
Paediatric Study Notes

Edited by David Tripp

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Disclaimer: These are study notes I compiled to get me through the 2004 Diploma of Child Health (DCH), offered by the University of Otago. I share them in the hope they may help others. However, this has not been peer reviewed in any way. I take no responsibilities for mistakes. Learn from them – don't repeat them!

I am very grateful to sources too numerous to name from which I have compiled, cut and pasted in order to learn everything I must know....

These notes are based on the paediatrics chapter of my 5th year medical school notes, and incorporate further material from the DCH course. These notes, and others I have compiled over the years, can be downloaded from www.sites.google.com/site/davidtrippnotes.

Happy Hunting!

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Epidemiology

Measures of Child Health

- Measures of death/disease:
 - Mortality
 - Disease specific mortality/morbidity (eg SIDS)
 - Hospital Discharges
 - Disparities: ethnic, gender, age, location, etc
- Measure of health interventions:
 - Immunisation coverage
 - Well child checks
- Measures of health or its determinants or impacts:
 - Breast feeding at 3 months, 6 months
 - Participation: early childhood education, school, sport, etc
 - BMI: marker of appropriate nutrition
 - Self-report (eg questionnaires)

NZ Statistics

- Numbers of children:
 - 23% of NZers are aged 0 - 14
 - Maori and PI children are about double adult proportions as percentage of total population – 1/3 of Maori and PI people are under 15, compared with 19% of Europeans
 - Until 2050, fall in the number of children, and fast fall in their proportion of the total population (from 23 → 16%) → future conflict over resources: “principle of first call” – essential needs of children should be given high priority in the allocation of resources
- Socio-economic status:
 - Children with no parent participating in the labour force (1996): European 13%, Maori 42%, PI 37%, Asian 30%
 - Proportion of children in one-parent families (1996): European 15%, Maori 43%, PI 27%, Asian 12%. Increased over all groups from 1986 to 1996
 - Maori and Pacific Islanders also more likely to not have a car, share a household, less likely to leave school with a qualification
- Mortality:
 - Under 5 mortality currently around 500 per annum
 - Age specific rates: 7/1000 live births for 0 – 1 years, 0.4/1000 for children 1 – 4 years, 0.2/1000 after this
 - All child mortality rates in NZ have declined by 1/3 over the last 15 years, but this is slow in comparison with other countries. Our OECD ranking for under 5 mortality has fallen from 6th to 15th. If we had had the same fall as Sweden 194 children would not have died.
 - Major causes of death:
 - < 1 year: SIDS (29%), Congenital abnormalities (28%), Perinatal conditions (27% - prematurity, neonatal infection, hypoxia, etc)
 - 1 – 4 years: Injury and poisoning (46%), Congenital abnormalities (18%), Cancer (11%).
Maori injury and poisoning rate 3.5 times Non-Maori
 - SIDS rate has fallen from 4/1000 in 1989 to 1.5/1000 (UK is 0.6/1000). Rate 4 times higher in Maori than non-Maori. Low income 3 times higher income (independent of ethnicity) – due to risk factors of maternal smoking, teen pregnancy, single parenthood, etc
- Morbidity:
 - Under 1’s most likely to be admitted: NICU, respiratory GI and infectious
 - Ethnicity patterns same as for mortality: Maori rates range from 1.7 – 4.6 times higher
- Conclusions:
 - Despite improvements, New Zealand hasn’t made the gains that other countries have
 - Ethnic and socio-economic disparities are growing
 - Improvements in curative medicine are unlikely to have an impact on this inequality

Health Systems

- Determinants of child health:
 - Biology: genetic, development in utero
 - Socio-economic:
 - Housing
 - Income
 - Education (especially maternal – key issue in 3rd world)
 - Environment:
 - Social
 - Physical
 - Health Behaviours
 - Health Services
- Impact of determinants:
 - Perinatal complications and family adversity have an *independent* impact on cognitive ability
 - Social adversity is a bigger factor in mild mental retardation than biological factors
 - Variations in family social background is a:
 - Weak determinant of specific problems
 - Pervasive determinant on generalised vulnerability to a wide range of problems
 - Large differences in absolute income have little or no effect on mortality. Small increases in income equality have a large effect
- Improving health equality through health services:
 - Population based measures:
 - Resource allocation
 - Intersectorial collaboration
 - Community development
 - Data collection on deprivation
 - Salaried GP services for deprived areas
 - Individual health services:
 - Site and mode of provision of services
 - ↑Communication with consumers
 - Targeting of preventative services

International Agreements

- Alma Ata Declaration on Primary health Care:
 - Declaration to protect and promote the health of all people
 - ‘Health for all by the year 2000’ through quality primary care
 - Defined primary care and gave principles for health services
- Ottawa Charter on Health Promotion:
 - Building health public policy
 - Creating healthy environments
 - Strengthening community action – power and control to communities to identify and solve their problems
 - Helping people to develop their own skills
 - Reorientating the health system: balance between preventative and curative services
- Jakarta Declaration on Health Promotion in the 21st Century:
 - Comprehensive approaches (using all 5 Ottawa charter principles) are the most effective
 - Settings offer practical opportunities for the implementation of comprehensive strategies
 - Participation by the community is essential
 - Health learning fosters participation
- United Nations Convention on the Rights of the Child:
 - Principles and standards by which governments, organisations and families can be measured
 - Ratified by NZ in 1993
 - Rights:
 - Article 18.2: “render appropriate assistance to parents and legal guardians in the performance of their child-rearing responsibilities
 - Article 23: Rights of mentally or physically disabled children: “full and decent life.... Dignity ... self-reliance Active participation in the community”

- Article 24: Right of all children to “the highest attainable standard of health and to facilities for the treatment of illness and rehabilitation of health”
- Most recent assessment of NZ raised concerns about:
 - Fragmented approach to the rights of the child
 - Insufficient data collection on the effects on children of economic reform
 - “Extensive delegation of support services to children and their families” – but quality remains the responsibility of the state
- NZ Child Health Strategy 1999 had 6 future directions:
 - A greater focus on promotion, prevention and early intervention
 - Better co-ordination
 - Development of a national child health information strategy
 - Child health workforce development
 - Improve child health research and evaluation
 - Leadership in child health

Difference Between Adult and Child History Taking

- Kids may not be able to tell you, teens may not want to
- Concerned with nutrition: what normal and current feeding practices are, micro-nutrients
- Growth: weight, height and head circumference
- Developmental perspective:
 - Gross motor: rolling, sitting, crawling, walking, running, stairs, sports
 - Fine motor: hand skills, co-ordination (assessed through play → art → writing)
 - Vision
 - Speech/language and hearing
 - Social development: bonding → parents vs strangers → peers
- Immunisation: 'Are your immunisations up to date' – usually meaningless. Need to be more specific
- Family history:
 - Congenital abnormalities
 - Genetic factors
 - Parental age and experience
 - Impact of chronic illness on family
- Social history:
 - Abuse and neglect
 - Living circumstances – overcrowding, smoke exposure
 - Education settings, eg day care
 - Peer support for kids (eg in adolescence)
 - Adolescence: HEADSS Assessment (see page 178)

History Outline

- General data: name, DOB, Ethnicity, where they live
- Presenting Complaint
- History of presenting complaint:
 - Chronological and including symptoms across all systems
 - Treatments so far
 - Contact history
 - Family history of the complaint
- Paediatric Past Medical History:
 - Antenatal
 - Birth/perinatal: Gestation, delivery, weight, APGARS, any special care, complications
 - Feeding (breast, formula, solids) – detailed if relevant (eg which formula, how much, which solids, how much)
 - Weight – growth history, where relevant growth and puberty in family members
 - Immunisations
 - Milestones – including relevant milestones for the child now: Cover Gross and Fine motor, receptive and expressive language, social, play and self care skills. See Development Chart: normal development from 0-60 months, page 22
 - Past medical history
 - Social/school
- Medications
- Allergies
- Family History: ages and health of parents and grandparents. Ages, names and health of siblings
- Social History:
 - Parent's occupations
 - Who cares for the child
 - Schooling/childcare, performance at school
 - Behaviour at home/school
 - Sleeping arrangements and home circumstances
 - Financial circumstances

- Alcohol, smoking
- Pets
- Problems/stresses at home
- Systems enquiry (OHCS, p 173):

	Neonate	Toddler	Older Child
Cardiorespiratory	Tachypnoea, grunts, wheeze, cyanosis	Cough, exertional dyspnoea	Cough, wheeze, sputum, chest pain
Gastrointestinal	D&V, jaundice, stool frequency	D&V, stool frequency	D&V, abdominal pain, stool frequency
Genitourinary	Wet nappies (how often?)	Wet nappies (how often?)	Haematuria, dysuria, sexual development
Neuromuscular	Fits, odd attacks, jitters, feeding ability	Fits, drowsiness, hyperactive, vision, hearing, gait	Headaches, fits, odd sensations, drowsiness, academic ability, vision, hearing, co-ordination
ENT and teeth	Noisy breathing	Ear discharge, teeth eruption	Earache/discharge, sore throat

- General questions: fatigue, lumps, itch, fevers, bleeding tendency, family interaction

Examination

- Principles:
 - Leave nasty things till last
 - Observe
 - Get on floor and use games
 - Wait until child familiar with environment but start before bored
 - Don't touch child until rapport established
 - Use your own toys – they're novel
 - Get parents to undress them (or do anything else that is nasty)
 - Get them to draw pictures while taking the history
- They're likely to be scared (depending on previous experience). Build rapport, play games, talk with child not through parent. Don't wear stethoscope around neck
- Show them what you want rather than telling them
- Blood pressure:
 - Is important – **always do it**
 - Getting them calm is hard – usually anxious → artefacts common
 - Cuff: Bladder should nearly encircle the arm. Width is 2/3 length from shoulder to elbow
- Chest exam:
 - Percussion more sensitive than auscultation (won't show anything in the absence of respiratory signs/symptoms)
 - Percussion will tell you about hyperinflation, fluid, mediastinal shift
 - Auscultate heart early in the exam – but not first
- Differences in a baby:
 - More liver in the abdomen (2 finger breaths is normal). Don't press too hard – moves with respiration
 - Pelvic organs higher (eg bladder)
 - Pulses: Radial/ Brachial – take both sides. Must palpate femoral pulse. If feet aren't white don't take peripheral pulses
- Teenage girls: examine chest underneath clothes

Normal Values

Age	Breathing	Pulse	Systolic BP
< 1 yr	30 – 40	110 – 160	70 – 90
2 – 5 yr	20 – 30	95 – 140	80 – 100
5 – 12 yr	15 – 20	80 – 120	90 – 110
> 12 yr	12 - 16	60 - 100	100 - 120

- Stethoscope around your neck adds 10!

Examination outline + tips for exams

- Introduce yourself + start building rapport
- Wash hands
- Ensure good light
- Look around room: inhalers, O₂, lines, monitoring
- Expose relevant parts, cover lower half with a sheet
- Height, Weight and Head Circumference (and plot them) – use syndrome specific growth charts if appropriate.
- General inspection – “Can you stand up for me” – look from front, side, back:
 - Nutrition
 - Sick or well
 - Dysmorphic features
 - Obvious distress
 - Temperature
 - Colour/rashes/anaemia/cyanosis/jaundice
 - Hydration/perfusion
- Hands:
 - Clubbing (Shamroth’s sign)
 - Nails
 - Palmar erythema, pallor of palmar creases
 - Pulse + respiratory rate
 - Blood pressure
- Cardiovascular:
 - Pulses: radial, femoral, synchrony, sinus arrhythmia (normal in all children)
 - Blood pressure (NB use correct cuff size)
 - JVP: often hard to see
 - Eyes: pallor, jaundice
 - Mouth: cyanosis, dental hygiene, hydration
 - Chest inspection: Harrison’s sulcus, symmetry, pulsations: SCARS:
 - Thoracotomy scars – axillary or subscapular: operations outside the heart
 - Axillary scar: PDA banding, shunts or coarctation repair
 - Central thoracotomy scar: open heart surgery
 - Feel the cardiac impulse: Apex may be more lateral in children.
 - Palpate for thrills, parasternal and substernal heave, palpable P₂
 - Auscultation in 4 valve areas:
 - HS: splitting, loud P₂
 - Murmurs: position, timing, loudness, character, radiation, manoeuvres
 - Roll to left to accentuate diastolic murmur (mitral stenosis)
 - Sit up, lean forward, breath out – listen for diastolic murmur of aortic stenosis
 - Normal variants in heart sounds:
 - Venous hums: low pitched, base of heart, change with position of head/neck
 - Innocent murmur: musical murmur at L sternal edge, disappears in extension
 - Peripheral pulmonary stenosis: widely heard, often loud, often at back
 - Look at back for scars, percuss, and auscultate lung basis for murmurs & creps
 - Feel for sacral oedema
 - Remove pillow. Palpate liver (→ right ventricular failure) – enlargement, smooth, pulsatile. Spleen.
 - Feel femorals
 - Look in inguinal area for cardiac catheters
 - Peripheral oedema (Periorbital in babies)
 - Clubbing in toes
 - Fundus for Roth’s spots
 - Summary: X is a well looking 6 year old with complex congenital heart disease who has obviously had surgical repair. In terms of his murmurs... My exam does not reveal any signs of congestive heart failure, infective endocarditis or arrhythmias
- Respiratory:
 - Hands: Tremor, flap, clubbing, pallor
 - Pulse: bounding? (CO₂)

- Respiratory rate, effort and accessory muscle use, grunting, ability to talk in sentences
- Eyes: jaundice, pallor, cyanosis
- Sinuses
- Nose: patency, polyps
- Stand up, with hands behind head and on hips check for chest deformity: symmetry, scars, check from side for hyperinflation
- Check for expansion from behind
- Intercostal, sub-sternal and supraclavicular indrawing, hyperinflation, Harrison's sulcus (lower ribs pulled in → chronic airways disease), pigeon chest (⇒ chronic ↑ in AP diameter), tracheal tug, nasal flaring
- Tracheal tug
- Percussion, including cardiac dullness (⇒ hyperinflation)
- Auscultation: comment on air entry, breath sounds, crackles, wheeze, rubs. Don't forget axillae
- Sacral odema while sitting up
- Lie down: feel liver
- Check for pedal oedema, clubbing of toes
- Ears and throat, also have a good look around the mouth and comment on dentition
- Abdominal:
 - Remove pillow
 - Check for nutritional deficiencies:
 - Rickets: splayed radius, rickettary rosary, bowed legs
 - Gum health: vitamin C
 - Iron: conjunctival pallor
 - Mouth: Stomatitis, glossitis
 - Inspection, movements, scars, hernia
 - "Are you sore anywhere?"
 - Get child to suck in and push out tummy to check for tenderness – then you won't have to hurt them yourself.
 - Palpate
 - Palpate liver, feel for consistency of surface, measure span
 - Palpate and percuss for spleen – to palpate get them to breath in and out and feel gently on surface
 - Ballot kidneys
 - Percuss for ascites – if no dullness in flanks no need to roll patient over
 - Percuss for tenderness
 - Clues for peritonism: lies still, hurts with coughing/going over bumps, won't puff abdomen in and out
 - Bladder
 - Roll child onto side, examine back for scars, spina bifida, biopsy sites, spinal tenderness
 - Front inguinal regions – scars, hernias
 - External genitalia + Tanner staging
 - Examine anus (PR rarely required)
- Lymph nodes: Check anterior nodes from behind, posterior and submandibular from in front. Check anterior and posterior cervical chains, subhyoid, sub-occipital, sub-mandibular, sub-lingual, axillary, inguinal and epitrochlear
- Neurological:
 - Developmental assessment: See Child Development, page 21
 - Neurological Exam: See Neurological Exam in Children, page 93
- Joints
- Skin

When is a child really sick?

- Factors which are **not** on their own discriminating between mild and severe:
 - Temperature: spikes easily
 - Pulse: variable, eg ↑↑ if crying
 - Blood pressure: hard to measure, and if shocked is still maintained till very late. As soon as they have *any* hypotension they're the same as an adult with no recordable BP
- Factors from **history** which discriminate:
 - Intake:

- Refusal to feed \Rightarrow more severe
- Refusal to take solids but still taking liquids \Rightarrow not so bad
- Losses:
 - Vomiting:
 - Frequency and amount: if vomiting their whole feed then bad (vs a small spill)
 - Colour: Bile is bad. Yellow (from gallbladder), green (after bile has been in the stomach) or orange. Due to obstruction or $\uparrow\uparrow$ sympathetic discharge, eg due to pain (not necessarily abdominal – could be a torqued testicle)
 - Decreased urine output (wet nappies $<$ 4 per day)
 - Diarrhoeal losses
- Dysuria and pale extremities may be the only warning signs before they crash
- Factors which discriminate on **exam**:
 - Floppiness: \downarrow tone
 - Perfusion: pale, mottled or blue, cold. **Capillary refill $>$ 2 secs.** (ie Peripheral vasoconstriction)
 - Fitting
 - Cyanosis
 - Tachycardia
 - Respiratory rate: quality as important as rate
 - Rash if petechial/purpuric (?meningococcal septicaemia)
 - \downarrow pH
 - \downarrow **Weight** (dehydration)
- Toxic Appearance =
 - Decreased level of arousal
 - Circulatory compromise: pallor, tachycardia, cool + mottled limbs, hypotension
 - Respiratory impairment:
 - Tachypnoea, grunting respirations, recession, cyanosis
 - Due to \uparrow O₂ requirements + trying to blow off CO₂ from acidosis + pulmonary oedema from capillary leak
- Shock =
 - Clinical diagnosis of failure of the circulatory system to deliver sufficient O₂
 - Look for compensatory mechanisms which try to maintain perfusion of vital organs (\uparrow HR, peripheral vasoconstriction)
 - Causes of shock:
 - Capillary leak \rightarrow \downarrow cardiac output
 - Changed vascular tone
 - Impaired myocardial function
- Progression: Toxic \rightarrow Septic \rightarrow Shock
- Specific signs:
 - Meningism: bulging fontanel, rash, stiff neck
 - Pneumonia: chest sounds (not very sensitive)
 - Distended abdomen and guarding: obstruction, appendicitis
 - Lumps in the inguinal region (seen or felt): hernia \rightarrow obstruction \rightarrow acidotic
 - Blood in faeces: Intussusception
- Basic investigations:
 - Bloods: FBC, electrolytes, culture, ABG, (cross match)
 - X-rays: chest, abdomen if distended
 - Urine culture (bladder stab)
 - Maybe lumbar puncture

Neonates

- Check list for a neonate (clinical acumen less reliable):
 - Fever: consider full sepsis evaluation for any child $>$ 38 C
 - Feeding: if intake $<$ 50% normal
 - Urine output: $<$ 4 wet nappies in 24 hours
 - Peripheral circulation: pallor of recent onset, mottling, cold periphery, slow capillary return
 - Responsiveness: poor response to stimulation and a weak cry
 - Activity: \downarrow movement, \uparrow sleepiness
 - Breathing difficulty: signs of distress, cyanosis, RR $>$ 60

- Apnoea: pause in respiration > 20 secs. Central (eg premature) or obstructive (eg URTI) or mixed
- Vomiting: treat any vomiting in neonate seriously. Look for bile staining
- Cyanosis
- Seizures
- Severe jaundice: risk of bilirubin encephalopathy

Talking with Children

- Do:
 - Engage them
 - Explain who you are and why you are seeing them
 - Use language and concepts that are age appropriate
 - Reassure if seeing separate from parents
 - Outline confidentiality issues with older children or adolescents
- Don't threaten:
 - The child's sense of loyalty to their family
 - Their defences against unbearable emotional pain
- Interviewing preschoolers (3 – 5 years):
 - Get down to their level, use simple language
 - Take things at their pace
 - Can use play, drawings and stories
 - Ask about everyday world
 - Watch verbal and non-verbal communication
 - See with parent
- School age children (6 - 11 years):
 - Can be structured
 - Ask about feelings (sadness, anger, etc) as well as daily life
 - Ask about family, school, friends, problems, worries
 - Wishes, hopes for the future
 - Very abstract, open-ended questions can be confusing
- For adolescents see Talking with Adolescents, page 177

Parent and Adolescent Education

- Aim is to change behaviour. Changing behaviour requires:
 - **Knowledge**: necessary but not sufficient
 - **Skills**: to manage the change
 - **Motivation**: Involves striving towards a goal, not just 'trying'. The goal must be:
 - Important to the person – 'I want this'. Make it attractive. May need their goals to come before yours.
 - Achievable – 'I can do this'. Believe in them
 - Not too unpleasant. 'I don't mind doing this'. Make it easy
- Good counselling technique:
 - Open-ended questions: "tell me about..."
 - Active listening: "Hmm, I see..."
 - Reflection: reflect facts **and** emotions
 - Summarising: "Let me see if I've got this straight..."
 - *Don't* ask leading questions: eg "You don't do that, do you?"
- Take a history using open-ended questions, reflecting, summarising:
 - Help parent or adolescent clarify exactly what it is they want to know
 - **Knowledge**: what do you understand about...? Where did you find that out? How convinced are you?
 - **Attitudes/fears**: are you worried about anything in particular?
 - **Practices**: What have you actually done so far?
 - **Barriers**: What's stopping you from doing this?
- Then:
 - **Validate/reinforce** knowledge they already have: "That's terrific – you already understand a lot..."
 - **Education** to correct incorrect beliefs/address fears
 - **Encourage** them to find their **own solutions**: "So, what do you think you could do?"
 - **Reinforce** safe practices and responses

- See also Neonatal and Infant Anticipatory Guidance (Parent Education), page 38

Families

- Ref: notes from Lorraine Christie, Clinical Psychologist

Unuhia te rito o te harakeke
Kei whea te komako?
E hua whakatairantitia
Rere ki uta, rere ki tai
Mau e ki mai He aka te mea nui o te ao?
Maki e ki atu, He tangata, he tangata

If you pluck the young shoot of the flax bush, where will you find the bellbird?
It will be fluttering about flying to the beach and sea!
What is the greatest thing in the world? I tell you, it is people, it is people

- The task of childhood is development:
 - Social relationships
 - Emotional maturity
 - Sound set of values and beliefs
 - Sound thinking patterns
 - Knowledge
 - Of body and skills
- Self esteem and self respect laid down in the first 7 years are influenced by: Gender, race, culture, sexuality, temperament, ordinal position, IQ, physical characteristics, creativity, did you arrive in the family at an okay time
- Families are systems with:
 - Structure
 - Various roles
 - Authority
 - Channels of communication
- Five characteristics of healthy families:
 - The marital relationship is the strongest relationship and the greatest focus of power
 - Communication is open and honest and permits spontaneous interruption
 - Warmth and caring predominates over anger and hostility
 - There are known problem solving techniques which can be instigated quickly
 - Movement towards independence for all members of the family
- The family life cycle: constant reorganisation and change
 - Two form a couple:
 - Take on husband/wife/partner roles
 - Substantial reorganisation of boundaries: family, friends, togetherness vs autonomy
 - Work through differences
 - Structuring a relationship: complimentary vs symmetrical
 - First baby:
 - Parenting roles
 - Ensure spouse relationship remains the strongest
 - Reorganised boundaries to allow for grandparents, friends, interests
 - Preschoolers:
 - Protect spouse relationship: contact, support, being together
 - Parent/child subsystem: affection, encouragement of appropriate autonomy, boundaries for child, individuating siblings from each other
 - Continued reorganising of boundaries: grandparents, outside world, work
 - School age children:
 - Integrating school and family systems
 - Allowing age appropriate autonomy
 - Children less egocentric, more sensitive to other's needs, develop sense of fairness and gender awareness
 - Adolescence:

- Issues of proximity and distance
- Extending boundaries to allow independence
- Continual renegotiation – autonomy vs control
- Sibling individuation from each other
- Young Adult: between families
 - Differentiating self from family of origin
 - Leaving home, career development
 - Intimate peer relationships, courtship
 - Parents: at height of careers, retirement looming
 - Grandparents: needy, mobility, loss of sight, death
 - Parents take on grandparent roles

Behavioural Issues

- Behaviour doesn't exist outside an environmental context

Behaviour Management

- History Taking:
 - Antecedent: what sets him off?
 - Behaviour: describe exactly what he does?
 - Consequence: What do you do about this?
- Principles:
 - Remove (time out). Somewhere safe and boring, and where you don't mind the child disliking (ie not the toilet if toilet training or bedroom if sleep training). Leave a minute for every year of age. Be specific about what they're going in for. At the end, remind of the behaviour you want, and then forget the incident.
 - Anticipate/avoid situations where conflict is likely
 - Ignore minor things, particularly tantrums
 - Distract
 - Example (set a good one)
 - Reward acceptable/wanted behaviour
- Reward Systems:
 - Star chart if young, more sophisticated and discrete if older
 - *Agreed* between parents and child. Child has to own it (can they help make it?)
 - *Planned*: don't have to make a decision when the time comes
 - *Anticipated*: known about in advance – **When** you this, **then** you will have ...
 - *Consistently* applied: no matter where he is or who he is with
 - *Immediate*: not at the end of the week or when dad gets home from work
 - Strong *reward* component
 - *Meaningful* to child:
 - Young child: cuddles, praise and attention
 - 8+ years: if you ... you can choose what we have for desert/which video, etc (choice is powerful)
 - 10+: money
- Referral options:
 - Special Education Service (SES): Resource teacher for learning and behaviour (RTLB) or Behaviour Support Team through SES
 - Child psychiatry service (CAFS) if severe psychiatric symptoms (anxiety, depression, OCD, PTSD, sexually abused, ADD), persistent family dysfunction or resistant to simple management strategies
 - Paediatrician if medical issues
 - CYFS if abuse

Risk and Resilience

	<i>Good</i>	<i>Bad</i>
<i>Internal</i> (child factors)	Resiliency: High self esteem, happy temperament, high IQ, problem-solving skills, coping strategies, humour	Vulnerability: poor social skills, low IQ and self esteem, hopelessness
<i>External</i> (factors in the environment)	Protective: caring and supportive adult, reasonable structure and limits, being believed in	Risks: Family discord, hostility, lack of warmth and understanding, lack of friends

- Attribution theory: Are their successes and failures due to an internal locus of control ('I passed the test cause I did the work' → high self-esteem) or an external locus of control ('It doesn't matter if I study or not, it won't make any difference' → low self esteem)
- Individual traits that build resiliency:
 - Insight: recognition of ones' distressed condition with subsequent action to overcome barriers
 - Independence: gaining emotional distance and autonomy amid chaos
 - Initiative: achievements that foster self confidence and constructive activity
 - Relationship building: Protective and nurturing connections with at least one supportive adult
 - Creativity: facilitates healing and positive activity in a difficult environment
 - Humour: focuses on hope not harsh realities
 - Morality: commitment to fairness and compassion
- Family traits that build resiliency:
 - Commitment: loyalty, determination to work things out, sacrifice for mutual benefit
 - Cohesion: togetherness, respect for the individual, interdependence
 - Adaptability: flexible, stress coping skills
 - Communication: listening and speaking skills
 - Spirituality: shared purpose and values
 - Connectedness: Support within and beyond family, attitude of service
 - Effective resource management: Competent use of money, time, etc
 - Coherence: Optimism and self reliance
- Intervention strategies:
 - Provide opportunity for and encourage contributions
 - Enhance decision making skills → ↑feeling of control
 - Encourage and give positive feedback
 - Develop self discipline: involve child in setting the rules and consequences

Toddler Behaviour

- Approach:
 - History, including:
 - Antecedents, behaviours and consequences
 - Social context
 - Collaborative history if necessary
 - Psychiatric history from mother (is the child the problem?)
 - PMH: A B F W I M P S
 - Exam: especially developmental
 - Education
- Most difficult behaviour does not indicate a serious disturbance. Indicators of serious disturbance include:
 - Deliberate self harm or messing
 - Wandering off
 - Running away
 - Age inappropriate sexual behaviour
- Developmental sequence of everyday habits:
 - Feeding
 - Sleeping
 - Eating
 - Toilet

- Going to bed and getting up
- Dressing and undressing
- Washing and cleaning teeth
- Aim is to achieve regular habits and routines:
 - To start with need to insist on regular routine and time schedule. Once achieved can be more flexible
 - Failure to achieve routine: daily hassle and distress
 - Regular routines → ↑security of child, ↓argument with parents
- Factors which ↓behaviour problems:
 - Routine and regularity
 - Clear limit setting
 - Unconditional love and affection
 - High level of supervision
 - Consistent care and protection
 - Age appropriate disciplines and rewards
- Tantrums:
 - Want their way. Purpose of tantrum is to get their way. Giving in reinforces the behaviour
 - Must be consistent. If you say no, will have to stick with it ⇒ choose your battles
 - Options for managing a tantrum (see Behaviour Management, page 14)
 - Ignore it: eg leave the room
 - Time out
 - Distract
 - Avoid problem areas (eg supermarkets)
 - Things will get worse before they get better. Once they realise the boundaries are consistent they will stop testing them
- Sleep Management: Sleep Management, page 41
- Other points:
 - Check parent's are not expecting too much of the child (eg 3 year old boy not wetting at night)
 - Keep no for important things
 - All parents make mistakes

