumonia • May mimic military TB
Adult type TB	• Usually in adolescence • Involvement of apices + cavitation • Typically reactivation TB
Pleural Effusion	• Unusual in young children • Common in adolescents with primary infection

℞ = INH, RMP, PZA (± EMB) } 2 mo (bactericidal)

then

RMP, INH } 4 mo (cont. phase)

1. HISTORY

a. Contact Hx: person with smear + PTB living in same house or having frequent contact with child
b. Chronic cough or wheeze > 3/52
c. Fever > 38 for 14 days (malaria & pneumonia excluded)
d. Weight loss of FTT (check growth chart)
e. Kwashiorkor or marasmus
f. Failure to recover after any acute illness, but esp. resp tract infx.

CLINICAL FINDINGS

no specific findings pathognomonic of PTB, but some signs, are highly suggestive of extrapulmonary TB

a. painless lympanenopathy + fistula formation
b. Gibbus; painless enlarged joint;
c. signs of tuberculin hypersensitivity (e.g. phlyctenular conjunctivitis, erythema nodosum).

2.1 CXR FINDINGS:

Only hilar lymphadenopathy & a military pattern are "diagnostic" of PTB; other features:

❑ Segmental leasion	56%
❑ Bronchopneumonia	30%
❑ Pleural effusion	16%
❑ Cavitation	14%
❑ Normal	2%
❑ Calcification	1%

NB: must have PA & lateral to clarify whether hilar adenopathy is present

2.2 TUBERCULIN TESTING: Mantoux = only test that has been standardised. TO be interpreted, child must have received BCG

INDURATION	INTERPRETATION
0 - 4 mm	negative
0 - 9 mm	Atypical mycobacteria or BCG
10 – 14 mm	BCG or M.tuberculosis
< 15 mm	M.tuberculosis
* > 5 mm	Positive in HIV infected children

Method: inject 0,1 ml of PPD (5IU) on dorsal aspect of forearem. Measure size of induration at 48-72 hours.

DIAGNOSTIC APPROACH

1. History & clinical findings
2. Special Investigations
   - ❑ CXR
   - ❑ Tuberculin Test
   - ❑ Bacterial diagnosis
3. Management
   - ❑ Protection
   - ❑ Contacts
   - ❑ Case management.
4. Drug treatment

2.3 BACTERIAL DIAGNOSIS

NB: kids have paucibacillary disease!
Appropriate samples include

- ❑ Sputum: kids >5yrs
- ❑ Early morning gastric aspirates
- ❑ Induced sputum
- ❑ CSF
- ❑ Pleaural & ascitic fluid
- ❑ fna's of lymphnodes
- ❑ ear swabs in chronic ottorhoea

NB kids < tend to swallow their sputum & NGA are preferred.
Send specimens for MC&S for M.Tb & AFBs

OTHER TESTS:
Serological and PCR tests are not currently recommended for routine diagnosis of childhood TB
► No EBM supporting routine usage

3.1 COMMUNITY PROTECTION

- ❑ Improve socio-economic conditions
- ❑ Health promotion & education
- ❑ Pasteurization of milk
- ❑ BCG vaccination

3.2 TRACING OF CONTACTS

All contacts ≤ 5 years must receive chemoprophylaxis
Rifampicin/isoniazid 60/30mg daily po for 3/12 OR
Isoniazid, 5mg/kg po daily q6/12

3.3 CASE MANAGEMENT

Case Finding: (4 methods)

- ❑ Passive detection at clinics
- ❑ Active tracing of contacts
- ❑ Screening at risk groups
- ❑ Mass CXRs (not cost effective)

Treatment Principles:

- ❑ Treatment must be supervised – DOT programme
- ❑ Use combinations of drugs
- ❑ Treatment involves intensive and continuation phases
- ❑ Treatment is given 5x per week

4. FIRST LINE DRUG MANAGEMENT

DRUG	MOA	DOSE mg/kg		S/E	COMMENTS
Isoniazid (I)	Bacteriocidal to dividing orgs. bacteriostatic for resting orgs. ?Inhibits mycolic acid synthesis	5x /wk 4-6	3x /wk 8-12	Peripheral neuropathy, Liver damage (rare <20yrs) ↓ metabolism of AEDs	Acetylation of INH is under genetic control. Cx occur 7x more commonly in slow acetylators. Give pyridoxine 5mg/100mg INH to prevent neuropathy
Rifampicin (R)	Inhibits DNA-dep, RNA synthesis; bacteriocidal Also a broad spectrum antibiotic	8-12	8-12	Commonest : skin eruptions, fever & GIT ; Liver damage is rare	Assess liver functions before starting treatment. Fatal liver failure can occure if underlying liver disease exists: in these pts check AST & bili weekly for 8/52, then monthly
Pyrazinamide (P)	Inactive at neutral pH Tuberculostatic at acid pH inside macrophages NB role in sterilizing lesions	20-30	30-40	Gout, GIT upsets, malaise & fever. Liver toxicity	Hepatotoxicity rare but there is no prodrome prior to severe dysfunction, as with other 1st line agents
Streptomycin (S)	Aminoglycoside- inhibits protein synthesis	12-18	12-18	Neurotoxic to CN VIII→ vestibular Cx, ataxia	Perfom Romberg, hee-toe walking etc regularly to assess vestibular fx
Ethambutol (E)	Only works on mycobacteria MOA uncertain. bacteriostatic	15-20	25-35	GIT upset common Optic neuritis- dose related; worse if renal impairment	Monitor colour vision with long term Rx since R/G blindness is the first sign, followed by ↓VA NB: ethambutol is safe in kids at a dose of 20mg/kg

Points to remember: drug toxicity is less common in kids. Hepatotoxicity is especially rare. If jaundice occurs, stop hepatotoxic drugs, substitute with S, E and a quinolone (2nd line agents) and consult an expert!

There is a 10x ↓ in TB bacilli in the 1st 2 days of Rx. Thus cross infection is negligible after 14days Mx.

TREATMENT RESPONSE: appetite returns, weight gain, apyrexial and normal CXR after 60 days (this may take up to 180 days)

Table 3 Recommended treatment regimens for children in each TB diagnostic category

TB diagnostic category	TB cases	Regimen[a]	
		Intensive phase	Continuation phase
III	New smear-negative pulmonary TB (other than in category I). Less severe forms of extrapulmonary TB	2HRZ[b]	4HR or 6HE
I	New smear-positive pulmonary TB	2HRZE	4HR or 6HE[c]
	New smear-negative pulmonary TB with extensive parenchymal involvement Severe forms of extrapulmonary TB (other than TB meningitis – see below) Severe concomitant HIV disease		
I	TB meningitis	2RHZS[d]	4RH
II	Previously treated smear-positive pulmonary TB: relapse treatment after interruption treatment failure	2HRZES/1HRZE	5HRE
IV	Chronic and MDR-TB	Specially designed standardized or individualized regimens (see treatment guidelines for MDR-TB (4) and Annex 3)	

MDR TB = TB resistant to INH & rifampicin..

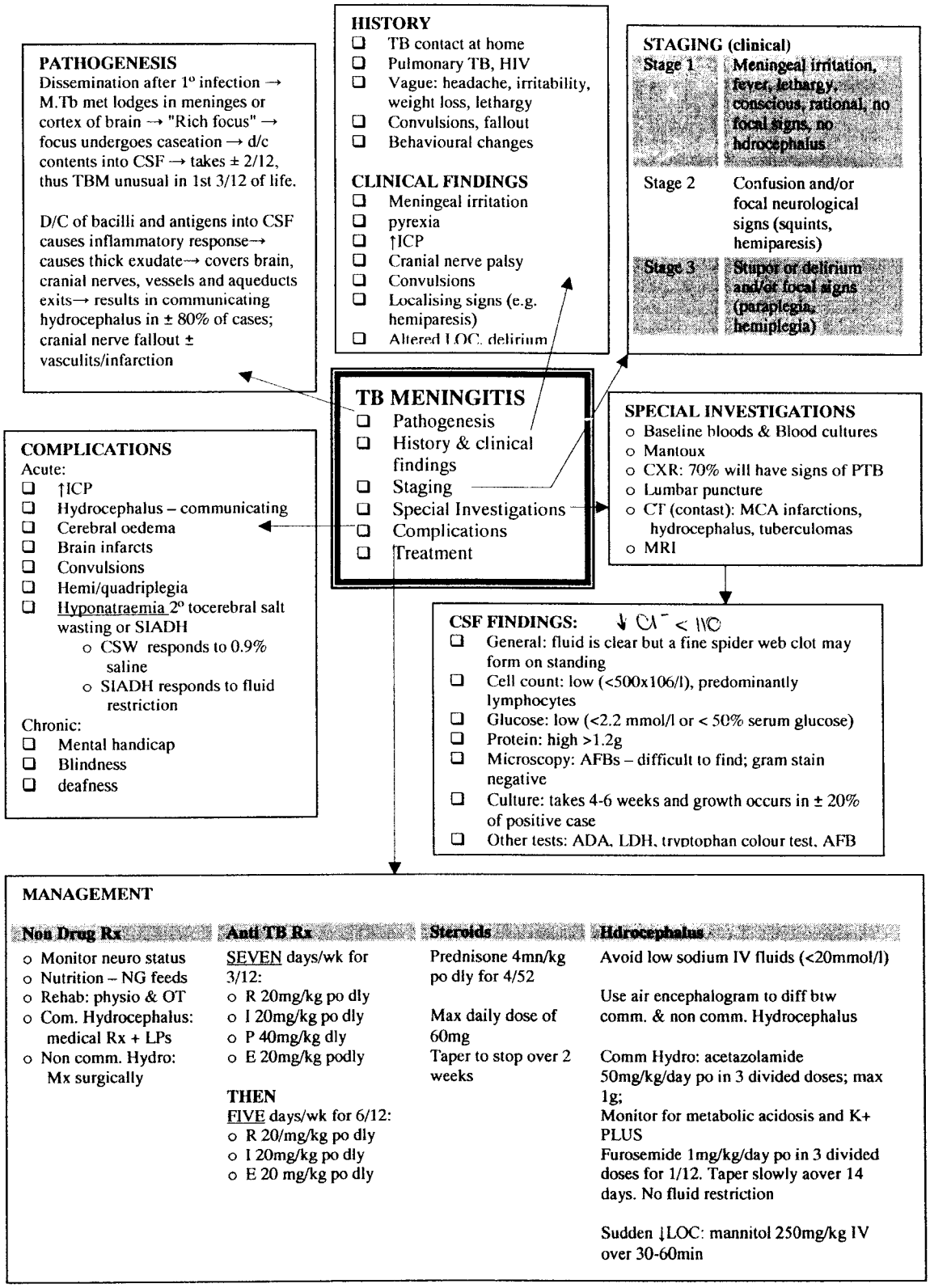
Note: these are the WHO 2006 guidelines

E, ethambutol; H, isoniazid; R, rifampicin; S, streptomycin; Z, pyrazinamide.
[a] Direct observation of drug administration is recommended during the initial phase of treatment and whenever the continuation phase contains rifampicin.
[b] In comparison with the treatment regimen for patients in diagnostic category I, ethambutol may be omitted during the initial phase of treatment for patients with non-cavitary, smear-negative pulmonary TB who are known to be HIV-negative, patients known to be infected with fully drug-susceptible bacilli and young children with primary TB.
[c] This regimen (2HRZE/6HE) may be associated with a higher rate of treatment failure and relapse compared with the 6-month regimen with rifampicin in the continuation phase.
[d] In comparison with the treatment regimen for patients in diagnostic category I, streptomycin replaces ethambutol in the treatment of TB meningitis.

Table A3.1 Second-line anti-TB drugs for treatment of MDR-TB in children

Drug	Mode of action	Common side-effects	Recommended daily dose	
			Range (mg/kg body weight)	Maximum (mg)
Ethionamide or prothionamide	Bactericidal	Vomiting, gastrointestinal upset[a]	15–20	1000
Fluoroquinolones[b]		Arthropathy, arthritis		
Ofloxacin	Bactericidal		15–20	800
Levofloxacin	Bactericidal		7.5–10	–
Moxifloxacin	Bactericidal		7.5–10	–
Gatifloxacin	Bactericidal		7.5–10	–
Ciprofloxacin	Bactericidal		20–30	1500
Aminoglycosides		Ototoxicity, hepatotoxicity		
Kanamycin	Bactericidal		15–30	1000
Amikacin	Bactericidal		15–22.5	1000
Capreomycin	Bactericidal		15–30	1000
Cycloserine or terizidone	Bacteriostatic	Psychiatric, neurological	10–20	1000
para-Aminosalicylic acid	Bacteriostatic	Vomiting, gastrointestinal upset	150	12 000

XDR TB = TB resistant to ≥ of the 2nd line drugs in this table


HISTORY
❑ TB contact at home
❑ Pulmonary TB, HIV
❑ Vague: headache, irritability, weight loss, lethargy
❑ Convulsions, fallout
❑ Behavioural changes
CLINICAL FINDINGS
❑ Meningeal irritation
❑ pyrexia
❑ ↑ICP
❑ Cranial nerve palsy
❑ Convulsions
❑ Localising signs (e.g. hemiparesis)
❑ Altered LOC, delirium
PATHOGENESIS
Dissemination after 1° infection → M.Tb met lodges in meninges or cortex of brain → "Rich focus" → focus undergoes caseation → d/c contents into CSF → takes ± 2/12, thus TBM unusual in 1st 3/12 of life.
D/C of bacilli and antigens into CSF causes inflammatory response→ causes thick exudate→ covers brain, cranial nerves, vessels and aqueducts exits→ results in communicating hydrocephalus in ± 80% of cases; cranial nerve fallout ± vasculits/infarction
STAGING (clinical)
Stage 1 Meningeal irritation, fever, lethargy, conscious, rational, no focal signs, no hdrocephalus
Stage 2 Confusion and/or focal neurological signs (squints, hemiparesis)
Stage 3 Stupor or delirium and/or focal signs (paraplegia, hemiplegia)
TB MENINGITIS
❑ Pathogenesis
❑ History & clinical findings
❑ Staging
❑ Special Investigations
❑ Complications
❑ Treatment
SPECIAL INVESTIGATIONS
o Baseline bloods & Blood cultures
o Mantoux
o CXR: 70% will have signs of PTB
o Lumbar puncture
o CT (contast): MCA infarctions, hydrocephalus, tuberculomas
o MRI
COMPLICATIONS
Acute:
❑ ↑ICP
❑ Hydrocephalus – communicating
❑ Cerebral oedema
❑ Brain infarcts
❑ Convulsions
❑ Hemi/quadriplegia
❑ Hyponatraemia 2° tocerebral salt wasting or SIADH
o CSW responds to 0.9% saline
o SIADH responds to fluid restriction
Chronic:
❑ Mental handicap
❑ Blindness
❑ deafness
CSF FINDINGS: ↓ Cl⁻ < 110
❑ General: fluid is clear but a fine spider web clot may form on standing
❑ Cell count: low (<500x106/l), predominantly lymphocytes
❑ Glucose: low (<2.2 mmol/l or < 50% serum glucose)
❑ Protein: high >1.2g
❑ Microscopy: AFBs – difficult to find; gram stain negative
❑ Culture: takes 4-6 weeks and growth occurs in ± 20% of positive case
❑ Other tests: ADA, LDH, tryptophan colour test, AFB
MANAGEMENT
Non Drug Rx
o Monitor neuro status
o Nutrition – NG feeds
o Rehab: physio & OT
o Com. Hydrocephalus: medical Rx + LPs
o Non comm. Hydro: Mx surgically
Anti TB Rx
SEVEN days/wk for 3/12:
o R 20mg/kg po dly
o I 20mg/kg po dly
o P 40mg/kg dly
o E 20mg/kg podly
THEN
FIVE days/wk for 6/12:
o R 20/mg/kg po dly
o I 20mg/kg po dly
o E 20 mg/kg po dly
Steroids
Prednisone 4mn/kg po dly for 4/52
Max daily dose of 60mg
Taper to stop over 2 weeks
Hdrocephalus
Avoid low sodium IV fluids (<20mmol/l)
Use air encephalogram to diff btw comm. & non comm. Hydrocephalus
Comm Hydro: acetazolamide 50mg/kg/day po in 3 divided doses; max 1g;
Monitor for metabolic acidosis and K+
PLUS
Furosemide 1mg/kg/day po in 3 divided doses for 1/12. Taper slowly aover 14 days. No fluid restriction
Sudden ↓LOC: mannitol 250mg/kg IV over 30-60min

AETIOLOGY/PATHOGENESIS

- ❑ Vector borne parasitic infection
- ❑ 4 species:

Species	*Type of malaria*
P.falciparum	***Malignant tertian***
P.vivax	*Tertian*
P.ovale	***Ovale***
P.malariae	*Quartan*

- ❑ Life cycle in 2 hosts:
- ❑ Asexual phase → (schizogny) humans
- ❑ Sexual phase→ (sporogony) anopheles mosquitoes
- ❑ CYCLE:
- ❑ Anopheles mosquito injects porozoites into blood stream
- ❑ Parasites then devlop in liver parenchymal cells (pre-erythrocytic phase)
- ❑ Invasion of bloodstream/RBC's occurs
- ❑ Multiplication & maturation within RBC's leads to cell rupture & the cycle repeats(eruthrocytic phase)
- ❑ Cycle lasts 48hr for *falciparum, vivx & ovale*; 72hrs for *malariae*
- ❑ *4 forms oif the parasite in this cycle are:*
- ❑ *Ring form →trophozoite →shizont →merozoite →*

Erythrocytic cycle/schizigony

- ❑ Attacks of fever correspond with the end of each erythrocytic cycle.
- ❑ After cycle has repeated a few time, gemtocytes of the parasite appear & are sucked up by mosquitos to start the sexual cycle in the insect host
- ❑ In *P.vivax, oval & malaria*, an exo-erythrocytic cycle occurs where parasites re-enter liver cells from the blood after the erythrocytic cycle has ceased. Subsequent re-invasion on the blood results in relapses of malaria (often years later)
- ❑ Ig level rise after infection→initially IgM →later IgG in chronic infection\transplacental passive immunity from mother to fetus explains rarity of congenital malaria
- ❑ Repeated exposure to infection leads to tolerance called 'premunity' but never complete immunity
- ❑ G6PD deficiency &/ sickle cell trait protect against P.falciparum infection
- ❑ Infection induces hyperactivity of the RES, with HSM, as well as anaemia & jaundice 2° to RBC destruction. Infected RBC's adhere causing small vessel thrombosis & infarction

SEVERE DISEASE

- ❑ This is a MEDICAL EMERGENCY
- ❑ Cerebral malaria (unrousable coma)
- ❑ Severe anaemia (Hb <5 g/dl)
- ❑ Parasitaemia > 10 000/µl
- ❑ Renal railure
- ❑ Pulmonary oedema
- ❑ Circulatory collapse
- ❑ Hypoglycaemia
- ❑ Spontaneous bleeding or DIC
- ❑ Repeated generalise convulsions
- ❑ Metabolic acidosis
- ❑ haemoglobinuria

Any 1 of this list present = severe malaria

MALARIA:

- ❑ Aetiology & pathogenesis
- ❑ Clinical manifestations
- ❑ Complications
- ❑ Severe disease

CLINICAL MANIFESTATIONS

P.falciparum:

- ❑ Incubation 7-12 days
- ❑ Abrupt onset, typically mane
- ❑ Fever (periodic)
- ❑ Classically Attack: headache, arthralgia and myalgia rapidly progress to shivering & rigors. Then flushing, N&V, severe headache, delirrium→ profuse sweating with relief of symptoms
- ❑ Convulsions may occur in young children, but cold, hot & sweating stages are rare
- ❑ Anaemia, HSM 7 mild leucopaenia are typical
- ❑ NB falciparum may present atypically

P.vivax,ovale, malariae

- ❑ Incubation 10-30days
- ❑ Otherwise as above

COMPLICATIONS

- ❑ Cerebral malaria :most dangerous, often fatal
  - Apathy, coma, disorientation, psychosis, focal or extrapyramidal Sx, convulsions, meningism
- ❑ GIT: vomiting, abdo pain, distension, diarrhoea ± dysentery
- ❑ Liver: necrosis with ⇈jaundice
- ❑ Renal failure: oliguria, anuria, 2[ ] to hypotension or coagulopathy →IV haemolysis + haemoglobinuria + oliguria = "blackwater fever"
- ❑ Haematological: anaemia ± cardiac failure, ↑ osmotic fragility, purpura and submucosal bleeding with DIC
- ❑ Pulmonary: severe refractory hypoxaemia 2[ ] tocapillary congestion & oedema
- ❑ Algid Malaria: resembles gram negative shock
- ❑ Chronic malaria: pts with inadequate or no treatment; ⇈splenomegally ± hypersplenism ± rupture is common

RBC BC. BURST → Hb in BLOOD +urine → KIDNEY failure

BLACK URINE

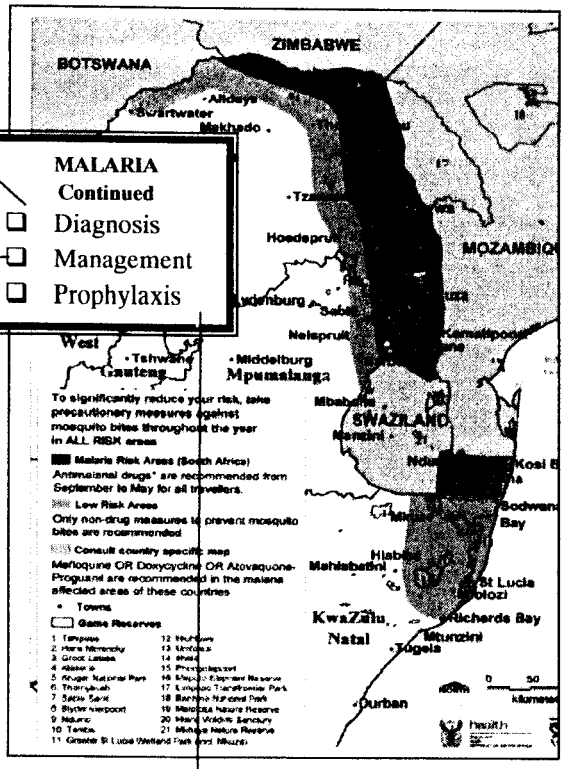
Special investigations:

- ❑ Hyperparasitaemia: >5% of RBCs infected
- ❑ Blood glucose <2.2 mmol/L
- ❑ Acidosis: lactate >5mmol/l, HCO_3< 15 mmol/l
- ❑ Thrombocytopaenia < 50 x 10^9/l

DIAGNOSIS

- ❑ Gold standard = identification of malaria parasites on thick & then blood smears
- ❑ Single negative smear does not exclude malaria
- ❑ High levels of parasitaemia (>4% or ≥3+) should be treated as severe malaria in non-immune patients.
- ❑ If severe malaria suspected, commence Rx & repeat smears after 6-8hrs
- ❑ Dipstick antigen tests: detect *P. falciparum* histidine rich protein (HRP), ➔ secreted by infected red cells. This antigen can be detected in red blood cells, serum, plasma cerebrospinal fluid and urine of patients infected with *P. falciparum* . Dipstix may also detect LDH or aldolase
- ❑ Other tests include: the Quantitative Buffy Coat (QBC) and Polymerase Chain Reaction (PCR).

MALARIA
Continued
- ❑ Diagnosis
- ❑ Management
- ❑ Prophylaxis



Treatment: UNCOMPLICATED malaria

Children < 1 year:

Quinine, oral 10mg/kg/dose 8 hrly for 7 days

- ❑ Clindamycin, po, 10mg/kg/dose bd for 7 days

Children > 1 year

- ❑ **Atemether –lumefantrine (coartem), po with fat containing food/milk (first choice)**
- ❑ 1st dose STAT, then 2nd dose after 8hrs, then bd for 2 days
- ❑ 1tab contains 20mg artemether + 120mg lumefantrine

Weight	Dose (tabs)	Total tabs
10-15kg	1	6
15-25kg	2	12
25-35kg	3	18
>35kg	4	24

OR

- ❑ Quinine, oral 10mg/kg/dose 8 hrly for 7 days PLUS
- ❑ Clindamycin, po, 10mg/kg/dose bd for 7 days if <8 yrs

OR

- ❑ Docycycline, po, 4mg/kg STAT, then 2mg/kg/daily with meals for 7 days MASKS TEETH BLACK

Treatment of COMPLICATED/SEVERE MALARIA

- ❑ Quinine IV, dilute in 5-10 ml/kg 5%DW or 0.9%NS ➔20mg/kg over 4 hrs, then 10mg/kg over 4-6hrs 8hrly until able to tolerate oral
- ❑ 2-3 days after starting IV quinine, quinine po 10mg/kg/dose 8hrly to complete 7-10 day course
- ❑ NB: quinine is cardiotoxic ➔monitor HR/ECG

PLUS

- ❑ Clindamycin, po, 10mg/kg/dose bd for 7 days if <8 yrs

OR

- ❑ Doxycycline, po, 4mg/kg STAT, then 2mg/kg/daily with meals for 7 days
- ❑ If concurrent bacterial sepsis:
- ❑ Ceftriaxone IV, 100mg/kg daily for 10 days
- ❑ Fever: paracetamol po, 10-15mg/kg/dose 6 hourly

NON drug treatment of SEVERE disease

- ❑ Admit to HC/ICU
- ❑ Avoid over hydration
- ❑ Monitor blood glucose & correct hypoglycaemia
- ❑ PRC 10ml/kg over 3 hrs if Hb <7g/dl
- ❑ Control convulsions
- ❑ Ventilation prn
- ❑ Nutritional support

PROPHYLAXIS

- ❑ IF > 5kg Mefloquine po, initiate Rx 8days before entering malaria area, continue through stay & for 4/52 after leaving
- ❑ IF > 8years, Doxycycline po, start 24hrs prior to entering malaria area, continue thru stay & for 4/52 after leaving
- ❑ Atovaqoune-proguanil: This combination is not recommended for children under 11kgs in weight due to lack of data.

Malaria prophylaxis (EDL)

Weight (Kg)	Mefloquine weekly	Doxycycline weekly
5-20	62.5mg	Contra-indicated
21-30	125mg	Contra-indicated
31-45	187.5mg	2mg/kg
>45	250mg	100mg

IMMUNIZATION

NEW EPI SCHEDULE 2010

AGE	VACCINE	SITE
Birth	❑ BCG ❑ Polio 0	❑ R arm ❑ Oral
6 weeks	❑ Polio 1 ❑ RotaVirus 1 ❑ Pneumococcal 1 ❑ Hep B 1 ❑ DTaP-IPV/HIB 1	❑ Oral ❑ Oral ❑ R Thigh ❑ R thigh ❑ L thigh
10 weeks	❑ DTaP-IPV/HIB 2 ❑ Hep B 2	❑ L thigh ❑ R thigh
14 weeks	❑ Rotavirus 2 ❑ Pneumococcal 2 ❑ Hep B 3 ❑ DTaP-IPV/HIB 3	❑ Oral ❑ L thigh ❑ R thigh ❑ L thigh
9 months	❑ Measles 1 ❑ Pneumococcal 3	❑ L thigh ❑ R thigh
18 months	❑ DTaP-IPV/HIB 4 ❑ Measles 2	❑ L arm ❑ R arm
6 years	❑ TM	❑ L arm
12 years	❑ TDap	❑ L arm

Missed Opportunities:

- ❑ A lapse in immunization does not require restarting of whole schedule
- ❑ Remaining doses must be given as if lapse had not occurred
- ❑ **NB no pertussis vaccine over 2 yrs of age**

HIV/AIDS:

- ❑ Full schedule, except:
- ❑ No **BCG** in kids with **AIDS;** but they can get live OPV

CONTRAINDICATIONS

- ❑ **Egg allergy:** measles, mumps, yellow fever, influenza
- ❑ **Immunosuppression:** no live vaccines – BCG, measles, MMR, OPV (give inactivated polio)
- ❑ **Pertussis:** not in kids with progressive CNS disease or if there was a severe reaction to previous dose (shock, collapse, anaphylaxis, screaming for > 4 hrs, fever > 40.5, convulsions or encephalopathy within 7 days)
  Note: pyrexia of 38.5, local induration & tenderness are normal
  NB no pertussis vaccine over 2 yrs of age
- ❑ Admin of plasma± immunoglobulin: defer MMR for 3 months

CONDITIONS with NO C/I to IMMUNIZATION

- ❑ minor illness with low grade fever, diarrhoea, URTI
- ❑ malnutrition
- ❑ breastfeeding
- ❑ prematurity – start at same chronological age as term infants
- ❑ family Hx of convulsions
- ❑ Hx of non-specific allergies, asthma, hayfever or rhinitis
- ❑ Dermatoses, eczema, localised skin infections
- ❑ Allergy to antibiotics except neomycin & streptomycin (contained in some vaccines)
- ❑ Soreness, redness, or T< 40 following previous DTP
- ❑ Treatment with antibiotics
- ❑ Children using topical, inhaled, short term (< 2 weeks) or low dose maintenance steroid therapy for a conditions that is not immune suppressive
- ❑ Static neurological disorders like cerebral palsy or Down syndrome

PASSIVE immunization

- ❑ **Measles:** give contacts IG 0.25 ml/kg (for imm-comp kids give 0.5ml/kg) with max 15ml within 6 days of exposure
- ❑ **Hep A:** contacts get IG 0.02ml/kg within 2/52 of exposure
- ❑ Hep B: newborns of mothers with acture or chronic Hep B- HBIG within 12hrs of delivery = PEP
- ❑ **Rabies:** rabies immunoglobulin 20IU/kg post exposure
- ❑ **Tetanus IG:** hyperimmune tetanus immunoglobulin (HTIG) 500IU for newborns, 2000 IU for children; 75-250 IU prophylaxis for severe wounds if incompletely immunised.
- ❑ **Varicalla Zoster IG:** 0.15ml/kg for susceptible children within 96hrs, & for newborns of mothers who contracted chickenpox btw 5 days pre- and 2 days post- delivery.

1. FEVER Overview:

- ❑ Common presenting symptom of both infectious & non infectious disease.
- ❑ In healthy individuals, body T° maintained in range ± 3° C, by # level of control
- ❑ Receptors in skin, SC & hypothalamus
- ❑ 'thermostat' in hypothalamus determines set point
- ❑ Effector channels to retain or release heat to maintain set point- metabolic rate, vasoconstriction/dilatation, sweating & behavioural responses
- ❑ Changes to set point: this occurs in febrile states where set point ↑↑. Fever is maintained by ↑ heat production (metabolism, shivering) and ↓ loss by vasoconstriction.
- ❑ Set point ↑ by: bacterial products which stimulate macrophages & PMNs to release IL-1, TNF, IFN etc; ACTH and endorphins are also released.
- ❑ IL-1 ↑ production of prostaglandins in the hypothalamus & these rest the hypothalamic thermostat by an unknown mechanism
- ❑ T° > 41°C are typically bacterial
- ❑ However, newborns, preterm & malnourished children are often **anergic** & do not manifest high T° despite severe infection

COMMON PROBLEMS OF INFECTION

1. Fever (overview)
2. PUO/FUO
3. SEPSIS/SIRS

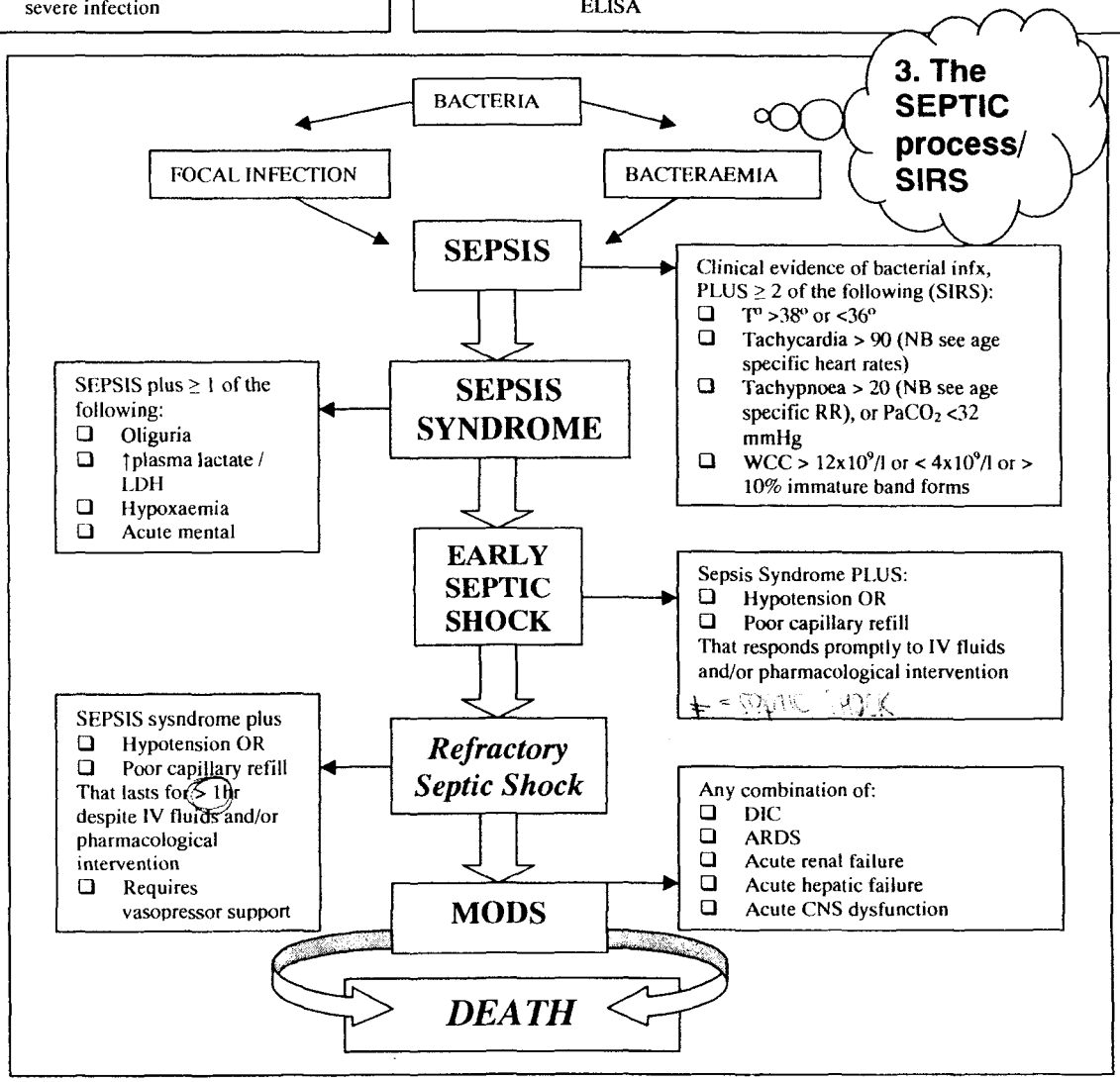
Treatment of FEVER:

- ❑ Physical methods: tepid baths, sponging, remove excess clothing, ensure hydration
- ❑ Drugs: paracetamol 5-10mg/kg 6 hourly.
- ❑ Avoid Aspirin in febrile children because of the risk of Reye's syndrome

2. PYREXIA OF UNKNOWN ORIGIN: no diagnosis after ≥ 1/52 of appropriate investigations in hospital. Aetiology varies according to age & geographical location

Common causes of PUO in developing countries

Disease	Clinical features & diagnostic tests
TB	Fever, anorexia, FTT +- contacts, mantoux, CXR, NGA for AFB
Typhoid	TOXIC, swinging fever, relative bradycardia, diarrhoea, rose spots, splenomegaly,, delirium, death → leucopaenia, widal test (antibody test), blood cultures (most NB), stool culture
Malaria	Febrile episodes, anaemia, jaundice, splenomegaly → blood film, malaria antigens
Amoebiasis	Bloody stools, RLQ pain, anaemia → leucocytosis, serology, stool exam
UTI	Fever, suprapubic pain → urine dipstix & MC&S
Osteomyelitis	Fever, bone pain → blood culture
Endocarditis	Fever, changing or new murmur, splenomegaly, petechiae → leucocytosis, blood culture, echocardiography
HIV	Wasting, lymphadenpathy, chronic diarrhoea, chronic RTI → ELISA



Serious systemic infections presenting with signs of sepsis ± septic shock

	Gram - & + Infections presenting with sepsis	Staph Infx presenting with "localised sepsis"	Toxic Shock Syndrome	H. Influenza Infections
Organisms	❑ *Gram - : E.coli, Klebsiella Spp, H. Influenza, N. meningitides, Salmonella spp* ❑ *Gram +: staph aureus,Strep. pneumonia*	*Staph. aureus* *Staph epidermidis*	❑ *Staph. aureus* producing endotoxin (TSS Toxin-1) ❑ Occasionally endotoxin producing *streptococci*	❑ H. influenza = gram negative pleomorphic rod ❑ Six serotypes (a to f) identified based on capsular polkysaccharide ❑ Type B = major cause of invasive bacterial disease worldwide
Clinical features	Non specific (see sepsis diagram); sometimes a focus of infection is obvious. Cutaneous manifestations → petechiae, ecchymosis, peripheral gangrene may be present	❑ Localised sepsis → tropical myositis, acute bacterial endocarditis, tracheitis and pneumonia ❑ Toxin producing staphs → scalded skin syndrome, food poisoning ± 2° sepsis	Pyrexia, hypotension, abdominal pain, vomiting, diarrhoea, and an erythematous rash	❑ Sepstic shock ❑ Meningitis ❑ Septicaemia ❑ Arthritis ❑ Cellulites ❑ Epiglottitis ❑ Pneumonia
Diagnosis	Blood culture, bacterial antigens in urine, CSF or plasma; evidence of DIC, metabolic acidosis, anaemia, neutrophilia/paenia, hypoglycaemia	❑ Isolation from septic focus ❑ Blood cultures	Clinical: ❑ Fever ❑ Diffuse macular erythematous rash which desquamates ≥ 2 weeks ❑ Hypotension ! Plus 3 of the following: ❑ Renal:↑ U & Cr ❑ Liver: ↑ AST, ALT, bili ❑ Blood: thromobcytopaenia ❑ CNS: ↕ LOC ❑ GIT: diarrhoea, vomiting ❑ MSK: myalgia, ↑ CK	Definitive Dx based on isolation from: ❑ CSF ❑ Blood ❑ Pleural fluid ❑ Joint aspirate Antign detection (immune electrophoresis or latex agglutination) in: ❑ CSF ❑ Urine ❑ Serum joint aspirates pleural fluid
Treatment	Empiric antibiotics ❑ Ampicillin + gentamycin ❑ 3rd Gen cephalosporin Change according to MC&S ❑ Hypoxia – Oxygen ± ventilation if hypventilation ❑ IV fluids ± vasopressors ❑ DIC: ffp, platelets, cryoprecipitate ❑ Heparin & steroids controversial	Cloxacillin = drug of choice ❑ Fusidic acid & vancomycin are alternatives for drug resistant infx ❑ Deep infections like arthritis & endocarditis – Rx for ≥ 6 weeks	❑ Rx with β-lactamase resistant anti-staph agent (cloxacillin) for ≥ 10 days. ❑ If streptococcal: penicillin plus IVIG ❑ Drain focal septic lesions ❑ Shock treated as for column 1	Antibiotics: ❑ Ampicillin plus chloramphenicol OR ❑ 3rd generation cephalosporin ❑ Rx for 10-14 days ❑ Shock, meningitis, pneumonia and epiglottitis according to protocols Primary Prevention: ❑ HiB vaccine at 6w, 10w, 14w, & 18 months Secondary Prevention: ❑ Rifampicin 20/mg/kg/day x 4/7 as chemoprophylaxis
Differential Diagnoses			❑ Scarlet fever ❑ Severe measles ❑ Kawasaki syndrome ❑ Rocky mountain Spotted Fever	