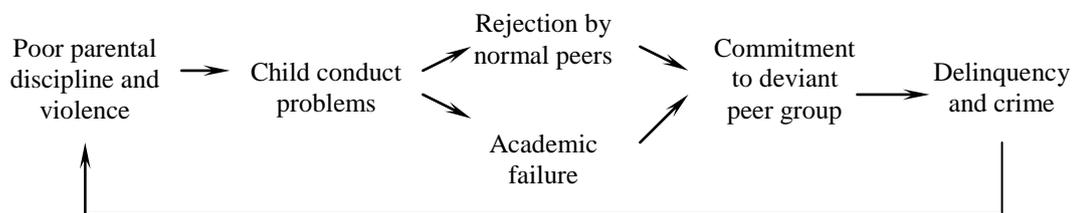
Problems at School

- For ADHD see Attention Deficit/Hyperactivity Disorder (ADHD), page 171
- Symptoms:
 - Normal IQ, no disabilities, but fail to develop academic potential
 - Difficulty with peer relationships, loners, act out, difficult behaviour
 - Develop associated problems: psycho-somatic, ↓self-esteem
- Usually multifactorial:
 - Constitutional factors: May have subtle defects in:
 - Receptive or expressive language
 - Auditory sequencing: can't remember verbal sequence (eg instructions). Can have a pervasive effect on schooling
 - Visual sequencing: difficulty reading/spelling
 - Motor problems (eg clumsy)
 - ↓Attention (may be secondary to the above)
 - Health, hearing, vision, etc
 - Environment: cultural, socio-economic status, family disruption, nutrition, etc
 - School related factors:
 - School factors associated with ↑antisocial behaviour: poor morale, high turnover, inconsistent standards, undervaluing children's work, bullying
 - Kids spend 15,000 hours at school – so can have a big impact (just as family does)
- Assessment:
 - History: parents
 - Information from teachers
 - Physical and neuro exam
 - Sensory exam: vision and hearing
 - Neuro-developmental, educational and psychological assessment
- For School refusal, see Separation Anxiety Disorder, page 173

- Management of Truancy:
 - Educational programme appropriate for the child's needs
 - Monitoring child through the day
 - Assist with the learning process

Child in Trouble with the Authorities

- History:
 - Interview child and caregiver separately
 - Presenting complaint
 - History of presenting complaint:
 - Describe behaviour: Antecedents, Behaviours, Consequences
 - Social Context in which behaviour occurs: Relationships within family, school, and peers. Physical or sexual abuse
 - Collaborative history: parents, teachers, sports coaches – this is important
 - Psychiatric history: look for depression, anxiety, attentional problems
 - Formal assessment of learning if academic problems
- Exam: especially dysmorphisms, stature, neurocutaneous lesions, observations of reading, writing and relationship with parents, vision and hearing
- Development of antisocial behaviour:



- Possible differentials:
 - Attachment disorder
 - Developmental delay
 - Behaviour conduct disorder
 - Psychiatric illness: Depression, ADHD, PTSD (may appear to be daydreaming)
 - School refusal/Truancy
 - Neglect or other abuse
 - Domestic violence
- Management plan:
 - Keep in him school if at all possible: prognosis plummets if expelled or regularly truant
 - Ensure a thorough developmental assessment
 - Referral for psych assessment and counselling
 - Management: accentuate the positive, minimise the negative
 - Referral to other services

Attachment Disorder

- See also Anxiety Disorders, page 172
- Attachment:
 - Starts in utero and is an ongoing process
 - Securely attached infants:
 - Are able to seek and obtain comfort from familiar caretakers
 - Are willing to explore and master their environment
 - Insecurely attached infants (eg due to long separation from parents and multiple carer-givers in hospital) appear:
 - Anxious: clingy without obvious stress
 - Avoidant: angry, distrustful of parents, won't be comforted after brief separations
 - Indiscriminately affectionate: won't show preference for parents
- Concepts:
 - Separation: the process by which a child develops an identity separate from their parents. Promoted by secure attachment. At risk when the parents perceive the child is 'vulnerable'
 - Autonomy: Development of independence (→ social competence)

- Mastery: increasing sense of competence over the physical environment
- Together autonomy and mastery lead to an internal locus of control. Struggles for autonomy and mastery produce normal tantrums
- Types of Attachment Disorder:
 - Disinhibited type: will go to anyone. No stranger awareness and constant, insatiable need for attention. Likely to be due to neglect. Also see it in chronic hospitalisation
 - Withdrawn: frozen watchfulness, fearful. Likely to be due to abuse
- Test by observing child when parent leaves (separation), when a stranger comes in, and when parent returns (reunion)

Domestic Violence

- Has significant health consequences: injury, psychiatric, chronic pain, drug and alcohol abuse
- Is common (some studies report up to 20% of women being hit in the last year), but often missed by doctors
- Domestic violence starts with a cycle of increasing control and disempowerment. Violence is used to reinforce this
- Screening questions:
 - ‘I have seen many people who come to see me with problems like yours. In my experience, many of these women are being hurt in some way by their partner. Is that happening to you?’
 - ‘A lot of tension and violence can be due to relationships within the family – often with a partner. Is your partner being violent toward you?’
- Management:
 - Ensure mum and the children are safe. If not, refer to police/CYFS
 - Refer to Women’s refuge – be aware of the local services available
 - Educate: eg the cycle of violence, it won’t stop without help
 - Avoid victim blaming (‘it’s not your fault’)
 - Take careful notes (explain to the women why you are going this)
 - Display information in your waiting room – signals a willingness to discuss it

When Parents Separate

- Responses to parent’s separation – all signs of distress:
 - Withdrawn
 - Clingy
 - Regression
 - Difficult behaviour
- Helping the child:
 - Accept the separation – then the child will too
 - Make sure the child knows you love them
 - Avoid conflict in front of the child
 - Allow them to express their feelings
 - Rely on other adults not the child for support
 - Tell the kids they’re not to blame
- Things to avoid:
 - Don’t abuse their loyalty and trust
 - Don’t use them as messengers
 - Don’t use them to spy on other parent
 - Don’t continue to be angry at partner in front of them
 - Don’t let outings/gifts take the place of normal parenting
 - Don’t force kids to take sides
 - Don’t force a clash of loyalties

Preparing a child for surgery

- Talk to the child about what is going to happen and why. Read books about hospital
- Reassure your child that you will be there too
- Answer your child’s questions
- Use simple terms that the child can understand
- Take a favourite toy. It can have bandages too
- Be honest without scaring: ‘It will hurt for a bit – but we’ll try and make it better’

- Tell the child he/she will be going home with you when it's finished
- Don't be surprised if the child gets angry with you: this is normal

The Dying Child

- Anticipated: preterm infants, congenital abnormalities, metabolic disease, etc
- Unanticipated: often older, trauma, SIDS
- Issues:
 - Relief of child's pain → pain management, including neonates
 - Clear and simple information: check their understanding, explain prognosis, explain disease process, avoid information overload or jargon, respect silence
 - Decisions about when to withdraw treatment
 - Before death: time and space, preparation of child
 - After death: viewing the body, coroner, funeral options, surviving siblings, other families, availability of health care team

Children's Grief

- It is not possible not to communicate to children (ie not telling them is not an option)
- Help should start at the time of diagnosis
- Talk about what won't change as a result of the illness
- Maintain things that are important in a child's life (e.g. routines)
- Talk about practical concerns
- Provide extra stability, order, routine and physical affection
- They need to know who will take care of them if key people leave or die
- Offer reassurance
- Children often assume responsibility for what has happened and feel very guilty
- Offer clear, simple, truthful information: repeat, repeat, repeat
- Don't use euphemisms (e.g. asleep – explain death, body stops working)

Signals for attention from a grieving child

- Marked change in behaviour: illegal behaviour, persistent aggression (> 6 months), tantrums, withdrawal, drug abuse
- Inability to cope with problems and daily activities
- Many complaints of physical ailments
- Persistent depressions, panic attacks
- Change in school performance
- Fearfulness for self, or for loved ones

Helping Families

- Listen effectively
- Foster communication
- Engage siblings
- Check social supports
- Address symptoms
- Provide constant factual data
- Help build positive memories
- Don't take offence

Autopsy of a Child

- Family concerns:
 - What is an autopsy – like a major operation
 - Will my child be cut – yes
 - When will it be done – soon
 - How long will it take – max 3 hours
 - What will my baby look like afterwards – won't see chest incision if dressed. Vertical cut down back of head
 - Can I take my baby home afterwards – yes
- Autopsy may provide:
 - A cause of death – but may take time

- Identify unacceptable iatrogenic lesions
- Quality control for a neonatal unit
- Assist medical knowledge
- Information that may help other babies
- Common reasons for refusal:
 - Concerns about disfigurement and further suffering
 - Lack of information
 - Objections from family members
 - Religious beliefs
 - Interference with funeral arrangements
- Must refer to the coroner:
 - Where death certificates cannot be signed
 - Thought to be related to an invasive procedure
 - ?Birth asphyxia
 - Deaths thought to be related to an instrumental delivery

Child Development

- Represents the interaction of heredity and the environment:
 - Heredity: potential of the child
 - Environment: extent to which potential is achieved. Requires:
 - Physical needs: warmth, clothing, shelter, food, health, activity with rest
 - Psychological needs: security, personal identity, self-respect, independence, opportunity to learn, play, affection and care
- Areas of child development:
 - Gross motor
 - Fine motor
 - Language (expressive, receptive, non-verbal)
 - Social (interaction, play, self-care)
 - Cognitive: all of the above
- Requirements for development (need all of them):
 - Hardware (neurons, muscles, etc)
 - Motivation (often driven by frustration – a child can't do what it wants to)
 - Nurturing environment
- Types of assessment:
 - Developmental screening: point in time snapshot
 - Developmental surveillance: following over time
 - A formal assessment will yield a Developmental Quotient. $< 100 \Rightarrow$ delay. $> 100 \Rightarrow$ advanced.

Developmental assessment

- Indirect assessment of the acquisition of life skills
- Establish rapport: use names a lot, 'thanks for coming', etc → more valid assessment
- **History:**
 - Current development and time course of development
 - Order of questions should be:
 - When asking about milestones, start with things he is likely to be able to do and work up. Get better rapport than starting at the upper limit and working down
 - Hearing: What things can he hear?
 - Have you been concerned about his hearing?
 - What makes you confident of that?
 - Vision: What small things does he see?
 - Have you been concerned?
 - What makes you confident of that?
 - Gross motor: roll, sit, crawl, pull to stand, walk, run, scoot, pedal (progression: head → trunk → limbs)
 - Fine motor: pincer, feeding self, spoon, drawing, blocks
 - Expressive language: coo, babble, words with meaning, combinations (most common area of delay – usually focal not global)
 - Receptive language: Responds to familiar voice, to own name, one or two step instruction, knows name, gender, address, prepositions, pronouns
 - Social: smile responsively, laugh, stranger aware, play with peers, name friend
 - Self care: manage cup, spoon, undress, toilet, dress, laces
 - Get history of influences on development:
 - Miscarriages, still births
 - Pregnancy: toxins, alcohol, infections
 - Birth: APGAR (usually means brain was vulnerable before birth), gestation, birthweight
 - Neonatal congenital abnormalities, feeding, jaundice, infections
 - Early milestones (smiling, sitting, walking, first words)
 - Illness (eg CF, heart/renal disease, epilepsy)
 - Hearing (→speech delay), vision (→ good verbal, poor motor)
 - Nutrition, constipation (especially if mobility problems)

- Current development, especially social, self-care
- Behaviour problems (sleep, tantrums)
- Family stress
- Family history, especially of development
- History from other sources (eg kindy teacher)
- Review previous rate of development: may get slowing before loss
- Past Medical History: ABFWIMPS
- **Observation:** Look systematically across each of the 6 areas. Use toys as tools.
- **Examination:**
 - On lap first (stranger shyness from 8 months)
 - Dysmorphisms: eyes, head shape, body proportions
 - Height, weight, head circumference – plot them
 - Vision (do first, affects motor): following, hundreds and thousands
 - Localise to noise (do before language): if concerned then formal testing
 - For each of gross motor, fine motor, expressive and receptive language, social and self care on the table below:
 - Ask open-ended questions to establish the floor (eg I notice he's walking, what other clever things is he doing)
 - Then use closed questions to establish a ceiling (eg can he walk backwards, throw over arm)
 - Then summarise: So he can but is not yet Have I got that right?... Therefore he is at age X for that domain
 - Summary: age for each domain is X, Y, X. Therefore, overall, he's developmentally around age [Average for X, Y, Z]
 - Other:
 - Skin pigmentation (eg tuberous sclerosis – seen under Woods lamp)
 - Ears, eyes, heart, abdomen, puberty
 - Neurologic exam
 - Relationship with parents
- **Plan:** for areas of weakness
 - If significant delay then early intervention
 - If some delay then anticipatory guidance – 'what could you do to help' – use Knowledge, attitudes/fears, practices, barriers framework
 - Always pitch safety advice at the level of gross motor skills

Development Chart: normal development from 0-60 months

- Ref: Dr Russell Wills
- Red flags:
 - Not smiling by 2 months
 - No eye contact by 3 months
 - Not reaching for objects by 5 months
 - Not sitting unaided by 9 months
 - Not walking unaided by 18 months
 - Not using words by 18 months
 - No 2 – 3 word sentences by 30/12 months

	Gross Motor	Fine motor-adaptive	Expressive language	Receptive Language	Personal-Social/Play	Self Care
6 wk	Clear face prone	Follow 180 horizontal Follow O vertically	Cry, coo	Quiets to voice	Smiles spontaneously	
3 m	Up on elbows. No head lag on pulling up	Grasp Retain 1 block	Reciprocal vocalisation, laugh	Turn head to voice Localise bell/keys (horiz)	Reciprocal smiles	
6 m	Up on hands Sit supported	Take 2 blocks Transfer hand to hand	Consonants Mono-syllabic babble	Localise bell/keys (vert)	Lifts arms for pick up	
9 m	Sit stable Pull to stand Crawl	Object permanence: Take 3 blocks 1 at a time and hold onto them Bang, index point	Vocalise to communicate Poly-syllabic babble Jargon	Understands no, ta Localise bell/keys (diag)	Peekaboo	Cooperate with dressing
12 m	Move around furniture	Good pincer	Few words with meaning	Shake head for no Copy bye bye	Push toy car Clap hands	Independent with cup
15 m	Walk independent	Stack 2 blocks Mark paper with pencil, fist grip	Several words	Understands object names Wave bye bye on request Understands pointing	Push large wheeled toy Casting prom.	Remove item Indep with spoon
18 m	Walk backward Kick Throw over arm	Stack 3 blocks Vigorous straight scribble 3 shape board	Prom. jargon, two wants Many common objects Family names, own name	Few body parts Hands familiar named object on request	Symbolic play single step 21 mo: likes books Play alone	Remove hat, shoes, socks
2 yrs	Run Throw ball in bin Seat self at table	Imitate vertical line, then horizontal; dagger grip for pen Stack 6, train 3 3 shape board rotated	Combinations; Echolalia; Songs/rhymes 30 mo: Questions: what, who? 3-5 word comb; I, we, you Verbs: eat, kick, gone	Prepositions: on, in 2 step commands: shut door Object uses Several body parts; 30 mo: fetch	Imitative play single step 28 mo: ringaringarosy 30 mo: imit sequence	Indicate wet/dirty nappies Dress some
3 yrs	Walking: tandem, heels, toes Jump feet together Pedal; Up stairs adult	Stack 9, 3 stairs/bridge Imitate circle, cross, tripod grip 42 mo. Count bricks	Echolalia resolved Full name Plurals	Prepositions: under, up, down Mime complex gestures 42 mo: fetch several items	Imagine play seq Gender Name friend Share, turns	T-shirt Shoes and socks
4 yrs	Hop 3 steps/ Jump off 2 Catch big ball Down stairs like adult	5 brick gate 4 part man, ladder, square. Cut with scissors; Higher, longer	Intelligible to strangers Tenses Constant Qs: where, why...	Prepositions: between Opposites: big/little What would you do if?; Comparisons	Gives age Co-op play, hide n seek, snap Dressups, pretend	Undress indep.
5 yrs	Run up stairs Gallop Bounce and catch	6 part man, triangle, pencil grip 6 brick stair, castle Count 15 bricks	Grammatical sentences – conj. & except. Ask meanings of words	What things are made of: wood, metal Enjoy jokes, riddles	Knows address Names best friend	Dress indep.

- Older kids:
 - Gross motor: bike (can ride without trainer wheels at 5), sport (running, kicking), clumsiness
 - Fine motor: computer, play station
 - Cognitive: don't ask if does OK at school – everyone does OK these days! Instead, does he do age appropriate work, need extra tuition, etc

Cognitive Development

- Overall process:
 - Autonomy: dependent on parents → peers → independent
 - Abstract thinking (what if?): concrete → mature
 - Future consequences of present actions
 - Gratification: immediate → delayed
 - Satisfaction with body image
 - Black and white → comfort with shades of grey
- Infancy (birth – 2 years): Developmental issues:
 - Later develop goal directed activity
 - Learn to distinguish between self and surroundings
 - Develop object permanence
 - Need secure attachment relationship with parents
 - Separation, individuation in toddler years
 - At 2: trial and error problem solving, planned and purposeful play but limited content, egocentric, parallel play
- Preoperational (3 – 5 years):
 - Egocentric world view (I made it happen, so it's my fault)
 - Use of magical thinking, difficulty distinguishing real from symbolic (if I wish it, it will come true)
 - Trial and error problem solving only
 - One aspect of a problem at a time
 - Cannot order a series of events
 - Cause and effect thinking: I did X, then Y happened, therefore $X \rightarrow Y$
 - Imaginative play
 - Gradually move from parallel play to interactive play with peers
 - Separation and autonomy
 - At 5: symbolic thought (imagination), classify by colour/shape, curiosity, magical thinking, social values, rules internalised but fixed, turn-taking, cooperative play, other's perspective, increasing independence
- Concrete Operational (6 - 10 years):
 - Black and white thinking, right and wrong
 - Capable of simple logic and problem solving
 - Can order things in a chronological sequence
 - May have difficulties with multiple perspectives
 - Peer relationships increasingly important
 - Sharing games, competition
 - Analogy, metaphor, figures of speech being
 - Able to concentrate for longer, delay gratification, predict personal and social consequences of actions, plan ahead
- Formal Operations (10 – 13 years):
 - Better memory, concentration, forward planning
 - Social skills refined
 - Still concrete and literal (black/white, good/bad, right/wrong)
 - Limited abstraction: eg what if I didn't do this? (Contrary-to-fact abstraction)
 - Dramatic changes to body → constant comparisons and normal anxieties
 - Need to conform with peer norms
 - Difficult to take others perspective's
 - Difficult to understand complexity
 - Difficult to apply rules to own situation
 - Lack future orientation/forward thinking
 - Clear consequences
- Middle Adolescence (14 – 16 years):

- Developing abstract and complex thought
- Beginning to see other's perspectives, starting to cope with shades of grey
- Increased self consciousness
- Easily swayed – not certain of own view
- Still difficult to integrate conflicting ideas
- Narcissistic (feels good/what I want → therefore its right → impulsiveness)
- Less need to conform to peer norms, try alternative beliefs and philosophies
- Need limits to be secure, limit testing
- Late adolescence
 - Adult memory and concentration
 - Mature abstractions, problem-solving, self reflection and long range planning
 - Weigh up multiple information
 - See multiple meanings, complex relationships, different points of view, tolerant of shades of grey
 - Able to think hypothetically and plan for possible events
 - Remains more difficult to use new abilities in challenging situations
 - Autonomous: able to leave home and return for counsel, rely on own opinion

Developmental Delay

- Constant slow development leads to widening gap
- Investigations: hearing, vision, chromosomes, DNA screen (eg Fragile X, Angelman, Prader-Willi), thyroid, metabolic, mucopolysaccharide screen, CK (Duchenne's), brain imaging, EEG
- Type of Diagnosis:
 - Functional Diagnosis:
 - Mobility, communication, learning, self-care, socialising, etc
 - What does the child need to achieve age-appropriate function
 - Pattern diagnosis:
 - Autism: see Autism, page 28
 - Cerebral palsy: see page 104
 - Other syndromes
 - **Biological diagnoses:** DNA disorders, brain injury
- IQ scores:
 - < 20 profound intellectual disability
 - 20 – 35 severe
 - 35 – 50 moderate
 - 50 – 70 mild
 - 70 – 85 borderline
 - Definite or highly probable cause in majority < 50. Cause in about half < 70
- Management:
 - Objectives:
 - Maximising function
 - Preventing and treating secondary problems
 - Supporting carers
 - Referral: paediatrician, geneticist, psychologist (eg cognitive testing), SLT (speech, swallowing, play), physiotherapist (gross motor problems), OT (fine motor, self care, aids and equipment), early intervention groups, VNDD (Visiting Neurodevelopmental therapist), support groups
 - Medical assessment of a diagnosed, disabled child
 - Always consider new illnesses
 - Look for syndrome specific health problems
 - Feeding difficulties, nutrition
 - Constipation
 - Medication
 - Carer Stress
 - Access to services and allowances

Tamariki Ora (Well Child) National Schedule

- Covers:
 - Health education and promotion
 - Health protection and clinical assessment

- Family/whanau care and support
- Health education/promotion topics to cover at appropriate stages
- Prevention:
 - Types:
 - Primary: shifting the whole population curve → improves the overall standard
 - Secondary: identifying risk factors → early or targeted intervention
 - Tertiary: minimising impact of established disease
 - Benefits of prevention: ↓adult sequelae: injury, child abuse, delinquency and arrest rates
- PPV of parental concerns about delay is about 80–90%. Must act or refer on parental concern

Causes of Developmental Delay

- Causes of abnormal development:
 - Environmental
 - Genetic (eg chromosomal, metabolic)
 - Disability (vision, hearing, motor)
 - Brain injury (hypoxia, trauma, toxins, infection, prenatal & post-natal)
 - Illness, nutrition
 - Unknown

Hearing

- See Hearing Loss in Hearing Loss, page 110
- 1 in 500 has significant permanent hearing loss → receptive and/or expressive language delay
- All infants babble, even hearing impaired
- Suspect deafness when:
 - Parental concern
 - At risk babies (should be routinely screened):
 - Family history
 - Inter-uterine infection: rubella, CMV
 - Defects of ENT: cleft palate, external ear
 - Low birth weight
 - Neonatal distress
 - Poor response to sound
 - Not using words by 15 months
 - General developmental delay
 - Poor speech, comprehension and hearing failure
 - Following brain trauma, infection, neurotoxic drugs
 - Recurrent or persistent ear infections
- Normal development: See Development Chart: normal development from 0-60 months, page 22
- History:
 - Can he hear – how do you know?
 - Previous development: first word, use of consonants, etc. Check Well Child Book
 - Ear infections
 - Antenatal: rubella, prematurity, jaundice, drugs
 - Family history of hearing problems, developmental delay, neuro problems
- Exam:
 - Dysmorphic features: cleft palate, external ear, skin, heart murmurs, liver enlargement, normal genitalia
 - Basic neuro exam, gait, symmetry of movement (including face), eye movement
- Investigations:
 - Tympanogram
 - Don't do distraction testing – hard unless you're well trained. Send straight for audiology
 - Others depending on clinical findings: eg if regression then EEG, brain scan, check stressors, chromosome problems, CK for Duchenne's
- Differential of language delay:
 - End of normal range
 - Deafness
 - Isolated language delay (usually expressive more delayed than receptive – but not necessarily)

- General delay or mild intellectual handicap → formal cognitive testing
- Autism
- Epilepsy: absence seizures – especially if fluctuates or regressive
- Possibly poor environment with little stimulation – but would also expect ↓socialisation and ↓self care
- Congenital problems: cleft palate, macroglossia (eg Down's)
- Rare isolated CNS or motor problems
- Management: Speech language therapist, early intervention service, multidisciplinary team if problems over other domains. GP to support and co-ordinate, anticipate problems – especially at transitions (eg school, moving) and checking for comorbidity (eg behavioural problems, ↓self esteem)
- Prognosis: Good if early intervention – but maybe problems with higher language function (eg essay writing)

Down Syndrome

- Trisomy 21: 47XY + 21
 - Accounts for 95% of presentations of Down Syndrome. Usually (80%) non-disjunction at first meiotic division
 - 5% have different karyotypes:
 - Mosaic Down: 3 %
 - Robertsonian translocation t14:21: 4.8%
- Epidemiology:
 - Overall incidence is 1 in 700
 - At least 20% still born
 - Incidence increases with ↑maternal age: at 16/40 gestation, 1 in 300 at 35 years, 1 in 22 at 45 years
 - Accounts for 25% of children with IQ < 50
 - ¼ of all chromosomal abnormalities. Chromosomal anomalies represent 15% of congenital anomalies
- Risk:

Maternal age at birth	Down in live births
25 - 29	1:1100
30	1: 900
35	1:350
37	1:200
40	1:100
43	1:50
45 and over	1:25

- Neonatal Screening:
 - Only 30% of children with Down are born to women over 35. Widespread screening of those > 35 will have only minor increase in detection rate
 - Screening with the triple test will increase detection, but at the expense of significantly higher rates of invasive testing as there is a high false positive given the low incidence in younger women
- Neonatal signs:
 - Hypotonia
 - ↓Moro reflex
 - Joint hyper-extensibility
 - Excess skin at the back of the neck
 - Flat facial profile
 - Misshapen low set ears
 - Protruding tongue
 - Blunt inner eye angle
 - Single palmer crease in 50%
 - Clinodactyly (incurving) of little fingers in 50%
 - Big 'saddle' gap between big and 2nd toe
- Complications:
 - IQ generally from 45 – 55
 - Congenital heart malformations in ~50%: VSD, ASD, patent ductus
 - Susceptible to respiratory infections
 - Duodenal atresia
 - Also cataracts (2%), epilepsy (10%), hypothyroidism (3%), acute leukaemia (1%)

- Development:
 - Most will walk and develop simple language
 - Puberty is often delayed and incomplete
 - Average adult height is 150 cm
 - Pre-senile dementia (similar to Alzheimer's disease) supervenes after age 40
 - 8% live past 40 years

Autism

- Onset before age 3 of:
 - ↓Ability to form social relationships: slow to smile, don't enjoy being cuddled, indifferent eye contact
 - ↓Use of both verbal and non-verbal communication: slow onset, echolalia, no use of gesture, poor pragmatics (eg turn taking and eye contact in conversation). If it was isolated language delay you would expect compensation in other areas (eg social) but here that's affected as well
 - Restrictive and repetitive behavioural repertoire – dislike change
- May start from birth, or regress after normal development
- Other behavioural problems: outbursts, sleep problems, distractibility, poor toileting
- Rare: 2 – 4/10,000. Boys = 3 * girls
- 75% show some degree of general intellectual impairment
- Asperger's Syndrome:
 - Symptoms overlap with autism
 - Social interaction and behavioural problems similar to autism but not associated with significant language or intellectual delay

Effect of Chronic Disease on Development

- See also Chronic illness and disability in Adolescents, page 181
- 10 – 15 % of children have some chronic health condition. 1 – 2% are severe enough to interfere with their ability to take part in normal activities
- Chronic illness can effect development by:
 - Direct effect: eg deafness → language delay
 - Effect of treatment: eg neuro-radiation
 - Indirect effect: reduced energy in cystic fibrosis
 - Social environment: sense of differentness → withdrawal or bullying
 - Transaction: impact on parents (eg maternal depression) → affects child's adaptation
- May lead to failure to develop independence (self control) and competence (control over their environment), leading to self-doubt or indecision.
- Issues to consider:
 - **Burden of care:** don't give them more helpful ideas if they're already over-stretched!
 - **Unpredictable future:** Clear idea for the parent and child of what the future might hold
 - **Social/school:** Watch for 'vulnerable child syndrome', bullying, and access issues.
 - **Cost:** check relevant benefits received
 - **Respite care:** do parents need a break? Deal with feelings of guilt and indispensability
 - **Activities of daily living:** the daily routine will be revealing
 - **Multiple professions:** check these are co-ordinated and organised around the family's needs
 - **Psychological:** consider impaired attachment, depression, stress, family dysfunction
- Also ensure:
 - Information for child and parents
 - Access to services
 - Access to consumer groups
 - Equipment and transport needs
 - Environmental modification
 - Vocational training for an adolescent

Infants

- Effect on parents of congenital malformation:
 - Shock, disbelief, upset, problem solving processes slowed
 - Adaptation over time

- Grief reaction similar to death of a child (must mourn the loss of a 'normal' child) – but parent must also attach to the living child
- Management:
 - Support good bond-enhancing practices before and immediately after birth:
 - Normal preparation for birth (learn about routines, processes, options, etc)
 - Time to establish rapport with paediatrician and visit NICU
 - Long periods together in first few days and breast-feed if possible. Is any separation really necessary?
 - Avoid criticism – a very sensitive time
 - Watch for signs poor attachment. See Attachment Disorder, page 17
 - For the toddler:
 - Watch for 'vulnerable child syndrome': continued parental concern after child has recovered → adverse affects on child. Problem is parents' expectations, not attachment. More complicated when some ongoing vigilance is required
 - Support appropriate attitudes and plans
 - Mobilise family support
 - Remain optimistic
 - If in hospital, use separations to reinforce that parents will return. Limited number and consistency in nursing staff

Pre-schooler

- Social and emotional development may be limited through lack of opportunity to achieve goals in play and by limited peer interactions
- Management:
 - Refer for early intervention, especially low socio-economic and disabled children
 - Promote normal development: separation, appropriate discipline
 - In hospital: encourage rooming in, maximum contact with families
 - Warn parents to anticipate behavioural problems especially if hospitalisation is prolonged or frequent

Head injured child

- Initial crisis: grieving put on hold, waiting to see if things improve, child still looks the same, swinging between hope, despair and disbelief
- Restructuring:
 - Reassign tasks in the family
 - Move out of crisis reorganisation into long term reorganisation
 - Inclusion of outside help into family
 - Appropriate time for husband/wife/other children
 - Time for self
- Grieving:
 - Allow for grief and acknowledge the loss
 - Avoid dichotomy of one person (eg mother) taking hope position and others despair
 - Promote openness. Devastation of silence
 - Denial can also be a coping mechanism
- Develop an acceptance of a new identity through the crisis:
 - Seeing how the child is different
 - Finding positives in this new identity and helping the family value these
 - Achieve a sense of movement through the crisis. Mark positives and achievements of the family
- Encouraging compliance:
 - For the highly compliant: teaching, directions
 - For the non-compliant (those who respond 'yes – but...'): general indirect messages, metaphor/story telling
- Subsequent learning disabilities: may have problems with learning from then on – but may not show up till those skills are needed (eg trouble reading when they start school)