STAPHYLOCOCCAL & STREPTOCOCCAL SKIN CONDITIONS

IMPETIGO:

- ❑ Common, superficial, contagious
- ❑ Caused by staphs & streps
- ❑ Source: nose & other kids
- ❑ Starts in nostrils, spreads to face with 2° spread..
- ❑ Lesions: superficial blisters that spread forming round, moist, eroded/crusted areas.
- ❑ Management:
  - o Local ontibiotic ointment:
  - o Polysporin (polymyxin B & Bacitracin) or Terramycin (oxytetracycline)
  - o Oral systemic antibiotics if widespread: Flucoloxacillin, oral 12,5-25mg/kg/dose 6 hrly for 7 days OR erythromycin, oral, 10mg/kg/dose 6 hrly for 7 days

IMPETIGO NEONATORUM

- ❑ Neonates very susceptible to staph. aureus
- ❑ Tend to develop generalised infection
- ❑ Lesions: superficial blisters that enlarge rapidly – pus filled
- ❑ Management:
  - o Swabs for MC&S
  - o Serology for syphilis
  - o Antibiotics:
  - o Cloxacillin, IV, 50mg/kg/dose 6 hrly for 5 days
- ❑ Diff Dx:
  - o Epidermolysis bullosa
  - o Congenital syphillis

STAPHYLOCOCCAL SCALDED SKIN SYNDROME

- ❑ Clinically resembles superficial burns
- ❑ Due to toxin causing erythema and desquamation
- ❑ Source of infection: nose, eyes skin
- ❑ Management
  - o Cloxacillin, IV, 50mg/kg/dose 6hrly for 5 days OR
  - o Flucoloxacillin, oral 12,5-25mg/kg/dose 6 hrly for 7 days

SKIN ERUPTIONS 2° to STEPTOCOCCAL TONSILLITIS (group A β haemolytic streps)

Possible manifestations:

- ❑ Desquamation of skin- especially palms & soles! OR
- ❑ Fine rash with small, diffuse , superficial papules OR
- ❑ Guttate psoriasis OR
- ❑ Seborrhoeic dermatitis OR
- ❑ Urticaria
- ❑ NB: any unusual rash in kids► rule out streptococcal infection
- ❑ **NB strep skin infx may precipitate glomerulonephritis**
- ❑ Management:
  - o Phenoxymethylpenicillin, oral, 12,5 mg/kg/dose 6 hrly for 10 days OR
  - o benzathine benzylpenicillin (depot), IM, 600 000 – 1,2 million units, 2 doses given 5 days apart, OR
  - o erythromycin 10mg/kg/dose po, 6hrly for 10 days.

STREPTOCOCCAL SCARLET FEVER

- ❑ Group A streps implicated (± C & G)
- ❑ Acute onset fever +sore throat +strawberry tongue
- ❑ 24hrs later diffuse sandpaper rash in groing, axillae, neck, cubital fossa
- ❑ Blanches under pressure
- ❑ Classically circumoral pallor
- ❑ Common in school age kids
- ❑ Disease lasts 7 days, with rash disappearing in 7-10 days.
- ❑ Residual petechial rash in antecubital fossa = 'Pastia's sign' (for Belinda & kobus!)
- ❑ Management
  - o Symptomatic PLUS
  - o Penicillin OR
  - o amoxycillin OR
  - o erythromycin

EPIDERMOLYSIS BULLOSA

- ❑ Inherited kin disorder with blistering due to abnormal keratin, collagen, laminin &/or integrin
- ❑ 3 main types
- ❑ Base of lesions NOT erythematous
- ❑ Nails may also be lost

CAUSES of BLISTERING @ BIRTH:

- ❑ Impetigo
- ❑ Herpes simplex
- ❑ Bullous Icthyosis
- ❑ Epidermolysis bullosa
- ❑ Bullous Congenital syphilis
- ❑ Incontinentia pigmenti

Chicken Pox

1. PATHOGENESIS

- ❑ Varicella zoster virus
- ❑ Incubation: 10-21 days
- ❑ Infectivity: 24hrs prior to rash until vesicles have crusted over (typically 6-7 days)
- ❑ Transmission rate = 85% in household contacts via respiratory secretions & vesicle fluid
- ❑ 1° infections usually results in lifelong immunity
- ❑ Maternal infection in 1st/2nd trimester→ cong abnormalities (low bith weight, CNS abn., digit/limb abn, cutaneous scarring, eye defects) \maternal inf 5 days prior & up to 2 days post delivery can lead to neonatal varicella

2. CLINICAL PRESENTATION

- ❑ After incubation, a mild prodrome occurs (fever, malaise, headache), lasting 24-48 hours
- ❑ Then red papules appear → develop into clear vesicles
- ❑ Within 24hrs, become cloudy, umbilicate and dry to scabs
- ❑ Vesicle erupt in crops for 3-4days, starting on trunk→ face, scalp, conjunctiva & mucous membranes
- ❑ "teardrops on an erythematous base"
- ❑ At height of eruption, all stage (macules, vesicles & crusts) present at same time
- ❑ Pruritis is severe
- ❑ Rash lasts 8-10 days & heals without scarring, unless 2° infection
- ❑ Systemic reaction typically minor
- ❑ Mucous membranes may be involved

CHICKEN POX

1. Pathogenesis
2. Clinical presentation
3. Differential Dx
4. Complications
5. Management
6. Shingles

3. DIFFERENTIAL DIAGNOSIS

- ❑ Popular urticaria
- ❑ Bullous impetigo
- ❑ Scabies
- ❑ Molluscum contagiousum

4. COMPLICATIONS

COMMON:

- ❑ 2° sepsis due to staphylococci/ streptococci

RARE:

- ❑ thrombocytopaenia,
- ❑ pneumonia, myocarditis,
- ❑ hepatitis,
- ❑ glomerulonephritis,
- ❑ **Encephalitis: ataxia, vomiting, seizures, coma**
- ❑ Guillain-Barre,
- ❑ cerebellare ataxia

Complications = common exam question!

REYE'S Syndrome: **if salicylates** given for pain

- ❑ acute hepatic encephalopathy & non-inflammatory fatty infiltration of liver & kidney
- ❑ mitochondrial injury
- ❑ 40% mortality

ADULTS: tend to develop encephalitis charactrised by convulsions, ↓ LOC and focal signs

AIDS/ MALIGNANCY

- ❑ Severe disseminated disease- often fatal

5. MANAGEMENT

Non drug treatment

- ❑ Isolation
- ❑ Isolate neonates until mother is non contagious
- ❑ Maintain adequate hydration

Drug Treatment

- ❑ Antiviral therapy: immunocompeten patient with Cx & all immunocompromised patients
  - o Acyclovir 40mg/kg po 8 hrly daily for 5 days max dose 800mg.dose OR
  - o Acyclovir 500mg/m^2/dose IV 8hrly over 1 hour for 7-10 days
- ❑ Fever: paracetamol: 10-15mg/kg po 8 hrly for 5 days
- ❑ Pruritis:
  - o Mild: calamine lotion, topical tds prn
  - o Promethazine 0.25-0.5 mg/kg/dose 6 hrly for 24-48hrs
- ❑ 2° skin infection:
  - o Amoxicillin po 30mg/kg/dose 8hrly for 5 days PLUS
  - o Flucloxacillin po 12,5-25 mg/kg/dose 6 hrly for 5 days

Prophylaxis

- o Neonates: varicella zoster immunoglobulin, IM, 100U given with 96hrs of exposure OR
- o Acyclovir po 20mg/kg/dose 8hrly for 10days
- o Immunocompromised contacts: Acyclovir po 20mg/kg/dose 8hrly for 10days

6. SHINGLES/HERPES ZOSTER

Reactivation of latent infection with VZV

Virus remains dormant in dorsal nerve roots → Uncommon in normal chidren → Spreads from sensory ganglia along nerves to skin

Clinical features:

- ❑ Pain & paraesthesia over a sensory dermatome (spinal/cranial), followed 2-4days by local vesicular eruption
- ❑ Cx: meningitis, encephalitis, post-herpetic neuralgia

Management:

- ❑ IV acyclocir for severe disease, HIV/AIDS, malignancies
- ❑ Antibiotics for 2° infection
- ❑ Prophylaxis: Zoster immune plasma: 10mg/kg IV for susceptible contacts

↑ shingles

Measles

Measles▶ acute, highly contagious disease caused by RNA paramyxovirus→ *morbilivirus.* Outbreaks typically affect 5-14y/o age group. Transmission is by droplet spread.

Barking cough!!
r/o LTB, croup, pertussis

1. PATHOPHYSIOLOGY

- ❑ Communicable for 7 days from onset of prodrome
- ❑ Inhaled droplets enter body in URT & spread to subepithelial & local lymphatic tissue
- ❑ Virus multiplies in lymphoid tissue for next few days (incl spleen)
- ❑ 1 week later viraemia occurs with dissemination to epithelial sites
- ❑ Respiratory symptoms then occur as resp epithelium is thin
- ❑ ± 10 days post infection, pt develops acute illness with fever, coryza, cough & conjunctivitis
- ❑ 2 days prior to generalised maculpapular rash, the patient develops Koplic spots on the buccal mucosa
- ❑ Morbilliform Rash starts on face, then descends over entire body for next 3 days
- ❑ Not contagious 4 days after onset of rash
- ❑ Clinical expression of infection/complications depend on immune status & vitamin A status.
- ❑ Both vitamin A deficiency and measles cause epithelial damage

Inccubat period ± 10davs

Prodromal Phase lasts 3-5 davs

2. CLINICAL FEATURES

After incubation (± days) prodrome of cough, coryza, conjunctivitis & fever occurs.

- ❑ Koplik spots (enanthem) occur 2 days before muculopapular skin rash (exanthum).
- ❑ Rash begins on face, spread to trunk, arms & reaches legs by third day
- ❑ Then starts fading by third day in order of appearance
- ❑ Fever subsides by third day, convalescence ensues & child is no longer infectious
- ❑ If feverish beyond third day- suspect complications

MEASLES
1. Pathophysiology
2. Clinical features
3. Diagnosis
4. Management
5. Complications

3. DIAGNOSIS

- ❑ Clinical findings & history of progression
- ❑ Serology for measles IgM

4. MANAGEMENT

Primary prevention:
- ❑ Live attenuated vaccine at 9 & 18 months according to new EPI schedule

Non drug treatment:
- ❑ *Notify provincial EPI manager*
- ❑ Admit High risk patients:
  - o < 6/12 old
  - o Immune compromised
  - o Severe malnutrition
  - o Complications
- ❑ Minimal exposure to light
- ❑ Isolate (infectious for 4 days from onset of rash)
- ❑ If pneumonia + hypoxia: give oxygen
- ❑ Cleanse eyes with warm saline

Drug treatment:
- ❑ All patients get Vit A po, single dose for 2 days
- ❑ <1y/o 100 000 units
- ❑ >1y/o 200 000 units
- ❑ Fever paracetamol po 10-15mg/kg/dose 6hrly
- ❑ *Complications: treat according to protocols*

Contacts:
- ❑ Immunise children > 6/12 if unvaccinated & <72hrs post exposure
- ❑ Btw 3-6 days post exposure & contacts < 6/12 : Gamma globulin, IM 0,25 ml/kg
- ❑ Immunodeficient: Gamma globulin, IM 0,5 ml/kg
- ❑ Immunise all kids > 6/12 if outbreak occurs

5. COMPLICATIONS

- ❑ Secondary bacterial infections: laryngotracheo-bronchitis (LTB), bronchopneumonia, otitis media, croup
- ❑ Diarrhoea: common, reduces food intake & contributes to negative nitrogen balance. Due to cellular damage of absorptive surface of bowel, with protein losing enteropathy
- ❑ Life threatening → post measle pneumonia, croup & disrrhoea
- ❑ Seconday viral infections: pneumonia (adenovirus), LTB (adenovius, para influenza, herpes simplex)
- ❑ Corneal ulceration & blindness
- ❑ Malnutrition
- ❑ **Post measles immune suppression** with FTT, recurrent infections, persistent pneumonia & diarrhoea
- ❑ **Encephalitis (1:1000): ataxia, vomiting, seizures, coma**
- ❑ Subacute Sclerosing Pan-encephalitis (1:100,000): slow measles infection of brain manifesting years later, characterized by preogressive cerebral deterioration, myoclonic jerks, fatal within 6-12 months.

RUBELLA jaundice

- ❑ Rubivirus
- ❑ Incubation: 14-21 days
- ❑ Infectivity: 7 days pre-rash to 5 days post rash
- ❑ Droplet spread
- ❑ Diagnosis: serology IgM
- ❑ Clinical features:
  - o Prodrome- non-specific- coryz, conjunctivitis, tender lymphadenopathy
  - o Rash: **maculopapular** on face→ entire body; pruritic, disappear by 4th day
  - o Congentical rubella sysndrome: cataracts/glaucoma, congenital heart disease, purpura (bluberry muffin baby), HSM, jaundice, microcephaly, developmental delay, radioluscent bones
- ❑ Management:► Symptomatic
- ❑ Prognosis:
  - o Excellent in acquired disease
  - o Irreversible if congenital
- ❑ Complications
  - o Arthralgis/arthritis
  - o encephalitis

INFECTIOUS MONONUCLEOSIS (IM)

- ❑ Caused by EBV→ infects B lymphocytes
- ❑ Infections occurs early in RSA
- ❑ Seldom recognised as a clinical entity in kids
- ❑ Clinical Features:
- ❑ Classical IM syndrome:
  - o Young adults: insidious, malaise, nausea → 2/52 later fever + pharyngitis ± tonsillar exudates & petechiae on palate; epitrochlear & cervical lymphadenopathy + HSM; ± **maculo-papular rash**; CHRONIC FATIGUE
  - o Infants/young kids: usually subclinical; otherwise: URTI, hepatitis, guillian barre, thrombocytopaenia, haemolytic anaemia, transverse myelitis, meningo-encephalitis
  - o Reactivation may occur!
- ❑ Diagnosis:
  - o FBC: leucocytosis; atypical lymphocytes (20-40%), downey cells, thrombocytopaenia, anaemia
  - o Serology: IgM, IgG
- ❑ Differential: CMV, tocoplasmosis, hepatitis, strep throat, diphtheria, rubella
- ❑ Supportive; AVOID ampicillin ►precipitates skin eruption

MORE VIRAL RASHES

- ❑ Rubella
- ❑ Infectious Mononucleosis
- ❑ CMV
- ❑ HSV-1

CYTOMEGALOVIRUS (CMV)

- ❑ CMV has the characteristics of herpes & causes IM like disease.
- ❑ Virus excreted in urine, faeces, milk, saliva, respiratory tract\transmitted from any of above sources
- ❑ \infection typically subclinical in immune-competent kids
- ❑ Infants may present with **petechial rash**, pneumonia, HSM, polyneuritis
- ❑ Immunocompromised kids► pneumonia, colitis, hepatitis, chorioretinitis
- ❑ Diagnosis by viral isolation
- ❑ Differential: EBV, hepatitis A or B
- ❑ Management:
  - o Gancyclovir if available

CMV

→ cytomegalo inclusion disease (congen)
- jaundice
- HSM
- petechial rash
- multi organ involvem.

also blueberry muffin

HERPES SIMPLEX- 1 skin + mouth

- ❑ Infants protected for a few months by maternal antibodies
- ❑ Primary infx at 1-5 years of age
- ❑ Spread by saliva or close personal contact
- ❑ **Vesicular lesions** on skin & mucous membranes
- ❑ Viraemia & dissemination in immunosupressed kids.
- ❑ High risk patients ►IV acyclovir

Clinical syndromes
- ❑ gingivostomatitis► commonest cause of stomatitis is kids; fever, salivations & refusal to eat. Vesicles, THEN Shallow, painful ulcers with thin red margin and yellow-grey base →lips, gums, tongue, buccal membranes
- ❑ Eczema herpeticum: widespread infx of eczematous skin ►crops of vesicles occur for 7-10 days; systemic reaction with high fever common
- ❑ Conjunctivitis
- ❑ Meningo-encephalitis (typically HSV-2 in neonates, HSV-1 in older kids)
- ❑ Disseminated infections ►immunecompromised

Management:
- ❑ Non drug Rx: hydrate with oral/NG/IV fluids
- ❑ Drugs:
  - o Chlorhexidine 0.2%, 10ml as mouthwash or gargle, 12hrly (DO NOT SWALLOW)
  - o Acyclovir, IV, 5-10mg/kg/dose 8 hrly for 7-14 days if severe infection/ immune compromised ►change to oral ASAP 10-20mg/kg/dose 4-6hrly
  - o Amoxil 35-45 mg/kg/dose po 8hrly if suspected super infection

MENINGOCOCCAL DISEASE

- ❑ Caused by Neisseria meningitides
- ❑ Gram negative diploccocus
- ❑ Droplet spread

Risk Factors
- ❑ Overcrowding
- ❑ Crèche/institution attendance
- ❑ Immune deficiency (esp. C3-8 deficiency)
- ❑ NOTIFIABLE CONDITION (meningitis)

Clinical features
- ❑ Incubation period 2-4days
- ❑ 35% meningitis; 15% septicaemia, 50% both!
- ❑ Pneumonia , enopthalmitis also occur (rare)
- ❑ SEPTICAEMIA (case fatality 25%):
  - o NB: onset of sepsis ABRUPT (hours) : fever, chills, prostration **with rapidly evolving petechial or purpuric rash**
  - o Involves MUCOUS membranes, as well as palms, soles
  - o DIC, purpura, & shock may be followed by coma & death in HOURS
  - o Poor predictive factors: rapid onset, shock, acidosis, coma, seizures, DIC and the ABSENCE of meningitis
- ❑ MENINGITIS is indistinguishable from other causes of meningitis (case fatality 5%)

Diagnosis:
- ❑ **Non blanching purpura in a sick, febrile child is virtually diagnostic of meningococcaemia**
- ❑ Diagnosis may be confirmed by blood culture & organisms may be seen on skin scrapings/biopsy
- ❑ Cautious LP as most patients have raised ICP→ treat empirically if signs of ↑ ICP
- ❑ Can also detect antigens in CSF, urine, serum: latex agglutination or electrophoresis.

Differential:
- ❑ Henoch Schonlein purpura, viral haemorrhagic disease, ITP.