Learning Disability

- History:
 - Start with things he is likely to be able to do and work up

- Questions over traditional domains for learning:
 - Reading: ‘what is she reading now’, ‘can she read three letter words’
 - Spelling
 - Numeracy
 - Writing
 - Drawing, art, craft
 - Social skills
- Strengths
- Collaborative history:
 - Previous assessments, IQ tests
 - Talk to the teacher
- Comorbidity screen:
 - Is the norm in developmental paediatrics
 - Can be:
 - Primary: eg biological morbidity such as learning and co-ordination difficulties, ADHD and clumsiness
 - Secondary: eg acquired psychological and behavioural problems such as loss of self-esteem, non-compliance, etc
 - Differential:
 - Behaviour: aggression, attention seeking, school refusal
 - Mood, anxiety, attention
 - Relationship with peers, teasing, bullying
 - Family issues – get good social history
 - If adolescent then HEADDSS assessment (See page 178)
 - School factors: teacher skills, interest/ability to manage the child’s needs, available skills
 - Parental insight: are they helping or hindering
 - Use questionnaires: eg Child Behaviour Checklist (screen for anxiety, depression, etc) or Connor’s (specific for ADHD) to provide diagnostic information and provide a pre-treatment baseline
- Exam:
 - Screen for gross and fine motor delay
 - Refer for vision and hearing tests
- Possible differentials:
 - Hearing and vision
 - Medical: hypothyroid
 - Intellectual disability
 - Specific learning disabilities
 - Head Injury
 - Psycho-social: Abuse, stress, etc
 - Psychological: depression, anxiety, ADHD
- Principles for management:
 - Review and follow-up (eg 3 monthly), especially at times of transition (eg changing schools)
 - Multidisciplinary approach: OT, Physiotherapist, SLT, VNDT, Educational Psychologist
 - Excellent communication between professionals
 - Helping parents to create realistic goals
 - Dealing with normal parent grief
- Strategies for management:
 - Demystify: Explain strengths and weaknesses to the child, parents and teacher. Removes guilt, pejorative labels (eg lazy), gives optimism
 - Bypass strategies: adjust rate, volume, complexity, format or use devices to make the task easier
 - Remediation of skills: focus on study skills, organisation, use strengths to remediate weaknesses
 - Developmental therapies: Eg speech therapy, gross and fine motor, etc. More effective when skill deficits reflect lack of opportunity, and when instituted earlier
 - Modify the curriculum: Eg drop subjects they’re not succeeding in
 - Strengthen strengths: sport, art, mechanics, etc
 - Individual/family counselling: especially with behaviour management, family dysfunction
 - Advocacy
 - Medication
 - Longitudinal case management

- Check whether parents get the child disability allowance. Can get a needs assessment done for respite care, home help, etc

Child Development Team

- For children with:
 - An identified disability
 - Developmental delay
 - At risk of developmental delay/difficulties
- Involves multi-disciplinary assessment and intervention
- Social Worker:
 - Conducts individual, marital and family social assessments
 - Liaises with other community services (CYFS, WINZ, etc) and facilitates brokerage of services
 - Provides emotional support
 - Teaching parenting skills
 - Advocacy
- Psychologist:
 - Assessment: neuropsychological, development, behaviour/emotion, family function
 - Intervention: For individual, skills for parents, family relationships
- Physiotherapist:
 - Functional assessment of gross motor skills: delay, abnormal muscle tone, loss of range of movement, gait abnormalities, mobility, co-ordination
 - Therapy (including hydrotherapy) and home/school exercise programmes
 - Assessing for standing frames, walking aides and wheelchairs (with OT)
- Occupational therapist:
 - Assessment of fine-motor skills (use of hands):
 - Tool manipulation: grasp and grip, bilateral hand use, release, eye-hand co-ordination (eg stacking)
 - Pre-writing: lines, circles, picture of a person
 - Handwriting: grip, control, sizing, closure, hand dominance, speed
 - Cutting with scissors: accuracy and co-ordination
 - Assessment of self-care skills:
 - Undress/dress, buttons, zips, shoelaces, etc
 - Feed themselves
 - Sit unsupported in the bath
 - Toileting, including sitting on toilet, pulling pants down, wiping
 - Clean teeth, brush hair, wipe face
 - Assess for equipment to help with the above
- Speech language therapist: assesses and assists with:
 - Receptive language: understanding
 - Expressive language: including gesture and facial expression
 - Phonology: sound system
 - Pragmatics: social rules – sharing, turn taking, eye contact (biggest problem in autism)
 - Feeding: transition from tube to oral feeding, behavioural feeding issues
 - Voice: vocal nodules due to abuse (eg screaming)
 - Fluency: stammering
- Visiting Neurodevelopmental Therapist:
 - Home based assessment
 - Family support
 - Liaison with other health professionals and community agencies
 - Assessment for equipment
- Developmental paediatrician:
 - Developmental assessments/examination for suspected delay
 - Investigates cause
 - Review of developmental progress

Growth

- Growth velocity = change in height over time. Declines till about 4, levels out, spike at puberty then zero
- Factors influencing growth:
 - Genetic potential
 - Psychosocial factors (eg psychosocial dwarfism)
 - Nutrition (including in utero): adequate calories, balance of nutrition
 - Diseases in major systems: uses energy (eg ↑ respiratory effort) and nutritional effects (eg GI)
 - Hormones and Growth factors
- Measurement:
 - Height:
 - Method: Use stadiometer, fixed to wall, feet together, knees straight, lift mastoid processes
 - Accuracy and reliability:
 - SD of a single measurement ~ 0.25 cm. In a 5 year old this can cause a range in growth velocity from 10th to 50th centile
 - Taller in morning than at night
 - Minimising error: Same measurer, calibrate regularly, careful measurement, don't look at last measurement, measure at beginning and at end of exam
 - Plot parents height (diff between average male and female = 12.6 cm)
 - Check for disproportionate growth (eg problem with long bones) by sitting on a box of a known height and measuring seated. Subtract standing from sitting to get sub-ischial leg length. Measure span to confirm abnormalities of long bones
 - At no stage of childhood should a child grow less than 5 cm per year
 - Weight
 - Head circumference
 - Bone Age: Left hand x-ray

Short Stature

- Definition:
 - > 2 standard deviations below the mean = below 5th centile
 - Reduced growth velocity
- Exclude failure to thrive
- Growth pattern is more important than height
- Normal variants:
 - Familial (genetic) short stature
 - Constitutional delay of growth and development. Presents mid to late childhood

	Familial (genetic) short stature	Constitutional Delay
Height	< -2 SDs	< -2 SDs
Bone age (hand xray)	= Chronological	< Chronological. Delayed through childhood, accentuated when peers reach puberty
Puberty	Normal	Delayed
Growth velocity	Normal	Normal
Final height	Short	Normal/Tall (keep growing for longer)
Parents	Short	Normal
Family History	Positive (for short stature)	Positive (for constitutional)

- Pathological causes:
 - Systems: eg subclinical GI or renal disease (reflux, coeliac, malabsorption, CF, etc). Always check:
 - Coeliac disease – do screen
 - Ask about headaches (especially early morning): ?CNS tumour
 - Snoring: Obstructive sleep apnoea – as prevalent in kids as in adults but usually caused by large tonsils/adenoids
 - Psychosocial
 - Genes:
 - Turner syndrome: webbed neck, wide nipples, wide carrying angle
 - Skeletal dysplasia: eg achondroplasia

- Syndromes
- Hormones: Thyroid or GH deficiency, glucocorticoid excess
- Drugs: Steroids (eg high dose inhaled steroids)
- Assessment:
 - History:
 - Height: measured accurately and over time
 - Mid-parental height: assessment of genetic potential (adjusted so both parents are same sex as child. Male = female + 13 cm or average their centiles)
 - Family history: eg constitutional delay
 - Systems
 - Psychosocial. Check for teasing/bullying as result of being small. Only severe deprivation will cause growth delay.
 - Development
 - Examination:
 - Growth parameters
 - Dysmorphic features → ?syndrome
 - Proportions: limbs vs trunk, eg arm span vs height, or upper segment (head to pubic bone) vs lower segment (pubic bone to floor)
 - Neuro-cutaneous lesions
 - Blood pressure (?renal disease)
 - Fundi and visual fields (?pituitary tumour)
 - In a child < 3 look at buttocks thighs for nutritional status
 - Markers of specific deficiencies: anaemia (Fe), rickets, etc
 - General
 - Investigations:
 - Bone age: accurate to about 3 months
 - Specific depending on history/exam, eg renal → creatinine, coeliac → antibodies, inflammatory markers, FBC (?anaemia – FE deficient or chronic disease), ALP for Rickets, Thyroid Fn (TST and T4 to check both thyroid and pituitary)
 - Karyotype in girls
- Treatment:
 - Treat cause
 - Growth hormone:
 - Effective in GH deficiency and Turner's syndrome
 - May help in chronic renal failure, intrauterine growth retardation and severe idiopathic short stature
 - Androgens: consider in constitutional delay – won't influence final height but get there faster.

Tall Stature

- Arbitrary definition
- Associated stigma (females more often seek help)
- Causes:
 - Familial/genetic
 - Over-nutrition
 - Syndromes (eg XXY, Marfan's, Homocystinuria)
 - Precocious puberty (tall early, but stop growing → eventually short)
 - Growth hormone excess is extremely rare

Growing Pains *

- Occurs frequently: 15% of children with peak age of 11
- Diagnosis of exclusion – no organic pathology usually found. ?Child more vulnerable to pain and stress-induced exacerbations
- Occurs at least monthly for a three-month period. Between times the child is well
- Differential:
 - Orthopaedic disorders
 - Collagen vascular disease
 - Infection
 - Neoplastic disorders

- Management:
 - Reassure, even if you can't find a cause
 - Symptom diary (also check for psycho-social stressors)
 - Symptomatic relief

- Neonatal is < 4 weeks

Examination of the Newborn

History

- Maternal history:
 - General health and well-being: past medical history and social history (partner, planned pregnancy, etc)
 - Pregnancy: mediations, alcohol and other drugs, complications, infectious illness (toxoplasmosis, rubella, etc), EDD, scan findings, parental blood groups
 - Family history: perinatal deaths, paediatric deaths, congenital problems (especially congenital dislocated hip)
 - Delivery history: length of labour, infection, resuscitation, APGAR, any concerns
 - Post-natal history: feeding, colour changes (blue, jaundice), behaviour, stools, urine
 - Have you any concerns about your baby?

Examination

- Initial assessment immediately after birth to check adaptation to extra-uterine life (eg APGAR) and to look for major congenital anomalies, especially:
 - Dysmorphic features
 - Choanal atresia
 - Major limb defects
 - Spina bifida
 - Anal atresia
 - Genital abnormalities
 - Birth trauma: bruising, cephalhaematoma
- Examine on Resuscitate. Check all equipment carefully first.
- APGAR assessment – at one minute, then 5 minutes then every 5 minutes till a score of 10:
 - Heart rate: 2 for > 100, 1 for < 100, 0 for not present
 - Colour: 2 for pink, 1 for blue, 0 for pale
 - Respiration: 2 for regular or strong cry, 1 gasping intermittently (may be bad sign – secondary hypoxia), 0 for none. May slow due to maternal drugs (eg pethidine)
 - Tone: 2 for active movement, 1 for limb flexion
 - Response to stimuli: On suction, 2 for coughs well, 1 depressed
- Apnoea:
 - Primary Apnoea: pulse < 60 and cyanosis. Give O2 and wait a minute
 - Secondary Apnoea: pulse < 60, pallor and floppiness: suction, ventilate, intubate
- General inspection:
 - Dysmorphisms: eyes, ears, mouth, cry
 - Colour: central, peripheral
 - Respiratory effort: grunting, indrawing, flaring nostrils
 - Posture and movements:
 - Normal: hips abducted, partially flexed, knees flexed, arms adducted, flexed at elbow, hands closed (not tightly), fingers over thumb
 - Abnormal: hypotonia, irritability
 - Skin: colour, rashes
- Systemic examination:
 - Head:
 - Skull: fontanelles, sutures, birth trauma
 - Eyes: red reflex, opacities, conjunctivitis
 - Nose: patency
 - Mouth: palate and suck
 - Ears: hearing, tags
 - Neck: upper airway

- Chest: shape, deformities, respiratory distress, cardiac auscultation, peripheral pulses, respiratory auscultation
- Abdomen: cord, 3 vessels (2 arteries and a vein), shape, liver, spleen, kidneys, bladder, genitalia, urine stream, anus, passage of meconium, femoral pulses,
- Limbs and other bones: upper limbs, digits, palmar creases, clinodactyly, grasp, lower limbs, digits, hips, talipes (club foot), spine
- Neurological status: cry, jittery, spastic, grasping, activity, irritability, symmetry of movement, tone, neonatal reflexes
- Neonatal reflexes: stepping, walking, Moro, grasp, rooting
- Also:
 - Growth: weight, length, OFC → plot
 - Offer vitamin K IM as prophylaxis against Haemorrhagic disease of the new born
 - Cord blood for blood typing and Rhesus -ive, and also measure Cord pH (from artery) – measure of hypoxia
 - If baby has patches of yellow ⇒ sitting in meconium for a while → stain
 - If uncertainty about gestational age then formal assessment
 - Re-examine at end of the first week of life, especially for signs of congenital heart disease. Takes ~ 48 hours for ductus to close
- Other observations:
 - Micturition: usually soon after birth, infrequent for first 24 hours
 - Bowel: 99.9% passed meconium by 48 hours/ Otherwise ? Cystic Fibrosis, Hirshsprungs
 - Jaundice: 40% develop it, but transient, resolves by day 5
 - Vomiting: a little is common. Green is bad (= bile)
 - Temperature: rectal best. Same range as adults when dressed appropriately
 - Weight: 1st 3 – 5 days may lose 5 – 10% of birth weight. Should regain it in 7 – 10 days
 - Haemoglobin: at birth: 170, day 5: 200, 12 weeks: 120 (lower limit of normal is 90 – 100)
- In first week:
 - Immunisations: if mother HBsAg +ive then Hep b Vaccine and HBIG
 - Guthrie card. See Genetic Testing, page 150

Outcome after Preterm Birth

- At 27 weeks, 90% survive to discharge
- Definitions:
 - Prematurity: < 37 = weeks Preterm, < 33 = weeks Very preterm
 - Birth weight (?relevance to Pacific Island Babies – usually heavier):
 - < 2.5 Kg: LBW
 - < 1.5 Kg: VLBW
 - < 1.0 Kg: Extremely low birth weight
- Factors affecting prognosis:
 - Prenatal: Socio-economic, maternal smoking, infertility
 - Antenatal: multiple birth, IURG, maternal illness, smoking, steroids before delivery
 - Birth: time of transfer, method of delivery, APGAR, resuscitation
 - Postnatal:
 - Size of NICU, surfactant, breast feeding
 - Hypoxic-Ischaemic Encephalopathy (HIE): ↓O₂ delivery to brain → becomes oedematous over next 24 – 48 hours
- Assessment of outcome: lots of problems with cohort studies: which population, admission, length of follow-up, what's measured, etc
- Issues for mothers of NICU babies:
 - How they perceive health workers
 - Postnatal Depression
 - Visiting family commitments
 - Breast feeding: often expressing

Complications of Preterm Birth

- Anaemia:
 - Miss out on the 'iron loading' that happens through 3rd trimester
 - Haemorrhage: fetomaternal, twin to twin, placental, cephalhaematoma, etc

- Haemolysis: Rhesus disease, ABO incompatibility, spherocytosis, G6PD deficiency
- Infection: CMV, rubella, septicaemia, UTI
- Bleeding disorder: haemorrhagic disease of the new born
- Respiratory Distress Syndrome:
 - =Hyaline Membrane Disease
 - Inversely proportional to gestational age and birth weight, also diabetic mothers, asphyxia, cold stress, etc
 - Surfactant deficiency → alveolar collapse → haemorrhage/protein leaking → hyaline membrane
 - Signs: indrawing and expiratory grunt
 - CXR: ground glass appearance with air bronchogram. See Chest Radiology, page 53
- Broncho-Pulmonary Dysplasia (BPD):
 - Follows ventilation for respiratory distress
 - Aetiology: O₂ toxicity, barotrauma (volume trauma), intubation, sepsis, inflammation
 - Definition requires O₂ dependency at 36 weeks
 - Histology: necrotising bronchiolitis and alveolar fibrosis
 - CXR: patchy collapse and fibrosis with areas of cystic change and over-distension
 - Incidence < 28/40 ~50%, 28 – 31/40 ~15%
 - Not improved by antenatal steroids (RDS and severe IVH is)
 - Mortality 40%
 - Long term: airways obstruction, airways hyper-reactivity and hyper-inflation
- Intraventricular Haemorrhage (IVH): small haemorrhages into the germinal layer lining the lateral ventricles with hypoxia. May → hydrocephalus. Most have no serious long term sequelae
- Parenchymal Haemorrhage:
 - Into brain, not IVH
 - Incidence 1 – 2 % of preterms
 - Most are unilateral
 - Outcome depends on site
 - Varies from nil to severe hemiplegia
- Periventricular Leukomalacia:
 - Incidence 4% of preterms
 - ?Associated with maternal infection
 - Frontal, usually watershed lesion
 - Cysts long term → spastic diplegia (legs worse than arm)
- Retinopathy of Prematurity: Abnormal vascularisation of retina following exposure to high O₂ concentrations. Screen all babies < 31 weeks or 1500 g
- Necrotising Enterocolitis:
 - During first 3 weeks (up to 3 months in VLBW infants). Rare in term babies
 - Aetiology uncertain:
 - Hypoxic damage to bowel wall (?umbilical catheterisation, apnoeic spells, septicaemia)
 - Colonisation with certain bacteria: Clostridium perfringens, E Coli, S Epidermidis, Rotavirus
 - Necrotic segment of intestine with Pneumatosis Intestinalis ('string of pearls' sign on X-ray plus portal gas seen in liver) → perforation, sepsis, etc
 - Presentation: sepsis, bloody stools, bile stained vomiting
 - Pathogenesis:
 - Necrotising inflammation of the small and large intestine
 - Mucosal oedema → necrosis → gangrene, perforation, peritonitis
 - Sequelae: malabsorption, strictures, short bowel syndrome
- Skin easily irritated (eg alcohol, tape, drips) → long term scars
- Also:
 - Jaundice more common
 - Hypoglycaemia more common
 - Failure of closure of patent ductus (give anti-PGs, eg endomethacin)
- Problems associated with Intrauterine Growth Retardation:
 - Immediate:
 - Hypoglycaemia (see Hypoglycaemia of the New Born, page 42)
 - Polycythaemia (eg due to placental insufficiency) → heart failure (due to ↑viscosity), pulmonary hypertension, NEC. Treat with exchange transfusion (eg or saline) → ↓Hb
 - Hypocalcaemia (test ALP)

- Jaundice
- Plus others (eg Cerebral Palsy)

Neonatal and Infant Anticipatory Guidance (Parent Education)

- See Parent and Adolescent Education, page 12
- Consider the topics for discussion about a neonate:
 - Vision and hearing: Can your baby hear and see (how do you know?)
 - SIDS prevention: sleep on back, ↓smoke exposure, breast feeding, nothing over head when sleeping (see Sudden Infant Death Syndrome (SIDS), page 41)
 - Immunisation: the schedule, genuine and non-genuine contra-indications, common myths, benefits and risks (see Vaccination Practice, page 85)
 - Maternal mental health: screening and assessing for post-natal depression
 - 6 week screening: dysmorphic features, cleft lip and palate, growth, eyes, heart, hips
 - Contraceptive advice
 - Smoke cessation
 - Always ask why has the mother *really* presented
- When neonatal and/or later, consider the following:
 - Recognition of illness, emergency contacts
 - Feeding: breast feeding and maternal nutrition, introducing solids, nutrition
 - CPR
 - Parenting skills: eg management strategies for sleep and toddler behaviour, toileting, eating
 - Injury prevention: seat belts, fire safety, falls, hot water, sun exposure, poisoning, safe child care, pools, playgrounds, road
 - When to expect which developmental milestones. Reassure for parents. Also early identification → early intervention
 - Developmental needs of kids: play, language, nutrition, social etc

Breast-feeding

- Advantages:
 - Prevention of disease: passive immunity against gastro-enteritis and ↓otitis media due to better Eustachian tube drainage
 - Bonding
 - ↓PPH
 - ↓SIDS
 - Cheap and convenient (available, portable, sterile – cf bottle feeding, especially in 3rd world)
 - Contents vary with circumstances (fore milk has ↑ water content)
 - No constipation
 - Less spilling, less irritating than bottle feeding
 - Supply matches demand
 - Smells nicer at both ends. No constipation with breast milk
 - ?Higher IQ
 - Males can't do it!
- Disadvantages:
 - Discomfort establishing
 - Limits mum
 - Limited iron and phosphate, Vit D and C if preterm (cf bottle feeding which can be fortified)
 - Can't easily measure intake
 - Leaking
 - Maternal drugs are included (eg lithium)
 - Breast milk jaundice
- Establishing breast feeding:
 - Babies don't feed much for 1st 48 hours
 - Breast milk comes in around day 3 (especially 1st baby) ⇒ baby's hungry on day 3
 - Can hurt – usually uncomfortable
 - Growth is best way of proving adequacy
 - Very hard to overfeed compared with bottle feeding (sucking is a reflex → will keep bottle feeding even if they don't want the feed)

- Ongoing issues:
 - If feeding too regularly then baby will be getting CHO rich foremilk but not fat rich hind milk → hungry
 - Attachment to the breast is key: Is the baby nipple feeding or breast-feeding. If nipple then repeated trauma → pain, cracked nipples, etc
 - Mastitis or blocked duct → express lots (try it in the bath)
- Establishing bottle feeding:
 - Day 1: average intake 60 ml/kg (= 40 calories/kg)
 - ↑ by 15 ml/kg/day until average of 150 ml/kg/day
 - If too much then ↑stools, ↑vomiting, ↑misery
 - Alternatives to cows milk: goat (but no folate), soya, hydrolysed (if allergic to everything else)
 - Allergy: eczema reaction mainly to casein proteins, but can also be allergic to whey protein
 - Can be intolerant (ie non allergic reaction) to:
 - Lactose (galactose + glucose): ↓lactase → osmotic diarrhoea + ↑fermentation by bacteria → ↑gas → frothy acid stools → acid burns round perianal skin. More common as secondary intolerance (eg following Rotavirus). Breast milk has lactose too.
 - Fructose (eg fruits)
 - Sorbitol (artificial sweeteners)

Failure to Thrive (FTT)

- = Failure to gain weight normally (< 3rd percentile, or falling serial measurements) [cf Stunted growth = failure to gain height]
- If also failure of linear growth ⇒ long standing problem (weight always falls first, then length, then head circumference falls last)
- History:
 - What goes in (diet):
 - What and how much (and does it actually go in, or is it just offered?). Milk, other drinks, meat, fruit and vegetables, cereals and breads, lollies
 - Assess parents knowledge base
 - Feeding difficulties: appetite, behavioural, structural, swallowing
 - What comes out (poos) – especially steatorrhoea. People usually overestimate vomit
 - Chronic illness: cardiac, renal, neurological
 - PMH: ABFWIMPS
 - Development
 - Social history: especially PND, other psych stresses, violence, drugs and alcohol
- Examination:
 - End of bed: fat, thin, energy, pallor, well/sick, dysmorphisms
 - Muscle and fat stores – look for scraggy buttocks
 - Signs of abuse and injury
 - Signs of chronic disease:
 - Cyanosis due to heart: L → R shunt *and* heart failure or cyanotic lesion (R → L shunt)
 - Respiratory: clubbing, nasal polyps (CF, asthma)
 - Gut: coeliac (not if breast feed) – distended abdomen and thin legs
 - Renal: blood pressure
 - Assess suck, chew, swallow
 - Rickets (↓vitamin D), anaemia (↓Fe), Bruising (↓vitamin K), dermatitis & neuropathy (↓Vitamin B) (all late signs)
- Differential:
 - Parent's expectations: In the 2nd year of life: ↓appetite, ↓rate of growth, ↑activity are all normal. Parents may need reassurance
 - Non-organic failure to thrive:
 - Inadequate parenting/poor nutrition the most common cause (will feed and gain weight well while in hospital).
 - Usually complex situation: eg young mum, unwanted pregnancy, obstetric problems, poor bonding, bottle feed, maternal depression, etc.
 - Is the milk being made up properly, any strange stuff (eg tea, Milo, etc)
 - To check for attachment: observe mum chatting to baby while they dress – is she talking to the baby

- Organic causes:
 - ↓Intake secondary to:
 - Underfeeding (eg engorged breasts → poor latching on, inverted nipples)
 - Congenital abnormalities (eg cleft palate)
 - Dyspnoea (eg chronic heart failure, CF, chronic URTIs)
 - Neurological lesions (eg pseudobulbar palsy)
 - Behavioural factors (eg alert, restless)
 - Abnormal losses:
 - Vomiting: need to be severe and persistent to → FTT. Eg pyloric stenosis, chronic UTIs, renal disorders
 - Stools: diarrhoea, steatorrhoea
 - Urine: eg diabetes, renal failure, diabetes insipidous, adrenal insufficiency
 - Failure of utilisation:
 - Chronic infection (eg Tb, UTIs, immune disorders)
 - Metabolic disorders (eg phenylketonurea)
 - Endocrine disorders (eg hypothyroidism)
 - Constitutional and genetic abnormalities: Short stature, Down's, Turner's, Achondroplasia
 - Increased requirements: Chronic lung disease, heart disease, etc
 - Macrosomic babies (ie mum diabetic) will loose excess weight after birth → looks like failure to thrive
- Management:
 - If non-organic failure to thrive, then educate regarding a baby's dietary needs. See Parent and Adolescent Education, page 12
 - Investigations: rarely necessary. Maybe Fe for anaemia

The Crying Baby

- A multifactorial problem
- Normal crying:
 - Babies cry their most at 6 weeks – just when the honeymoon period is over and all the supports have gone back to work/gone home
 - Normal range is ½ to 7 hours per day
 - It's their only means of communications
 - May be hungry, overfed, tired, pain, bored, hot, cold, inadequate burping, switching breasts too soon (→ low fat feeds), solids before 3 months.
 - Note especially babies cry when they're tired – common mistake is to stimulate and sooth them when they need to sleep
 - Not due to parental stress. Crying leads to stress not the other way round. Harder for older women and professional who worked up to delivery to cope with (→ ↑sense of isolation post delivery)
- Colic: definitions vary from crying lots to “well thriving baby who develops muscle spasms, flushing face, pulls up legs, screams. On and off every few minutes for several hours, loud tummy rumbles, relieved by flatus or passage of stool”
 - Theories:
 - Gut immaturity → disordered intestinal motility → GI pain
 - CNS immaturity → immature, disorganised response to stimuli → response to most things is to cry
 - Very unlikely to be lactose intolerance (rare before 3 months) or maternal cows milk consumption
- History:
 - Clarify what the parent wants to know – address their issues
 - HPC: How often, when, associated behaviours, timing, pattern
 - Vomiting and bowel patterns
 - Feeding and sleep patterns
 - PMH: ABFWIMPS
 - Maternal social history: attitude to baby, supports, PND, drugs and alcohol
- Exam and investigations:
 - Check growth
 - Exclude physical causes:
 - Acute: otitis media, intestinal cramping/diarrhoea, corneal abrasion, incarcerated hernia

- Chronic: gastro-oesophageal reflux
- Nutritional intolerances from mother's diet (rare)
- Issues:
 - Baby's safety: Is mum is at breaking point?
 - Feeding problems: sore nipples, nipple infection (eg thrush)
 - Maternal mental state: depression, lacking support, sleep deprived, anxious
 - Maternal nutrition: is she eating well?
- Management:
 - Acknowledge strain
 - Reassurance: "I have looked carefully for physical causes and there are none that I can see". "Baby is growing well so is getting the food they need"
 - Things to try: rocking, pram, vacuum cleaner, ride in car, dummy, massage, warm bath
 - Feeding: not too often, burp well, having enough?, no solids till 4 – 6 months, maternal diet (↓caffeine, cabbage, onions, experiment with what causes baby to cry)
 - Optimistic outlook: from 6 weeks to 3 – 4 months amount of crying normally reduces significantly
 - Active advice: plan what mum can do to make it easier
 - Referral to Plunket nurse or Plunket Karatane centre and/or lactation consultant

Sleep Management

- Principles:
 - Sleep is a learned process – you train your baby to do it
 - After 6 months a night feed becomes a reward for waking up → trained night waker
 - Parents also need time for themselves
- For babies:
 - Night feeds: quick, quiet, dim light
 - Leave the baby to cry for a while
 - Wrap them well, then not woken by their own reflexes (eg startle reflex when lightly asleep)
- Toddlers:
 - Evening routine: won't harm toddler if you're firm with bedtime routines. No energetic games beforehand
 - Approach to Sleep Training:
 - Agree with partner/family what you are going to do
 - Plan in advance (eg start on a long weekend). Warn neighbours
 - Tell the child how it is going to be and why
 - Quiet bedtime routine every night
 - Put in bed, say good night, walk out
 - If they come out, return them to bed with no reinforcement or eye contact
 - If they cry, wait 5 minutes, then 7 minutes, then 9 minutes, etc. When going in, no reinforcement
 - Stick with it. May get worse before it gets better. Should see improvement by 5th night