Management:
- ❑ MEDICAL EMERGENCY (if SEPSIS)
- ❑ DRUGS: (from EDL)
  - o benzylpenicillin (Pen G), IV, 100 000 U/kg/dose STAT, then 4hrly PLUS
  - o hydrocortisone, IV, 4-6mg/kg/dose, STAT, then 4-6hrly
- ❑ NON DRUG: monitor vitals, capillary refill, IV fluids; Mx shock according to protocol
- ❑ MENINGITIS (from EDL)
  - o Cefotaxime, IV, 25-50 mg/kg/dose, 6-8hrly OR
  - o Ceftriaxone, IV, 50mg/kg/dose 12 hrly
  - o Dexamethasone, IV, 0.15 mg/kg 6hrly for 3 days
  - o Paracetamol po for pain & fever

Contacts: (from EDL)
- ❑ Ceftriaxone, IM STAT dose
  - o <12 years 125mg
  - o >12 years 250mg OR
- ❑ Ciprofloxacin, po, 10mg/kg STAT dose
  - o 6-12 years 250mg
  - o >12 years 500ng
- ❑ Rifampicin, oral
  - o 3-12 months 5mg/kg bd x 2 days
  - o > 1 year 10mg/kg bd x 2 days
  - o Adults 600mg bd x 2 days

Prevention
- ❑ Vaccine against groups A,C,Y and W135
- ❑ Group B is the predominant strain in RSA

DISEASES associated with **PETECHIAL/PURPURIC** Rashes
- ❑ Meningococcal Disease
- ❑ Rickettsial Infections

RICKETTSIAL INFECTION

- ❑ Obligate intracellular organisms
- ❑ Pleomorphic coccobacilli
- ❑ Transmitted to humans from animals via arthropod vectors (tick, lice, flea, mites)
- ❑ Small vessel endothelium is invaded
- ❑ There is subsequent proliferation of cells resulting in thrombosis ± plasma leakage
- ❑ Changes occur in:
  - o Skin
  - o Meninges
  - o Brain
  - o Myocardium
  - o Kidneys
  - o Lungs
- ❑ Clinical entities (only common South African ones)
  - o Tickbite Fever
  - o Epidemic Typhus
  - o Q-fever (rickettsia-like organism: coxiella burnetti)

DIAGNOSIS
- ❑ Rule OUT: meningococcaemia, typhoid, measles, meningitis, encephalopathy
- ❑ Look for the ESCHAR!
- ❑ Labs: indirect immunoflourescent Ab assay

TREATMENT
- ❑ Chloramphenicol & tetracyclines ▶bacteriostatic only.
- ❑ 50 -100 mg/kg/day po in 4 divided doses OR 30-40 mg/kg/day IV in 3 divided doses OR
- ❑ Docycycline 2-4 mg/kg dly po
- ❑ Supportive measure
- ❑ Continue until apyrexial for 48hrs

TICKBITE FEVER

- ❑ R. conorii, R. australis
- ❑ Pyrexia, headache which reach peak intensity & respond poorly to symptomatic Rx
- ❑ A small ESCHAR at the bite site found in most patients, with regional lyphadenopathy
- ❑ **Rash (appears on DAY 2): either maculopapular ornon-blanching purpuric rash of of vasculitis**
- ❑ **Involves trunk, limbs, palms, soles**
- ❑ Significant CVS, respiratory or CNS symptoms typically absent
- ❑ Rarely meningo-encephalitis picture

EPIDEMIC TYPHUS

- ❑ R. prowazekii
- ❑ Transmitted by infected faeces of human body lice
- ❑ Occurs in times of war or overcrowding with poor hygiene
- ❑ Incubation period of 14 days
- ❑ Then sudden onset pyrexia, headache, malaise.
- ❑ Rash (DAY 4-7): blanches on pressure but may be haemorrhagic in severe cases
- ❑ Severe disease: stupor, delitium, collapse, renal failure, pneumonia
- ❑ Revovers in 3 weeks if untreated

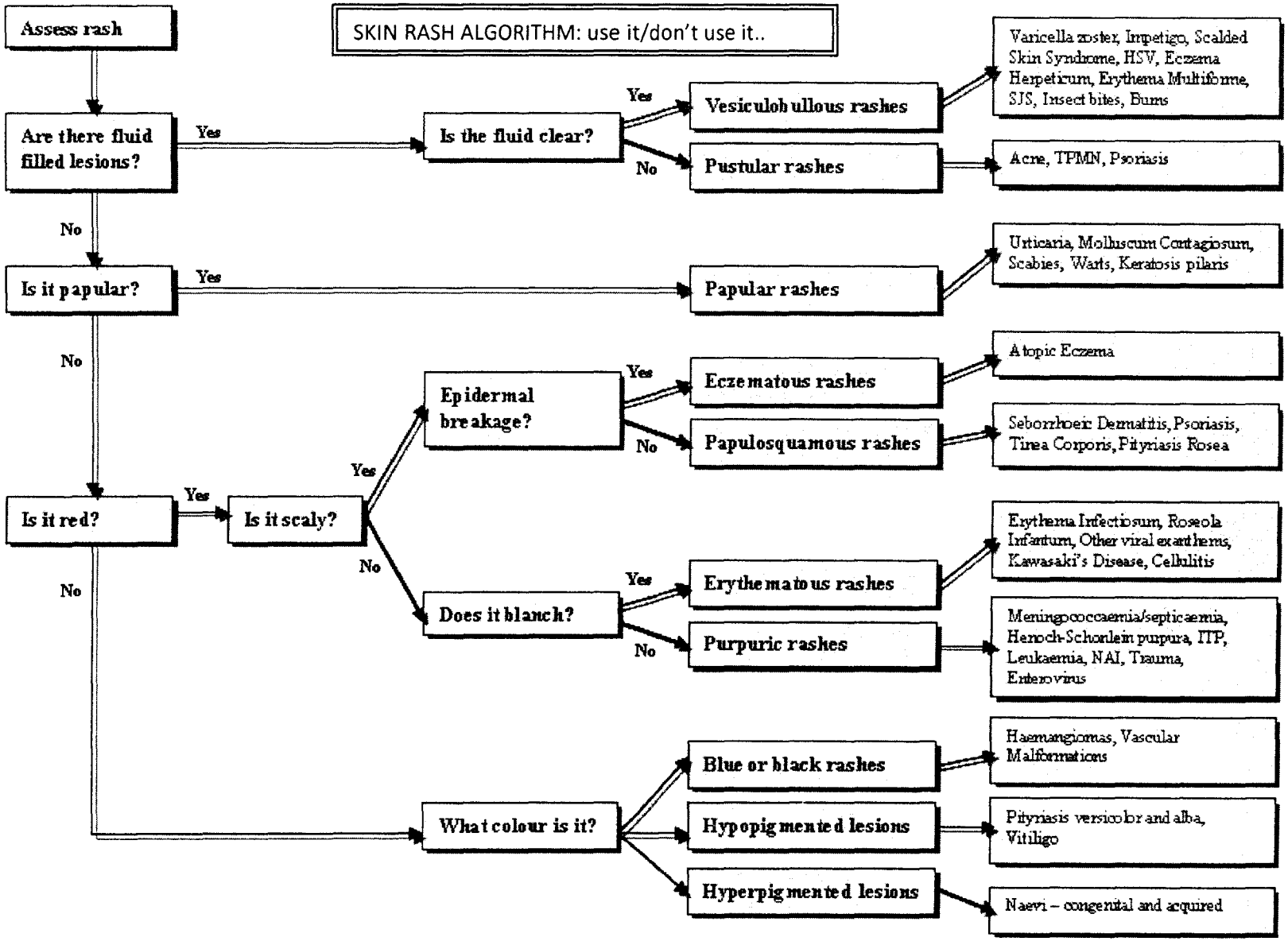

SKIN RASH ALGORITHM: use it/don't use it..
Assess rash
Are there fluid filled lesions?
Yes
Is the fluid clear?
Yes
Vesiculobullous rashes
Varicella zoster, Impetigo, Scalded Skin Syndrome, HSV, Eczema Herpeticum, Erythema Multiforme, SJS, Insect bites, Burns
No
Pustular rashes
Acne, TPMN, Psoriasis
No
Is it papular?
Yes
Papular rashes
Urticaria, Molluscum Contagiosum, Scabies, Warts, Keratosis pilaris
No
Is it red?
Yes
Is it scaly?
Yes
Epidermal breakage?
Yes
Eczematous rashes
Atopic Eczema
No
Papulosquamous rashes
Seborrhoeic Dermatitis, Psoriasis, Tinea Corporis, Pityriasis Rosea
No
Does it blanch?
Yes
Erythematous rashes
Erythema Infectiosum, Roseola Infantum, Other viral exanthems, Kawasaki's Disease, Cellulitis
No
Purpuric rashes
Meningococcaemia/septicaemia, Henoch-Schonlein purpura, ITP, Leukaemia, NAI, Trauma, Enterovirus
No
What colour is it?
Blue or black rashes
Haemangiomas, Vascular Malformations
Hypopigmented lesions
Pityriasis versicolor and alba, Vitiligo
Hyperpigmented lesions
Naevi – congenital and acquired

Table 13.4 Blistering and vesicular skin rashes

Disease	Description of rash	Other features and complications	Cause
Chickenpox	Crops of vesicles develop on red papules, spread from trunk, become turbid and umbilicate	Mild fever, secondary infection of ruptured vesicles. Ataxia, encephalitis, pneumonia	Varicella zoster virus
Herpes zoster (shingles)	Vesicles develop in the distribution of dermatomes	Uncommonly localized pain. In immunocompromised patients may disseminate	Varicella zoster virus
Herpes simplex gingivostomatitis	Thin-walled superficial blisters rupture early, inside of mouth and lips, extend to skin around mouth, may spread	Fever and irritability	*Herpes simplex* virus
Eczema herpeticum	Thin-walled superficial blisters clustering in areas of eczematous skin	Fever. Risk of dissemination and secondary infection	*Herpes simplex* virus
Hand, foot and mouth disease	Ulcers on tongue and buccal mucosa, vesicles on dorsal surfaces, palms and soles of hands and feet	Fever. Rarely aseptic meningitis, encephalitis	Coxsackie A 16 and Enterovirus 71
Impetigo	Vesicle on traumatized skin develops into honey-coloured, crusted plaque; oozes	No fever or constitutional symptoms. Regional adenopathy	Streptococci or staphylococci
Staphylococcal scalded skin syndrome	Localized bullous impetigo or generalized erythematous tender skin which closely resembles severe burn	Fever, irritability, skin tenderness. Secondary sepsis	*Staphylococcus aureus*
Papular urticaria	Various stages between erythematous wheals and oedematous red-brown papules	Pruritus. Secondary infection (impetigo)	Flea or insect bites Hypersensitivity reaction
Stevens Johnson syndrome	Macules, vesicles, bullae, desquamation, haemorrhagic crusting on face, trunk, extremities. Erythema multiforme, target lesions. Involvement of two or more mucosal surfaces	Corneal ulceration, scarring and strictures, pneumonia, myocarditis, hepatitis, renal failure	Mycoplasma pneumoniae Drugs (Sulphonamides, NSAIDS, anti-convulsants)
Toxic epidermal necrolysis	Skin erythema and inflammation leads to full thickness skin loss in flaccid bullae. No target lesions. Conjunctivae and mouth often involved	Worst end of spectrum of erythema multiforme. Fever and constitutional symptoms.	Infection and drugs Hypersensitivity phenomenon

Table 13.3 Erythematous maculopapular rashes

Disease	Description of rash	Prodrome	Other features	Complications	Cause
Measles	Generalized maculopapular starting behind ears and face, spreading to trunk and limbs, becomes confluent	Fever, cough, conjunctivitis, Koplik spots	Post-measles staining	Pneumonia, croup, eye complications, diarrhoea, suppression of immunity	Morbillivirus
Rubella	Fine generalized discrete maculopapular rash	Mild fever	Suboccipital adenopathy, arthralgia	Rarely encephalitis, thrombocytopenia	Rubivirus
Non-polio enterovirus	Measles-like, may be petechial	Abrupt onset	Common under 5 years. Associated herpangina often	Many including meningo-encephalitis, gastro-enteritis, myopericarditis, others	Coxsackie and Echo viruses
Infectious mononucleosis	Generalized maculopapular, usually precipitated by ampicillin treatment, may become purpuric	Malaise, headache, fever, sore throat, adenopathy, splenomegaly	Few clinical features under 4 years. Lifelong latent infection established.	Haemolytic anaemia, thrombocytopenia, hepatitis, oncogenesis	Epstein-Barr virus (HHV 5)
Erythema infectiosum	'Slapped cheek' flushed appearance, then lace-like macular rash on trunk and limbs	Unusual	Afebrile, generally well. Palms and soles are spared	Arthritis, arthralgia, transient aplastic crisis, chronic hypoplastic anaemia	Parvovirus B19
Roseola infantum	Rose-coloured discrete lesions spread from trunk to face and proximal extremities	Upper respiratory signs, then high fever, irritability, some with febrile convulsions	Rash appears as fever subsides	Rare encephalitis	Herpesvirus 6 and 7
Scarlet fever	Punctate erythema on face or generalized. Circumoral pallor	Fever and sore throat	'Strawberry' tongue	Nephritis, rheumatic fever	Group A beta-haemolytic streptococcus
Toxic shock syndrome	Diffuse macular erythroderma with subsequent desquamation on hands and feet	Unusual	High fever, hypotension, myalgia, 'strawberry' tongue, diarrhoea	Renal failure, ARDS, circulatory failure	Toxin producing *Staphylococcus aureus*
Kawasaki disease	Diffuse maculopapular, scarlatiniform or erythema multiforme	Fever	Bulbar conjunctival injection, mucosal erythema and strawberry tongue, cervical adenopathy, desquamation of fingers, palms and soles	Coronary aneurysms	Unknown
Drug reactions	Usually morbilliform	Antibiotic exposure often for febrile illness	Rash unrelated to fever, pruritus, improves on drug withdrawal	Unusual	Antibiotic exposure often for febrile illness

Table 13.5 Petechial or purpuric rashes

Disease	Description of rash	Associated features and complications	Cause
Meningococcal septicaemia	Maculopapular, petechial or purpuric with ecchymoses, occasionally vesicular	Fever, pharyngitis, weakness and headache. Rapid progression to shock, DIC, coma. May develop pneumonia, myocarditis, arthritis, meningitis	Neisseria meningitidis
Disseminated intravascular coagulation	Petechiae and ecchymoses, also areas of skin necrosis can develop	Severe predisposing systemic disease process, bleeding from puncture sites, haemolytic and blood loss anaemia	Excessive activation of clotting in sepsis, shock, acidosis, snakebite, rickettsial infections, incompatible blood transfusions
Rickettsial diseases	Discrete pale red blanching maculopapular rash on limbs, palms and soles spreads to whole body, may become purpuric	Fever, headache, myalgia, can develop DIC, meningoencephalitis, myocarditis, pneumonia	Rickettsiae
Viral haemorrhagic fevers	Maculopapular rashes on face and trunk become petechial, associated red enanthem on palate common	Prior fever, headache, myalgia, vomiting. DIC universal, leads to haemorrhagic tendency	Several viruses: Ebola, Marburg, Lassa, Dengue, Rift Valley, Congo
Acquired cytomegalovirus infection	Petechial rash occasionally	Subclinical in most, some with fever, pneumonitis, hepatitis, hepatosplenomegaly, adenopathy. Severe in immunocompromised	Cytomegalovirus
Henoch Schönlein purpura	Pink maculopapules blanching on pressure progress to palpable purpura on dependent areas (buttocks, legs, arms)	Mild fever, arthritis, abdominal pain, proteinuria	IgA-mediated vasculitis of small vessels
Idiopathic thrombocytopenic purpura	Petechia and purpura (non-palpable), also in conjunctivae and mouth	Preceding viral infection, otherwise well. Risk of intracerebral haemorrhage low	Platelet auto-antibodies triggered by virus infection

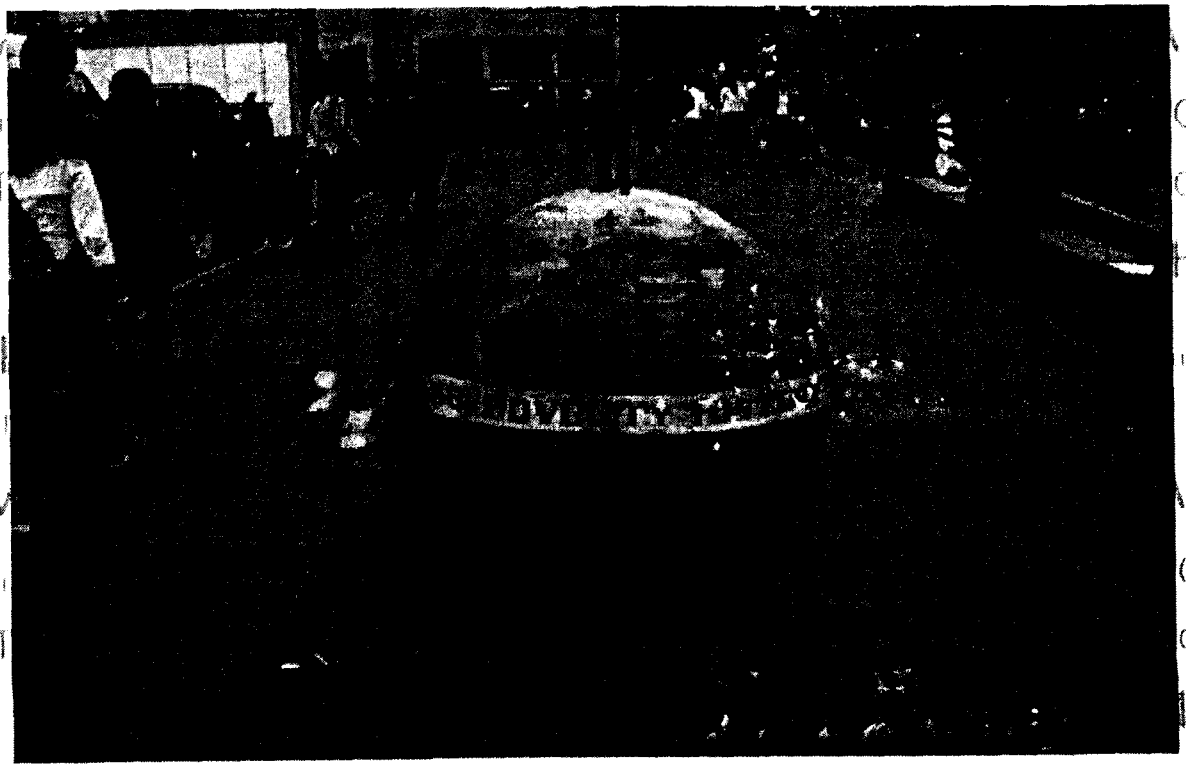
COMMUNITY PAEDIATRICS & CHILD HEALTH

LEANE

LEVELS OF CARE
CHILD HEALTH PRIORITIES IN SA
IMCI
CHILD HEALTH SURVEILLANCE

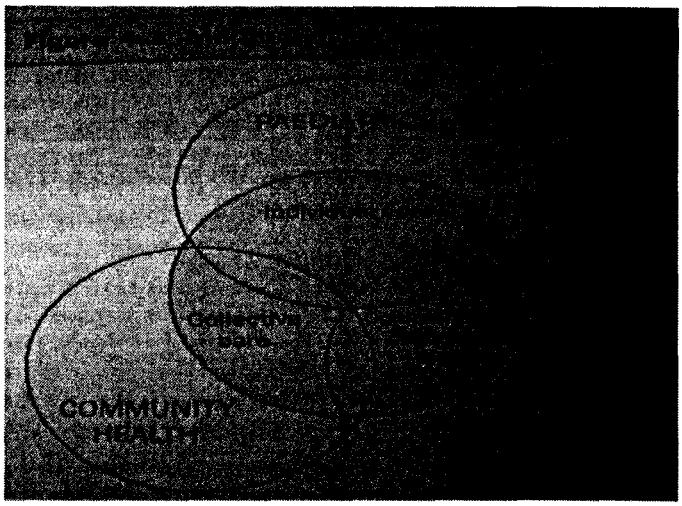
COMMUNITY PAEDIATRICS AND CHILD HEALTH

Levels of care
Child Health Priorities in SA
IMCI
Child Health Surveillance



COMMUNITY PAEDIATRICS AND CHILD HEALTH

Community paediatrics is practised within community setting and combines the individual perspectives of clinical paediatrics with the collective or population-based perspectives of community health. The role of community paediatricians may vary in different health care settings and there is some overlap with disciplines like community health and primary care.



Generalists handle common and important child health problems. The communities in SA differ enormously and some community paediatricians provide largely individual care in community settings for children with problems of child abuse ,handicap, neurodevelopmental delay, learning and behavioural difficulties and other chronic disorders of childhood. The discipline of community paediatrics lies within a framework for the delivery of community child health services and programmes.

LEVELS OF CARE

Primary level of care: health service facilities thru which a patient makes first contact with the health care system eg. clinics, office of GPs, opt patient departments in level 1,2,3 hospitals.

Secondary level of care: facilities staffed by either generalists or specialists to which patients are referred to.eg. level 1 but mostly level 2 and 3 hospitals.

Tertiary level of care: facilities staffed by specialists or super specialists for patients who have been referred by level 2 hospitals eg. Level 2 and 3 hospitals

HOSPITAL LEVELS

Level 1 hospitals: district, community, or non-specialist hospitals located in small towns or rural areas, staffed by medical generalists providing 24 hour in=patient care for common conditions.

Level two hospitals: regional or specialist hospitals located in larger towns, staffed by general specialists like paediatricians, obstetricians, radiologists etc..providing specialist care.

Level three hospital: super specialist or teaching hospitals located in metropolitan are.

LEVELS OF CHILD CARE

- Primary health care: first contact
- Comprehensive child care: combines promotive, preventative, curative and rehab activities within a single health care setting
- Ambulatory child care: In settings other than hospital beds
- Community child care: promotes health of groups of children>Community-based child health programmes refer to child health promotion activites eg. Home based care for HIV-infected children, programmes at school etc

FRAMEWORK FOR CHILD HEALTH SERVICES AND PROGRAMMES

- The National health system

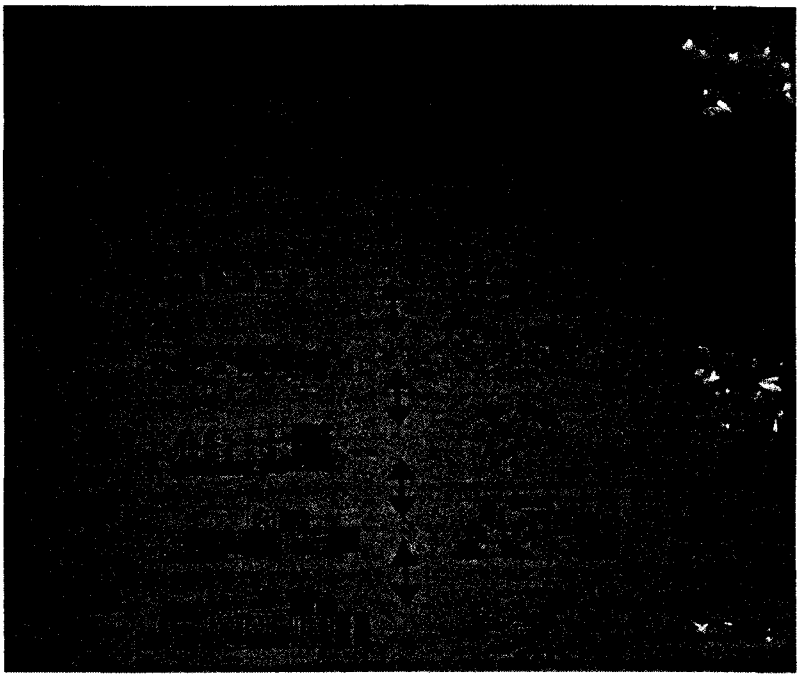
Child health services are located within 3-tiered, unitary, national health system .Consists of:

1. First-tier national health authority or ministry of health, responsible primarily for national policy guidelines, priority settings and resource location
2. Second-tier provincial health authorities , subdivided into regions, and mainly responsible for policy guidelines and resource allocation at a provincial level
3. Smaller geographic and administrative entities or districts at the 3rd tier. The distric takes responsibility for implementation of all child health services and programmes.

A) DISTRICT HEALTH SYSTEMS

Districts are seen as the appropriate level within the health system where top-down government policies and directives meet bottom-up development initiatives involving communities. They are regarded as the key to implementation of a health system based on the primary health care approach. There are maternal and child health services in these clinics and their aim is to provide as a minimum, the following services and emergency cover on a 24 hour basis:

- Antenatal, postnatal care for uncomplicated pregnancies
- Immunization, growth monitoring and support for breast feeding and oral rehydration during infancy and early childhood
- Simple curative services, essential drugs
- Management of peads emergencies
- Family planning counselling



In many districts a similar range of service is provided by mobile health teams at non-permanent visiting points throughout the community. Polyclinics are found more commonly in urban or per-urban districts.

B) REGIONAL LEVEL

This should ideally be 2nd level hospital within each region staffed by a team of specialist paediatricians and supported, at a minimum, by teams of obstetricians, community physicians, and anaesthetists. They must be able to supervise and assist districts in the running of child health services and in the development of child health programmes. Districts should be regularly visited by a regional paediatricians.

C) PROVINICAL LEVEL

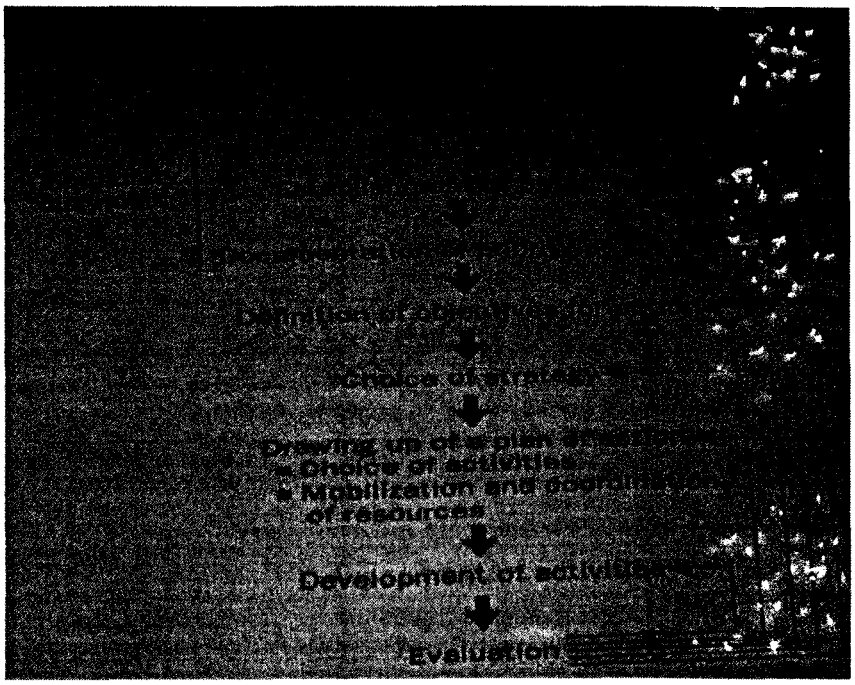
Provincial departments of health provide administrative and support for child health services at all health facilities within their borders(all facilities from 3rd level hospitals to residential clinics). They also promote and support the implementation of national child health programmes at the district level. Although 3rd level hospitals, linked to academic health science faculties, are based in particular provinces, they are regarded as national resource ws serving all the provinces.

D) NATIONAL LEVEL

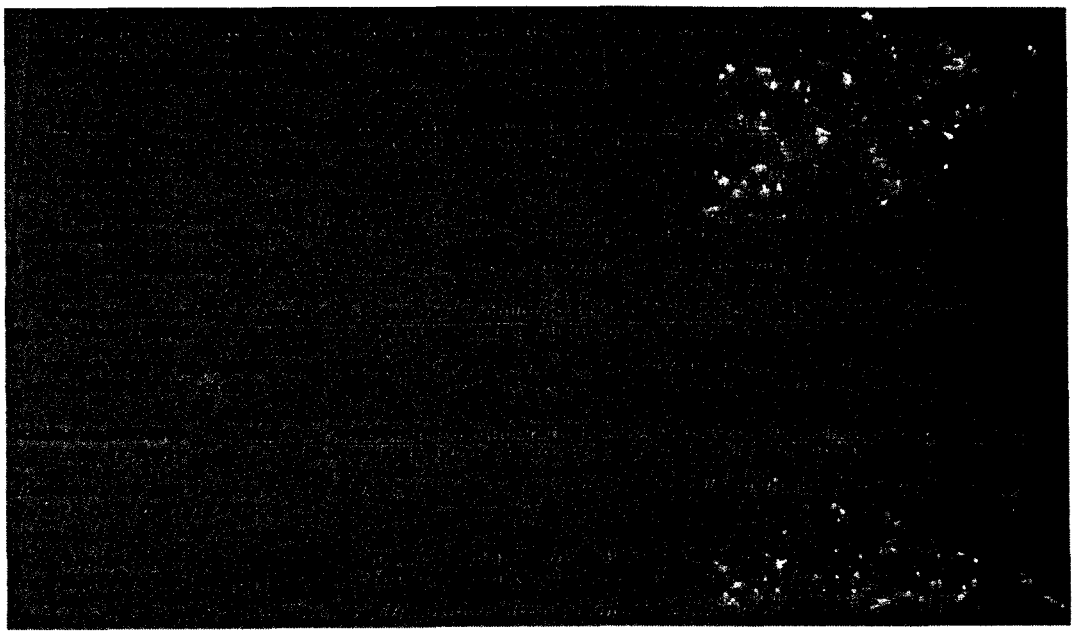
The national ministry of health provides policy guidelines and allocates resources for all health service activities. It identifies certain health issues as national priorities and specifically promotes national health programmes to address them. Many of these programmes are directly concerned with the health of children eg:

- National programme of action(NPA)
- Intergrated Management of Childhood illnesses(IMCI): a global WHO initiative to achieve integrated management of incidental and comprehensive health needs of children
- Primary school nutrition programme

COMMUNITY DIAGNOSIS – is the process of identifying the health problems and health needs of a community and the available resources to address them.



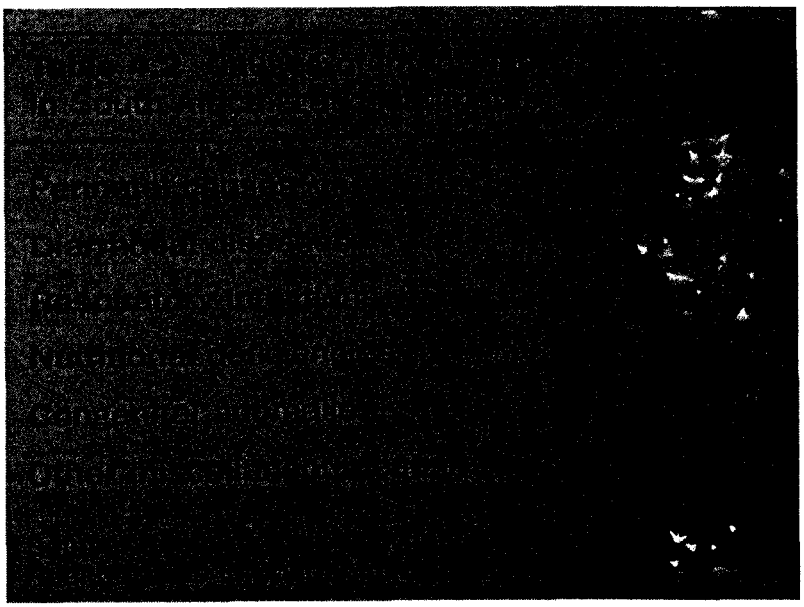
Specific indicators or rates to describe the health status of children and their coverage with child health services are especially useful in setting defined objectives and later on, evaluating the impact of the programme.



CHILD HEALTH PRIORITIES IN SA

- Children under 5 years

(U5MR)Under 5 mortality Rates, provide an overall indication of the health status opf young children in any district, region or country. Most available estimates of mortality rates in SA infants put the national figure at some where between 40 to 60 per 1000 live births.White infants have similar rates to those found in industrialised countries while black infants have much higher rates. In SA 34 % of all under-3 deaths occur in the first month of life, 76% occur in the first year of life, 24% occur in the residual 4 years(6 % per year). This emphasises the importance of targeting programmes at children in the first one to two years of life. Perinatal problems are the single largest cause of deaths in infants and young children and this mirrors the trends in other developing countries. Programmes focusing on pregnant women and their newborn children are therefore considered to be a major priority in countries like SA that are undergoing this health transition.



GLOBAL STRATEGIES FRO CHILDSURVIVAL AND DEVELOPMENT

GOBI-FFF: Most of the elements in the GOBI-FF strategy has always been core components of basic maternal and child health services and programmes.

- Growth monitoring :Road to health chart
- Oral rehydration therapy : 8 level teaspoons of sugar plus a ½ teaspoon of salt added to 1 litre of boiled water.
- Breastfeeding :promotion of exclusibe BF for 6 months and continued BF for at least 1 year is very important in child survival and development. HIV-AFASS criteria.
- Immunization : simplest most powerful and coast effective of all child health interventions
- Family spacing :at least 2 years is reccommended
- Food supplementation
- Female literacy

IMCI: the latest WHO stategy aims to integrate previous programmes and treatment strategies and includes a strong emphasis on health worker training and ongoing support. This strategy, like the preceding GOBI-FFF approach, specifically aims to lower morbidity and mortality from the 5 most common conditions in infancy after the first few months of life:

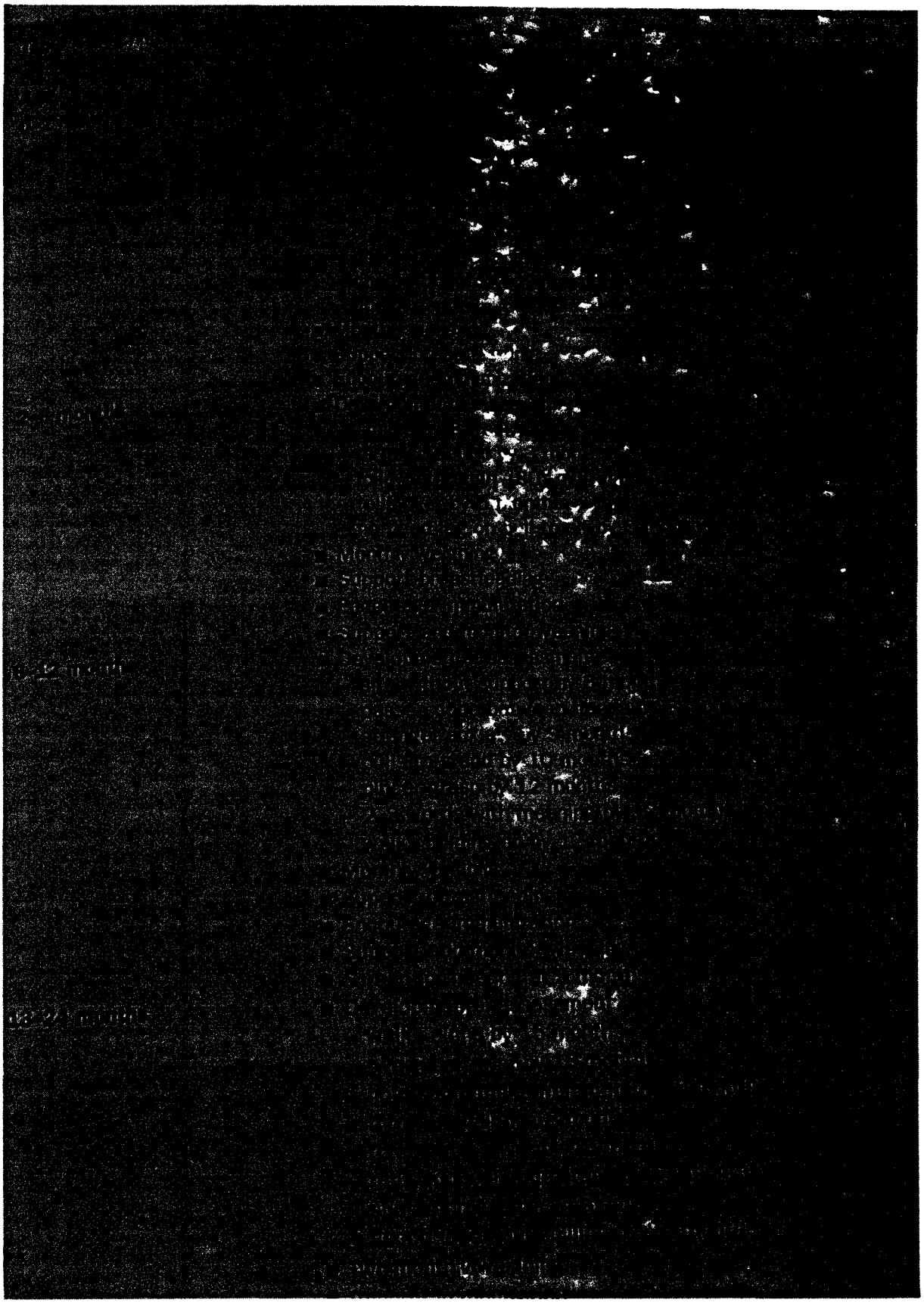
1)Diarrhoeal disease
2)Acute resp infection
3)vaccine preventable diseases esp.measles
4)malaria
5) malnutrition

CHILD HEALTH SURVEILLANCE

This is a strategy within primary child care settings to screen individual children for deviations from normal. Screening plays an important role to distinguish those children who probably have a condition from those who probably don't. Screening may take the form of mass screening programmes or group screening programmes(smaller). Opportunistic screening on an age –appropriate basis is the most common context for child health surveillance.

Schedules for surveillance:

- Birth to 8 weeks
- 8 weeks to 6 months
- One to two years
- Two to five years



Links between education and child health

HEALTH EDUCATION: This happens either one-to-one contact between health professionals and parent or patient in a health care setting. School health services vary in their sophistication in different settings but the objective is early identification and management of health problems at the school eg. Medical problems of school going age; learning problems; difficulties with hearing and vision. Recently the concept of a health-promoting school has emerged with a combination of healthy policies eg. Healthy physical and social environment; health education in the classroom.

Pre-schools provide one of the few organized channels to reach this group of children between 3 to 6 years but the attendance at pre-schools is low in rural areas.This provides the opportunity for screening as the child with a developmental or specific learning disability can be identified at an early stage when corrective action will be most beneficial. However, the crowding together of these children at this age carry the well-known risk of transmitting infection, care must be taken to maintain hygienic conditions to minimize this problem.