Sudden Infant Death Syndrome (SIDS)

- Defn: death < 1 year, and still unexplained after autopsy, review of clinical history and examination of the death scene (in practice none of these is usually done well)
- Epidemiology:
 - 1990: approx 4.5 per 1000 live births
 - 2000: approx 1 per 1000 live births (about 70 per year). Pakeha lower, Maori about 4 per 1000
- Epidemiological risk factors:
 - Age (3 – 5 months)
 - Maternal smoking – now greatest modifiable risk factor given sleeping on back well established
 - Prone sleeping position
 - ?Bed sharing
 - Seasonal (winter worse)
 - Previously well
 - Race (eg higher in indigenous minorities)
 - Male
 - Low birth weight

- Low maternal age
- Low Socio-economic status
- Theories:
 - Re-breathing of expired gases (eg prone or bed sharing)
 - Hyperthermia
 - Co-sleeping (bed sharing)
- Differential diagnosis:
 - Child abuse (eg shaking injury, suffocation)
 - Metabolic disease
 - Cardiac disease (congenital or acquired)
 - Overwhelming sepsis
 - Accidental asphyxia (eg in bed) – requires good death scene exam and history
- SIDS follow-up:
 - Explanation of death
 - Explanation of grieving process
 - Follow up with next child
 - Screen for risk factors
 - Role of monitoring (no evidence of effectiveness but reassuring for parents)
- Prevention:
 - Supine sleep position
 - No smoking
 - Own cot
 - Avoid bed sharing or sofa if tired or smoker or alcohol intake or pillows
 - Dress for room temperature (ie don't let them get too hot, no hat in bed)
 - Make up bed so they can't slip under the covers (ie short-sheet the bed)
- Complications of prone position: Plagiocephaly (flat spot on skull). Prevent by varying position of the head when lying

Neonatal Acute Airway Problems

- Choanal Atresia: failure of formation of nasal passages. Baby goes blue until someone opens the mouth. Can't pass NG tube. Can be unilateral
- Congenital masses: nasal encephalocele and nasal dermoid. Care with nasal intubation. Beware the midline lesion
- Pierre Robin Sequence: short jaw, cleft palate and tongue falls back and obstructs. Nurse prone. Associated with oligohydramnios
- Subglottic Stenosis: due to intubation trauma in a preterm baby

Hypoglycaemia of the New Born

- Not a big deal, but needs to be recognised and managed
- Causes (either big babies or small babies):
 - Hyperinsulin: Child of poorly controlled diabetic mother. \uparrow Maternal glucose \rightarrow \uparrow fetal glucose \rightarrow \uparrow fetal insulin (important growth factor in utero) \rightarrow fatter and larger baby, \uparrow haemoglobin
 - Small babies: Double whammy: lack of substrate and \uparrow requirements (eg cold quicker)
 - If septic or otherwise sick (may also go hyperglycaemic due to cortisol and adrenaline)
- Symptoms:
 - Usually none. Can be asymptomatic at < 1 mmol/litre of glucose [would cause convulsion in adult]
 - May be jittery (but most common cause is difficult delivery)
 - Convulsions or floppy (post-ictal) – late sign
- Prevention:
 - Identify at risk babies and monitor blood glucose
 - Feeding is usually required (normal babies can go 48 hours without a feed)
 - May need IV glucose
 - Prevent hypothermia. If they're small and get cold they will become hypoglycaemic

Jaundice

- Key question is why, not by how much (although this is important too)
- Two types of bilirubin:
 - Unconjugated:
 - If ↑↑ unconjugated → kernicterus: cerebral palsy, deafness, ↓IQ
 - Can die acutely (seizures, bilirubin encephalopathy)
 - If survive: deaf, athetoid cerebral palsy (snake like movements – the harder they try to move the harder it becomes), normally intelligent
 - Conjugated: water soluble, conjugated in liver by glucuronyl transferase
- Early onset (in **1st 24 hours**):
 - Always pathological
 - Causes:
 - Haemolysis of any cause (eg Rhesus, ABO blood incompatibility, spherocytosis, G6PD deficiency etc)
 - Sepsis: respiratory distress + jaundice (not common)
 - Exam:
 - Maybe hydrops fetalis, large liver, large spleen (site of haemopoiesis in newborn)
 - Sepsis: especially breathing (indrawing and difficulty) – if in doubt then culture and stat antibiotics
 - Prevention:
 - Expect ABO if they've had it before
 - Check for Rhesus disease
- Jaundice in **1st week**:
 - Emphasis on extent of the jaundice (as well as consideration of the cause)
 - Treatment:
 - Phototherapy (visible light at the blue end of the spectrum – not UV)
 - Exchange transfusion
- **Persistent** jaundice:
 - If it doesn't got away by 10 – 14 days then revisit
 - Causes:
 - ↑Conjugated bilirubin (go green) – needs treatment
 - Liver obstruction abnormalities (eg biliary atresia, secondary to liver damage from infection, toxins, etc)
 - Hepatitis/liver inflammation
 - Unconjugated (yellow) – needs treatment if high. Eg Breast milk jaundice – progesterone in breast milk delays maturation
 - Diseases picked up on Guthrie card causing jaundice:
 - Hypothyroidism
 - Galactosaemia
 - Cystic Fibrosis
- Aside: ABO Blood Incompatibility
 - Maternal antibodies from mother with type O blood attack fetal blood cells if type A, B, or AB. Not isoimmunisation – it's an existing immune response. Doesn't get worse with subsequent pregnancies
 - Transfusion:
 - Want to transfuse type O RBCs – aren't antigenic to anyone
 - Want to transfuse type AB plasma – won't contain antibodies to either type A or B blood

Other Neonatal problems

Infant Drug Withdrawal Syndrome *

- For first 2 weeks after delivery if mother is abusing heroin, methadone, other narcotics:
 - Jitteriness
 - Sneezing
 - Yawning
 - Poor Feeding
 - Vomiting

- Diarrhoea
- Weight loss
- Seizures

Child of Diabetic Mother *

- Maternal complications: polyhydramnios, preterm labour, still birth near term
- Fetal: ↑malformations, macrosomic, growth retarded
- After birth: hypoglycaemia, hypocalcaemia, respiratory distress (surfactant doesn't ↑ till later in gestation), polycythaemic (venous haematocrit > 0.65, looks plethoric. May require exchange transfusion to remove RBCs)

Transient Tachypnoea of the Newborn *

- Occurs in both term and prem babies
- ?Delayed absorption of amniotic fluid from lungs
- Risk factors: C-section, perinatal asphyxia, excessive analgesia, hypothermia
- Presentation: subcostal recession, grunting, and cyanosis all seen but not prominent
- CXR: inflated lung fields, perihilar opacities, ↑vascular markings
- Treatment: O₂ for several days, respiratory failure uncommon. Penicillin if ?congenital pneumonia

Meconium Aspiration *

- Hypoxia during labour → gasp → aspirate meconium (+/- vernix, meconium, blood)
- →Patchy lung collapse and over inflation (ball valve effect)
- Complications: pneumothorax and pneumomediastinum (→ angel wing appearance on CXR due to air under the thymus)
- Treatment: suction via ET tube

- For cardiovascular exam see Examination, page 7

The Blue Baby

- Foetal circulation:
 - IVC (Oxygenated) is streamed to foramen ovale → LA → Aorta → brain
 - SVC is streamed to right ventricle → ductus → umbilical artery
- Neonatal adaptation:
 - With first breath:
 - Alveolar oxygen tension increases
 - Pulmonary bed dilates
 - Ductus arteriosus starts to constrict
 - Cord clamp:
 - ↑ In LV and LA pressure
 - Functional closure of foramen ovale
 - Ductus: closes at 24 – 48 hours. A murmur may be normal. Can open or close it with drugs (NSAIDS close, prostaglandins open)
 - Replacement of HbF with HbA from 24 weeks (90%) to birth (70%) to 6 months (trace)

Clinical Signs of Heart Disease

- Clinical warning signs:
 - Early murmurs in a clinically well baby
 - New-born who becomes hypoxic
- Classifying:

	Cyanotic Heart Disease	Acyanotic Heart Disease	Respiratory Disease
Cyanosis	Severe	Variable	Variable
Tachypnoea	Mild	Variable	Variable
Respiratory Effort	Not major	Variable	Variable
Heart Murmur	+/-	+/-	None
When	First 1 – 7 days	First 1 – 4 weeks (effect of failure – takes longer)	Often at birth

Causes of Cyanosis

- Respiratory Causes:
 - Hypoventilation: Central apnoea from drugs, apnoea of prematurity, sepsis, metabolic (eg hypoglycaemia), seizures
 - Mechanical interference with lung function: airway obstruction, abdominal distension, pneumothorax, thoracic and sternal deformities, etc
 - V-Q mismatch with lung disease:
 - Infection (Gp B Strep, G – ive): pneumonia on X-ray hard to distinguish from wet-lung of early respiratory distress. Have high index of suspicion, low threshold for antibiotics
 - Respiratory Distress Syndrome: X-ray appearance: Ground glass + air bronchogram ⇒ ↓surfactant. If maternal diabetes, are deficient in surfactant until later in gestation
 - Aspiration: meconium, milk, blood
 - Pulmonary oedema, hydrops fetalis (⇒ in heart failure before delivery. Used to be due to Rhesus negative disease prior to Anti-D treatment, now numerous other causes)
 - Lung haemorrhage: complication in premature
 - Primary lung disease
- Cardiac causes of cyanosis:
 - R to L shunt: Cyanotic heart disease or pulmonary hypertension
 - L to R shunt *and* Heart failure
- Differentiating Heart and Lung Disease:
 - History and exam:
 - When did it start
 - Relationship of cyanosis to birth. If heart, pink to start with then go blue as ductus closes (blood gets to lungs via reverse flow through ductus if right heart not functioning well)

- Check respiration:
 - If apnoea ⇒ heart. If heart problems, won't work so hard at breathing
 - Respiratory distress and ↑ effort ⇒ airway or lung problem
- By investigations:
 - CXR (heart size, lung fields)
 - ABG/O₂ saturation monitoring. If lung disease, may have ↑CO₂
 - Hyperoxia test: put in 100% O₂ – if heart disease then won't change PO₂ as gas transfer is not the problem
 - Echocardiography
- Also consider sepsis and anaemia

Congenital Heart Disease

Summary

- Congenital
 - Acyanotic: Ventricular septal defect (VSD), Atrial septal defect (ASD), Atrioventricular Septal Defect (AVSD) and Patent ductus (PDA)
 - Cyanotic:
 - Decreased Pulmonary Flow (→ dark lungs on X-ray):
 - Critical pulmonary atresia/stenosis, critical aortic stenosis (if not critical then acyanotic)
 - Tricuspid atresia
 - Fallot's Tetralogy (commonest congenital cyanotic problem, presents about 3 months of age, no murmur)
 - Increased pulmonary flow:
 - Transposition of the great arteries (TGA): Fine till birth, goes blue as ductus closes
 - Total anomalous pulmonary venous drainage (TAPVD): very rare
 - Other: Coarctation
- Acquired: Rheumatic fever
- Arrhythmia: Long QT, SVT, Pre-excitation, VT
- If chronic leads to developmental delay and clubbing
- Associated with:
 - Chromosome disorders: Trisomy 21 (40% have cardiac lesion – mainly AVSD), 18, 13 and Turners (coarctation)
 - Numerous syndromes
- Physiological classification (a la Esko Wiltshire) – as cyanotic/acyanotic distraction blurred in practice:
 - L → R sided shunt of any cause: size depends on size of lesion and relative pressure
 - L sided obstruction: coarctation, hypoplastic L heart, aortic stenosis (Eg Williams syndrome)
 - Pulmonary venous congestion and oedema (due to ↑↑ pulmonary flow): TAPVD (pulmonary veins connect to R heart → SOB shortly after birth)
 - ↓Pulmonary blood flow: eg Tetralogy
 - Transposition streaming
 - Mixing: univentricular heart

Aetiology

- Genetic causes:
 - 6 – 8/1000 live, full term births (higher in premature and still born). Second most common congenital malformation after the brain
 - Chromosomal eg Down Syndrome
 - Single gene eg Marfan's (prolapsing mitral valve)
- Environmental:
 - Infection (eg Rubella)
 - Maternal (eg Diabetes)
 - Substance abuse (eg alcohol)
 - Drugs (eg phenytoin, thalidamide)
- Usually leads to an abnormality in tissue migration

Incidence

- Pathology not Paediatrics' numbers!:

Acyanotic (L-R Shunt)	Ventricular Septal Defect	25 – 30%
	Atrial Septal Defect	12 – 20%
	Patent Ductus	10 – 15%
Cyanotic (R-L Shunt)	Tetralogy of Fallot	8 – 15%
	Transposition of Great Vessels	8 – 10%
No Shunt	Coarctation of the Aorta	5 – 7 %
	Pulmonary Stenosis	5 – 7 %
	Aortic Stenosis	4 – 5 %

Ventricular Septal Defect

- Epidemiology: 12/10,000
- Types are muscular, perimembranous and outlet (affects aortic valve)
- 90% involve membranous septum which grows down to meet muscular wall
- Clinical:
 - Wide range: from asymptomatic through life to fulminant heart failure in infancy
 - Signs:
 - Pansystolic murmur (often with thrill): bigger lesion → ↓murmur but early failure
 - Mitral diastolic murmur
 - Features of heart failure if large or prolonged lesion
 - Features of pulmonary hypertension if large: ↑JVP, sternal heave (↑RH work), loud P2
- Investigations:
 - ECG: LV hypertrophy (due to volume loading)
 - CXR: ↑heart size, ↑vascular markings in lungs
 - Echo: size and location of defect
- Management: Treat only if large: surgical repair
- Prognosis dependent on size of defect:
 - Small defects often close over spontaneously
 - Large defects → Eisenmenger's syndrome (see below, pulmonary hypertension, shunt reversal and cyanosis)
 - Endocarditis risk even with small lesion. Prophylaxis if dental work

Atrial Septal Defect (ASD)

- Epidemiology: 6 – 8/10,000 live full term births. F:M = 2:1. Higher in stillborn and premature births
- Aetiology unknown in > 90% of cases
- Pathogenesis:
 - Septum primum closes foramen primum at week 5. Failure to form completely results in a low ASD adjoining AV valve
 - Septum secundum closes over foramen secundum at week 4 – flap forms a one-way valve. If it fails to reach far enough leads to fossa ovalis ASDs. Common in trisomy 21
 - Defects in primitive sinus venosus lead to ASD near vena cava ostia
- Clinical:
 - A L → R shunt increases pulmonary flow but if < 1 cm may be asymptomatic through life
 - Larger defects → arrhythmias and/or murmur in 3rd decade. Evaluate for corrective surgery to prevent pulmonary hypertension, heart failure or arrhythmia
 - Chronic pulmonary flow 2 – 4 times normal → pulmonary hypertension with R → L shunt and heart failure in less than 10% of cases
 - Signs:
 - May have evidence of RH overload: ↑JVP, sternal heave, loud P2
 - Ejection systolic murmur in pulmonary region (flow murmur due to ↑flow, not due to defect). This is not specific – many children have it, especially when ill ⇒ P2 important in differentiating benign from ASD
 - Fixed splitting of S2
- Investigations:
 - ECG: RV hypertrophy and Right axis deviation (due to RV volume loading)
 - CXR: ↑heart size, prominent pulmonary artery with ↑ vascular markings
 - Echo to confirm diagnosis
- Management: Surgical or percutaneous closure by age 5 - 10 (unless small and spontaneous closure). Catheter umbrella if shunt > 2 : 1

Patent Ductus Arteriosus

- Aetiology: Occurs as an isolated lesion or in combination with other abnormalities (eg Tetralogy of Fallot). Association with Rubella. 90% isolated defects. Incidence 1 in 2000. Can be same size as pulmonary artery
- Pathogenesis:
 - Connects aorta to left pulmonary artery (acts as R → L shunt in foetus).
 - ↑Oxygenation at birth → ↓PGs (lungs metabolise them) → muscular contraction → functional closure at 2 days, anatomic closure at 2 – 3 months. Forms ligamentum arteriosum.
 - If hypoxia at birth then it can remain patent. A patent ductus allows up to 75% of LV output to flow from the aorta to the pulmonary artery (ie becomes a L → R shunt)
 - If persistent then → pulmonary hypertension → becomes a R → L shunt (Eisenmenger's Syndrome – see below), deoxygenated blood flows more to legs than arms → clubbing in toes not fingers
- In term infants: Delayed closure due to ↓PaO₂: pulmonary disease (eg meconium aspiration), pulmonary hypertension, high altitude
- In preterm:
 - ↑Incidence with ↓ birth weight
 - ↓ Sensitivity to PaO₂ and ↑ sensitivity to PGE₂
 - Haemodynamic effect of hypoperfusion and hypotension: associated with intraventricular haemorrhage and necrotising enterocolitis
- Clinical:
 - Murmur at left sternal edge, 2nd or 3rd intercostal space: loudest in systole (can be heard in first few hours in many infants). Silent if high pulmonary artery pressures. Systolic only in prem as still have ↑ R pressures
 - Active precordium with bounding pulses and wide pulse pressure (collapsing pulse). Can feel foot pulses
 - Hepatomegaly
 - LV failure: apnoea, bradycardia
 - → LV hypertrophy, RV hypertrophy secondary to pulmonary hypertension if persistent
- Investigations:
 - ECG usually normal. LV and/or RV hypertrophy if large and persistent
 - CXR: cardiomegaly and pulmonary plethora
 - Echo: diagnostic
- Management:
 - Requires closing:
 - NSAIDs (anti-PG) → promote closure
 - Surgical or device closure
 - Small risk of endocarditis if closed

Persistent Pulmonary Hypertension

- Failure of pulmonary capillary bed to dilate sufficiently after birth
- Return to fetal circulation (ie R→L shunt): ↓ pulmonary flow
- Causes:
 - Aspiration, eg meconium
 - Hypoxia: asphyxia
 - Infection: Group B strep
 - Hyaline membrane disease
 - Lung hypoplasia

Coarctation of the Aorta

- Preductal or infantile form:
 - Narrowing of the arch of the aorta between the left subclavian artery and ductus arteriosus. Association with patent ductus and ASD (?due to low flow in this area after birth) and with bicuspid aortic valves. Associated with Turner's Syndrome. Severe consequences: LH failure, cyanosis in lower half of body
 - Variety of presentations:
 - Pink or blue baby (if pulmonary congestion)

- Problems occur from day 2 – 3 when ductus closes (often supplies distal to lesion). Heart failure in infancy (LV hypertrophy due to pumping against an ↑ load). If severe, ↓ blood flow to kidney → acidosis → ↓ LVF → poor perfusion → ↓ blood flow to kidney etc
- Hypertension in child/young adult
- Asymptomatic murmur
- Exam:
 - ↓Femoral pulses, radio-femoral delay
 - Upper limb hypertension
 - Systolic murmur heard anteriorly and posteriorly (in back). Continuous murmur if severe
- Investigations:
 - ECG shows evidence of LV hypertrophy
 - CXR:
 - Rib notching due to collaterals (> 5 years of age)
 - Abnormal aortic arch contour: look for faint post stenotic dilatation below cardiac knuckle
 - Echo: shows lesion – harder to see descending aorta
 - Cardiac catheter: assess haemodynamics
- Treatment: surgery, balloon angioplasty and ongoing management (hypertension, risk of dissection, etc)
- Post ductal or adult form: Less severe narrowing with possible post-stenotic aneurysm due to turbulence. Ductus is closed. May be asymptomatic. Possible LV hypertrophy. Left intercostal artery provides collateral flow. (NB Proximal aneurysms occur in Syphilis, Coarctation and Marfan's)
- Left and right pulses may be different (left bounding)

Tetralogy of Fallot

- Malformation during closure of the interventricular septum leads to:
 - Transposition/over-riding aorta
 - High VSD
 - Pulmonary obstruction (valve stenosis/atresia) or infundibulum hypertrophy
 - Right ventricular hypertrophy (giving a boot shaped heart – diagnose on X-ray)
- Survival requires a patent ductus
- Clinical: RH failure, commonly endocarditis with subsequent brain abscess. Present several months after birth with episodic blueness. Death likely at puberty if not corrected

Transposition of the Great Arteries (TGA)

- Aortic and pulmonary arteries transposed → 2 separate circuits
- Requires a patent ductus +/- VSD
- OK until birth. Go blue as ductus closes

Pulmonary Valve Stenosis

- Similar effect to pulmonary atresia
- → ↓ Right heart development. Blood has always got to LA via foramen ovale, rather than through lungs

Other

- Truncus arteriosus: congenital malformation in which the pulmonary artery and aorta have failed to separate from their common precursor truncus

Arrhythmias in Children

- Relatively uncommon in paediatric population
- Often not associated with other heart disease (cf adults, associated with structural disease)
- Clinical
 - Asymptomatic or palpitations
 - If prolonged may → growth retardation, heart failure, arrest, etc
- Classification:
 - Bradycardias:
 - AV Block:
 - 1st and 2nd degree: normal variant
 - 3rd degree: Associated with AVSD, post-surgical, Rheumatic fever, myocarditis, maternal mumps, etc. Acute treatment: Isoprenaline/Atropine → ↑HR. Long term: pacemaker.

- Sinus node dysfunction
- Tachycardias:
 - Supra-ventricular tachycardia (SVT): due to re-entrant pathway associated with A-V node.
 - Generally paroxysmal. Regular rhythm. Normal QRS duration. 1:1 A-V conduction.
 - Management: Terminate tachycardia: vagal manoeuvres (ice on face, valsalva – breathing against closed glottis, carotid massage), IV adenosine (A-V node blocker), β -blockers, DC cardioversion. Prevent recurrences with β -blockers or ablation of re-entry pathway
 - Ventricular Tachycardias: rare in children. Cardiovert.
- Pre-excitation Syndromes: Wolfe-Parkinson White. Accessory pathway from atria to ventricle \rightarrow short PR interval, wide QRS and delta wave. Risk of cardiac arrest and sudden death. Treatment: ablation
- Long QT syndrome: due to congenital (K^+ channel abnormality) or acquired (drugs, hypocalcaemia). Susceptible to arrest, torsades and bradycardias. Consider in unexplained syncope or convulsions. Treatment: cardiovert if arrhythmia, β -blockers or pacemaker to prevent

Complications of Congenital Heart Disease

Heart disease with failure

- Definition: inability of myocardium to meet metabolic needs of the body
- Causes:
 - Congenital Heart disease:
 - Lesions with left to right shunt: Large VSD (\rightarrow \uparrow blood to pump \rightarrow overloaded heart), AV canal defect, Patent ductus
 - Left outflow obstruction: Hypoplastic left heart, Coarctation of the aorta, aortic stenosis
 - Arrhythmia: usually SVT
 - Cardiomyopathy: Usually ischaemic, due to birth asphyxia
- Incidence by age:
 - Infants: congenital heart lesions, rarely arrhythmias (eg SVT)
 - > 1 year: cardiomyopathy, right heart disease, dysrhythmias
- Symptoms of heart failure: \uparrow respiratory effort, sweating, poor feeding (no energy to suck), failure to thrive (hypermetabolic state and poor feeding due to breathlessness)
- Signs of heart failure: Tachycardia ($\uparrow 160$ in infants), tachypnoea (intercostal indrawing, wheeze), gallop rhythm, hepatomegaly
- Features *not* found in children:
 - \uparrow JVP
 - Peripheral oedema
 - Crepitations in lung fields
- Key differential to acute onset is sepsis
- Treatment:
 - Rest
 - Diuretics
 - Digoxin
 - O₂
 - Adequate calories: fortified feeds
 - Treatment of underlying cause (arrhythmias, infections, anaemia, etc)

Eisenmenger's Syndrome (Pulmonary Hypertension)

- Sequelae of large L \rightarrow R shunt with untreated VSD, PDA or ASD. Rare – as they are usually corrected
- Usually present in 3rd to 4th decade
- \uparrow Pulmonary flow \rightarrow oversupply of blood \rightarrow pulmonary capillary hypertrophy \rightarrow \uparrow resistance \rightarrow pulmonary hypertension \rightarrow :
 - Reversal of shunt (R \rightarrow L) \rightarrow development of cyanosis
 - RH hypertrophy and failure
- Also abnormal flow \rightarrow mural thrombosis \rightarrow endocarditis (as in most congenital defects)
- Clinical:
 - Signs of pulmonary hypertension: RV heave, loud P2, hepatomegaly
 - Little or no murmur
 - Cyanosis, clubbing

- Prognosis:
 - Arrhythmias and sudden cardiac death
 - ↑Hb due to cyanosis (polycythaemia) → ↑viscosity → clotting problems
 - ↑Risk of systemic emboli (lungs don't act as a filter for emboli)
 - Haemoptysis due to pulmonary infarct/haemorrhage
- Treatment: Supportive or heart-lung transplant

Rheumatic Fever

- Incidence:
 - Incidence has declined over last 50 years, from death rate of about 20/100,000 to 6/100,000
 - 1 per 1000 people in NZ (highest rate in western world).
 - Annual incidence in NZ per 100,000: European 0.1 – 0.3, Maori 6.1 – 11.3 (ie 50 – 100 times), Pacific Island 13.3 – 24.3 (ie 100-200 times). Pockets in Porirua, South Auckland
 - Acute attacks occur mainly between 5 – 15 years of age. Peak 10 – 11 years.
- Aetiology:
 - Incidence following strep throat is 0.3 % (sore throat for 1 week) to 3% (sore throat for 3 weeks)
 - ?Some at ↑ risk due to longer carriage of strep → ↑ antibody response
- Pathogenesis: Group A β-haemolytic streptococci infection (eg Streptococcus Pyogenes) → cross reactive antibodies – substances in myocardium similar to strep antigens lead to significant inflammatory reaction in cardiac muscle → acute rheumatic fever 1 – 5 weeks following infection (average 19 day latent period).
- Clinical features:
 - Carditis:
 - Endo +/- myo +/- peri
 - Usually mild in first attack
 - New left sided diastolic murmur: mitral and/or aortic regurgitation
 - Tachycardia
 - PR prolongation (may be buried in T wave)
 - Cardiomegaly: due to valve dysfunction (→ dilation) or myocarditis
 - Arthritis:
 - Migratory polyarthritis – as one joint starts to recover another flares
 - Usually large joints. Can be red and swollen
 - Dramatic response to aspirin
 - Never permanent joint damage
 - Chorea (St Vitus Dance):
 - Sudden or gradual onset. Acute onset chorea in a child only occurs in RF
 - Usually generalised, although can be focal
 - Deterioration at school, eg writing
 - Stops during sleep
 - Increased by anxiety/stress
 - Rare symptom. Always resolves, after 2 – 3 weeks
 - Erythema Marginatum:
 - = Red rash around edge, centre normalises as it expands
 - Evanescent (comes and goes quickly)
 - Not itchy, blanches with pressure, mainly on trunk and proximal limbs
 - Subcutaneous nodules: small, painless, over bony prominences, RARE
- Investigations:
 - Throat swab: +ive for strep in 15%
 - ESR usually > 100 (not if chorea or CHF)
 - CXR: looking for cardiomegaly
 - ECG: prolonged PR in 14%
 - Echocardiogram
 - Streptococcal titres
- Diagnosis:
 - Acute Phase: Jone's criteria: evidence of strep throat (↑serum titres) plus 2 major or 1 major/2minor:

- Major criteria: carditis, migratory polyarthritis of major joints (75%), erythema marginatum (non pruritic, non painful), subcutaneous nodules, and chorea (later)
- Minor criteria: fever, arthralgia, ↑acute phase proteins, c-reactive protein, ESR, ↑PR interval
- Also watch out for murmurs, arrhythmias (from focal fibrosis)
- Very difficult to diagnose. Always consider as differential in pyrexia of unknown origin
- Chronic Phase: recurring attacks magnify cardiac injury. Mitral and/or aortic stenosis progresses to congestive heart failure. Recurrent attacks make it worse → ?long term prophylaxis
- Treatment:
 - Eradicate streptococcus
 - Aspirin: 75 mg/kg/day (↓inflammation)
 - Bed rest if cardiomegaly or CHF, others should avoid rigorous exercise
 - Steroids for acute treatment – but doesn't affect long term prognosis
 - Diazepam for chorea
- Course:
 - Acute phase lasts 6 – 8 weeks, monitored by ESR
 - Dental check
 - Ongoing management:
 - Will get it again if they get another strep infection, and more severe
 - Penicillin prophylaxis: 4 weekly IM benzathine penicillin until 18 if no cardiac damage (for life if damaged).
 - Regular dental care. Prophylaxis for deep dental work (erythromycin or clindomycin – won't have any penicillin sensitive organisms on board)
 - Valves can recover
- Macroscopic appearance:
 - Acute (exudative and proliferative) phase: Pancarditis grossly visible in valves and pericardium. Valve leaflets have evenly spaced small 1 – 2 mm sterile/inflammatory (not infective) 'verrucae' – small vegetations resulting from deposition of fibrin along edges of valve. Verrucae resolve but Aschoff bodies (areas of necrosis surrounded by macrophages) organise and fibrose. Mitral valve always involved. Pericardium show non-specific serofibrinous (bread and butter) pericarditis similar to uraemia or acute MI
 - Chronic (healed) phase: Heals with organised fibrosis → deformed valves (50% mitral, 50% mitral and aortic) and/or shortening/thickening/fusion of chordae tendinae. Subendocardial fibrosis → fibrous plaque (McCallum's patch). Characteristic "fish mouth" stenosis → atrial hypertrophy and LV atrophy. Aortic stenosis → LV hypertrophy. May lead to murmurs or arrhythmias
- Microscopic appearance:
 - Exudative phase: fibrinoid necrosis with neutrophils, lymphocytes, plasma cells and macrophages
 - Proliferative phase: Aschoff body in the myocardium is pathognomonic. Consists of central fibrinoid exudate/necrosis with aggregates of large mononuclear or multinuclear cells (Aschoff giant cells), fibroblasts, plasma cells, lymphocytes and oedema. Aschoff bodies may also be seen in perivascular spaces, joint capsules, tendons, subcutaneous tissues
- Susceptible to later valvular infection
- Treatment: Possible surgical replacement of deformed valves