SOCIAL PAEDIATRICS

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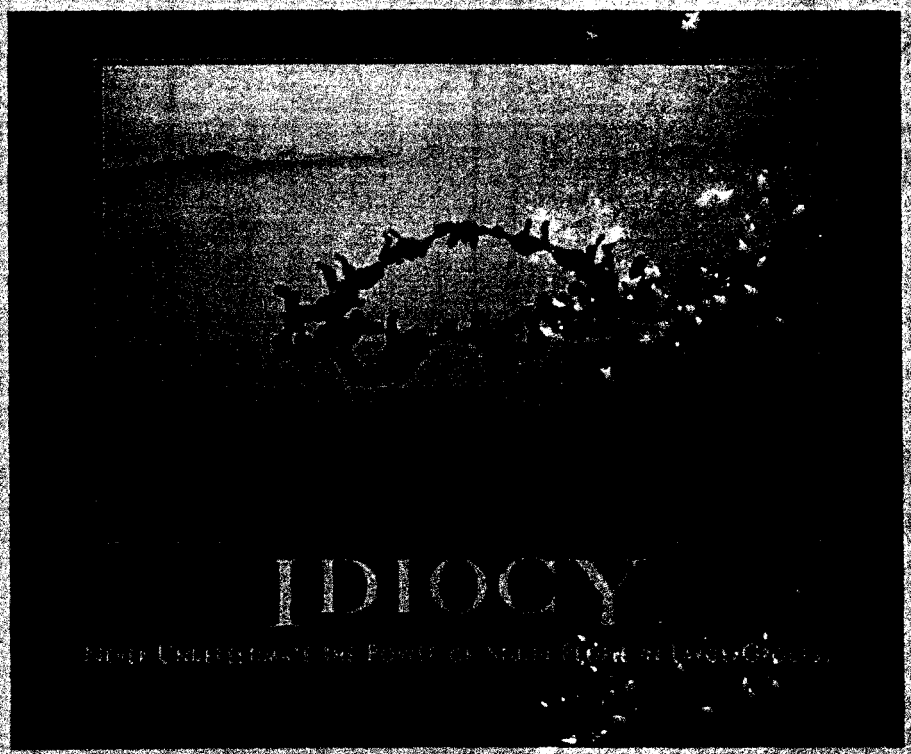
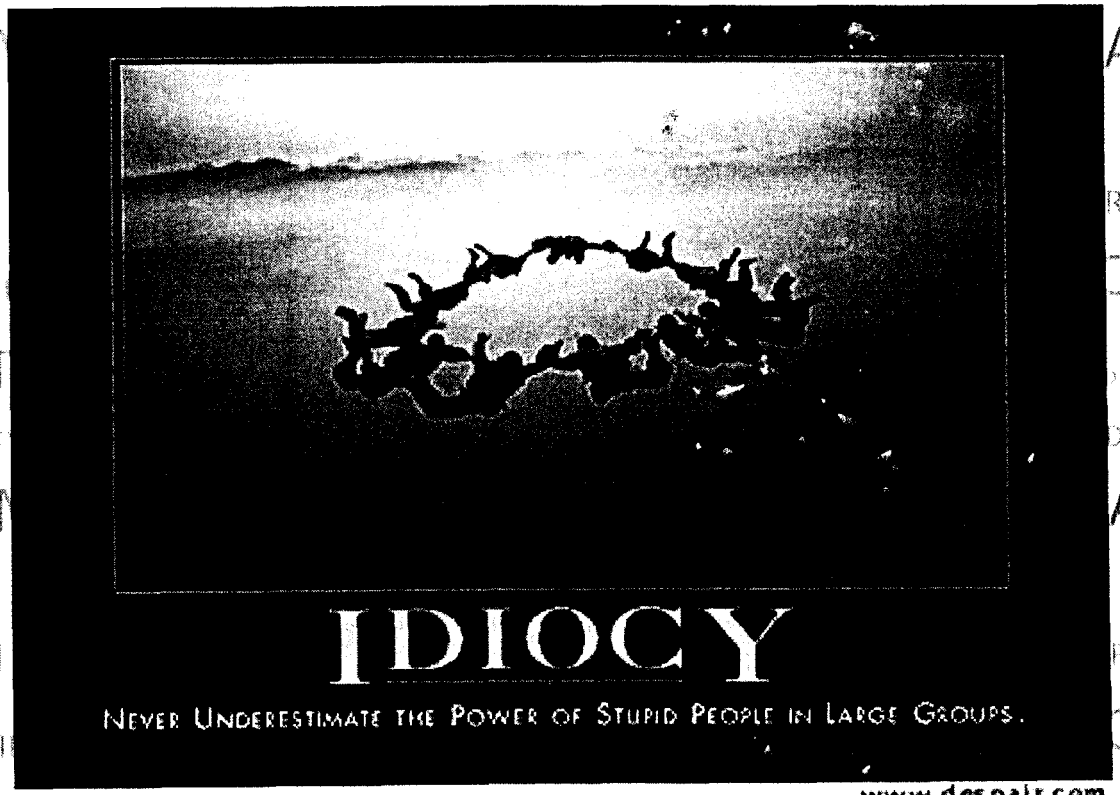
THE SOCIAL ENVIRONMENT
FAMILY FUNCTIONING
LEGISLATIVE FRAMEWORK AND SOCIAL SECURITY PROVISIONS
AFFORDABLE MODELS OF COMMUNITY CARE
CHILD ABUSE



SOCIAL PAEDIATRICS

- The social environment
- Family functioning
- Legislative Framework and Social Security Provisions
- Affordable models of community care
- Child abuse



SOCIAL PAEDIATRICS

- Growth and development = individual make up + physical environment + social environment
- Physical environment → primary impact on physical health
- Social environment → impact on psychological, emotional and educational well-being
- Social paeds = environment which kid is raised, consequences thereof and strategies to maintain good family function and protect kids in poor social circumstances.

The Social Environment

- **4 interacting systems:**
  - Mother's womb/care – impinging directly on the senses of the child
  - Family and home and patterns of interaction between family members
  - Other settings – spend extended periods of time there and significantly influence development e.g. day care, preschool etc
  - Local community/wider world – political/cultural/social influences impacting directly/indirectly on children through their effect on the family/members
- Social environment defined by make-up of each component and relationships between them.

- **The child:** own individual characteristics influencing growth and development

- **The Family**: definition:
  1. kinship ties (classical, cultural view)
  2. any group of people living together/in close proximity who provide mutual care, support and guidance (functional view)
  - Most NB social setting capable of shaping and influencing health and development of the child.
  - **Physical wellbeing:** determined by physical environment of the home, health risk behaviour and health-seeking practices of the parents.
  - **Emotional health:** determined by parental sensitivity to children's needs, expectations of children, degree and quality of the affective support children receive from their family.
  - Ability to develop and maintain **social relationships** and to take on certain **roles** within a unit are learned by children in their own families/households.
  - Whatever the composition of the family, **responsibilities** are the same:
    1. **Material support and supervision** (food, clothing, shelter, safety, supervision, hygiene, health care, education)
    2. **Affective functions** (love, companionship, social support, socialization, teaching of coping/life skills)

    The capability of parents to fulfill these responsibilities depends on their:
    - Standard of education
    - Childhood and adult experiences
    - Innate and accessible resources
    - Level of support from community
- **The community**: macro-environment with indirect impact on child exerted through the influence on the family unit. The relationship between the family and the community dependent on:
  - Tangible (child care, recreational and healthcare facilities etc)
  - Intangible (attitudes, beliefs, practices of the community)

FAMILY FUNCTIONING

- A functioning family has the **resources** and the **coping mechanisms** to deal with the **demands** and **stresses** with which it is regularly faced.
- Balance of demands vs. capabilities = ↓ sequelae of stress/tension
- **Demands:** (table 5.1 – examples)
  - **Stressors:** Acute – over defined time period
  - **Strains:** Chronic – vague, poorly defined
  - **Hassles:** Seemingly innocuous events of daily living
- **Capabilities**: **resources** + **coping mechanisms**
  - **Resources:** characteristics/competencies of social environment – tangible/intangible (table 5.2 – examples)
  - **Coping mechanisms**: behaviour response of a family to stress. E.g. redefining attitudes to a problem

- Sequelae of an imbalance of capabilities vs. demands are: **stress, family malfunctioning, and family breakdown** = increased:
  - Physical illness
  - Psychological symptoms
  - Disruptive and destructive behaviour
  - Depression and anxiety
  - Social and academic difficulties
- Majority of SA = poor = ↑ demands and ↓ capabilities = downward spiral
  - ↑ perinatal morbidity
  - ↑ prevalence of handicap
  - ↑ childhood deaths, child abuse
  - ↑ maternal mortality
  - ↑ children living in surrogate care
- **POVERTY** is most powerful negative influence on child development.

CHILDREN'S NEEDS AND SOCIAL SECURITY NETWORKS

Children's needs:

Physical	**Emotional**	**Social and cognitive**
Food	Security	Socialization and peer interaction
Clothing	Love and affection	
Shelter	Companionship	Coping skills
Supervision		Life skills
Safety		
Health care		

Reality: one parent households = many responsibilities = negative impact on parent = negative impact on family functioning = don't meet all needs → social networks need to be mobilized/established

- **Day care at home**: extended/multiple generation families or multiple family households
  - Pooling of financial/human resources = ↑ capability to meet child's needs
  - Reliance placed on elderly = problem → seldom able to be effective caregivers due to ↓ physical capabilities
  - Families with adequate financial resources – nanny/babysitter
    - Advantages:
      - Familiarity of environment and caregiver to child
    - Disadvantages:
      - Difficulties in finding a suitable caregiver
      - Lack of backup should caregiver be absent
      - Lack of adequate supervision of caregiver
      - Lack of suitable peer group interaction for child
- **Day care outside the home**
  - Play groups, family day care, centre based child care (day care centre, crèche, pre-school)
  - **Formal:** >6 children, registered with Dept of Education/Welfare/Local authority and meet requirements (facilities, child-to-staff ratios, staff training). Full day = crèche (Dept Welfare), half day = pre-school (Dept Educ).
    - **Advantages**: safe care, supervision, peer group interaction, socialization, stimulation, skills training.
    - **Disadvantages:** inability to address individual needs of each child, ↑ exposure to illness, lack of flexibility in the hours. '
    - However, good-quality child care (↓ child to staff ratios, training of caregivers) associated with improved social development. Acceptable alternative.
  - **Informal:**
    - ↑↑ in urban and peri-urban informal settlements - ↑ need for childcare and as channel for subsidized feeding in needy communities.
    - Seen as education opportunity for access to good schools by parents from disadvantaged communities
    - Convenient way for health authorities to reach children 2-5 years for health promotion activities
- **Surrogate and alternative care**
  - ± 20% of children don't live with mothers – 1/3 orphaned, 1/10 abandoned, rest mother is unable to give care (remarriage of mother, return to school, need to live in at work, lack of resources)

- Some in very poor circumstances – absorbed by informal support networks
- **Child Care Act** – children in need of care defined as children without parents/guardians or whose parents/guardians are unfit/unable to care for them
  - Regulates placement of children into surrogate care: adoption, foster care, residential care, place of safety.

Adoption:

- Permanent legal procedure = child of parent/couple other than biological parents
- **Suitable for adoption** = Orphaned, abandoned, parents unable to care, parents wish to give up legal claim
- **Adopting parent:** any adult screened by social worker and found to be physically fit, reputable and capable of maintaining and educating the child.
- **Consent** has to be obtained from parent, child (over 10 years) and adopters. Exceptions:
  - Parents abandoned their child
  - Natural parents died
  - Mentally incompetent to give consent
  - Parents have mistreated their child
- Adoptions may only be **rescinded** within **2 years** if:
  - Natural parent did not consent to adoption
  - Adopting parent fraudulently induced to adopt child
  - Or child has mental/physical problem present at adoption which was not disclosed to adopting parents.

Foster Care and places of safety

- Both are temporary placements to protect children in need
- Following investigation of a child's circumstances by police/social worker a Children's Court Inquiry (CCI) is held to arrange appropriate care of child.
- Pending completion of investigation/CCI a child may need to be kept in place of safety
- Outcomes:
  - Return child to parents under supervision by social worker
  - Foster care under supervision of social worker
  - Children's home or school of industries.
- Parents whose kids have been removed from their care **lose custody** but **retain guardianship** rights = their consent is still required for medical/surgical procedures.
- **Foster care:** placement into care of temporary parent/s under supervision of social worker for up to 2 years at a time.
  - May be extended CCI until home circumstances allow or child is 18.
- **Problems:** physical, mental, social wellbeing is poor and demonstrate numerous physical, intellectual, behavioural problems.
- Children are often moved from one foster home/place of safety → vulnerable to neglect, abuse and exploitation.

Children's Homes:

- Surrogate care in a residential facility
- More permanent than foster care/place of safety they are also temporary
- In terms of Child Care Act – all must fulfill certain requirements with respect to management, staff and structure and must be registered with Dept of Welfare

LEGISLATIVE FRAMEWORK AND SOCIAL SECURITY PROVISIONS

- **United Nations Convention on the Rights of the Child** – adopted Sep 1990, ratified June 1995
  - Preamble and 54 articles
  - Preamble = children need special care, including legal and other protections before birth and throughout childhood. Special emphasis on role of family caring for children and cultural values of a child's community
  - Defines any person under 18 as a child
  - Sets out wide range of rights
- **African Charter on the Rights and Welfare of the Child**
  - Developed by Organisation of African Unity
  - Better reflect African cultural concerns and address relevant issues not addressed ↑
  - Signed in Oct 1997, not yet in operation
  - Makes provision for:

        - Protection against harmful social and cultural practices
        - Children of imprisoned mothers
        - Responsibilities of child to his/her family and community
        - Education
        - Armed conflict
    - Therefore addresses issues such as female circumcision, child soldiers, literacy, and role of the family in adoption.
- **SA Constitution**
    - **Section 28:** various rights – name and nationality from birth; to family and parental care or appropriate alternative care when removed from family environment; basic nutrition, shelter, health care services, social services; to be protected from maltreatment, neglect, abuse, degradation, exploitative labour practices, in times of armed conflict; not to be detained except as a measure of last resort; not to be used directly in armed conflict.
- **Child Care Act**
    - Being reviewed
    - Will include provisions on parental roles and responsibilities, children in need of special protection, age of majority, surrogacy, artificial insemination, prevention and early intervention, early childhood development, partial care, health rights of children, rights of children as consumers
    - Principles of the new statute include the following objectives:
        - To make provision for structures, services and means for promoting the sound physical, mental, emotional and social development of children
        - To utilize, strengthen and develop community structures which provide care and protection for children
        - To prevent, as far as possible, any ill-treatment, abuse, neglect, deprivation and exploitation of children.
        - To provide care and protection for children who are suffering ill-treatment, abuse, neglect, deprivation or exploitation or who are otherwise in need of care and protection
        - Generally to promote the wellbeing of children
- **Statutes which impact on the wellbeing of children:**
    - Age of Majority Act – capacities of children at different ages
    - Services and Rights (Health Care Act, Schools Act)
    - Family and caregivers (Marriage Act, Divorce Act, Prevention of Family Violence Act)

In addition to the above, SA has a **social security network** to promote family unit/support children whose well being is threatened. Makes provision for support in 2 layers:

1. **General response: poverty alleviation**
    - Development of communities, establishing appropriate infrastructure and services, job creation activities
    - Direct monetary transfers:
        - **Old age pensions**
        - **Social relief**
            - paid by Dept of Welfare/local Magistrate's court
            - to those with NO MONEY and wouldn't survive without immediate help
            - three months only – issued in food or money
        - **Child support grant (CSG)**
            - Primary caretaker of any child under the age of 7
            - provided they live in a household with total monthly income less than R800 (urban) or R1100 (rural)
2. **Children with special needs**
    - **Foster care grant** for children in surrogate care
        - Foster parent/s of a child by court order because parents unable to care, abandoned or orphaned
    - **Care dependency grant** for children with disabilities
        - Parents of severely handicapped child between 1-18 years who pass a means test

AFFORDABLE MODELS OF COMMUNITY CARE

- Social structure of communities, status of family and well being of children seriously undermined by:

- Rapid urbanization
- High levels of unemployment
- Poverty and violence
- Escalating HIV/AIDS epidemic

- Expected to produce ↑ population of children whose families won't be able to care for them.
  - Essential that new models are developed which spread the burden/responsibility more widely between public, private and NGO sectors.
  - Within public greater collaboration needed, vertically and between 3 levels of Govt. and at each level between state departments (health, welfare, education, public works)
  - Dept of Welfare will have to move to development of communities to ↑ capacity of care for their own members and will be responsible for policy framework in which models of care must be developed and for financial/material aid to impoverished communities
  - District level welfare officers will have to work closely with and fund NGOs and CBOs involved in training, social support and delivery of care to the needy. Will need to enlist the support of private and international donors to finance these district based activities
    - Resources must be used to ID and strengthen existing informal models of care and form new, innovative alternative models of care in consultation with the recipient communities
  - District level: emphasis on development of community based structures (child care facilities/alternative residential care facilities)
    - Central to such models is concept of community child care committees (CCCC) responsible for monitoring and supervision of the children in need.
    - Activities of CCCC include:
      - Creation of home-visiting networks for early detection of high risk families/children
      - Regular supervision of child headed households by volunteers
      - Running of community homes
      - Supervision of group rather than family foster care
      - Implementation of various community rehabilitation services for care and support of children with special needs
    - Incentives need to be offered to families that care for children in need e.g.:
      - Eligibility for ↓ municipal service charges
      - Provision of free education to both the children in need and the other children living in that household
  - By shifting responsibility for the care of children to the community level while still supporting the community there will be an ↑ likelihood of establishing sustainable, alternative models of care which are affordable and acceptable to communities.

Children with special needs

- Refers predominantly to children with **physical or mental disabilities, learning disorders, emotional and behavioural problems who require greater physical care and supervision**. In turn, families require greater emotional and social support. ↑ demands on family.
- To cope families have to develop additional resources and coping skills
  - Supportive extended family and community network often required
    - E.g. family friends, church groups, support groups
  - Limited state support available (care dependency grant, remedial classes in normal schools, special schools for various categories of handicapped children, training centres, special residential care facilities)

The child with disabilities:

- WHO definitions:
  - **Impairment:** describes pathological process e.g. spina bifida
  - **Disability:** consequence of the impairment e.g. paraplegia
  - **Handicap:** social consequence of impairment or disability. (how the individual responds to his/her impairment/disability)
- **International Classification of Functioning, Disability and Health:**
  - ↑ emphasis on the **environment** → role on limiting the child's activities and restricting his/her full participation in society.
  - Views disability as a socially constructed problem requiring active steps to combat stigma and to fully integrate children into society instead of disease needing treatment by a professional. Ideal = combination.

- **Stats:**
  - Majority (±85%) disabled children in developing countries
  - 8% of children under 10 years will have a disability in an average community
  - Largest group: mentally handicapped
  - Next: physically handicapped
  - Less common: sensory handicaps (blind, deaf)
  - Categories may overlap in children. E.g. blind and paralysed
- Majority present with functional disturbance that may vary widely in cause, severity, clinical picture
  - Assessments deals with:
    - Diagnosis and establishment of cause
    - Determining the child's functional disabilities
    - NB for parents who struggle to come to terms with child's condition
  - **Needs to be addressed** when providing care for handicapped children and their families:
    - **Early ID of handicap:** ↑ likelihood of effective treatment and amelioration of handicap; improve understanding of causation; boost confidence of parents in health professional therefore improving effectiveness of counseling.
    - **Diagnosis:** elusive but should be attempted as influence risk of recurrence and content of counseling
    - **Assessment:** level of functioning is essential for management of child; age related and needs to be repeated periodically. Review in accepted categories: gross and fine motor functioning, vision, hearing, speech and language, perceptual and intellectual functioning, emotional and social development
    - **Immediate advice and counseling:** parent's first need = counseling; 2nd = practical advice how to deal with child. Will need to understand child has different needs at different ages but that they will occur later than non-handicapped. Early years = need to attain skills of daily living (feeding, continence, mobility, language). Need to follow programme to reach max potential and revised regularly.
    - **Management programme:** prevent/ameliorate secondary problems. Problems can be anticipated and avoided e.g. hip dislocation in CP. Needs will change with age: high school – prep for work
    - **Advice about child rearing:** may see all problems as due to disability and not just normal growing up. Encourage to see child as going through developmental phases like normal kids. May have access to normal forms of support – may go to normal school depending on degree of disability.
    - **Periodic reassessment:** clinical picture and functional development often unpredictable. Timing may be adjusted to needs of child but minimum of:
      - 4-5 years before entering school
      - 9-10 years in anticipation of puberty
      - 13-14 years to assess post school needs
      - Reassessment may involve whole family – remarriage, birth of child, employment change/problems, marital problems, health of other members may affect status and care of handicapped child

Community based disability programmes

- UK: multi-professional teams set up to support families and children with handicaps
  - Links the professional team to child-care staff, teachers, parents
  - Coordinates range of activities for children with handicaps that include assessment, training, surveillance and research.
- Developing world – simpler strategies (↓ resources and involving community members)
  - Community based rehabilitation (CBR) requires reorientation of services to a community based approach.
  - Professionals share skills with parents, community workers and disabled people
  - Address problems that have arisen western models (institutional care) → reaching only 5% of disabled children and causing serious problems with reintegration of disabled children into their communities.