Symptoms

- Differential of dyspnoea
 - Heart failure
 - Cerebral hypoxia
 - Metabolic acidosis
 - Respiratory Causes
- Differential of Stridor:
 - Retropharyngeal abscess: lymph nodes in midline behind pharynx, usually under 4 years, mainly strep, maybe staph aureus. Acute toxicity, hyper-extended, quiet stridor, CT diagnostic
 - Croup
 - Epiglottitis
 - Foreign Body
 - Angio-oedema
 - Peritonsillar abscess
 - Laryngomalacia: noisy breathing due to floppy larynx from birth, especially inspiratory, crying or exertion
 - Tracheomalacia: soft tracheal cartilages: Brassy cough (honking). May get severe obstruction
 - Adenoid and tonsillar hypertrophy: reaches peak at 8 – 10 years, but relatively largest at 5 – 6. Snoring and obstructive sleep apnoea. Acutely enlarged → stridor (eg in EBV – treat with steroids)

Chest Radiology

- Initially:
 - Name, date, and view
 - Orientation: L & R
 - Check exposure: lung fields and intervertebral discs
 - Centering: check rib length on each side for rotation (clavicles unreliable)
 - Lung field size:
 - 5 – 7 anterior ribs to the midline of the Right diaphragm
 - 7 – 9 posterior ribs to the spine
 - If too many, then hyperinflated: asthma, CF
 - If too few, then inspiratory film: hard to interpret
- Middle right lobe is against RH border – consolidation there will obscure border. No other consolidation will
- Staph pneumonia → pneumatocele (air filled cysts). Generally resolve
- Pneumo-mediastinum → ‘angel wing’ appearance as air lifts up thymus
- Chylothorax: lymph surrounding lung in the newborn → ?thoracic duct dysfunction
- Trachea: in an infant is floppy, so in an expiratory film can have a kink
- Lateral CXR:
 - Vertebrae should get blacker as go down
 - Retrosternal clear space: in infant whiter due to thymus
- Thymus on AP CXR:
 - Lots of variation – can look like large heart
 - Thymic notch: lower right or left edge as it abuts the heart
 - Thymic wave sign: contour down the side of the thymus
 - Thymic sail sign: sail-like shape sticking into the lung fields
- Respiratory Distress Syndrome:
 - = Alveolar collapse (not bronchi)
 - Xray: diffuse opacity, air bronchograms and small lung volume
 - Severity assessed by blurring of heart borders and diaphragm
 - Group B Strep infections in full term babies can look a bit like it
- Transient Tachypnoea of the Newborn:
 - Xray: Retained lung fluid, lung volumes normal to large, and pleural effusions

- Mild → recover
- Meconium Aspiration:
 - Xray: Diffuse, coarse lung field opacity (fluffy), hyperinflated (airway pathology not air space pathology → plugging and ball/valve effect)
 - Can get pneumothorax, pleural effusions due to the work of breathing
 - Mainly in term babies – they have the grunt to suck it down. Also, pre-term babies less likely to pass meconium when stressed

Respiratory Tract Infections in Children

- Reference: Mainly from Prof Grimwood's extensive infectious diseases handout
- Epidemiology:
 - Common: During the first 3 years of life, a child may have up to 6 episodes of otitis media, 2 episodes of gastro-enteritis and 6 respiratory infections per year. 10 – 15% have 12 colds per year.
 - Other risk factors:
 - Breast feeding is protective
 - Passive smoking
 - Exposure to infection: older siblings, day care, etc
 - Socio-economic status (multifactorial)
 - 95% of respiratory infections involve the upper respiratory tract and 90% are viral
 - But antibiotics prescribed in 70% of cases. Leads to:
 - Unnecessary adverse effects: rashes, diarrhoea, thrush, plus more serious ADRs
 - ↑Cost
 - Antibiotic resistance → major increases in cost. Especially *S pneumoniae* and *S. aureus*
 - Reduce unnecessary prescribing by developing guidelines, practitioner education, public relations and ↓OTC antibiotic sales (eg mupirocin)
- Pathogenesis: 60% due to rhinoviruses and coronaviruses, then RSV, parainfluenza viruses, influenza and adenovirus

Common cold

- Starts with nasal congestion, throat irritation → sneezing, watery nasal discharge
- Low grade fever, malaise, cough, headache
- After 1 – 3 days nasal discharge becomes thicker and mucopurulent. This is part of the natural history of URTI and does not indicate a bacterial super-infection
- Generally improved by day 10, although cough (in 30%) and nasal discharge (in 40%) may persist for > 2 weeks
- Numerous RCTs have consistently failed to show that antibiotics alter the course of the common cold

Acute Otitis Media

- = Infection of the middle ear cleft
- Presentation:
 - Eardrum opaque (not semitransparent), red, normal landmarks lost, bulging. But if kid is screaming, ear will be red regardless
 - Otagia, otorrhoea, hearing loss
 - Systemic signs: fever, irritability
 - If it ruptures, child will be instantly better (but parents will panic!). Acutely ruptured eardrum will heal in 24 hours
- Pathogens:
 - *S pneumoniae* (30 – 50%)
 - Non-typeable strains of *H influenzae* (20 – 30%)
 - *M Catarrhalis* (10 – 20%) – nearly all β-lactamase producing
 - Viral (10 – 20%) especially RSV
 - Mixed bacterial/viral infections account for 50% of antibiotic failures
- Treatment:
 - Without treatment, 70 – 90% of infections resolve spontaneously
 - Those least likely to respond are:
 - Aged < 2 years
 - Those with constitutional disturbance (eg > 39 C)
 - Where *S pneumoniae* is the pathogen

- Antibiotics:
 - Should be directed against *S pneumoniae*: it is the most common pathogen, the least likely to resolve spontaneously, and the most commonly associated with mastoiditis. Amoxicillin for 7 – 10 days (?5 days just as good) is the treatment of choice, even when there are non-susceptible *S pneumoniae* isolates. Good penetration of middle ear. Erythromycin/cotrimoxazole if allergic. Main reason for antibiotics is to prevent rare complications
 - For the 90 – 95% of otitis media that responds to antibiotics, 90% are due to spontaneous resolution
 - If < 2 years, constitutional disturbance and persistent symptoms > 48 hours:
 - Amoxicillin 15 – 30 mg/kg TID for 10 days (ie high dose).
 - If no improvement after 48 – 72 hours try Augmentin (cover H influenzae and Moraxella)
 - Main aim is to reduce the very small chance of suppurative complications
 - Treatment for Acute Otitis Media in children (NZ Guideline for Acute Otitis Media):
 - Main benefit from antibiotics is less pain on the 2nd or 3rd day in 1 in 17 kids, and failure to spread to other side in 1 in 17. No effect on pain on first day, prevention of recurrence or build up of middle ear fluid
 - Side effects of skin rash, vomiting or diarrhoea are as common as benefits
 - Recommendation: use Paracetamol, return to doctor if symptoms persist beyond 48 hours, and have ears checked in a month for persisting fluid (common in first several weeks) – this occurs in about 1 in 10
 - Alternative is the use of “safety net prescriptions” for use if symptoms persist after 48 hours
 - Oral cephalosporins and 2nd generation macrolides don’t penetrate the middle ear and/or have poor activity against *S pneumoniae*
- Complications:
 - Mastoiditis in 0.1%. Incidence is not increased by delayed treatment
 - Little evidence to suggest that untreated otitis media causes mastoiditis
 - Very rare: petrositis, labyrinthitis, facial palsy, subdural/epidural/brain abscess

Recurrent Acute Otitis Media

- Risk factors for recurrent acute otitis media: childcare centres, passive smoking, family history (twin studies show strong genetic component), reflux
- Management:
 - Ensure correct diagnosis
 - Reassure: spontaneous improvement in many after age 2 – 3 years and during summer
 - Limit passive smoking, discourage pacifier use
 - Encourage breast feeding in infancy
 - Antibiotic prophylaxis generally ineffective
 - Avoid unproven therapies: antihistamines, decongestants, chiropractic, homeopathy and naturopathy
- Refer to paediatrician/ENT surgeon if febrile seizures, antibiotic intolerance, hearing loss/speech problems, underlying facio-cranial abnormalities
- In the future, conjugate pneumococcal vaccines are likely to play an important role. Polysaccharide vaccines confer little benefit.
- In rarer cases lack secretory IgA, or perhaps IgG2 antibodies to the polysaccharide capsule

Chronic Otitis Media with Effusion

- = Presence of sterile *or* infected fluid in middle ear
- Chronic OME (=Glue Ear) if > 3 months. If it hasn’t cleared by then, less likely to clear spontaneously.
- Common up to age 5 or 6
- Symptoms:
 - Incidental finding in asymptomatic child
 - Hearing loss and its effects: speech delay, slurred speech, failing at school, irritable, poor balance, falling over. But delayed language and cognitive problems related more to genetic and SES than previous otitis media
- Pathogenesis: eustachian tube dysfunction (not just blockage)
- Sequelae of otitis media: Middle ear effusion:
 - In 70% after 2 weeks
 - In 50% after 1 month

- In 20% at 2 months
- In 5 – 10% after 3 months
- Associated with mild hearing loss.
- Treatment:
 - Effusion common after an ear infection. Watch and wait if child otherwise well
 - If bubbles behind ear drum then it's resolving itself
 - Drugs: antibiotics and decongestants not very effective
 - If persisting > 3 – 6 months:
 - Test hearing
 - Limit passive smoke exposure
 - Treat underlying allergic rhinitis/adenoidal enlargement with intra-nasal steroids
 - Refer after 3 – 6 months if hearing loss and:
 - Failure to respond to antibiotics
 - Recurrent acute otitis media
 - Persistent otalgia
 - Retraction pockets
 - Expressive/receptive language delay
 - Underlying cranio-facial abnormalities (eg Down syndrome)
 - ENTs say grommets (Ventilation Tubes) are the treatment of choice: Aerate middle air (→↓ CO₂ →↓ squamous metaplasia →↓ goblet cells →↓ effusion). Extrude over 18 months – 2 years. Take out if still there 5 yrs later. Short term benefits vary depending on degree of symptoms. If asymptomatic, little benefit likely. Little evidence of long term benefit from ventilation tubes. May take out adenoids at same time → ↑ eustachian tube function (Paediatricians say adenoidectomy is treatment of choice).
 - Precautions with grommets:
 - Plug ears when washing hair and bathing
 - Can swim in clean fresh water but no diving below the surface
- Chronic Suppurative Otitis Media – with hole in drum. Treatment: get rid of infection then surgical repair

Pharyngitis

- Almost 100% given broad-spectrum antibiotics. Inappropriate in 90% of cases
- Pathogens:
 - Viruses: Adenovirus, also rhinovirus, coronaviruses, RSV, Parainfluenza virus, influenza, enteroviruses, EBV
 - Bacteria: S Pyogenes (GABHS = Group A Beta-Haemolytic Strep) in about 20 – 30% of cases, predominantly in those over 4 years
- Differentiating (at best 70% predictive accuracy):
 - Exudative tonsillitis: Adenovirus, GABHS, EBV
 - > 4 years, enlarged tender anterior cervical lymph nodes and diffusely inflamed pharyngeal structures (+ exudates) suggests S Pyogenes
 - Diffuse, sandpaper-like red rash, accentuated in skin creases (Pastia lines) suggest Scarlet Fever. See Streptococcus Pyogenes (Group A, β Haemolytic), page 75
 - Nasal discharge, cough, hoarseness, conjunctivitis or diarrhoea +/- fever +/- tonsillar exudates suggests virus
 - Throat swabs: usually identify organism, but 10 – 50% are carriers
- Treatment:
 - Aim: Prevent acute rheumatic fever, suppurative complications (peri- or para tonsillar abscess) and hasten recovery
 - But
 - Only benzathine penicillin has been shown to reduce RF – and this was in military personnel
 - No convincing data which shows antibiotics reduce the risk of rare suppurative complications
 - Antibiotics reduce symptoms by 8 hours only
 - Reinforces the notion that antibiotics are effective and increases the likelihood of their future use for trivial illnesses
 - If high risk for RF (eg Maori, PI, > 4 years of age) take swabs or treat empirically. However, prescribing penicillin for sore throat hasn't altered the rates of RF, and many children with RF haven't consulted their doctor
 - S Pyogenes: penicillin, 500 – 1000 mg BID for 10 days (Allergy: erythromycin)

Acute Sinusitis

- Uncommon. Bacterial sinusitis complicates 0.5 – 5% of viral upper respiratory tract infections
- With most colds, nasal discharge and obstruction are improving after 2 weeks. Children with acute sinusitis will not be improving
- A minority present with high and persistent fever, periorbital swelling, facial and dental pain
- Imaging:
 - Plain x-rays don't differentiate well between common cold and sinusitis
 - CT more useful. Air-fluid levels, opacification, mucosal thickening > 4 mm
- Maxillary and ethmoid sinuses present at birth (although small). Frontal and sphenoid sinuses begin at 4 – 6 years of age
- Pathogens: S pneumoniae (30 – 70%), H influenzae (20%), M Catarrhalis (20%), virus alone (10%)
- Treatment:
 - High spontaneous cure (60% by 10 days vs 85% with amoxicillin)
 - Treat for S Pneumoniae in children with persisting symptoms which are not improving
 - Amoxicillin 15 – 30 mg/kg TID for 5 days. Higher limit if < 2 years, attend child-care, or have received antibiotics in the last month in areas with > 10% penicillin resistance
 - Consider Augmentin, co-trimoxazole, cefuroxime or ceftriaxone if no improvement after 48 – 72 hours

Bronchitis

- Inflammation in bronchial mucosa → productive cough
- Most cases are from viruses (eg RSV)
- Numerous studies have not found any evidence to support antibiotic treatment (but they're usually prescribed...)
- Production, colour or culture of sputum does NOT predict aetiology
- Consider treatment if:
 - Prolonged cough in older child: ?M pneumoniae → erythromycin
 - Pertussis and cough < 4 weeks: erythromycin (or co-trimoxazole) reduces infectivity
 - Cystic fibrosis/other chronic lung disease: tailored antibiotics
 - Prolonged cough (> 8 – 12 weeks and not from URTI): investigate for asthma, Tb, pertussis, CF, foreign body, Subacute-sinusitis, psychogenic cough

Croup

- = Laryngotracheobronchitis
- Pathogens: Usually viral: Parainfluenza 1 and 2 are the most common. Measles and influenza are the most severe. Don't give antibiotics
- Presentation:
 - Child < 5 years
 - Coryza and fever over 1 – 2 days
 - Then characteristic harsh “barking” cough, hoarseness +/- signs of upper airway obstruction (stridor, respiratory distress), *inspiratory* stridor
 - Worse at night, and peak on 2nd or 3rd night. Varies hour to hour (ie don't send them home just yet...)
 - Lasts 3 – 4 days then changes to sound productive. May last for another 2 weeks
- Differential:
 - Epiglottitis: Absent/minimal cough, low-pitched expiratory *snore*
 - Bacterial tracheitis: toxic appearing, older child, high fever, brassy cough, stridor, tender trachea
 - Laryngeal foreign body: sudden onset, unable to vocalise
 - Angioneurotic oedema: associated signs usually present
 - Retropharyngeal abscess: High fever, dysphagia, hyperextension of neck
- Assessment:
 - Severe if restless, anxious, pallor, lethargy, tachycardia, tachypnoea, indrawing, cyanosis or ↓breath sounds
 - Loudness of stridor is not a reliable guide to severity of obstruction
 - ↑Risk of obstruction if: pre-existing upper airway narrowing (eg sub-glottic stenosis) or Down Syndrome
- Management:
 - Avoid distressing the child, settle them on parent's lap

- Blood tests, pulse oximetry, O2 masks and nebulisers rarely needed
- Mild:
 - Not distressed, no stridor at rest
 - No treatment, management at home, return if signs of ↑obstruction, lots of comfort
 - Paracetamol
- Moderate:
 - Frequent barking cough, distressed, persistent inspiratory stridor, tracheal tug or sternal retraction at rest, but no signs of hypoxia
 - Observe or admit
 - Steroids (Dexamethasone or betamethasone 0.6 mg/kg orally or im, prednisolone 1 mg/kg) orally. May be repeated 12 – 24 hours later (but consider alternative diagnoses first)
 - Disturb child as little as possible
- Severe:
 - Signs of obstruction, hypoxia (restless, irritable, anxious, cyanosis), ↓breath sounds
 - ?ICU admission
 - Nebulise adrenaline (4mls of 1:1000, can safely repeat half hourly) + Steroids (Prednisolone 1 mg/kg/day)
 - Monitor closely

Epiglottitis

- Caused by Haemophilus Influenza Type B
- Incidence ~ 20 cases pa (dropped from 160 in 1992 prior to vaccination)
- Presentation:
 - Incubation for 2 – 4 days
 - Acute, febrile illness, toxic looking child
 - Snore, mouth always open, drooling, prefers to sit upright. Soft inspiratory stridor, louder expiratory stridor
 - No cough (cf croup)
- Management:
 - Blood cultures
 - Intubate first, then give iv antibiotics (if given first, pain → panic → respiratory arrest)
 - Cefotaxime 25 – 50 mg/kg/8hr iv (max 2g) due to ↑penicillin resistance
 - Amoxicillin 50 mg/kg/4 hr iv (max 2g) if penicillin sensitive
- Other illnesses caused by H Influenzae type B:
 - Meningitis: 5% mortality, 10% with sequelae (retardation, seizures, hearing loss, etc), 20 – 30% have functional disabilities (eg learning difficulties)
 - Also pneumonia, empyaema, septic arthritis, periorbital or facial cellulitis
- Vaccination:
 - Prior to immunisation was the most common cause of life threatening bacterial infection < 5 years of age.
 - Herd immunity now works well
 - Subunit vaccine is 95% effective. Few side effects (< 5% with local reactions)
 - Notifiable disease

Pertussis

- Bordetella Pertussis = Whooping Cough
- Epidemiology:
 - Highly contagious. Regular epidemics every 3 – 5 years in NZ
 - Incidence: up to 5000 cases a year (only a small proportion notified)
 - In first year of life 80% are hospitalised and 0.2% die
- Presentation:
 - Phases:
 - Incubation 2 – 3 weeks
 - Coryzal phase: ~ 1 week
 - Paroxysmal phase:
 - Develops into paroxysmal bouts: unprovoked cough followed by inspiratory gasp (whoop), apnoea, vomiting.

- Thick tenacious sputum → can't clear → coughing spasm. Whoop may be absent in infant. If severe may need suction
- In between paroxysms looks well, is afebrile and has no chest signs
- Median length: 6 weeks. Can be up to 12 weeks
- Infectious for 2 – 3 weeks of paroxysmal phase
- Persistent cough for 3 – 4 months (convalescent phase – bacteria cleared)
- Treatment: if < 4 weeks duration: *erythromycin*. Doesn't impact illness after paroxysmal phase is established, but will ↓ infectivity
- Admit if under 6 months and/or cyanosis or apnoea in paroxysms
- Complications:
 - Anoxic seizures in 1 – 3%
 - Encephalopathy in 0.1 – 0.3% → retardation, spasticity and seizure disorders. Rate of severe neurological complications of immunisation negligible compared with the risk of encephalitis from whooping cough
- Vaccine:
 - Whole cell vaccine effective in 60 – 90%, has higher efficacy for more severe outcomes, local reactions or fever in 50%. 1 in 1 million are associated with an encephalopathy (? No causal relationship established)
 - Acellular pertussis has higher efficacy and is better tolerated (< 10 – 15% adverse reactions) – now being introduced

Bronchiolitis

- Epidemiology
 - Classically RSV
 - Highly infectious acute viral respiratory illness in kids 2 weeks to 12 months of airways < 1 mm diameter
 - Epidemics every winter with RSV, also parainfluenza, influenza and adenoviruses
 - Major cause of URTI in kids: up to 50% of 1 year olds have had RSV infection
 - Seasonal in winter/spring
- Presentation:
 - Short incubation: 3 – 4 days
 - Contacts: older siblings will have had nothing more than a snotty nose
 - Difficulty with expiration (cf Croup – inspiratory)
 - Typical pattern: Starts as URTI - 1 day of runny nose, 1 day of cough, then wheeze. Illness/breathlessness worst on 4th day of wheeze (6th or 7th day of illness)
 - Low-grade fever, non-toxic, cough, wheezy, difficulty feeding, hyperinflated chest, diffuse fine inspiratory crackles and expiratory wheeze
 - If more severe then ↑irritability, pallor, pulse > 160/min, respiratory rate 50 – 70/min, expiratory grunt (not stridor), head nodding, more marked retractions
 - Respiratory failure in 1 – 2%: pallor, sweating, drowsiness, ↓respiratory effort, ↓breath sounds, apnoea. Cyanosis is a late sign
 - Feeding a good indicator of respiratory distress (and one which parents can monitor at home)
 - Recurrence common (?hypoplastic airways and smoke exposure)
 - Usual recovery is 7 – 10 days
 - Can get repeat viral illness – in which history suggests fluctuation – getting better, then got worse again, etc
- Distribution of LRTI from RSV:
 - Bronchiolitis: 40 – 90%
 - Pneumonia: 5 – 40%
 - Tracheobronchitis: 10 – 30%
- Risk factors for severe presentation:
 - < 6 weeks old
 - Older siblings
 - Maternal smoking
 - Preterm delivery
 - Underlying conditions: congenital heart disease, chronic lung disease of infancy, congenital abnormalities, immunodeficiency
- Differential:

- Recurrent bronchiolitis, history of eczema, strong family history of atopy ⇒ ?asthma. Trial of nebulised salbutamol.
- Persistent cough, failure to thrive ⇒ cardiac disease, cystic fibrosis, structural lung disease, aspiration, immunodeficiency
- Investigations:
 - Nasopharyngeal aspirate for culture and viral immunofluorescence
 - Bloods for culture and serology
 - Imaging: CXR shows hyperinflation, peribronchial thickening, often patchy areas of consolidation and collapse. Hyperinflation and wheeze differentiate it from pneumonia
- Treatment:
 - **Not** bronchodilators, steroids, ribavirin or antibiotics
 - Symptomatic treatment: O₂, rehydration, minimal handling
 - Can go home if they're feeding OK and don't need O₂
 - Admit if respiratory distress, difficulty feeding, or adverse social circumstances. If sending home early in the illness, arrange for review within 24 hours
 - Put on NG feeds: not hungry → ↓distress
 - If respiratory rate > 70/min and feeding poorly then IV or NG fluid at 50 – 75% of maintenance requirements (risk of SIADH)
 - If oximetry < 92% then O₂
 - If severe, monitor blood gases, consider CPAP or ventilation (especially chronic respiratory/heart disease)
 - Maybe wheezy for 2 weeks and a cough for 4 weeks

Pneumonia

- Epidemiology: Peak incidence in first 2 years, and in Maori and PI children
- Presentation:
 - Initial prodromal coryzal symptoms for a few days
 - Fever, cough, tachypnoea, signs of consolidation
 - Young children may present with predominantly systemic features: fever, lethargy, vomiting, abdominal pain
 - Older children may have headache, pleuritic chest pain, irritating cough, maybe abdo pain if lower lobe or even signs of meningism if upper lobe
 - Severe if:
 - Toxic: lethargy or ↓arousal, circulatory compromise, abnormal respiration (eg apnoea, cyanosis)
 - Respiratory distress: pallor, restless, agitated, nasal flaring, grunting, head nodding, chest wall recession, paradoxical abdominal movement, difficulty feeding
- Signs on exam:
 - In infants: may be few signs, usually limited to a few focal crackles
 - Older children: ↓chest wall movement, ↓breath sounds, fine crackles, later dull to percussion and bronchial breath sounds
- Pathogens:
 - Viruses are the most common cause in infants and young children:
 - RSV and Parainfluenza 3 most common
 - Suggested by: infant or young child, coryzal prodrome, mild or moderate constitutional disturbance, hyperinflation and diffuse inspiratory crackles, patchy consolidation on CXR
 - Rarely, infections with influenza A, adenovirus 3, 7 or 21 can be severe leading to death or severe lung damage
 - Bacterial:
 - S pneumoniae most common bacteria
 - S aureus uncommon but severe, H influenzae uncommon
 - M pneumoniae common in school age children, insidious onset including anorexia, headache, scattered fine inspiratory crackles, bilateral
 - S pyogenes: typically follows Varicella, influenza A or measles, protracted course and often empyema
 - Chlamydia: in 1st 2 months. Vertical transmission + eye infection in first 5 – 7 days. See Eye disorders in Children, page 107
- Investigations:
 - Imaging: CXR to:

- Confirm diagnosis
- Detect complications: pleural effusion, pyopneumothorax, lung abscess
- Exclude other causes: congenital lung lesions, lung abscesses
- Blood cultures before antibiotics
- Nasopharyngeal aspirate for RSV detection
- Serology for M pneumoniae or RSV
- Aspiration of pleural fluid (assists diagnosis, and is therapeutic – antibiotics won't penetrate a large effusion)
- Treatment:
 - Penicillin G is the treatment of choice for uncomplicated bacterial pneumonia (unless allergy). Despite 20% of S pneumoniae's showing reduced sensitivity, concentrations in the serum and lung tissue exceed the MIC by several fold. More treatment failures are associated with erythromycin and co-trimoxazole
 - Admit if any of:
 - < 2 years
 - Signs of toxicity, hypoxia, respiratory distress
 - Extensive consolidation or an effusion
 - Clinical or x-ray signs of Tb
 - Adverse social circumstances, no transport or no access to phone
 - If sent home, then review within 12 – 24 hours
 - For uncomplicated bacterial pneumonia: Penicillin G 25 – 30 mg/kg/6hr iv (max 2.4g)
 - If not afebrile within 24 hours on penicillin G, then review microbiology results, repeat CXR, consider other causes and treatments. Treatment failure: consider Viral, Mycoplasma, S aureus, resistant H influenzae
 - Supportive therapy: minimal handling, careful fluid management (max 50% of maintenance fluids if IV), O2
 - Management of pleural effusion. Before antibiotics do diagnostic aspiration and urgent gram stain. Discuss with paediatric surgeon:
 - Thin clear fluid: aspirate as much as possible
 - Thin purulent fluid: intercostal drain
 - Thick purulent fluid: loculates so drain won't work ⇒ thoracotomy (consider flucloxacillin +/- Cefotaxime)
 - Infected effusion = Empyema = pus in pleural cavity
 - Fibrous septae will form around empyema = loculated empyema

Tb Pneumonia

- Rarely presents as acute pneumonia
- Consider if:
 - Known exposure to Tb
 - Child or family born in an endemic area
 - > 4 week history of cough, especially if fever, sweats and weight loss
 - Refractory pneumonia
 - Suggestive CXR
- Nurse in respiratory isolation:
 - Virtually all child cases are primary and non-infectious with a small burden of disease
 - But adolescents, those with extensive or cavitating disease, or infected visiting family are infectious
- Investigations:
 - FBC, ESR, electrolytes, CR and LFT
 - Mantoux test
 - Specimen collection: sputum if available. Early morning gastric aspirates better than lavage. Also consider urines, pleural biopsy and LP
- Empiric treatment: isoniazid, rifampicin, pyrazinamide
- Notify to Medical Officer of Health

Differential of Wheezing in a Child

- Common:
 - Asthma

- ‘**Happy wheezer**’ (Transient infant wheeze):
 - Diagnosis of exclusion
 - Usually < 12 months
 - Chronic *daytime* wheeze and no cough
 - No atopic background
 - Child undistressed and no impact (ie feeding OK, not waking)
 - Less wheeze when asleep
 - Requires no therapy
 - ?Collapsible airways
 - Can become an ‘unhappy’ wheezer when they get a cold, in which case treat as for bronchiolitis
- **Bronchiolitis:** See page 59
- Uncommon:
 - **Inhalation:** If convincing episode of inhaling a foreign body (stridor, went blue, etc) should be bronchoscoped – even if they you think they brought it all back up. Signs: unilateral wheeze or stridor. May present months or years later with haemoptysis. CXR – will have hyperinflation *or* collapse on side of inhaled object
 - **Cystic Fibrosis:** [see Cystic Fibrosis (CF), page 65]
 - If breast feed can still thrive for a month or two
 - Respiratory symptoms often present with wheeze not cough
 - Heart Failure:
 - Sweat when feeding + poor feeding/cyanosis
 - Wheezy – sounds like bronchiolitis
 - Look for enlarged liver (but beware, bronchiolitis → hyperinflation → liver lower)
 - **Aspiration:**
 - If due to neurological problems, will cough and splutter when swallowing
 - Long vague history with unclear start
 - Gives acute onset of wheeze – eg OK when you put them to bed, but sudden coughing and wheezing later
 - If lying on back, most likely to aspirate into right upper lobe, but in practice they are wheezy everywhere
 - Hard to prove. Diagnosis of exclusion. Can do reflux probe to show they reflux often
 - **Immune deficiency:**
 - Rare. Only consider if lots of serious illnesses
 - Cilia dyskinesia: usually starts with ears (middle ear has respiratory epithelium with cilia), then lungs and sinuses. Associated with dextrocardia
 - Hypogammaglobulinaemia
 - Can confuse wheezing with soft **stridor:** eg laryngomalacia. Inspiratory sound
 - Rare congenital causes: cysts, tumours, lobar emphysema, tracheomalacia/bronchomalacia (not properly formed → floppy)
- Smoking contributes to all the above:
 - Prenatally, maternally smoking → ↓airway size
 - Post-natal → inflammation/irritation

Asthma in Young Children

- Epidemiology:
 - 3rd most common reason for admission (after Bronchiolitis and URTI/Otitis media).
 - Much much less common in < 1 years (NB bronchiolitis causes wheezing in young). Peak in 2 – 4 years
 - Peak flow very unreliable under age 7 (and most bad asthmatics diagnosed from 2 – 5) → *have to rely on history*
 - If neither parents have asthma, 15% risk by age 10, if both parents then 60% risk
 - If atopic dermatitis as a child then 40% risk by age 10, if none then 20% risk
 - Males 30% risk, females <20%
- History:
 - *Symptoms:* waking at night with cough/wheeze, after exercise, how often are attacks, had time off school/kindy as a result, how long does preventer last
 - *Environmental factors:* smokers, pets, damp, obvious triggers. Ask about:

- Living situation
- Occupation
- Allergies, any pets?
- Seasonal
- Cold air
- Irritants (eg fumes)
- Exercise
- Night cough
- History of atopy: eczema
- *Current treatment*: medicines, do the family understand the difference between reliever and preventer, assess technique and compliance, is spacer accepted by child and is it washed
- 2 patterns on history:
 - Episodic (intermittent): viral URTI → cough and wheeze. No interval symptoms
 - Persistent (with exacerbations): interval symptoms (with exercise, at night), exacerbations with viral infection, interferes with everyday life
- Symptoms in a toddler:
 - Cough, often worse at night
 - May vomit with cough (NB exclude pertussis: cough → choke → vomit → OK for an hour. In asthma, cough again straight away)
 - Usually wheezy with URT infection
 - Diagnosis difficult in an infant unless recurrent, strong immediate family history or evidence of atopy
- Physical findings in a toddler:
 - Often normal chest exam
 - If severe chronic symptoms:
 - Hyperinflated chest (↑ AP diameter)
 - Harrison's sulcus: dip in chest wall where diaphragm attaches
 - Eczema
 - Reduced growth (if severe)
 - Stethoscope can be confusing
- Possible investigations:
 - Symptom diary
 - Spirometry and response to bronchodilators
 - Initial CXR if more than mild episodic disease
 - Skin tests for atopy
- Diagnosis:
 - Diagnosis: "episodic wheeze or cough in a clinical setting where asthma is likely, and other rarer conditions have been excluded"
 - Cough is very common in kids (8 – 10 per year). But more during the day than at night. Won't slow them down when running
 - Is it asthma, bronchitis, bronchiolitis?
 - Trial of therapy (preventative as well as relievers) and review
- Criteria for admission:
 - Pulse rate > 1.5 * normal
 - Respiratory rate > 70 minute
 - ↑Chest movements
 - Restlessness/apathy/CNS depression or cyanosis/pallor [signs of exhaustion]
- Acute Severity assessment:

	Mild	Moderate	Severe	Critical
<i>Mental State</i>	Normal	Normal	Agitated	Confused/drowsy
<i>Accessory Muscle use</i>	Nil	Minor	Mod/marked	Maximal/exhaustion
- Interval symptom severity:
 - Physiological: cough (irritation) → wheeze (obstruction) → SOB (hypoxia)
 - Exercise performance: impaired requiring or in spite of reliever
 - Sleep: waking with or without breathlessness, ?frequency.
- Treatment:

- Avoid triggers: passive smoking, pets, house dust mite (dehumidifiers don't work), pollens, cold, exercise, damp houses, certain foods (overstated)
- Infrequent episodic asthma (75% of child asthma):
 - Consider no therapy, avoid triggers
 - If distressed with attacks: use bronchodilators + spacer only. Start during URTI phase. No preventative
- Frequent Episodic Asthma (only get it with a cold, 20% of childhood asthma):
 - Intervals between attacks < 6 weeks
 - Bronchodilator as needed with URTIs
 - Prophylaxis:
 - Sodium cromoglycate (Vicrom + spacer). ?Evidence of poor efficacy
 - Nedocromil (Tilade + spacer)
 - Inhaled steroids: if it makes no difference then stop
- Persistent Asthma (5% of childhood asthma)
 - Male: female = 4:1
 - Preventative. If mild try Vicrom or Tilade. Moderate or severe use inhaled steroids (takes 2 – 3 months for maximal effect). Titrates back once controlled
 - Bronchodilators as required
 - Poor control: consider ↑dose, check inhaler device and technique, poor compliance, environmental triggers
 - DON'T double steroid inhaler with a cold - ineffective
- Other treatment options:
 - Long-acting β-agonists: salmeterol (Serevent), formoterol (Foradil, Oxis)
 - Theophylline (Nuelin, Theodur): 3rd line, gut ache → poor compliance
 - If severe: *alternate* day oral prednisone treatment – reduced side effects (short and fat), and reasonable asthma control
- Protocol for an acute attack:
 - Salbutamol dose: up to 5 years: 6 puffs via spacer. Over age 5: 12 puffs via spacer
 - For severe add ipratropium (Atrovent)
 - For moderate and severe, give doses at 0, 20, 40 and 60 minutes and review at 75 minutes
 - Oral steroid for all except minor attacks: 1 mg/Kg/day → ↓relapse. For 3 days – but stop earlier if well

Medication

- Inhaled Corticosteroids:
 - Action: Anti-inflammatory and ↓hyper-reactivity
 - Effect: ↑lung function, ↓symptoms, ↓admissions (only drug to do this)
 - If using a β-agonist most days then should be on an inhaled steroid
 - Doses:
 - 200 to 400 µg/day of Beclomethasone Dipropionate (BDP/Becotide) or Budesonide (BUD/Pulmicort), or
 - 100 to 200 µg/day of Fluticasone Propionate (Flexatide - only difference is potency, not efficacy, ↓systemic side effects at large doses)
 - Back titration: in stable patients back titration may be attempted. ½ dose as a one off. If cut too far too fast can rebound within a month. Stopping treatment altogether is likely to cause a relapse
 - Biggest mistake in little kids is to give too low a dose of steroids – poor technique means less gets in than an older child
 - Takes 3 months for maximal effect
 - Side-effects: Dose dependent redistribution of fat, electrolyte disturbances, hypertension (ie Cushing's features), stunted growth in children – initial drop in growth velocity when commencing inhaled steroids – but they will catch up.
- Bronchodilator:
 - Reliever. Short acting inhaled β agonist.
 - Potent and rapid bronchodilator and a relatively low toxicity. Relaxes airway smooth muscles (plus other effects, e.g. ↓release of mast cell mediators). Adverse effects: muscle tremor and tachycardia common. Use as needed – not regularly – then becomes a guide to severity

- Long acting agonists for more severe asthmatics: Salmeterol and Eformoterol (similar effect but ↑ potency). Peak effect 2 – 4 hours, duration 9 – 12 hours. More effective in symptom control than increasing Beclomethasone over 400 mcg/day.
- Others:
 - Sodium cromoglicate: non-steroidal preventer – less effective than steroids but fewer side effects. Single dose good for prevention of exercise induced asthma
 - Anti-leukotrienes: Leukotrienes → ↑vascular permeability, ↑mucus production, ↓mucus transport, etc. Oral montelukast → 15 % ↑ in FEV1, ↓use of β agonist. Place in therapy still uncertain
- Follow-up (eg good liaison with GP) following emergency admission is critical to preventing recurrence

Inhalers

- Advantages: minimum possible dose, highly targeted, patient controls therapy
- Inhaled steroids → deposition in mouth. If not using spacer, **need to rinse, gargle and spit** otherwise risk of thrush and croaky voice. At best, 10% gets to lower airways without spacer
- Metered dose inhalers (MDI):
 - Autohaler: shake, push lever up, suck. Lower level of suck needed than powder inhalers – but still require good suck to get lower airways deposition. As expensive as powder inhalers. OK from age 8 upwards
 - Standard MDI: (cheap, light and rapid delivery of drug, but co-ordination difficult). From age 12 onwards. Instructions for use:
 - Shake an inhaler between each puff
 - Remove cap
 - Hold it upright and pointed backwards
 - Breath out
 - Fire during 1st 25% of long slow inhalation
 - Hold breath
 - Breath out after removing inhaler from mouth
- Inhalers through a spacer:
 - Requires lower dose, less time, 1/10th the price and more mobile
 - As effective as a nebuliser. Increases LRT deposition by 4 times
 - Eliminate oral deposition of steroids and ↑ lung deposition of both preventers and relievers
 - If child crying then ↓↓ lung deposition
 - Breath-a-tech with a facemask up to 6. Remove mask as soon as you can (stops nasal filtering – try at age 4 - 5). Need smaller spacer as they have a small tidal volume
 - Volumatic without facemask. Need to be able to mouth breath well (ie try from age 2 – 3 onwards)
 - Need to inhale within 30 seconds of a puff into the space
 - One puff at a time
 - But plastic spacer → static charge → particles stick. So wash in detergent once a week and do not rinse bubbles off (→ microfilm of detergent)
 - If using a new spacer without washing, need to prime it (10 puffs). Don't do this in front of patient
- Dry Powder Inhalers: ↑↑ oral deposition (30% lungs, 70% upper airways). Use from age 5 up (good for use at school when they don't want to lug a spacer around but their MDI technique is inadequate). Advantages: light, quick delivery, don't need co-ordination, CFC free. Disadvantages: cost, require high respiratory flow
 - Accuhaler: 60 doses, easy to use, has dose meter
 - Disk haler: 6 doses
 - Turbohaler: easier to use than disk haler. Red mark inside indicates when its empty

Cystic Fibrosis (CF)

- Autosomal recessive. 1 in 25 are carriers
- Disease of epithelial lined organs:
 - Lungs: mucus plugging → chronic inflammation → necrosis, adjacent pneumonia, bronchiectasis. Leads to chronic infection, emphysema and pseudomonas colonisation. Eventually → Cor Pulmonale
 - Pancreas: fibrosis around ducts, dilated ducts, islets cells relatively preserved. → pancreatic insufficiency

- Gut → meconium ileus, biliary cirrhosis, recurrent RLQ pain
- Bile ducts obstructed
- Middle ear
- Vas deferens → infertility
- Presentation:
 - Newborn screening (80% will turn out to be carriers, not diseased)
 - Neonates: Meconium ileus:
 - > 90% with meconium ileus will have CF → obstruction at birth. Occurs in 15% of those with CF
 - Presentation: bilious vomiting, palpable bowel loops, distension if perforated
 - CXR: distended bowel loops with thickened walls
 - May also have associated volvulus, small bowel atresia, perforation, neonatal meconium peritonitis secondary to perforation
 - Treatment: enema + IV fluids or surgery
 - Failure to thrive
 - Sibling with CF
- Pathogenesis:
 - Abnormality of cAMP dependent chloride transport due to mutation of the CFTR protein. Less water gets out → thicker mucus ⇒ obstruction and ↓ciliary clearance
 - In 70% of the mutations, the protein is not glycosylated normally → not transported to site
 - Most common is DF508/U (on chromosome 7)
 - Not all mutations are pathogenic. Milder mutations are dominant over severe ones
 - Genotype is best predictive of pancreatic function, lung disease is more multifactorial
- Testing:
 - Guthrie card for Immuno Reactive Tripsin (IRT). Tripsin leaks into blood from pancreas if pancreatic duct blocked. Samples in top 1% considered positive. Sensitivity high (~95%) but not at all specific. 8% false negative. 92% false positive. If positive then → gene screen
 - If IRT positive then 3 mutation screen (covers 98% of CF population – ie false negative for 2%). If positive for one or two mutations then referred to a regional CF paediatrician who will organise:
 - Sweat test (lack of Cl channel → can't reabsorb NaCl from isotonic secretions → salty sweat): Need > 75 ml sweat. >30 mmol/L Cl is suspicious, > 60 is definite CF
 - Extended mutation screen: 20 mutations screened for, accounting for >98% NZ CF cases. False positives extremely rare. False negatives possible ⇒ genetic testing cannot rule out CF
 - Pancreatic function test: 3 day fat balance is useful but unpopular. Faecal elastase level on a spot sample is highly correlated with pancreatic function. Some mutations allow sufficient function for normal absorption, but because of continuing pancreatic secretion in the presence of blocked ducts are susceptible to acute/chronic pancreatitis
 - Notification does not state whether one or two mutations found because those with only one found may have a rarer second gene. Need definitive sweat test.
 - Diagnosis:
 - Definite: abnormal duplicate sweat test or 2 mutations found
 - Possible: 1 mutation + suspicious sweat test
 - Probably carrier: 1 mutation + normal sweat test
 - Each year 564 babies are IRT +ive, 12 CF cases are diagnosed and 1-2 missed
- Advantages of early diagnosis: ↑lung function, ↑nutrition, less traumatic diagnostic process
- Post-natal management:
 - Good nutrition: enzyme replacement, high calories, fat soluble vitamin supplements
 - Antibiotics for URTI
 - Grommets at 2 years
 - Multidisciplinary approach
- Possible monitoring tests:
 - CXR, lung function tests, sputum culture (esp pseudomonas lung infection – key prognostic indication)
 - FBC, electrolytes, total protein, albumin, Vit A, D, E, blood glucose
 - Faecal elastase (testing pancreatic insufficiency)
- Complications include bronchiectasis (CF is commonest cause): bronchi dilated and filled with purulent secretions. Like CF requires regular physio

Cervical Lumps

- 99% of general neck lumps are lymph nodes:
 - Large nodes can take up to 6 – 8 weeks to go down
 - Three types:
 - Reactive hyperplasia: due to infection, not painful
 - Acute lymphadenitis:
 - Acutely tender, erythematous mass with accompanying fever, usually settle with rest/analgesia
 - Results from URTI, cellulitis or other skin infections
 - Cervical lymphadenitis: *S aureus* or *S pyogenes*
 - Management: antibiotics and/or drainage
 - Lymph node abscess: lymphadenitis may progress to abscess. Doughy feeling. Overlying skin erythematous. If raised, red and soft →? Staph abscess → flucloxacillin (always on an empty stomach). Augmentin for Strep abscess. Otherwise excise and drain under GA
 - Rare causes of lymph node enlargement: Consider if subacute, minimal tenderness, fixed or overlying skin changes consider:
 - Cat-scratch disease
 - Toxoplasmosis
 - Cutaneous Tb (collar stud abscess, bruised looking, no systemic symptoms, non-tender, tethered to skin, 6 mths to 5 years): excision, Mantoux and chest x-ray
 - Hodgkin's Lymphoma: Child > 5, rapid enlargement, rubbery spherical lymph non-tender nodes, night sweats, fever, weight loss, lymphadenopathy elsewhere, splenomegaly
- Lateral Neck Lumps: branchial cysts and branchial sinuses
- Midline Neck Swellings:
 - Thyroglossal Cysts:
 - 80% of midline cervical lumps
 - Peaks in pre-school child and young adulthood
 - Swelling near the hyoid that moves with swallowing or when pokes tongue out
 - May trans-illuminate
 - Early referral: get it out before it becomes infected
 - Treatment: surgery (Sistrunk procedure) and excision of the tract
 - Submental nodes: Usually superficial and anterior. Check mouth for primary infection (eg ulcer)
 - Dermoid Cysts: Common at the corner of the eyebrow (external angular dermoid). In the cervical region they are subcutaneous and mobile, and appear yellowish. Require excision
 - Ectopic Thyroid: Rare. May be only thyroid tissue. Tend to become hypothyroid

- See also:
 - Respiratory Tract Infections in Children, page 54
 - Gastroenteritis, page 129
 - Epstein Barr Virus, page 80
 - Herpes Simplex Virus (HSV), page 78
 - Varicella Zoster, page 79
 - Streptococcus, page 75
 - Bacterial Meningitis, page 69
 - Polio and Rubella in Vaccine Preventable Diseases, page 86

Fever in Children

- Most fevers caused by respiratory tract viral infection, are self-limiting, and require only symptomatic treatment.
- Kids have 6 – 8 viral infections each year → they are common
- Role of doctor:
 - Identify source of infection
 - Counsel caregivers and child
 - Manage the illness
 - Identify and refer those with potentially serious illness
- If no focus found:
 - Consider UTI, occult pneumococcal bacteraemia, meningitis
 - Consider non-infectious causes: rheumatic fever, poisoning, drug fever, more rarely leukaemia and other autoimmune diseases (eg Kawasaki's Disease)
 - On exam, pay attention to:
 - General appearance: activity, perfusion, colour
 - Vital signs: pulse, respiration, blood pressure
 - Exclude: fontanelle, neck stiffness, respiratory distress, abnormal chest signs, ears, throat, lymphadenopathy, hepatosplenomegaly, abdominal distension, bone or joint tenderness/swelling, skin rashes
 - At greater risk: neonates, immunocompromised, congenital abnormalities, toxic appearance, epidemiological ↑ risk (eg Maori)
 - WBC are unreliable for detection serious infection
 - Review within 24 hours and parent education
- Advice for parents:
 - Light clothing
 - Small, frequent drinks of water or fruit juice diluted 1:4, 5 – 7 mls/kg/hr
 - Paracetamol, 15 mg/kg/6 hourly, max of 90 mg/kg/day for 2 days
 - Return to doctor if refusing drink, pale or floppy, difficulty breathing, headache/neck stiffness/photophobia, doesn't improve in 48 hours
- Clues for predicting serious illness (even over the phone):
 - Responsiveness and activity
 - Feeding
 - Urine output
 - Breathing
 - Colour

Potentially Serious Infections

- See When is a child really sick?, page 9
- Sepsis: leads to systemic inflammatory response syndrome (SIRS). Also get it in major trauma, pancreatitis, etc. Mass release of cytokines. (Cf bacteraemia: bugs in blood but no major systemic reaction)
- Sepsis + focus (pneumonia, kidneys, joints/bones):
 - Neonate: Gp B Strep (*S agalactiae*), E Coli, *S aureus*

- Infant/older child: S Pneumoniae, N Meningitidis, S aureus (complication of skin infections), S pyogenes, [HIB]
- Meningitis: infection of CSF via choroid plexus – see Bacterial Meningitis, page 69

Infections of the CNS

Bacterial Meningitis

Signs and Symptoms

- Rapid onset of:
 - Meningism: Headaches, photophobia, stiff neck. Kernig's sign: Pain on straightening knee with hip flexed
 - ↑ICP: Headache, irritable, drowsy, vomiting, fits, ↓pulse, ↑↓BP, ↓LOC, pin-point pupils, papilloedema (late sign), tense fontanelle
 - Septicaemia: fever, arthritis, DIC, ↓BP, ↑pulse, tachycardia, rash (ultimately 80% will have a purpuric rash, 10 – 15% will have a maculo-papular or urticarial rash, 5 – 10% will have no rash)
- In different age groups:
 - Infants/toddlers: fever, lethargy, poor feeding, vomiting, toxic (drowsy, pallor), rash. Only 30 – 50% have signs of meningism ⇒ absence doesn't exclude. Bulging anterior fontanelle – but if vomiting may be normal or reduced
 - Children > 3: fever, headache, vomiting, photophobia, stiff neck, confusion (may be combative), non-blanching rash (initially blotchy macular rash that rapidly becomes petechial or purpuric)
 - Adolescents: may present as acute mania or appearance of drug induced psychosis

Pathogenesis

- Organisms:
 - Neonates: E. Coli, β-haemolytic streptococci Group B (eg streptococcus agalactiae – normal vaginal flora), rarely listeria
 - Children < 14 years: H. Influenza (if < 4 and not immunised), Neisseria Meningitidis Type B, Strep Pneumoniae, Tb
 - Adults: Neisseria Meningitidis Type B, Strep Pneumoniae, maybe staph aureus or Cryptococcus neoformans
 - Elderly, Immunocompromised: Pneumococcal, Listeria, Tb, G –ive, Cryptococcus Neoformans
- Pathogenesis:
 - Pathology: inflammation of pia mater and arachnoid
 - Most common are N Meningitidis and S pneumoniae
 - Nasopharynx→blood→subarachnoid space (via choroid plexus): N meningitidis, HIB, S. pneumoniae
 - Middle ear→blood→subarachnoid space: S Pneumoniae, HIB
 - Congenital abnormalities (eg spina bifida): coliform bacilli, pseudomonas, Strep agalactiae
 - Trauma: Skull fracture + CSF leak, CNS surgery, shunts: Staph aureus
 - Depressed immunity: listeria monocytogenes, cryptococcus neoformans
 - Neonatal meningitis from vaginal flora (especially with prematurity, prolonged ROM, delayed 2nd stage): Strep agalactiae, coliforms (E coli), listeria monocytogenes
- If recurrent:
 - Consider immunosuppression (eg hypogammaglobulinaemia or complement deficiency)
 - Look for lumbosacral defects, especially if enteric bacteria or S aureus

Investigations

- Do blood culture before presumptive treatment if possible, but NOTHING should delay presumptive treatment. Tell lab about antibiotics
- Must do:
 - Blood cultures
 - CSF via lumbar puncture unless contraindicated (see below)
 - Urine: supra-pubic aspiration or catheter
 - If antibiotics have already been administered:
 - Needle aspirate purpuric lesions for gram stain and culture
 - Throat swab

- Bloods:
 - Blood Glucose sample – may be hypoglycaemic [ABC = Airway, breathing, circulation. DEFG = Don't Ever Forget Glucose]
 - FBC, electrolytes, clotting time, ABGs
- Lumbar puncture:
 - Contraindicated if:
 - Signs of ↑ICP (all meningitis will have ↑ICP) causing cerebral herniation (eg very ↓LOC, very bad headache, focal signs including abnormal papillary reflexes, tonic seizures, decerebrate or decorticate posturing, irregular respirations, bradycardia, papilloedema). If in doubt then CT
 - Severe cardiovascular compromise with DIC/coagulopathy (eg fulminant sepsis)
 - Infection over the injection site
 - Tests of CSF: Gram stain, Tb, cytology, virology, glucose, protein, India ink (Cryptococcus), culture (if clear then ?virus), antigen testing (especially if partially treated)
 - May be normal, repeat if symptoms persist
 - Typical CSF (lots of variation):

	Pyogenic	Tb/Fungal	Viral ('aseptic')	Normal
Main cell seen	Polymorphs	Mononuclear	Mononuclear	< 5 * 10E6 WBC/ml (< 20 in neonates)
Glucose	↓↓	↓	- or ↓	2.5 – 5.0 mmol/l
Protein	↑↑	↑	Mildly ↑ or ↓	< 0.4 g/l
Bugs seen	Yes	No	No	

- NB: early viral meningitis may have predominantly polymorphs
- RBCs: None. If there are then either traumatic (more in 1st of 3 tubes) or bleed (new if red, yellow if old – zathachromia)
- Appearance on Gram stain:
 - N Meningitidis: G –ive diplococci
 - H influenzae: Pleomorphic G –ive bacilli
 - S pneumoniae & S agalactiae: G +ive diplococci
 - Listeria: G +ive bacilli
 - TB: Acid fast bacilli very scant – take at least 5 mls of CSF
 - Cryptococcus neoformans: Indian ink stain shows capsules
- Imaging: To identify subdural collections, abscess, hydrocephalus, thrombosis and infarction. Only if LP contraindicated and suspected mass lesion or persistent or focal neuro signs

Management

- See When is a child really sick?, page 9
- Management (based on protocol for a child):
 - Standard infection control precautions plus surgical mask when examining throat, intubating etc
 - ICU if:
 - Coma
 - Circulatory collapse
 - Persistent, recurrent seizures
 - SIADH with cerebral oedema or seizures
 - Shock or ↑ICP is what kills
 - Maintain perfusion:
 - Colloid bolus (20 – 40 ml/kg 4% albumen iv), then colloid + glucose
 - Inotrope eg dobutamine (10 µg/kg/min)
 - Watch for ↑ ADH secretion → hyponatraemia and cerebral oedema if too much fluid given
 - Check Na 6 – 12 hourly. If Na < 135 mmol/l then ↓iv rate. If Na > 145 then ↑rate
 - Respiratory support:
 - O2
 - Early elective intubation if persistent shock (but may exacerbate hypotension due to vasodilation and ↓sympathetic drive)
 - Immediate intubation if ↑ICP, hypoxia and/or respiratory failure, pulmonary oedema or hypotension (uncompensated shock)
 - Correct abnormalities: anaemia, hypoglycaemia, coagulopathy (FFP), acidosis (NaHCO3), hypokalaemia

- Seizures: anticonvulsants
- Watch for ↑ICP:
 - ↓Conscious state, focal neuro signs, abnormal pupils, hypertension and relative bradycardia.
 - Treatment: ICU, ↓PCO₂, diuretics (Mannitol, frusemide), head up, deep sedation, inotropes. But priority is to correct the shock (CBF = MAP – ICP)
- Weight and measure head daily in an infant
- Isolate patient, ensure analgesia
- Dexamethasone treatment controversial (most benefit in HIB). Not routinely used. Reduces fever and gives misleading impression of clinical improvement
- **Antibiotic regimens:**
 - Empiric antibiotic treatment:
 - Neonate – 3 mths: Amoxicillin 50 mg/kg (for listeria) + Ceftriaxone 50 mg/kg (E coli and Strep). 2 weeks for G +ive, 3 weeks for G –ive.
 - Older child:
 - Cefotaxime 50 mg/kg/6hr, max 2 g, iv for 7 – 10 days or
 - Ceftriaxone 50 mg/kg/12hr, max 2 g, iv for 7 – 10 days or
 - Penicillin G 50 mg/kg/4hr iv for 7 – 10 days
 - If strep pneumonia suspected: *Vancomycin 15 mg/kg/6hr, max 500 mg, iv + cefotaxime/ceftriaxone – synergistic, necessary due to ↑resistance to 3rd generation cephalosporins*
 - If still failing consider adding Rifampicin
 - Specific Treatment according to culture and susceptibility results:
 - N Meningitidis, S agalactiae: Penicillin (Cefotaxime if allergic to penicillin) for 5-7 days. For meningococcaemia only can use penicillin or cefotaxime
 - S pneumonia:
 - Penicillin susceptible: penicillin (but 20% are resistant) for 7 – 10 days
 - Penicillin resistant, 3rd generation susceptible: Cefotaxime
 - Penicillin and 3rd generation resistant: Cefotaxime + Vancomycin
 - H Influenza: Cefotaxime, Ceftriaxone
 - L Monocytogenes: amoxicillin
 - Staph Aureus: Flucloxacillin
 - M Tuberculosis: Rifampicin, Isoniazid, Pyrazinamide, Ethambutol
 - Coliforms: 3rd generation Cephalosporin (ie Cefotaxime, Ceftazidime)
 - Pseudomonas: Ceftazidime
 - Cryptococcus Neoformans: fluconazole or amphotericin B
 - NB: Erythromycin and gentamycin don't have good CSF penetration
 - If not responding, or non-susceptible strain of pneumococci or receiving dexamethasone then repeat LP after 24 – 48 hours
- Complications:
 - Seizures:
 - First suspicion should be hyponatraemia (also hypoglycaemia):
 - SIADH (Na < 130 and urine Na > 20) → exacerbates cerebral oedema
 - Prevent by restricting fluids to 50% of maintenance
 - Treatment: severe fluid restriction (10 ml/kg/day), in an emergency consider hypertonic saline, Mannitol or frusemide
 - Hypoventilation can further ↑ ICP → hypoxia, hypercapnea, acidosis
 - Anticonvulsants can also exacerbate these metabolic changes
 - Management options: diazepam, clonazepam, phenobarbitone, dextrose to control hypoglycaemia, intubation and ventilation
 - Major disability in 15%: *Deafness*, brain damage, peripheral necrosis, etc. All cases should have audiologist check within 6 – 8 weeks of discharge
 - Death in 5%, 10 –15% pneumococcal meningitis, 20% in fulminant meningococcaemia

Meningococcal Disease

- Cause: Neisseria Meningitidis
- Epidemiology:
 - 10-year epidemic started in 1990 with about 50 reported cases. Since then 3696 cases and 163 deaths. Current case fatality rate is 3 – 5 %
 - Leading infectious cause of death in children

- 500 reported cases in 2000. NZ rate is 13.3 per 100,000. UK rate is 4 per 100,000
- Regional variation: East Cape and Central North Island the highest
- Rates per 100,000 < 1 year olds:
 - Pacific Island: 570
 - Maori: 230
 - European: 80
- Healthy people can be carriers
- Transfer via respiratory secretions
- Kids and teenagers more susceptible than adults
- Not a cause of Otitis media
- Pathogenesis: endotoxins (lipopolysaccharides in the cell wall) activate complement and release of PAF causing endothelial injury → immune activation and ↑vascular permeability
- Notifiable to public health (as is HIB)
- Prophylaxis to stop nasal carriage of the bug – not to cure incubating illness. Nasal carriage higher in adults than children
 - Rifampicin: 4 doses, 600 mg bd for adults, 10 mg/kg bd for kids (very high dose). Broad spectrum antibiotic
 - Offer to index case (if only treated with penicillin), all intimate, household and day-care contacts during last 10 days
 - Contraindications: pregnancy (use single dose ceftriaxone), liver disease.
 - Side effects: nausea, vomiting, diarrhoea (GI effects), turns urine/tears/sweat orange/red (will stain contacts)
 - Interactions: asthma, blood clotting and oral contraceptives (continue pill, use barrier method until 7 days after antibiotics finished)