Children and the HIV/AIDS epidemic

- Socio-economic consequences have greatest impact at household level = ↑ vulnerability children in affected communities

- 3 categories can be defined within the relationship between children and the epidemic
  - HIV-infected children
  - Children living in an HIV infected household – orphans and vulnerable children
  - Children of uninfected households living in an affected society
- Magnitude of children affected hard to quantify. Observations are:
  - Peak seroprevalence amongst pregnant women should stabilize between 25 - 30% 5-10 years into the epidemic
  - No of Orphaned children will peak 10 -15 years after seroprevalence peaks and then level out at slightly lower plateau
  - The more rapid the progression to AIDS the earlier the peak and the lower the plateau
  - 20 years into the epidemic over 25% of all children will have lost their mothers
  - Mean age of a child orphaned by AIDS is at least 2 years younger than any other cause
- During early stages of HIV parents can still meet all demands → disease progresses → productivity ↓ as need ↑ for physical, psychological, health support = restructuring of household functioning, impacting the children. As disease worsens, children take on ↑↑↑ adult responsibilities. Older children frequently leave school to ↓ household expenditure and work or stay home to care for sick parents/younger siblings. Older rural children may migrate to urban centres in search of work → lose all family contact as they merge with other children living on streets. **Net effect = physical and psychological trauma aggravated by social stigma attached to HIV infected people and their households.**
- Unless arrangements are made to cater to children's needs before parents' death, the trauma, grief and guilt compounded by uncertainty of their future, relocation in extended family and at expense of breaking up support offered by sibling group
- Alternative models of care are needed to provide for these additional vulnerable children
  - Innovative shifts in welfare policy
  - Increase in social security grants
  - Greater recognition of roles of NGOs and extended family
- All children affected by epidemic not only those with HIV infected family member:
  - Day to day contact with peers experiencing personal tragedies
  - Sharing homes with orphaned children
  - Participating in community programmes to address needs of infected and affected community members
  - Indirect contact with socio-economic sequelae of the epidemic
    - Deteriorating levels of service provided within education, health and welfare sectors
- All children will be affected and minimizing impact on children lies with each individual in our society

CHILD ABUSE

The infliction, or permitting of infliction, of physical or mental harm on a child by a person who is responsible for that child. Includes:

- Physical abuse or non–accidental injury (NAI)
- Emotional trauma
- Sexual abuse
- Neglect
- Administration of drugs or alcohol to children

Prevalence: rapidly growing problem, increasingly common.

Risk factors: each alone is of low predictive value

- Families/parents at risk
  - ↓ SES status
  - Families under stress
  - Young parents/teenage mothers
  - Single self-supporting parent
  - Psychiatric illness (chronic depression) in mother
  - Parental drug dependence
  - Parents who were abused or in institutional care as children
  - Maternal illness
  - Interference with mother-child bond at birth
- Children at risk
  - Step or foster child
  - Premature baby
  - One of twins
  - Child with mental or physical defect

Physical abuse or Non-accidental injury (NAI)

- **Any injury inflicted on a child by a responsible caregiver, irrespective of intent or justification, that produces anything more than erythema or redness and involves any area besides the buttock or hand**
    - Any physical punishment administered to child <1year or child with physical/mental disability
    - Extreme definition deliberately sets out to state acceptable limits of physical punishment at home and corporal punishment at school
    - Essential to regard overzealous punishment by parents/teachers as abuse and to report these incidents to induce them to learn more acceptable, alternative modes of discipline

Clinical picture:

History:

- complete and careful description of injury including when, where and how it occurred, who was present and the time elapsed between injury and presentation.
- Age and development must be recorded, current health status, past history of injuries
- Social and family history describing composition of household, SES conditions, family support structures must be taken
- Following points should suggest NAI:
    - No/inadequate explanation of injuries
    - Delay in seeking medical help
    - Changing explanation for the injury
    - Different explanations from different people
    - Recurrent injures in child or sibling

Examination: always try answer 2 questions:

- Is injury compatible with alleged cause/circumstances as provided by caregiver?
- Is injury compatible with the child's stage of development?

- Must include precise description of all injuries:
    - Length, shape, colour, position on the body, degree of demarcation and whether injuries are bruises, scratches, abrasions or burns
- Comment on child's general appearance, cleanliness, state of clothes
- Note child's mood and affect e.g. apathy, frozen watchfulness, irritability
- Observe interaction between parent and child (parental concern, warm parent-child relationship)
- Note if marked disparity between state of hygiene, dress or nutrition of caregiver and child
- Measure and plot weight and length of child against previous measurements if available
- Examine whole child and look specifically for:
    - Hair loss
    - Bruised/swollen ears and torn tympanic membranes
    - Retinal haemorrhages
    - Damage to the gums or torn frenulum
    - Bruising of neck
    - Evidence of injury to anus or genitalia

Signs suggestive of NAI: (raise index of suspicion if present)

- Bruising or abrasions with any of the following characteristics:
    - Multiple bruises at different sites
    - Bruises at different ages (table 5.4)
    - Well demarcated linear bruises indicating imprint of well known objects
    - Parallel "tram track" lesions from whipping with a stick or cord
    - Black eyes, especially when bilateral (blood from scalp injury may track down to soft tissues around the eyes)
    - Teeth marks producing crescenteric bruising
    - Bruises on legs of child who is not yet walking
    - Bruises on face and neck
- Burns with any of following characteristics:
    - Glove and stocking scalds to hands and feet (suggests forced immersion into hot water)
    - Well demarcated, circular "cigarette burns" usually on the back of hands, wrists, face
    - Any well demarcated burn without an adequate explanation
- Multiple scars, abrasions, or scratches in different stages of development
- Circumferential injuries of ankles, wrists and neck
- Subconjunctival, anterior chamber, and retinal haemorrhages
- Unexplained impaired level of consciousness

- Signs of ruptured abdominal viscus
- Multiple or unusual fractures – children under one year of age cannot generate the momentum needed for a fracture
- Very few absolute diagnostic criteria – suspicion is sufficient basis for reporting the case to the relevant authorities

Investigation:

- X-rays often provide supportive evidence for diagnosis
- Long bones most commonly affected and diaphyseal # 4x more common than metaphyseal-epiphyseal # though the latter are much more specific for NAI
- Spiral # indicate torsional/rotational injuries such as occur when swung by their arms
- Full skeletal X-rays should be done on children <2years suspected of NAI
- In children with multiple bruises, bleeding disorders must first be ruled out, however, NAI can occur in children with bleeding disorders

Sexual abuse

- Involvement of a child in sexual activity to which he does not consent; that he does not understand on the basis of his developmental age; and which violates the norms of society
- **Four categories:**
    - **Mild sexual abuse:** all activities which don't involve physical contact between a naked child and perpetrator
    - **Moderate sexual abuse:** activities which involve physical contact of naked participants but without penetration of the body
    - **Severe sexual abuse:** involves penetration of any body orifice, oral, anal or vaginal, by finger, penis, or any other object
    - **Suspected but unconfirmed** sexual abuse of an undetermined nature
    - Relationship of perpetrator and abused child fall into one of 4 categories:
        - Unknown – where child too young/frightened to disclose identity of perpetrator
        - A family member – brother, father, grandfather, uncle or cousin
        - Family acquaintance – friend, lodger, neighbour, teacher
        - Stranger
- Mother may be aware or have suspicion she may be immobilized by fear of dissolution of family
    - May have been abused as child and fail to find inner strength to bring affair to light
    - Denial on part of mother not unusual phenomenon
    - Often involves family member characterized by secrecy, guilt, loss of trust, lack of self esteem on part of victim
    - This explains why it usually takes a long time to surface and why it usually presents indirectly

Clinical picture:

- Common presentations: STDs, UTIs, genital trauma causing difficulty walking, vague psychosomatic complaints.
    - Gonorrhoea and syphilitic sores in prepubertal girls are concrete evidence
    - Condylomata accuminata should be regarded with grave suspicion
    - Late onset enuresis, UTIs and dysuria in the absence of infection should raise the possibility
    - Vague lower abdominal pain, unexplained headache have a similar origin but lower predictive value
- **History**
    - Guidelines should be followed when interviewing children who are victims of suspected abuse:
        - Note child's words verbatim
        - Avoid repetitive histories – distorts final version, discredits subsequent testimony in court
        - Wherever possible child should be interviewed by experienced and skilled interviewer
- **Examination**
    - Thorough to look for associated evidence of abuse and to lessen focus on the genital area
    - Exam of genitalia can usually be carried out with little difficulty if done sensitively with tact and patience

- Girls <3 years best examined on mother's lap with heels drawn up to buttocks. Good exposure is obtained on complete abduction of the knees
  - Bruising and other injuries of the vulva, perineum, thighs should be noted and following gentle retraction of the labia majora, the introitus and hymen should be thoroughly inspected
  - Sexual penetration causes a midline tear of the hymen
  - Non-sexual and less forceful penetration increases the size of the hymenal orifice (>0,7cm)
  - Inspection of the perineum and anus for evidence of sodomy is imperative:
    - Bruising
    - Superficial tears
    - Dilated veins
    - Patulous anus
  - Speculum exam, where deemed necessary, should only be performed under anaesthesia
- Older girls can be examined in a similar way, lying supine and appropriately draped.
- Laxity of the pubo-coccygeal muscle is further evidence of sexual activity

- **Investigation:**
  - Specimens of any discharge on moist sterile swabs for MC&S
  - Sperm detected up to 12 hours after abuse
  - Semen up to 24 hours
  - DNA occasionally can be recovered 106 hours after abuse
  - Medico-legal/forensic specimens should be taken from all children who present within72 hours of having been sexually abused
  - Medical specimens include a pus swab if there is a vaginal discharge and blood to exclude syphilis, hep B and HIV.
  - Further specimens should be taken after 6 and 12 weeks if the initial serology was negative

Management of child abuse

- Should involve a wide range of professionals to address the needs of the abused child and family.
  - Traditionally social workers coordinated teams
  - Management involves 6 basic steps:

1. **Detection of possible abuse**
   - See above
2. **Investigation of possible abuse**
   - Ensure adequate management of child as well as protection from ongoing abuse
   - Physical or psychological state of child and social circumstances of child and family
   - Physical abuse:
     - Document nature and extent of injuries and to exclude an organic cause e.g. nutritional disorders, blood disorders and bone diseases → osteogenesis imperfecta
     - Attention to emotional sequelae especially PTSD
     - Investigation of social circumstances to assess if whether constitutes correct setting for child abuse and also to ensure that precipitating factors and underlying family dynamics are addressed as part of the overall management of the child and family
   - Sexual abuse:
     - Minority present to hospital <48hours → there is usually no urgency to examine them and examination can be deferred until someone who is competent to do so is available
3. **Validation**
   - ASAP – team decision based on all info whether child was abused or not
   - legal obligation of medical practitioners, in terms of section 42 of child care act, is to report all cases of suspected child abuse to a social worker/SAPS, usually through child protection unit and to notify the regional director of the Dept of Welfare
4. **Steps to protect the child**
   - Continued safety main priority
   - Ideal: at home in care of responsible and caring parent
   - Child at risk of ongoing abuse and above not possible: child or abuser must be removed

- Temporary measure: admit child to hospital while alternative solutions pursued.

5. **Treatment of the child**
   - Physical problems are easily identified and should be treated appropriately
   - **Prophylaxis**:
     - **Physical abuse:** (tetanus) ATT 0,5 cc IMI STAT (only if penetrating injuries present)
     - **Sexual abuse:**
       - Metronidazole 15 mg/kg/day in divided doses for 7 days
       - Ceftriaxone 125mg IMI STAT
       - If HIV negative on rapid testing 28 day course of AZT and 3TC should be given
       - Girls who have started menstruating/obviously entered puberty need a pregnancy test – if negative – must receive abortifacient – Ovral 2 tablets STAT and 2 tablets after 12 hours
   - PTSD - common complication. May need acute crisis intervention, follow up and long term support.
6. **Rehabilitation of the child and family**
   - Family therapy and reintegration of child into family responsibility of mental health professionals.
   - The doctor responsible for ensuring these needs have been met

Unusual manifestations of child abuse

- E.g. forced ingestion of drugs such as alcohol or cocaine; intentional microwave oven burns; forced ingestion of pepper, resulting in aspiration and fatality; water deprivation resulting in hypernatraemia
- Munchausen's by proxy:
  - Caregiver/mother induces factitious illness in the child, often with serious/tragic consequences
  - Mothers present repeatedly to hospitals and doctors with refractory complaints that disappear when mother and child are separated
  - Mother derives gratification from medical and nursing attention that results from placing their child under medical investigation
  - Mothers have history of abusive childhoods, abnormal illness behaviour themselves and possess a variety of unusual personality traits

Child neglect and abandonment

- Failure on the part of the parents/caregivers to meet child's basic needs.
- Differs from abuse in that harm to child is due to parent's omission
- **Majority**: inability of caregiver to provide necessary conditions through lack of physical/personal resources rather than from deliberate intent e.g.:
  - Parents extremely poor
  - Low intelligence
  - Cultural belief that has adverse effect on child's health
- Difficult to manage. Substitute care not practicable except where malevolent neglect is demonstrated
- Various forms – failure to meet needs of children (see needs of children)
- Features of neglect include:
  - Delayed development, especially of speech and language
  - FTT → objective evidence of growth failure in the absence of any organic cause
  - Characteristic physical features:
    - Dental caries
    - Pallor
    - Impetigo and contact dermatitis
    - Chronic suppurative otitis media
    - Hair loss over the occiput
  - Disorder of affect such as:
    - Avoidance of eye contact in infancy
    - Lack of stranger anxiety as a toddler
    - Poor interaction with peers at pre-school
- Most extreme: abandonment of child
  - Other reasons e.g. unwanted pregnancy, handicapped/chronically ill children
  - Neglect and abandonment must be prevented by:
    - Promoting bonding and ensuring adequate support during the postnatal period

        - Identifying those at risk ASAP e.g. mother displays little interest in her pregnancy/baby, uncooperative during birth, loathe to breastfeed
    - Management: see surrogate/alternative child care

Special problems of the adolescent:

- Difficult and confusing transition phase – pre-existing problems compounded by new ones.
- Impulsive and anti-social behaviour, depression and suicidal behaviour, eating disorders, drug dependency, sexual experimentation, teenage pregnancies.
- **Sexuality and adolescent pregnancy:**
    - Increased awareness of own sexuality
    - Sexual activity in teenagers is becoming increasingly prevalent in younger ages in virtually all communities
    - Majority of teen pregnancies occur within first year of becoming sexually active
    - In SA 330/1000 estimated teen pregnancy rate and up to 20% of women giving birth below 19 years of age in many state hospitals.
    - Teen pregnancies often occur in dysfunctional families – low levels of education and inadequate supervision
        - Therefore closely related to a culture of poverty and deprivation and other risk factors such as smoking and alcohol use
        - Other risk factors: single parent households, family history of teen pregnancy in mother or older sister, inadequate knowledge of sexuality, contraception and pregnancy
    - **Problems:**
        - Sexual feelings not appropriately dealt with – driven underground. Communication about healthy development of sexuality in affectionate and responsible relationships is more difficult
        - Sexual activity carries with it the inevitable risk of unwanted pregnancy, STDs/HIV
        - Adolescents least prepared to prevent or deal with these consequences
        - **Consequences for teenage girl:**
            - Late ANC booking (shame, ignorance of pregnancy signs etc)
            - Young women who haven't reached full physical maturity almost 3 times as likely to die of birth complications
            - Risk for 10-14 years much greater than for 15 – 19 years
            - Higher risk for infection, PIH, PTL
            - Subsequent health problems include stunted growth following early epiphyseal closure and 23% likelihood of another pregnancy in a year
            - ↓ likelihood of finishing school
            - Early entry into workforce at lower level, with fewer skills and poorer long term prospects
            - ↑ rate marital instability
        - **Consequences for baby:**
            - ↑ risk for infant for poor development and ill health
            - Lower chance of survival
            - ↑ LBW = ↑ susceptibility to illness and infection in babies of adolescents
            - ↑ perinatal and infant mortality rates in mothers under 20
            - ↑ SIDS, hospitalizations, accidents, burns, poisonings, superficial injuries.
            - Most reflect inadequate supervision by an immature mother
            - Psychosocial and material wellbeing compromised
                - Born outside marriage = stress and poverty
                - Very young mother = poor care → mother has needs of child
                - Ameliorated by extended family but often not available.
    - **Interventions**
        - Mother educated in basic knowledge of infant care
        - If she is clearly incapable of caring for baby adoption must be offered to her
        - NB Increased awareness by doctor of teen sexuality and inquiry into activity and contraceptive use
        - Promote early attendance at ANC
        - Increase support and nutritional supplements during pregnancy
        - NB High risk babies, infants and children of teenage parents recognized and wellbeing closely monitored

        - Support groups for young mothers
        - Education in schools, churches etc of life skills, parenting, sex ed

Drug dependence (addiction/abuse)

- Child is particularly vulnerable, traffickers try to exploit the young and gullible
- Levels of drug abuse: (3 levels)
    - Infant born to drug dependent mother. Profound handicap – may present as FAS or drug withdrawal syndrome. Physical and emotional neglect
    - Families of those involved in illicit production/trafficking of drugs are frequently deprived of adequate education, nutrition, domestic stability
    - Child/adolescent deprived of parental love/creative outlets at risk
- **Aetiology/epidemiology:**
    - Abusers can be:
        - In search of exciting experience because life appears dull
        - In search of oblivion because life lacks any joy or luster
        - In search of new personality because their life is filled with anxiety and indecision
    - Breakdown of social environment leaves adolescents with few capabilities in face of anxiety, conflicts, and temptation.
    - Males/urban ghettos ↑ risk of abuse
    - Most NB factors: lack of parental love and understanding and unrealistic expectations
- **Clinical features:**
    - Marijuana
        - Moderate use: euphoria, inattentiveness, loss of memory for recent events, ↑ suggestibility, nausea, vertigo
        - Higher doses: depersonalization, hallucinations and anxiety states
        - O/E: tachycardia, conjunctivitis, dry mouth, ataxia
        - Used to evade stress/confrontation
        - Interrupts normal psychological growth process – preventing emotional maturation – no skills for everyday life
    - Solvent and glue sniffing:
        - Hydrocarbons/toluene → euphoria, hallucinations and vertigo
        - Liver, kidney and CNS damage
        - Permanent brain damage may occur with ataxia, personality change or irreversible peripheral neuropathy
        - Consider in: unexplained coma, seizures, ataxia or behavioural disturbances
        - Precursor of major drug dependence
        - Sign of emotionally distressed child
    - Alcohol:
        - Suggests emotional instability or distress
        - Coma ensues earlier in child, high risk in infants
        - Hypoglycaemia = coma because of direct inhibition of gluconeogenesis.

Child rights and child advocacy

- Responsibility of child is with parents – civil society must ensure this is done properly
- 1959: Declaration of the Rights of the Child adopted by United Nations
- 1989: Convention of the Rights of the Child adopted by United Nations
    - Establishes children as equal and vital members of families and communities with inalienable rights
    - Needs should be met by identifying underlying reasons within society = breakdown of care and nurturing
    - Requires child health professionals to take on the role of child advocates – especially those in very difficult circumstances.
    - Advocates will need to change the world – using the convention as a tool and supporting processes which change the world.