TB Meningitis

- Rare
- Most common < 5 years
- Slow onset: malaise and fever progressing to drowsiness, neck stiffness and seizures over 2 weeks
- Mantoux testing may be normal, and CXR normal in ½ of cases
- Investigations:
 - Gastric lavage, urine and CSF for Acid fast stain and culture
 - CT
- Treatment: isoniazid, rifampicin, pyrazinamide
- Notifiable disease

Viral CNS Infections

Viral Encephalitis

- Herpes Simplex:
 - Clinical: usually short history, fever, headache, confusion, ataxia, focal convulsions → coma (if clouding of consciousness consider encephalitis in addition to meningitis)
 - CSF: raised leucocyte count, predominantly mononuclear
 - Diagnosis: PCR test of CSF for Herpes Simplex antigen
 - Treatment: Acyclovir 10 mg/kg iv 8 hourly for 10 days. Low threshold for treatment
- HIV:
 - Most AIDS patients have a subacute encephalitis caused by direct brain infection
 - Symptoms: mood changes, depression, lethargy, confusion, dementia
- Other viruses: Mosquito born (Murray Valley Encephalitis, Japanese Encephalitis), Rabies virus
- Management:
 - Full blood screen: Cr, electrolytes, glucose, LFT, ABG, urine drug & metabolic screen, blood and urine cultures, ammonia, cortisol, coagulation screen, ECG
 - Serology and viral cultures
 - LP if not contraindicated – may be normal in up to 50% of cases
 - Consider empiric acyclovir + cefotaxime – at least until HSV is excluded
 - CT (MRI better still) for focal lesions
- Consider differential:
 - Head injury
 - Toxic or metabolic encephalopathy

- Hypoxic insult
- Supportive treatment:
 - Fluid restriction
 - Control of seizures
 - Cardio-respiratory support
 - Maintenance of nutrition

Viral Meningitis

- Causes:
 - Most due to non-polio enteroviruses:
 - Faecal → oral ⇒ little kids at risk
 - ECHO viruses, Polio, Coxsackie A & B
 - Mumps
- Presentation: fever, headache, malaise, photophobia, abdominal pain and vomiting. Neck stiffness in older children. Maybe a macular or even petechial rash
- Differential diagnosis of lymphocytic (aseptic) meningitis
 - Viral meningitis (eg ECHO, Mumps, Coxsackie)
 - Viral Encephalitis (eg Herpes Simplex, CMV, Varicella Zoster)
 - TB meningitis
 - Fungal meningitis (eg Cryptococcus neoformans)
 - Neurosyphilis
 - Acute Leptospirosis
 - Cerebral toxoplasmosis
 - Neoplasm
 - Cerebral sarcoid
- Lab tests:
 - CSF Culture: Enteroviruses, mumps, fungi, TB
 - Throat culture and Faeces for enteroviruses
 - CSF Antigen tests: PCR for Herpes Simplex, CMV, VZV, TB, Toxoplasmosis
 - Serology: antibodies to Treponema pallidum, Leptospira, Toxoplasma gondii
- Admit if:
 - Diagnosis in doubt
 - Antibiotics are being considered
 - IV Rehydration is needed
- Ensure good analgesia

Post-Infective Encephalitis

- Immune hypersensitivity reaction to host cells containing viral antigens
- Late onset – 7 – 10 days after acute illness
- Viruses involved: Morbilli (Measles), Mumps, Rubella, Varicella-Zoster

Other

- Spongiform encephalopathies:
 - Caused by Prions (Proteinaceous infectious particles)
 - Histology: vacuolation of brain tissue, deposition of amyloid plaques
 - Eg: Kuru (in PNG), Creutzfeldt-Jakob Disease (CJD), Variant CJD
 - Symptoms: Insidious onset of ataxia, dysarthria and dysphagia. Progressive dementia
- Slow virus infections:
 - SSPE (Subacute sclerosing pan-encephalitis): Measles like virus affecting children and adolescents
 - PML (Progressive Multifocal Leukoencephalopathy): Affects adults from 40 – 70, Polyoma virus implicated.
- Neonatal Encephalitis:
 - TORCH Complex: Toxoplasmosis, Rubella, CMV, Herpes Simplex
 - Usually accompanied by disseminated disease
- Reye's Syndrome: post-infectious encephalopathy with associated acute liver failure. Most common antecedent infection is Influenza virus

Common Paediatric Viruses

- Reference: Prof Grimwood's extensive Paediatric Infectious Diseases Handout

Measles

- Highly contagious paramyxovirus spread by coughing and nasal droplets
- Epidemiology:
 - Overall mortality 0.5%
 - Risk of infection 100% if not immunised
 - Epidemics occur every 7 years
 - Incidence up to 3000 notifications in epidemic years. Lab confirmations drop in epidemics as high incidence → high PPV of clinical diagnosis. Very few cases in non-epidemic years will actually be measles
- Presentation:
 - Incubation 10 – 14 days
 - Fever, ALWAYS a cough ("measles bronchiolitis), coryza, conjunctivitis for 2 –3 days
 - Then red maculo-papular rash beginning on face and spreading to rest of body. White spots on cheery-red buccal mucosa for 24 hours before rash (Koplik's Spots) – pathognomonic
- Treatment: Supportive, antibiotics for 2ndary infection
- Complications:
 - Otitis media (10%)
 - Pneumonia (1 – 5%)
 - Encephalitis (0.1%): 15% die and 25% left severely disabled. 1 in 100,000 develop the fatal grey matter degenerative disorder Subacute Sclerosing Panencephalitis (SSPE)
- Vaccine:
 - Live attenuated virus. Now MMR2 given at 4 years to ↑ time between epidemics and address 2 – 5% chance of primary vaccine failure in first dose
 - Mild fever, malaise or rash develops in about 1% 7 – 10 days after vaccination
 - 1 in 1 million develop encephalitis (1,000 fold less likely than if infected with wild virus)
 - Contraindicated during pregnancy and in immunocompromised hosts

Mumps

- Contagious paramyxovirus spread by saliva and droplets
- ~ 80 notified cases per annum. Used to be 3 – 4 year epidemics, now longer
- Presentation:
 - Incubation 2 – 3 weeks
 - 70% develop fever and swelling and tenderness of salivary glands
 - 15% have aseptic meningitis
 - 0.2% develop encephalitis
 - 20% of post-pubertal males have painful orchitis
 - Case fatality is 0.02% - usually from encephalitis
- Infective 1 week before and after parotid swelling starts
- Vaccine: Live attenuated virus (contraindicated during pregnancy and immunosuppression). Efficacy 95%. Only introduced because it can piggy back other vaccinations

Non-Polio Enteroviruses

- Include Coxsackie A and B, echoviruses and enteroviruses
- Cause: non-specific febrile illnesses, pharyngitis, gastroenteritis, viral meningitis, encephalitis, pericarditis, myocarditis, hepatitis, haemorrhagic conjunctivitis, etc
- Viral exanthem: macular rashes, maculo-papular, vesicular and petechial rashes
- *Hand, Foot and Mouth Disease*: Coxsackie A16. Mild illness, low-grade fever and sore throat. Scattered vesicular lesions in the mouth with similar lesions surrounded by erythematous areolae on the hands and feet.
- Incubation for 3 – 6 days, infectious for at least 1 week after onset of symptoms
- Diagnosis: culture (including from faeces – if isolates persist for several weeks may be unrelated to illness), possible PCR for blood and CSF. Serology difficult

Human Herpes 6 and 7 (Roseola Infantum)

- Acute febrile illness of *young* children for several days with occipital adenopathy, then reduced fever and appearance of a fine red maculo-papular rash over the trunk and arms for 1 – 2 days (confused with antibiotic rash)
- 70% of 2 year olds are sero-positive. Serology and PCR problematic due to latent infection
- Incubation 5 – 15 days
- Rare complications: encephalitis or benign intracranial hypertension

Erythrovirus (Parvovirus) B19

- = Erythema Infectiosum or Slapped Cheek Syndrome
- Mild illness, fever in 30%, bright red rash on cheeks for 2 – 3 days
- A few days later, a maculo-papular, then lace-like rash may appear on arms, then trunk, buttocks and thighs. May recur over following weeks after hot baths
- Incubation 4 – 14 days
- Infectious period is before the rash appears
- Complications: Adolescents and adults may also have polyarthralgia/arthritis, aplastic crisis if chronically anaemic (eg immunocompromised)

Orbital and Pre-Orbital Cellulitis

- Want to determine if its:
 - Orbital cellulitis:
 - Eg spread from anterior ethmoid sinus
 - Proptosis (eye pushed forward) and/or ophthalmoplegia (limitation of movement) and/or ↓ visual acuity
 - Surgical emergency: discuss with ENT, ophthalmologists, radiologist re imaging (CT not MRI)
 - Bugs: *S Aureus*, also *S pneumoniae*, *S pyogenes*, HIB
 - Cefotaxime and flucloxacillin
 - Complications: intracerebral extension (lumbar puncture contra-indicated until this is ruled out)
 - *Periorbital cellulitis*: in superficial fascia around the eye but not into the orbit. Fever and local tenderness
 - Investigations: FBC, blood cultures
 - *S pyogenes* and *S aureus* especially if contiguous skin lesion, *S pneumoniae*, HIB if not fully immunised (can check urine for antigens). If HIB then ?HIB meningitis and Rifampicin prophylaxis for patient and family
 - Treatment:
 - If < 5 and not fully immunised: cefuroxime or Cefotaxime (50 mg/kg/6hr, max 2 g)
 - If > 5 or <5 and fully immunised: flucloxacillin (50 mg/kg/6 hr, max 2 g)
 - If no response after 24 – 48 hours, treat as for orbital cellulitis or underlying sinus disease
 - *Local allergic reaction*: eg just erythema without tenderness or temperature

Bacterial Disease

Streptococcus

Streptococcus Pyogenes (Group A, β Haemolytic)

- NB: Lancefield Groups only apply to β Haemolytic Streps
- Causes:
 - Commonly: acute pharyngitis, cellulitis, impetigo (also caused by group C)
 - Uncommonly: necrotising fasciitis (haemolytic strep gangrene), strep toxic shock syndrome, scarlet fever, erysipelas (= contagious skin infection with strep pyogenes), acute otitis media
 - Rarely: pneumonia, infective endocarditis
- Has remained sensitive to penicillin
- Identical strep can lead to a variety of infections:
 - Sore throat
 - Impetigo/Cellulitis. See Impetigo (School Sores), page 135

- Toxic Shock Syndrome
- Myositis
- Necrotising Fasciitis
- Infection via throat (mainly) or via skin (impetigo/wound infection):
 - Suppurative: tissue invasion
 - Non-suppurative (after 2 – 8 weeks):
 - Rheumatic Fever (See Rheumatic Fever, page 51)
 - Glomerulonephritis
 - Super antigens: pyogenic exotoxins – ability to avoid classical antigen processing by APCs
- **Scarlet Fever:**
 - Direct response to Streptococcal toxins (cf virus rash which is autoimmune and therefore delayed)
 - Presentation: fever, exudative pharyngitis, scarlatina rash (fine punctate rash with perioral sparing), desquamation
 - Skin feels like sandpaper then desquamates. May get purpura in flexures (Pastia's Lines)
 - Tongue affected – white then strawberry red
- **Streptococcus Toxic Shock Syndrome:**
 - First described in children. Now associated with Tampon use
 - Early (1 – 7 days): vague, viral like illness: fever, chills, myalgia, diarrhoea
 - Later: abrupt onset of pain (not necessarily associated with findings), redness, hypotension, renal failure, ARDS, coagulopathy. May lead to necrotising fasciitis. Also skin diffusely erythematous like sunburn, conjunctivitis
 - Desquamation a week later characteristic
 - Age group: 2- 50 year olds, no predisposing or underlying disease
 - Bacteriology:
 - Blood culture +ive in 60%
 - Swab or aspirate in 95%
 - M protein types 1 & 3: impedes phagocytosis by leucocytes, expressed on cell wall
 - Lab tests: Haematuria, ↑Cr, ↓albumin and ↓Ca, serum CK for deep tissue infections
 - Treatment: Ceftriaxone
- **Necrotising fasciitis:**
 - Diffuse swelling and mild erythema, followed by bullae filled with clear fluid. Spreads along facial planes
 - Infection of subcutaneous tissue → progressive destruction of fascia and fat but may spare the skin itself.
 - 25 cases per year in NZ
 - Requires aggressive surgical debridement
 - Causative bacteria:
 - Group A strep most common
 - Staph Aureus
 - C. Perfringens
 - C. Sceptica
 - Predisposing factors:
 - Diabetes
 - Peripheral vascular disease
 - Chicken pox
 - Minor trauma/surgical procedures
 - Use of NSAIDs masks inflammation and delays diagnosis

Streptococcus Lancefield Group B

- β Haemolytic Streps
- Eg Strep agalactiae: differential in neonatal meningitis. Normal vaginal commensal

Streptococcus Pneumoniae

- Is α haemolytic but not classified as a Viridians
- Causes:
 - Commonly: acute otitis media, acute sinusitis, febrile convulsion in infants, community acquired pneumonia, infectious exacerbations of chronic bronchitis, meningitis (nasty type)
 - Uncommonly: peritonitis (2ndary to chronic hepatic/renal disease or to infected IUCD)
 - Rarely: infective endocarditis

- Antibiotic sensitivity:
 - Parenteral:
 - Penicillin resistance in 1% blood isolates in adults and 11% in kids ⇒ Strep pneumonia penicillin resistance is not an issue in adults but is in kids
 - Ceftriaxone
 - Vancomycin (for penicillin resistant strains and MRSA)
 - Oral: amoxicillin, erythromycin, cefaclor, tetracycline (not kids or pregnant)
- Vaccination:
 - Pneumovax
 - Polysaccharide-based subunit vaccine containing 23 serotypes covering 90% of strains causing invasive pneumococcal disease
 - Contains T-cell independent antigens ⇒ non-immunogenic if < 2 years (and poor response for some serogroups up to age 6). Predominant IgM response without induction of memory. 5 yearly boosters recommended
 - Recommended for:
 - > 65 years
 - > 2 with asplenia, immunocompromised (including nephrotic syndrome) and chronic illness
 - Conjugate vaccines generating IgG response being worked on....

Viridians Streptococci (plus also Enterococcus faecalis)

- Causes UTI, abdominal wound sepsis, infective endocarditis (uncommon)

Staphylococcus

Staphylococcus Aureus

- Sources of bacteraemia:
 - Skin sepsis
 - Wound infection (esp hospital acquired)
 - Pneumonia (esp hospital acquired)
 - Osteomyelitis
 - Septic arthritis
 - Lines: Subclavian, IV drips (esp CVP)
 - Infective endocarditis
- See also Impetigo (School Sores), page 135

Staphylococcus coagulase negative (eg epidermidis)

- Sources of bacteraemia: IV lines – Hickman, CVP lines, premature neonates with IV lines

Haemophilus Influenzae

- Uncapsulated type (not type B which is capsulated)
- Causes:
 - Commonly: acute otitis media, acute sinusitis, acute infectious exacerbation of chronic bronchitis
 - Uncommonly: community acquired pneumonia (more CORD patients)
 - Rarely: meningitis
- Antibiotic sensitivity:
 - 5% of isolates produce penicillinase ⇒ resistant to amoxicillin
 - Augmentin
 - Cefaclor
 - Tetracycline (not kids or pregnant)
 - Cefuroxime (iv)
 - Is not sensitive to erythromycin

Moraxella Catarrhalis

- Previously known as Branhamella Catarrhalis
- Commonly causes: acute otitis media, acute sinusitis, acute infectious exacerbation of chronic bronchitis (same as Haemophilus Influenzae)
- Antibiotic sensitivity: 70% produce penicillinase, so use Augmentin, cefaclor, tetracycline or cefuroxime (iv)

Other G-ives

- Escherichia coli, Klebsiella aerogenes, proteus mirabilis, other Coliform bacilli
- Cause: UTI, Pyelonephritis, abdominal wound sepsis, peritonitis, biliary tract infection (gallstones) or obstruction

Anaerobes

- Bacteroides fragilis, Clostridium perfringens, anaerobic streptococci
- Cause: Abdominal wound sepsis, peritonitis, pelvic sepsis, septic abortion, puerperal sepsis

Mycobacteria

- Classification:
 - Tuberculosis complex: M. Tuberculosis and M. Bovis
 - Other mycobacteria: M. Avium-Intracellulare (MAC), M. Kansasii, M. Marinum
 - Leprosy: M. Leprae
- Resulting Diseases:
 - Tuberculosis Complex
 - Immunocompetent: In descending frequency: lung, lymph nodes, kidney, genital tract, CNS
 - Immunodeficient: Lung in > 70%, but extra pulmonary involvement > 70% in blood (25 – 40%), lymph nodes, faeces, CNS due to ↓cell mediated immunity
 - MAC:
 - Immunocompetent: Kids – cervical lymphadenitis, adults: chronic destructive lung disease (uncommon)
 - Immunodeficient: Infection common. Initial colonisation of GI tract, then spread to blood, lymph nodes, liver, spleen, less lung involvement but invariably fatal
 - Most strains of MAC are resistant to standard anti-mycobacterial drugs
- Drug treatment:
 - Standard drugs: Rifampicin, Isoniazid, pyrazinamide, ethambutol. Normally first 3, except if from Pacific Islands where use all 4 due to ↑isoniazid resistance. Rifampicin is the best, if resistant to this then poor prognosis
 - Most strains of M Bovis are resistant to pyrazinamide
 - Many strains of M Tb from AIDS patients in the US (especially NY) are resistant to Rifampicin and Isoniazid
 - Other anti-mycobacterial drugs: ciprofloxacin, clarithromycin, amikacin, rifabutin, clofazimine
- Vaccination: BCG:
 - Live vaccine
 - Indicated for high risk infants: household has individuals from endemic areas of with past or current Tb
 - Neonatal BCG is 60 – 90% protective for extra-pulmonary Tb and 65% for pulmonary Tb. Protection lasts 10 – 15 years
 - Adverse effects: local abscess in 1%. Treated conservatively. Some require excision

Herpes Viruses

- All Herpes viruses exhibit latency

Herpes Simplex Virus (HSV)

- Manifestations: systemic (fever, sore throat), gingivostomatitis (ulcers with yellow slough – cold sores), meningitis (uncommon, self-limiting), encephalitis (fever, fits, headache, dysphagia, hemiparesis – do PCR on CSF sample – refer urgently)
- Incubation: 2 – 25 days. Chronic infection is due to the virus remaining in the sensory nerve ganglia. Infectious period indeterminate → contact isolation
- Symptoms:
 - Blisters which become shallow painful ulcers, often preceded by itching or tingling
 - First episode may be accompanied by flu like illness, tender inguinal nodes and dysuria
 - Recurrences can be brought on by stress, fatigue, depression, immunosuppression and concurrent illness. Recurrences usually less severe and become less frequent
- Diagnosis: clinical suspicion. Swab the base of an unroofed ulcer and refrigerate in viral medium. This will be painful. Culture negative doesn't exclude HSV as timing and collection technique important. Serology possible, but not routinely used

- Pathogenesis. There are two antigenic types of Herpes Simplex Virus:
 - Type 1 is associated with lesions on the face and fingers, and sometimes genital lesions. Treat with zovirax (topical cream). Prevalence: 70% of population
 - Type 2 is associated almost entirely with genital infections, and affects the genitalia, vagina, and cervix and may predispose to cervical dysplasia. 10% of oral lesions caused by type 2. Prevalence: 10 – 15% of population (depends on population – more in high risk)

Type 1 Herpes Simplex Virus

- Infection of fingers or thumb leads to a whitlow (vesicles coalesce)
- Can infect eczematous skin → eczema herpeticum
- Children:
 - HSV1 the most common type in children.
 - Primary infection in childhood leads to gingivostomatitis – may lead to dehydration as child won't drink. May need NG tube
 - Dribbling can → perioral spread
 - Auto-inoculation can → conjunctivitis, genital lesions, skin infection with eczema (eczema herpeticum) can be severe
 - If neonate or immunocompromised can be life-threatening
 - Treatment: Oral analgesics (eg lignocaine) and Paracetamol. Acyclovir

Genital Herpes (type 2)

- Description:
 - Painful, recurrent condition.
 - Male – anus or penis – small grouped vesicles and papules + pain, fever, dysuria. Dysuria may be severe enough to cause urinary retention
 - 20% may have it, but 20% are asymptomatic and 60% mild or unrecognised
- 40% caused by type 1, 60% by type 2
- Transmission: spread through skin-to-skin contact, usually when skin is broken or lesions present, but asymptomatic viral shedding a possible route of transmission. Neonatal transmission is rare (1 in 10,000 live births), but carries risk of ophthalmic infection ⇒ caesarean section indicated if active blisters at delivery
- Prevention of genital herpes: Condoms with new partner (although doesn't eliminate risk). Avoid sex during an outbreak
- Can have extra genital lesions on thighs and buttocks. Can → radiculoneuropathy → urinary retention/constipation
- Treatment of Genital Herpes (type 1 or 2):
 - Acute: Acyclovir 200 mg 5 times daily for 5 days. Topical creams not effective. Symptomatic treatment: salt bathing, local anaesthetic creams, oral analgesia, oral fluids. Counselling and follow-up important – written information for patients and partners, Herpes Helpline (0508 11 12 13)
 - Suppressive Therapy: Where frequent outbreaks or psychological morbidity. Acyclovir 400 mg BD for up to a year. Can reduce viral shedding by up to 95%
 - Can be devastating. Refer to counselling at Sexual Health Service
- Complications:
 - ↑Risk of AIDS transfer
 - Erythema Multiforme
 - Neonatal Herpes: 1 % transmission but 50% mortality
 - In pregnancy:
 - If first primary episode: miscarriage, prem labour
 - If recurrent, tiny risk for baby
 - If lesions at delivery then Caesarean

Varicella Zoster

- Primary infection: Chicken Pox.
 - Macules → papules → vesicles → crusts
 - Incubation 10 – 21 days (usually 14 – 16)
 - Infectious for 1 – 2 days before rash appears until it crusts over
 - Highly infectious, in hospital requires strict respiratory/contact isolation
 - Complications:

- Commonly becomes super-infected (eg with scratching) with Staph aureus (or S Pyogenes) which leads to scarring
- If immunocompromised → overwhelming infection, pneumonitis, hepatitis, encephalitis (treat with Ig and acyclovir)
- Post-natal infection can be overwhelming
- Immune response can → encephalopathy with cerebellar ataxia
- Can lead to severe exacerbation of eczema
- Then remains dormant in dorsal root ganglia
- Treatment: Supportive, antipruritic lotion if itchy, cut fingernails short
- Prevention in acute situation: Live attenuated virus, or im Ig within 96 hours of exposure if at risk and susceptible (immunocompromised, pregnant, newborn, prem babies)
- Tests: culture – swab transported in viral medium
- Vaccination:
 - Live attenuated vaccine recently licensed for both children and adults
 - Not recommended for general use, but role in protecting non-immune adults (more severe illness)
 - Contra-indicated if immuno-suppressed or pregnant
- **Shingles:**
 - Reactivation of infection: affects 20% at some time. Elderly and immunocompromised are high risk
 - Symptoms: Dermatomal pain, then fever malaise for several days, then macule-papules + vesicles, especially in thoracic or ophthalmic division of trigeminal dermatomes. If sacral, then urinary retention may occur. Thoracic (50%), cervical (20%), trigeminal (15%)
 - Complications:
 - If shingles around eye (especially end of nose), then are likely to have a dendritic ulcer on cornea. Stain with Fluorescein and shine on blue light, corneal abrasions will shine green. Don't give steroid → blindness. Urgent referral to an ophthalmologist.
 - Post-hepatic neuralgia – especially in the elderly and trigeminal
 - Recurrence rare and suggests HIV (or Dermatomal Herpes Simplex)
 - Treatment if needed: acyclovir as early as possible, 800mg 5 times a day for 5 days. Pain relief – analgesic or low-dose Amitriptyline. Maybe prednisolone to reduce post-herpetic neuralgia. Report visual loss immediately

Epstein Barr Virus

- DNA virus
- One of Herpes Group
- Spread by respiratory secretions (e.g. sneeze, kiss)
- Pre-schoolers an important reservoir: usually just a non-specific URT infection. In later life (e.g. adolescent) get it more acutely plus hepatitis. 1 – 5% present as hepatitis

Clinical

- Highly variable course. Often asymptomatic if < 5 years
- Sore throat (often exudative)
- Fever
- Lymph nodes up
- Tender liver (liver involvement → ↓appetite and ↑feeling unwell), maybe big spleen
- Rash in 10%
- Doesn't resolve (especially after antibiotics)
- Will be tired for weeks/months
- Incubation 30 – 50 days
- Association with symptoms:

	Sore Throat	Lymphadenopathy	Atypical Mononucleosis
EBV	+++	+++	+++
CMV	-	+	+++
HIV	++	++	++
Toxoplasmosis	+	+++	++
Viral Hepatitis	-	+	++

Investigations

- Throat swab

- FBC: may be ↑ atypical mononuclear lymphocytes
- EBV serology

Treatment

- Symptomatic
- **Don't** give penicillin if risk of EBV: leads to rash that can be interpreted as penicillin allergy. (E.g. amoxicillin, rash in 80 – 90%)
- Infectious for months. No isolation required
- Steroids if upper airway obstruction in kids

Antibodies to EBV

- IgM Anti-VCA (Virus capsid antigen) and IgG Anti-VCA
 - Usually appear in blood 7 days after symptoms develop in acute primary EBV infection
 - IgM: usually persists for 2 – 4 months
 - IgG: usually persists for life
- Anti EBNA (Epstein-Barr nuclear antigen): Appears 2 months after primary infection and persists for life
- Profiles:

	IgM VCA	IgG VCA	EBNA
No infection	-	-	-
Acute Primary	+	+	-
Past Infection	-	+	+

(ie EBNA +ive rules out acute infection)

- Paul-Bunell now largely obsolete. Negative in 10 – 15 % of cases

Associated diseases

- Burkett's lymphoma
- Nasopharyngeal carcinoma
- Hodgkin's disease (EBV in 40 – 60% of cases)
- Chronic EBV may occur but is very uncommon (recurrent sore throat, cervical lymphadenopathy)

Cytomegalovirus (CMV)

- Transmission:
 - Blood: transfusions, intra-uterine, perinatal, needle sharing
 - Cervical secretions and semen
 - Saliva (eg close contact with kids)
 - Urine (eg infants to adults)
 - Organ donation (transplantation)
- Immunocompetent:
 - Kids:
 - Common in preschoolers, usually asymptomatic. May give URTI
 - Prolonged excretion in saliva and urine common
 - Adults:
 - Usually asymptomatic, if not then usually self-limiting
 - May be fever (up to 2 weeks, ie a differential of PUO)
 - Sore throat, cervical lymphadenopathy uncommon
 - Atypical mononucleosis on blood film
 - Differential: EBV, HIV, toxoplasmosis
- Pregnancy:
 - Congenital infection (ie crosses placenta) in 20 – 40%
 - > 90% show no signs at birth, but watch for long term neurological sequelae (eg sensori-neural deafness, retardation)
 - Severe cases: respiratory distress, jaundice, microcephaly, etc
 - Part of TORCH complex: Toxoplasmosis, Rubella, CMV, HSV
 - Perinatal infection (eg during vaginal delivery):
 - Full term: usually mild
 - Pre-term: may be severe
- Immunodeficient:

- AIDS: one of the most common infections → CMV retinitis (common), CMV encephalitis (rare), CMV colitis (rare)
- Transplant: greatest risk if they're CMV negative and CMV positive organ → interstitial pneumonia and hepatitis (in liver transplant)
- Transfusion: blood is not routinely screened for CMV antibody. Should give CMV –ive blood to prem babies (<1500 g) and seronegative transplant recipients with seronegative transplants
- Lab diagnosis:
 - Serology:

	IgG	IgM
No infection	-	-
Past infection	+	-
Acute primary or reactivated infection	+	+
 - Cell culture – slow (>7 days). Culture lung biopsy or peripheral blood leucocytes
 - PCR for CMV DNA on peripheral leucocytes, amniotic fluid, CSF (very specific, less sensitive, very expensive)
- Treatment:
 - Ganciclovir: bone marrow toxicity
 - Foscarnet (nephrotoxic)
 - Ganciclovir prophylaxis used for –ive patients with +ive organs

Parasitology

Toxoplasmosis

- A protozoa/parasite
- Main source: cysts in meat. Also kitten faeces (eg cyst in garden – pregnant gardeners should wear gloves)
- Presentation:
 - Immunocompetent:
 - Lymphadenopathy (eg unilateral)
 - Maybe: fever, myalgia, acute pharyngitis, hepatosplenomegaly, atypical mononucleosis
 - Usually self-limiting – may take months to settle
 - If persistent/recurrent lymphadenopathy → ?Need for treatment
 - Immunodeficient:
 - Acquired or reactivated
 - AIDS most common: CNS involvement (solitary space occupying lesion, encephalitis), also myocarditis, hepatitis
 - Less common in transplants and encephalitis
 - Ocular toxoplasmosis: most cases in adolescents and adults → reactivation infection. → Blurred vision, photophobia, multiple retinal lesions
 - Congenital Toxoplasmosis:
 - 29% fetal infection if mother has primary CMV infection
 - Highest risk in 3rd trimester (1st trimester may miscarry)
 - Complications: spontaneous abortion, premature, still birth
 - Surviving neonates: bilateral choroïdo-retinitis. In severe cases, TORCH type symptoms
- Lab diagnosis:
 - PCR test for toxoplasmosis: amniotic fluid, CSF (AIDS patients)
 - Lymph node biopsy → characteristic histology
 - Serology:
 - IgM antibody after 5 – 14 days, peaks at 2 – 4 weeks, traces for up to a year
 - IgG: high levels for up to 6 months, declines slowly over years
 - Avidity test: can differentiate between acute phase 'immature' IgG and 'mature' IgG
- Treatment:
 - Pyrimethamine (Gold standard, but gives bone marrow suppression + give folate) + sulphadiazine (not available in NZ)
 - Pyrimethamine + clindamycin (gives C. difficile diarrhoea)
 - Spiramycin (only one safe in pregnancy)