PSYCHOLOGICAL, EMOTIONAL, AND BEHAVIOURAL DISORDERS

LEANE

APPROACH TO THE PROBLEM & ASSESSMENT PROCEDURE
COMMON PSYCHOLOGICAL & BEHAVIOURAL PROBLEMS:
→BIRTH TO 5 YRS
→6 TO 10 YRS
→11 TO 14 YRS

PSYCHOLOGICAL, EMOTIONAL, AND BEHAVIOURAL DISORDERS

APPROACH TO THE PROBLEM & ASSESSMENT PROCEDURE
COMMON PSYCHOLOGICAL & BEHAVIOURAL PROBLEMS

- **-BIRTH TO 5 YRS:**
  - **→MR**
  - **→TEMPER TANTRUMS**
  - **→SLEEP DISORDERS**
  - **→PDD**
- **-6 TO 10 YRS:**
  - **→SCHOOL FAILURE**
  - **→LD**
  - **→ADHD**
  - **→SCHOOL PHOBIA**
  - **→ENURESIS**
  - **→ENCOPRESIS**
  - **→TIC DISORDER**
  - **→ATYPICAL STEREOTYPED MOVEMENT DISORDER**
  - **→STUTTERING**
  - **→CHILDHOOD SEXUALITY & MASTURBATION**
  - **→ANXIETY DISORDERS**
  - **→DEPRESSION**
- **-11 TO 14 YRS**
  - **→CONDUCT DISORDER**
  - **→SUICIDAL BEHAVIOUR**
  - **→PSYCHOTIC DISORDERS**
  - **→SUBSTANCE ABUSE**
  - **→TEENAGE PREGNANCY & ABORTION**
  - **→VIOLENCE, CHILDREN & MENTAL HEALTH**

PSYCHOLOGICAL, EMOTIONAL, AND BEHAVIOURAL DISORDERS

Prevalence rates of significant psychiatric disorders among children range from 6 to 25% : variation depends on the age of the children, location, and identification criteria. Boys are more frequently affected than girls. Childhood psychiatric disorders:

- Educational difficulties at school
- Developmental delay or regressed behaviour
- Physical symptoms
- Behavioural problems

Educational difficulties at school

A sudden deterioration in school performance points towards a physical illness, an emotional problem or interpersonal problems either at school or at home. Failure at school evokes great stress in the child and the family because of the considerable investment made in attaining a good education for the child. Therefore, this can be a common cause for the result of psychological or emotional problems.

Developmental delay or regressed behaviour

This includes features of slow speech, faecal soiling, enuresis, or other unacceptable behaviour at the child's age and must be differentiated from organic brain disorders

Physical symptoms

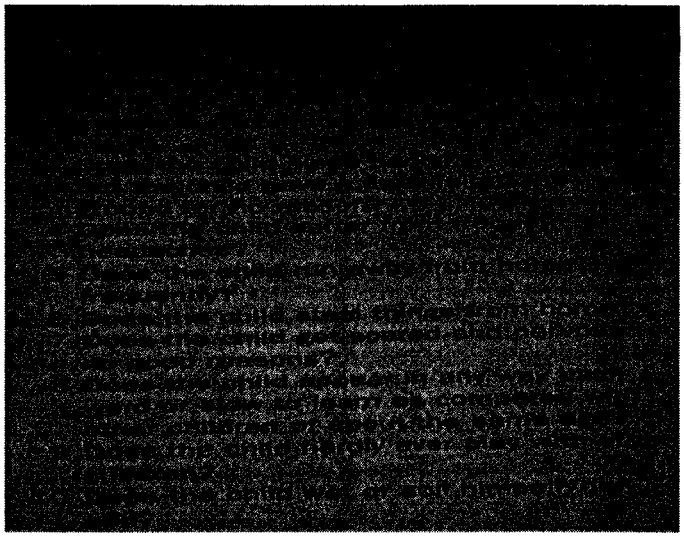
Like headaches, sore eyes, abdominal pains, diarrhoea, or pain in the legs are common complaints by children under stress(no organic cause).

Behavioural problems

Aggression, running away, stealing, generally indicate an unhappy child who has no other way of bringing his or unhappiness to the attention of adults.

APPROACH TO THE PROBLEM

WHO has complied a questionnaire to help professionals outline the problem area in the psychological domain.



Its NB to clarify the time span over which the child has had the presenting problem with particular attention to the following:

- Developmental milestones (regression)
- Child level of function(toilet training/peer relationships)
- Academic and social performance at school(teacher's report)
- Family history(3 generation genogram)
- Cognitive and education-related tests

The professional team: psychiatrists, psychologist, remedial teacher, OT, speech therapist BUT in developmental countries this is not always possible therefore a team of a doctor, nurse and teacher is extremely useful.

ASSESSMENT PROCEDURE

Comprehensive assessment: with particular attention to

- Development
- Family environment
- Cultural environment

CHILD DEVELOPMENT

In the absence of standardized psychological and other test the ESTIMATED LEVEL OF FUNCTION(ELOF) is a useful concept, its a rough estimate. The comparson should be made with other children in the family, class, or community in terms of speech, social interaction, play, tasks at home, performance at school, level of responsibility give to child, and the caregiver's and family's expectations of the child. Eg>10 year old with ELOF of 5...level of function of performance will be estimated at 50%.

FAMILY ENVIRONMENT

Permanent psychological and emotional scars are sustained by children who suffer the disruption by frequent change of caregivers and by exposure to family violence. However children differ widely in temperament.

CULTURAL ENVIRONMENT

Many families are in a state of transition eg. Moving away from cultural and religious norms of their parents. Change from town and country, migration, high social upward mobility and pressures of employment and unemployment are among factors which influence this transition.

PHYSICAL EXAM: emphasis on neurological system, hearing and vision is essential.

TESTING BASIC COGNITIVE TASKS: To confirm ELOF by estimating the child' intellectual ability and screening for possible specific learning disorders. Reading, calculations, writing, drawing are screened and age-appropriate responses measured. Clinical or educational psychologists can verify results.

TESTS FOR LATERALITY AND COORDINATION: As the child matures coordination develops(awareness of right and left), this can be observed which eye, hand or foot the child prefers.

DRAWING: Children usually love to draw. The way they draw and the content can be most informative. The Draw-a-person test gives a good indication of intelligence but culture and possible deprivation have to be considered.

PLAY: A play assessment is done by observing the child at play, which is useful in very young or grossly disturbed children. Some children are anxious, afraid and totally inhibited some will not use equipment appropriately at all; others are disinhibited and explore everything in a very short time.

FAMILY INTERACTION: in other words, the system in which the child is cared for. Ideally the patient is brought up by both parents but this is not common. The positive and negative relationships are assessed. Genogram is useful in showing relationships

PROBLEM ANALYSIS

The art of the assessment is being able to collaborate a great deal of info and give priority to main problem areas. Findings in the following heading:

- Child problems: The physical, psychological and educational aspects.
- Family problems
- Environment problems

TREATMENT

Factors which determine the choice of treatment includes accessibility and affordability in terms of time and cost as well as the severity of the problem. Possible treatment modalities may include medical treatment, psychotherapy, or special educational intervention, information or counselling for parents and family therapy. A report to the school is needed, especially if the child was referred by the school.

CHILD PSYCHIATRY IN DEVELOPING COUNTRIES: In most developing countries half the population is below 16 years, and due to the lack of psychological expertise ,the majority of these children and undiagnosed and untreated. There are few child psychiatrists and most of them work in the larger cities and private sector. There is an overlap of educational, physical and emotional concerns as well as mental and behavioural disorders in school going children.

COMMON PSYCHOLOGICAL AND BEHAVIOURAL PROBLEMS: birth to 5 yrs

1) MENTAL RETARDATION(MR): IQ

- **Mild (IQ 50-70)**
- **Moderate (IQ 35-49)**
- **Severe (IQ 20-34)**
- **Profound (IQ below 20)**

This is very common and mild degrees of retardation are not recognized. Causes include:

- Birth asphyxia; intrauterine infections ; intracranial infections ; congenital abnormalities ; brain damage due to epilepsy

Mental retardation results in a delay in achieving milestones. Regression of development is an indication of a progressive disorder.

EXAMINATION: Apart from ELOF a clinical examination is essential.

Eg. Dysmorphic features might suggest a chromosomal abnormality or hypothyroidism

Eg. Hearing and sight must be assessed, otherwise incorrectly labelled as being retarded.

It may be difficult to be sure of the degree of MR in a young child and a firm prognosis should be avoided.

MANAGEMENT OF MR:

- Treat child for any physical problems eg. Epilepsy/contractures
- Counsel parents about the condition and social benefits (the need for genetic counselling may be needed).
- Encourage appropriate stimulation of the child
- Discuss education, where appropriate
- Follow up

It is important to give the parents emotional support. Each milestone can be a major obstacle, demanding support and counsel for the parents. Dispel unrealistic expectations which parents may have. A MR child may put strain on the marriage relationship. Toilet training, personal care, and basic social skills are among the most important achievements for which to aim. It involves reinforcement of the positive aspects of intellect and behaviour. Behaviour problems may require special behaviour modification therapy or medication. Symptom-oriented medication used for short periods may be useful. The tendency towards polypharmacy needs to be resisted.

2) TEMPER TANTRUMS

Common problem in toddlers, ranges from the occasional outbursts to daily episodes associated with breath-holding attacks, cyanosis, and even minor seizures. Usually this is a passing phase with good response to counselling where parents as reassured and helped to cope with the tantrum. But this is difficult if parent control is lacking. Forms of epilepsy should be considered.

MX:

- Avoid frustrating situations by offering alternatives to the "forbidden fruit"
- Ignore the tantrum by walking away
- Avoid punishing the child for the tantrum
- Give full attention and approval when behaviour is acceptable
- Be CONSISTENT

3) SLEEP DISORDERS

Establishing a good routine with some children can be difficult.

MX:

- Exclude any physical abnormality eg. Epilepsy
- Consider child abuse
- Take a detailed Hx, looking at feeding patterns, routine in the family, stresses on the child, and parents attempts at solving the problem
- Be supportive, reassurance is important
- Find a way for the mother to sleep better to cope better
- Involve the father and other members of the family
- Assist in developing a bed-time routine
- Arrange family therapy for chaotic families
- Hypnotics for the child are NOT useful

4) PERVASIVE DEVELOPMENTAL DISORDER (PDD)

This includes children who have major impairment of social interaction and communication, and they are restricted in their activities and interests. Onset is during the first 3 years, although some have normal intelligence the majority are below average. PDD may be subdivided into

1)infantile autism 2)non-specific PDD

MX:

- Refer to psychologist/psychiatrists or paediatrician for diagnosis.
- Special intensive educational programmes

COMMON PSYCHOLOGICAL AND BEHAVIOURAL PROBLEMS: 6 to 10 years

1) SCHOOL FAILURE

- Detailed hx is essential

A sudden decline in performance, the cause is most likely physical or emotional, whereas a consistently poor performance may indicate mental retardation or specific learning problems.

2) LEARNINGS DISORDERS(LD)

These disorders must be seen as maturational and developmental problems and may occur despite a good educational foundation in a child with normal intelligence eg. Problems with reading, maths, expressive or receptive language coordination. DYSLEXIA is missed unless a careful Hx is taken and assessment is performed. An unrecognised LD may cause profound emotional difficulties.

MX:

- Define the problem by a remedial teacher or psychologist
- Explain the problem to parents and teacher
- Introduce a remedial-programme(preferably at home)
- Support the child emotionally
- Follow up and mentor

3) ATTENTION DEFICIT/HYPERACTIVITY DISORDERS

Problem: is the Inability to give sustained attention appropriate for the child's age. The lack of attention may occur with greatly increased activity. Child is distractable, disinhibited, and poorly organized with extreme over activity . However, hyperactivity is not always present especially when the child is in a strange environment.

MX:

- Early diagnosis
- Special education allowing for short attention and gross distractibility
- Behaviour modification techniques
- Medication with Methyphenidate, which is sometimes useful

4) SCHOOL REFUSAL (SCHOOL PHOBIA)

The longer the child stays away from school the more difficult it is to treat. It may be necessary for someone to accompany the child to school each day in the earlt stages of treatment.

MX:

- Full assessment of child with parents and teacher cooperating
- Establish areas of stress which may be remediated
- Get the child to go to school again with support ASAP
- Treat ongoing problems
- Medication is rarely necessary

5) BEDWETTING (ENURESIS)

Most children achieve day and night control of the bladder by 4 years. Nocturnal enuresis is considered a problem after the child has reached the mental age of 4 to 5 years. It may be an isolated developmental problem.

PRIMARY ENURESIS: child has never had total bladder control.

SECONDARY ENURESIS:

Occurs when child starts bedwetting again after attaining bladder control for several months. This is usually the result of emotional stress or a physical problem.

Enuresis tends to run in families.

MX:

If it is nocturnal and there are no other urinary symptoms, it can be assumed to be non-organic. There is a tendency to spontaneous cure. But when it is due to emotional stress symptomatic treatment is unlikely to be successful if the root cause does not receive attention.

- Educate the parents.
- Home programme:2 principles

1) Increasing bladder capacity, hold back urine as long as possible and then voiding into a container(30ml per year of age is a good guide to a reasonable bladder capacity). Increase capacity by drinking increased amounts and holding urine in as long as possible.

2) Self training to wake up when there is an urge to urinate.

- Reduction of fluid intake during the evening is advisable (use a reward star chart)
- IMIPRAMINE 25mg on retiring usually is enough and the dose can be doubled in the older child. Drug therapy must NOT be done in isolation!

6) ENCOPRESIS

A detailed Hx is necessary to obtain a clear picture of exactly when and under what circumstances the encopresis occurs. This may indicate the emotional causes. A clinical exam must be done to rule out organic causes eg. Megacolon which causes chronic constipation with 'overflow" incontinence. Rectal examinations must be done. Primary neuro deficits eg. Spina bifida or cord lesions must be excluded. Encopresis with late onset and with no neuro lesion is very likely to be the result of emotional stress. It is often accompanied by enuresis and infrequently by psychopathology.

MX: where possible try to correct the stress situation coupled with the introduction of behaviour modification techniques

7) TIC DISORDER (STEREOTYPED MOVEMENT DISORDERS)

These include: transient tics; chronic motor tics; Tourette's disorder ; and atypical tics and stereotyped movement disorder. The presenting features are rapid movements of a group of functionally related skeletal muscles or an involuntary production of noises and words. These characteristics distinguish them from other movement disturbances, such as choreiform movements. May be associated with emotional disturbances.

MX:

If the tic is of short duration:

- Anxiolytics
- Low doses of antipsychotics (haloperidol) are useful.
- Methylphenidate will worsen the tics.
- Parental counselling
- Comprehensive biopsychosocial assessment

8) ATYPICAL STEREOTYPED MOVEMENT DISORDERS

Eg. Head banging, rocking and repetitive hand movements. They are distinguished from tics in that they involve voluntary or non-spasmodic movement and the patient is NOT usually distressed by the symptoms. Incidence is high in children with MR, PDD and markedly inadequate social stimulation. It may also occur in the absence of a mental disorder.

MX:

- Detailed assessment of interaction between parents and child
- Increased contact with mom
- Parent counselling
- Increased stimulation of child
- Technique for controlling rocking and hand movements: try make these rhythmic motor habits purposeful by making music, dancing, hobby horses, see-saws, swings....

9) STUTTERING

This may be accompanied by jerks, blinks, or tremors. The onset is usually before the age of 12 years and there may be a family history. Over 50% of milder cases make a spontaneous and complete recovery.

MX:

Management remains controversial on what treatment is the best. Modern approaches are based on the concept that the disorder is a learned form of behaviour.

- Speech therapy
- Behaviour and individual therapy

10) CHILDHOOD SEXUALITY AND MASTURBATION

Parents are generally the main models of appropriate sexual roles. Children quickly discover the gratification which results from stimulation of the well-innervated external genitalia. Masturbation should be viewed with concern only if it becomes a persistent habit. In most children it is a passing phase. Emotional deprivation occasionally leads to the child expressing an uninhibited and affectionate attitude to relative strangers which may be interpreted as sexual precocity and has resulted in sexual abuse.

MX:

Take into account the developmental level of the child. Spontaneous remissions are common in infants, most of whom grow up to be normal unless the situation is mismanaged.

- Attitudes of parents needs to be assessed and they should be assured of the innocuousness of the habit
- Boredom must be considered a contributory factor and well as the irritability of tight clothing
- Increase the physical activity of child to reduce the childs energy levels
- Sex education for the child may be necessary, mention that masturbation is an infantile habit
- Correctly discourage masturbation
- Enquire about the emotional factors
- Parents should not overreact to their pre-school child's natural interest in the genitalia of the opposite sex(take a matter-of-fact approach)

11) ANXIETY DISORDERS (NEUROTIC AND EMOTIONAL)

These are common disorders in children In adolescence, somatoform disorders with physical symptoms are quite common.

MX:

- Detailed assessment is necessary, with careful exclusion of physical pathology.
- Therapy usually involves both parents and child
- Provide specific treatments of physical or educational problems
- Medication: short term with anxiolytics or long term with antidepressants
- Refer the child to a main centre for review if there is no improvement in a few months

12) DEPRESSION

This is part of the mood disorders. Children may present with different symptoms than adults. Mania is less common in young children than in adolescents. After puberty depressive disorder approaches the same prevalence as adults . This cause may be hereditary with a biological vulnerability.

MX:

- Make a detailed assessment with careful exclusion of physical pathology
- Treat the child for non-specific areas of stress
- Provide family counselling
- Anti depressant medication may be useful and should be maintained for several months(TCAs taken in overdose can be lethal)
- Where there is suicidal behaviour or there is no response to treatment within 5 to 6 weeks, refer child to an expert.

COMMON PSYCHOLOGICAL AND BEHAVIOURAL PROBLEMS:11-14 years

1) CONDUCT DISORDER

This disorder is characterized by antisocial behaviour. The problems must have existed for 6 months or longer. Conduct disorder is the most common psychiatric disorder in older children.

MX:

- Family and behaviour therapy
- Medication has a small place eg. In ADD

2) SUICIDAL BEHAVIOUR

Suicide threats and behaviour should never be taken lightly as just 'attention seeking' or ' a cry for help'. Depression and conduct disorder account for aprox. 50% of these children and adolescents.

The assessment should always include an evaluation of the risk of repeat, and possibly success, attempt.

- Disorders such as depression, psychosis, and drug abuse should be identified and attended to. In the evaluation, predisposing, precipitating, perpetuating, and protective factors must be identified for which an interview with the parents is essential.
- Facilitate communication

3) PSYCHOTIC DISORDERS

"Psychotic" means that the patient is out of touch with reality. Features: disorientation, memory loss, and inability to do simple intellectual tasks.

- Rule out organic causes, substance abuse, epilepsy
- Refer to a specialist

4) SUBSTANCE ABUSE-SEE SOCIAL PEADIATRICS

5) TEENAGE PREGNANCY AND ABORTION-SEE SOCIAL PEADIATRICS

6) VIOLENCE, CHILDREN AND MENTAL HEALTH

Violence has been a constant partner for this generation in SA throughout their development. Assault, hijacking and random brutal violence remain part of everyday experience in many areas. Children are unavoidably a part of this violence. This is not a normal society. The evil of racism, educational depreivation and years of violence has taken a tragic toll on the mental health of a generation.

MX:

- Establish community-based, easily accessible, walk-in child and family help centres where preventative and rehabilitation mental health principles will be applied; where educational problems can be addressed; and where help can be given to people to restore stable family life and to maintain the integrity of the FAMILY. Present structures can be used for this purpose. Professionals can join with lay people and YOUTH groups in mass action to provided DIGNITY and HOPE.
  I know that this is a bit random....for the few of you that actually read this chapter..the above paragraph has been my dream since a young age, to start a HEARTFELT HEALTH COMMUNITY CENTRE, based on my dad's property of Heartfelt Family Arena(big blue building across from One Mil hospital in Pta).....so watch that space..and if you ever want to volunteer you're BRILLIANT expertise ☺give me a ring or go visit me there!!! God bless to everyone for the exam! Leane Sebastiao