Other

Malaria

- Transmitted by mosquito and very rarely transfusion
- Clinical: Irregular fever – peaks on release of parasite from infected RBCs. May only be mild if person has immunity (ie previous exposure). Various strains have various periodicities, chills, headache, malaise, vomiting (20%), diarrhoea (<5%) – ie similar to Typhoid

Amoebiasis (Entamoeba histolytica)

- Diagnosis:
 - Intestinal amoebiasis: stool sample * 3, 48 hours apart, in PVA fixative
 - Cysts: frequently present asymptotically (carrier state)
 - Extra-intestinal amoebiasis (eg amoebic abscess of the liver) maybe months later. Serum antibody test
- Treatment:
 - Intestinal amoebiasis: metronidazole then diloxanide furoate
 - Extra-intestinal: metronidazole (surgical drainage may be necessary)
 - Asymptomatic: Diloxanide furoate

Giardiasis

- Diagnosis:
 - Stool examination for Giardia Lamblia cysts, 3 samples 48 hours apart
 - Duodenal aspirate and direct examination for trophozoites
- Treatment:
 - Tinidazole 2g stat or Metronidazole 400 mg 8 hourly for 7 days
 - Test for cure with repeat stool sample. Relapse not uncommon

Filariasis

- Commonest is Wuchereria bancrofti imported from Samoa
- → Elephantitis
- Diagnosis: Blood sample
- Treatment:
 - Ivermectin
 - Most cases are asymptomatic or low grade pyrexia and don't require treatment
 - If severe, surgical relief of major lymphatic obstruction may be necessary

Intestinal Worms

- Hookworm:
 - Ancylostoma duodenale, necator americanus
 - Diagnosis: stool sample * 3
- Roundworm:
 - Ascaris Lumbricoides
 - Diagnosis: worms passed in faeces, or stool samples * 3 and examine for Ova
- Pinworm:
 - Enterobius vermicularis
 - Diagnosis: sellotape swabs of anus
- Whipworm:
 - Trichuris trichura
 - Diagnosis: stools * 3
- Treatment: mebendazole 100mg BD for 3 days for Hookworm, Roundworm, Pinworm (treat whole family) and whipworm (only if severe)
- Strongyloides Stercoralis:
 - Diagnosis: Stools * 3
 - Treatment: Thiabendazole
- Tapeworms:
 - Taenia saginata, beef tapeworm
 - Diagnosis: Stools * 3, examine for worm segments
 - Treatment: niclosamide

Hydatid Disease

- Aetiology: *Echinococcus granulosus* (a flatworm). Infected from ova excreted in dog faeces. Dogs infected from eating raw sheep offal (ie liver) containing hydatid cysts
- Clinical: Often acquired in childhood, present in older age with solitary cysts (liver, lung, brain)
- Treatment: surgical drainage + alendazole as adjunct
- Diagnosis: Serology: haemagglutination test + complement fixation test

Cryptosporidium

- Common protozoan parasite
- Profuse watery diarrhoea for 48 hours. Very common cause of diarrhoea.
- Severe and persisting cases in AIDS
- Diagnosis: Stool microscopy with ZN stain for acid fast cysts
- Treatment: Paromomycin (an oral, non-absorbable aminoglycoside) has some efficacy

Pneumocystis Carinii

- Protozoan parasite probably part of normal respiratory flora
- Causes interstitial pneumonitis in immuno-compromised patients (transplant, leukaemia, AIDS)
- Diagnosis: Bronchial lavage or open lung biopsy
- Treatment: Cotrimoxazole (alternatively pentamidine). Relapse in 25%

Antibiotic Treatment

Vaccination

- Reference: Public Health Module Notes

Vaccination Principles

- Jenner first vaccinated using cowpox against smallpox in 1796
- Characteristics of immunity:
 - Specificity: response to specific antigen
 - Priming
 - Memory: brisk secondary response
- Results of vaccine:
 - Most stimulate serum antibodies (IgG, IgM)
 - Some stimulate IgA (eg polio, rubella)
 - A few promote cell mediated reaction (eg BCG)
- Types of vaccine:
 - Live attenuated vaccine (eg OPV, MMR, VZ, BCG): full and long lasting immunity after a single dose (except OPV which requires 3 doses)
 - Inactivated vaccines:
 - First dose gives a predominantly IgM response. Further doses raise IgG level (depending on potency of the vaccine, maturity of the immune system and time interval)
 - Inactivated whole bacteria or viral vaccines: IPV, Hep A, Whole cell pertussis (being replaced).
 - Modified toxins (toxoids) eg Diphtheria, Tetanus → antibody response to toxin not infective agent
 - Sub-unit vaccines: eg Hep B, HIB, Pneumococcus, Influenza – the main focus of modern vaccines – conjugated vaccines with fewer side effects and easy to grow from genetically engineered yeasts etc.
 - Also passive immunity available from injectable IgG. Immediate protection lasting from weeks to months
- Population protection:
 - Immunisation is delivered to individuals and provides individual protection and benefit
 - Also provides population protection (herd immunity):
 - Some level of immunisation protects unimmunised people who would otherwise have caught it ⇒ don't need to immunise those for whom its contraindicated (eg too young or sick)
 - ↑Virulence ⇒ ↑coverage necessary to get herd immunity
 - 'Free riders' – because they perceive costs (needles, hassle, side effects) to be greater than perceived benefits → weakens herd immunity

- Efficacy and effectiveness:
 - Efficacy: Does intervention provide a specific outcome (eg an IgG response) under ideal lab circumstances
 - Effectiveness: Does it work under normal clinical circumstances
 - Apparent paradox: as coverage ↑, so does the proportion of cases that have been vaccinated (but lower absolute numbers of disease), due to vaccination failure. Can create the illusion that the vaccine is ineffective
- Vaccine failure:
 - Primary vaccine failure: inadequate physiological response to the vaccine (eg freezing or overheating of the vaccine, or poor host response)
 - Secondary vaccine failure: waning immunity
- Degrees of protection:
 - Generally provides 80 – 95% protection (BCG 50%, Influenza 70%)
 - May protect against severe disease rather than infection (eg Diphtheria)
- Vaccination coverage:
 - = Proportion of a population who have completed a specific course of immunisation
 - In Northern Region in 1996, 63% by 2 years but only 45% for Maori and 53% for Pacific islanders
 - With measles: ↑coverage → ↑time between epidemics as need a pool of 130 – 150,000 measles susceptible children to sustain an epidemic. Each epidemic → 50,000 kids contract measles and therefore immune in future. 10,000 unprotected kids added to the pool each year.
 - Policy measures: revise schedule to reduce the number of visits, immunisation certificates on enrolment at school/early childhood centre.
- Surveillance: Generally poor systems
 - Disease surveillance: notifications, discharge and mortality database, outbreak investigations, disease modelling
 - Coverage surveillance: registers and periodic surveys
 - Adverse event surveillance
 - Cold-chain monitoring

Vaccination Practice

- Practical vaccination standards:
 - Ensure correct storage and transport: maintain the ‘cold chain’ at 2 – 8 C. Eg have dedicated fridge and check its minimum and maximum temperature daily
 - Check vaccines due for each patient: either age groups (neonates, children, adolescents, adults, elderly) or specific exposure situations (occupational, travel, post-exposure)
 - Discuss and obtain informed consent: Written consent only required for children if care giver not present
 - Check contra-indications
 - Administer vaccine
 - Manage adverse reactions:
 - Observe for 20 minutes afterwards
 - Local or systemic reactions (fever, rash, joint pains): symptoms of immune activation. Offer Paracetamol. Especially whole cell pertussis. MMR may be followed about 7 – 10 days later by a 2 – 3 day fever and rash (but the vaccine is not infectious)
 - Anaphylaxis: Distinguish from fainting (which is common). Treatment: ABC, Adrenaline 1:1000 IM injection, 0.01 ml/kg, O₂
 - Report to centre for Adverse Reaction Monitoring if serious (includes persistent screaming > 3 hours and > 5 cm swelling at injection site), but also convulsions, meningitis within 30 days
 - Manage records: practice notes, HBL claim record and immunisation certificate for parents
- Anti-immunisation views:
 - Risks outweigh benefits: some diseases now rare and specific vaccines have serious side effects
 - Alternative health views: disease part of growing up (so was death!) and natural infection develops immune system
 - Plus a variety of beliefs/values that will be hard to shift
 - Main reasons for non-immunisation is ‘passive rejecters’ – don’t get around to it
- Contraindications:
 - Acute illness or fever > 38 C: defer vaccine. Otherwise will blame the illness on the vaccine!
 - Living with an immune suppressed person: use IPV rather than OPV

- Reaction to previous dose: encephalopathy with 7 days of DTP vaccines or immediate severe allergic reaction. If true anaphylaxis seek specialist advice
- Immune suppression: don't give live vaccine. Likely to have reduced response to inactivated vaccines
- Pregnancy: theoretical risk from live virus vaccines
- If in doubt, refer to a paediatrician
- False contraindications:
 - Mild illness, URTI, fever < 38.5 C
 - Asthma, hay fever, eczema
 - Prematurity and low birth weight in an otherwise healthy child – these especially need vaccination
 - Previous clinical history of illness: no harm done from vaccinating and many clinically diagnosed cases of an illness are in fact something else
 - On antibiotics, inhaled or low dose steroids
 - Stable neurological conditions (cerebral palsy, Down)

Currently Vaccine Schedule

- Current Vaccination Schedule from February 2002:
 - Covers Hep B, Diphtheria (child dose = D, adult dose = d – smaller), Tetanus, acellular Pertussis, Polio (now all intravenous = IPV, not oral), Hib, Measles, Mumps, Rubella

	DTaP- IPV	Hib- Hep B	Hep B only	IPV	MMR	DTaP- Hib	Td	Influenza
6 weeks	✓	✓						
3 months	✓	✓						
5 months	✓		✓					
15 months					✓	✓		
4 years	✓				✓			
5 years								
11 years				If not had 4 doses			✓	
45 years							✓	
65 years							✓	✓

- For unimmunised adults:
 - Give jabs over same timeframe
 - Don't need HIB, don't give paediatric dose of diphtheria (too big) and more inclined to use IPV
- Additional vaccination in specific age groups:
 - Neonates:
 - Babies of HBsAg +ive mothers: Hepatitis B immune globulin (HBIG) and vaccine at birth, vaccine at 6 weeks, 3 months and 5 months. Also offer vaccination to household and sexual contacts.
 - BCG if possible Tb exposure
 - Women of child bearing age who are susceptible to Rubella should be offered MMR
 - Adults: Td (after injury and at 45 and 65 – used to be 10 yearly) + annual influenza
 - Elderly: annual influenza + pneumococcal (5 yearly)
- Specific exposure situations:
 - Splenectomy: Pneumococcal vaccine
 - Occupational: Health care workers (eg Hep B) or HAV to food workers
- Future Developments:
 - Inclusion of Varicella Zoster and pneumococcal for children
 - Research into Group B meningococcal (currently 10 year epidemic, 250 cases per year), Rotavirus and RSV, non-infectious diseases including cancer

Vaccine Preventable Diseases

- Measles and Pertussis are the main ones still happening that we shouldn't have
- **Hepatitis B:**
 - Most effective means of control: vaccination: Engerix B. 85 – 90% efficacy
 - Yeast derived subunit vaccine.
 - Number of notifications has dropped from 400 to 100 since introduction in 1988

- Suspension of synthetic HBsAg
- Doses at 0, 1 and 6 months → immune levels of Anti-HBs in 92%.
- Check for seroconversion 2 months later
- Booster every 2 – 3 years if high risk
- **Diphtheria:**
 - *Corynebacterium diphtheriae* → respiratory and cutaneous infection (grey membrane on throat). Exotoxin causes cardiac toxicity and ascending paralysis. Spread by nasal droplets
 - 1 imported case in last 20 years. Till 1945 killed 100 babies a year. High in USSR in 90s.
 - Vaccine: inactivated diphtheria toxoid, boosters every 10 years. > 80% efficacy
- **Tetanus:**
 - *Clostridium tetani* from soil and animal faeces → muscular rigidity due to neurone specific toxin, 10% mortality
 - 3 notifications per year (old ladies in the garden). Common in environment ⇒ no herd immunity
 - Vaccine: Inactivated toxoid, boosters every 10 years, 100% efficacy
- **Pertussis:** See Pertussis, page 58
- **Polio:**
 - Enterovirus spread by faeces and saliva
 - Presentation:
 - Usually asymptomatic or mild (fever, headache, nausea, vomiting)
 - Only 1% of infected get severe clinical disease: severe muscle pain, neck and back stiffness → flaccid paralysis
 - Last wild virus infection in 1962. Occasional imported and vaccine associated cases
 - Vaccine:
 - Live oral polio (OPV) > 90% protection after 3 doses. < 1% of recipients develop diarrhoea, headache or muscle pains. 1 in 2.5 million recipients or close contacts develop paralysis (more common in immunosuppressed) = Vaccine Associated Polio Paralysis (VAPP)
 - Inactivated polio vaccine (IPV) for immunocompromised (will be used more widely when it can be combined with other jabs)
- **Haemophilus influenzae type B (HIB):** See Epiglottitis, page 58
- **Measles:** See Measles, page 74
- **Mumps:** See Mumps, page 74
- **Rubella:**
 - Togavirus spread by nasal droplets
 - Presentation:
 - Incubation 2 – 3 weeks
 - Fever, headache, mild conjunctivitis, erythematous maculo-papular rash, lymphadenopathy (especially posterior triangle), arthritis, arthralgia
 - 50% develop the rash and lymphadenopathy
 - 50% of adolescents and adults have arthralgia or even frank arthritis
 - 1 in 5,000 have encephalitis
 - Complications:
 - Congenital rubella syndrome: 90% of embryos of mothers infected in 1st trimester will abort or have major abnormalities (severely retarded, seizures, deafness, cardiac defects). Frequent problems after birth
 - Rate of congenital rubella is 5 times the US rate
 - ~ 60 notifications per annum (1600 in 1995)
 - Vaccine:
 - 98 % protective
 - To protect the unborn child only – relies on herd immunity. Need to vaccinate guys as well otherwise they will maintain a population reservoir which women with vaccine failure will catch
 - 5% of adolescents and adults have arthralgia and 1% have non-infectious rash
 - Contra-indicated in pregnancy and immunosuppressed
- **Influenza:**
 - Virus types A (H3N2 and H1N1) and B
 - Causes Fever, rigors, headache, myalgia, protraction. Estimated 400 deaths per annum.
 - Vaccine: inactivated subunit vaccine for new strains (resulting from ‘antigenic drift’). 60 –90% effective. Contraindicated if egg allergy

- Pandemics result from 'antigenic shift'
- Tb: **BCG**: See Mycobacteria, page 78
- **Pneumococcal Disease**: See Streptococcus Pneumoniae, page 76
- **Varicella Zoster**: See page 79

Congenital Abnormalities

Cleft Lip and Palate

- Failure of fusion of maxillary and premaxillary processes during week 5. With cleft lip, the lesion runs from the lip to the nostril, can be bilateral
- Incidence: 0.8 – 1.7 per 1000
- Cause: genes, drugs (benzodiazepine, antiepileptics), rubella
- Treatment:
 - Feeding with special teats
 - Surgery: repair lip at 3 months old, palate at 1 year old
- Prognosis: Unilateral or incomplete → good results. Bilateral lesions → some residual deformity
- Complications: otitis media, aspiration pneumonia, speech problems (refer to SLT)

Developmental Dysplasia of the Hip

- Encompasses Congenital Dislocation of the Hip
- Occurs after birth. Covers a spectrum from instability through subluxation to dislocation
- Commoner on the left. 25% bilateral
- Incidence: 1 in 1000
- Risk factors: extended breech, females, positive family history, first child, post-maturity, oligohydramnios
- Clinical: From 12 months shortening of the limb, external rotation and asymmetrical skin creases. Delayed walking, Trendelenburg gait and OA in early 30s
- Diagnosis:
 - Ortolani's Test: Flex hips to 90° then abduct them → click as femoral head slips back into the acetabulum
 - Barlow's test: Test for instability. Fix the pelvis with one hand and try and press the head and neck of the femur backwards out of the acetabulum
- Investigations. Neonatal ultrasound. > 4 months then xray
- Treatment: achieve and maintain a stable reduction. Neonate Pavlik harness. Later: open reduction
- Prognosis:
 - The earlier the treatment the better the outcome. Otherwise degenerative changes in the femoral head (eg anteversion), acetabulum, capsule, altered alignment
 - Poor prognosis: boy, late detection, Ortolani's negative (ie doesn't reduce easily)
- Clicking: a common finding and rarely associated with CCH

Club Foot

- Congenital Talipes Equinovarus
 - Small foot at birth, plantar flexed (equinus), heel in varus, forefoot displaced towards midline, fore foot inverted and lateral border convex, ankle is fixed, calf is wasted
 - Incidence: 1 in 1000. Twice as common in boys. 50% bilateral. Associated with other abnormalities (eg myelomeningocele)
 - Aetiology: multifactorial inheritance
 - Treatment: early diagnosis, stretching and strapping then serial casting from 10 days. Surgery at 12 weeks if not right yet to release tight tissues (eg tendons) on inner side of foot. Raised outside of shoe when walking. Follow-up: prone to relapse
- Calcaneo-Valgus Foot: Dorsiflexed and heel in valgus

Tarsal Conditions

- = Peroneal Spastic Flat Foot (old term)
- An abnormal union between one or other of the bones of the hind foot
- Autosomal dominant failure of segmentation or maturation of the mesenchyme
- Incidence 1%
- Diagnosis: flat foot as child with increasing stiffness of the hind foot. Progressive onset of pain in adolescence
- Diagnosis: lateral and oblique x-rays. MRI

- Treatment: 6 weeks casting, rigid orthosis, resection of the bar if found early, otherwise fusion

Flexible flat feet

- Normal in 10 – 15% of the population
- Check medial longitudinal arch of the foot reconstitutes when stand on tip toes (generalised ligamentous laxity)
- If rigid then needs investigation

Internal Tibial Torsion

- Internal bowing of the tibia caused by inter-uterine positioning
- Exclude other problems of hip, knee and foot
- Usually self corrects by age 5

Femoral Anteversion

- ↑ Angle between femoral shaft and neck – normal is 15 degrees
- Often ligamentous laxity and flexible flat feet. Intoeing presents at 2 – 4 years. Often blamed for frequent falls – however this is common with children with normal gait as well.
- Exam: intoed gait and excess internal rotation of the hip. Egg-beater running style
- Exclude neuro deficit.
- Treatment: Trend to correct up to to 8 – 10 years. Avoid sitting with legs in internal rotation. Osteotomy if deformity is severe and does not correct
- No evidence of improved outcomes from braces or footwear.

Scoliosis

- Lateral spine curvature
- Types:
 - Non-structural or postural curves, eg due to limb length inequality (curve disappears on bending forward)
 - Structural curves: has lateral deviation *and* rotation of the vertebra. When child bends forward there is a hump to one side and curve is still present/exaggerated eg congenital, neuromuscular, miscellaneous
- Idiopathic types often present during adolescent growth phase
- Causes pain, deformity and impaired lung function
- Usually progressive. Follow carefully or active management (casts or surgery)

Other Congenital Skeletal abnormalities

- Congenital Torticollis: tightness of SCM. Early input to prevent plagiocephaly, otherwise surgery
- Neurofibromatosis: Can present with skeletal overgrowth, tibial bowing, thinning, fracture. See Neurofibromatosis, page 146
- Osteochondritis juvenilis (osteochondrosis): bony centres in children/adolescents become temporarily softened → deformity due to pressure → harden again in 2 – 3 years in deformed shape
- Skeletal dysplasia: achondroplasia, osteogenesis imperfecta, plus numerous others
- Soft tissue disorders: Marfan's, plus numerous others
- Chromosomal disorders: Trisomy 21, 13, 18
- Metabolic: Numerous, including Wilson's, haemophilia
- Neuromuscular: Charcot Mari Tooth, Duchenne, Cerebral palsy
- Spinal dysraphism

Gait

- To develop gait need head and trunk control and standing balance
- Pathological gait can be due to:
 - Muscle weakness
 - Structural bone and joint abnormalities
 - Neuromuscular disorders
- Adult knee alignment by 7 years
- Bow legs present at 18 months – 2 years. Pre-walking legs are bowed and this only becomes apparent on walking. Not of concern unless asymmetry, severe deformity of another pathology suspected (eg rickets)

- Knock Knees: presents at 3 – 5 years, especially in obese children. Will usually resolve. Measure angle and monitor.

Bone and Joint Injury and Infection

- Differential of joint swelling:
 - Acute rheumatic fever
 - Septic arthritis
 - Reactive arthritis
 - Henoch-Scholein Purpura
 - Juvenile chronic arthritis
 - Sero-negative arthritis
 - Rickets and vitamin deficiencies: A, folate, B12, C
 - Transient synovitis
 - Trauma
 - Haemophilia
 - Osteomyelitis

Supracondylar Humeral Fracture

- Most common fracture above the elbow, typically extension injury by fall on outstretched hand
- Type 1: undisplaced. Type 2: displaced but some cortical contact. Type 3: Completely displaced
- Complications: nerve palsy (usually resolves after 6 - 8 weeks), vascular injury (esp brachial artery), compartment syndrome
- Treatment: closed reduction and percutaneous pin fixation. Non-displaced fractures without collapse of the medial or lateral columns can be treated by immobilisation. Open reduction if unsatisfactory closed reduction, open fracture or if vascular compromise

Medial Epicondyle Fractures

- Often accompanied by dislocation. Bony fragment may be trapped in the joint preventing reduction
- Usually treated non-surgically

Fractures of the Forearm

- 75% are fractures of the distal radial metaphases. Loss of reduction in 1/3 of cases

Wrist and Hand Fractures

- Radius and ulnar fractures account for 45% of childhood fractures
- Scaphoid fractures account for only 0.45% of paediatric upper extremity fractures
- 75% of finger injuries are stable and can be treated with simple immobilisation (often little finger)
- In toddlers and young children, most common pattern of injury is a crush injury of the finger, leading to distal phalangeal fracture, nail bed laceration and/or distal tip amputation
- In teenagers, diaphyseal level phalangeal fractures are common, with malrotation most apparent with digital flexion
- In teenagers, fractures of the metacarpal neck are common (“Boxer’s Fractures”)
- Fingertip trauma may lead to complete or incomplete amputation. Various treatment approaches. For more proximal amputations, replantation is now standard over 1 year. Best prognosis with sharp injuries (more common in adolescents, crush more common when younger)

Transient Synovitis of the Hip

- Transient synovitis is common and self-limiting, often following URTI.
- Hip or knee pain, limp, decreased motion but normal xray
- Diagnosis of exclusion. Main differential: septic joint. If in doubt, aspirate

Perthe’s Disease

- Legg-Calve-Perthes Disease: Poorly understood.
- Avascular necrosis of the femoral head, usually *boys* aged 4 – 8.
- Softens bone then gradually reforms in a deformed shape. ?Due to interference with venous drainage of the femoral head. May present as an incidental finding
- Earlier onset and smaller areas are indicators of better prognosis
- Usually benign. Maintain motion. Treatment controversial
- If extensive then degenerative arthritis by 5th decade

Slipped Upper Femoral Epiphysis

- Most common disorder of the hip in early adolescence, especially overweight and boys
- 90% are chronic and stable (can bear weight) with limp for several months
- Pain on abduction, flexion, internal rotation
- Often pain refers to the knee – knee pain in an adolescent is SUFE until proven otherwise
- Treatment: Percutaneous fixation

Femoral Shaft Fractures

- Common, generally solid healing
- Various treatment options including spica casting and traction
- Subsequent limb overgrowth is common but not predictable

Knee

- Patellar dislocation:
 - Congenital, habitual (due to quadriceps contracture) or recurrent
 - Predisposed to by generalised laxity, genu valgum, rotational malalignment
 - Initial management with extension bracing for 4 – 6 weeks (?controversial) then aggressive quadriceps building

Limb Length Inequality

- Various causes: check for soft-tissue hypertrophy, vascular anomalies, etc etc
- Often idiopathic. If mild (< 1.5 cm) then monitor with serial exam and x-rays
- Treatment depends on severity – involves surgical, gait, etc

Other

- Knee injury:
 - Osteochondral fractures of the knee: associated with patellar dislocations
 - Osteochondritis Dissecans: Fragmentation or separation of a portion of the articular surface of the knee ?secondary to localised area of underlying avascular necrosis. Symptoms include vague pain, clicking, popping or effusion. Initial treatment is immobilisation
- Physeal fractures of the distal tibia

Neurological Exam in Children

General (ALWAYS do these)

- Are they well or unwell (esp toxic)
- Growth:
 - Weight, height, and head circumference
 - Head exam:
 - Anterior and posterior fontanels (while upright). Anterior closes ~ 18 months, posterior ~ 4 months
 - Sutures, shape of head,
 - Check for shunts (subcutaneous tubes behind the ears),
 - Auscultation over closed eyes and temporal area for bruit
- Dysmorphic features
- Neurocutaneous stigmata: marks on skin, port-wine stains, Cafe-au-lait spots, use Woods lamp to look for depigmented lesions if fair skinned

Higher cortical function

- Ask questions appropriate to child's age
- State
- Attention: serial sevens, repeat numbers
- Memory:
 - Dependent on attention, processing and storing, ability to access and ability to communicate
 - Immediate (repeat numbers), recent (3 items at 5 minutes but not visual things), remote (old teachers name)
 - Object permanence
 - Visual: geometric reconstruction
- Reading and spelling
- Speech: dysphasia and dysarthria
- Draw a man
- Draw a clock face
- Following instructions
- Behaviour
- Right left discrimination (crossed)
- Name objects – visual agnosia
- Construction of complex geometric figure
- Sequencing: palm, hand on side, fist, repeated quickly
- Cortical sensation
- Abstract thought
- Looking for:
 - Frontal lobe disturbances: personality changes, irritability, lethargy, sphincter incontinence, primitive reflexes such as rooting, grasp re-emerge
 - Temporal lobe disturbances: altered ability to read, write and understand speech, memory dysfunction
 - Parietal lobe dysfunction: sensory perception abnormalities, 2 point discrimination, graphesthesia, stereognosis, apraxia

Cranial Nerves

- 1: Olfactory: don't often test unless abnormalities in the same area. Rarely impaired. Check each nostril separately. Use chocolate, mint or vanilla essence.
- 2: Optic
 - Visual Acuity:
 - Babies: fix and follow, optokinetic nystagmus, blink reflex (50% of 5 months, 100% by 1 year)
 - Toddlers: offer toys of different sizes. Look in books for smaller and smaller things

- Visual Fields:
 - Screen first: objects in the periphery – make sure they can't follow your arm to your hand
 - If suspicious: test with wiggly finger ('look at my nose and grab the finger that wiggles')
- Optic disc:
 - Very important
 - Use low light and small aperture
 - Get mum or dad to make funny faces behind you
 - Stay still and wait for optic disc to come into view
 - Look for venous pulsations – take pulse to get rhythm. If still can't see them, push lightly on orbit – if veins collapse then OK. If no pulsations then ? ↑ICP.
- Pupils
- III, IV and VI: Oculomotor, Trochlear and Abducens
 - Ptosis: nerve II and sympathetic. One eye doesn't open as much as the other
 - III: down and out
 - IV: Up and out
 - VI: in
 - Sun-setting: paralysis of upward gaze = pressure on quadregeminal plate
 - Get them to follow an object past the limit of head turning – don't hold head
 - Hold them to your stomach and spin around with their head out. Nystagmus is normal
 - Dolls eye
- V: Trigeminal
 - Motor: temporalis – bulk, power, clenching, chewing. Get them to bite on a wooden spatula while you pull it away
 - Sensation: test from out of sight with feather
 - Reflexes: jaw and cornea (only if unconscious or other signs point to a problem)
- VII: Facial
 - Taste: anterior 2/3: very hard in children
 - Lacrimation and salivary glands
 - Motor:
 - Tickle nose with tissue (try and get them to wrinkle face up)
 - Close eyes/mouth open: look for asymmetry of facial creases
 - Watch when crying – emotional movements less affected than voluntary ones (helps localised to UMN/LMN)
- VIII: Vestibulochoclear
 - Ask parents
 - Testing: whisper words (ice-cream, Wiggles) – rub fingers next to other ear (→ white noise)
 - Spinning
- IX and X:
 - Symmetry of uvula and palate movement
 - Swallowing
 - Gag: only if really necessary
 - Taste on posterior tongue: too hard
 - Voice: nasal 'b', 'd' and 'k', hoarse
- XI: Accessory shrug shoulders, turn heads with resistance, test rotation of head with pressure over chin
- XII: Hypoglossus: stick out your tongue at me – bulk, fasciculation, power. Poke tongue through cheek and feel it

Motor

- Observe:
 - Abnormal movements: ticks, seizures, chorea, etc
 - Bulk
 - Scars
 - Contractures
 - Symmetry (eg small thumb nail on one side – contra-lateral parietal lesion)
 - Posture: eg frog leg posture in hypotonia, fisting
- Tone:
 - Must be relaxed. Lie on back and shake arms and legs to a song
 - Range of movements: passive and active

- Power:
 - Functional: Observe, including:
 - Gait: walking forward and backward, running, hopping, tandem gait (eg heel-to-toe), on tiptoes, on heels, on insides and outsides of feet (Fog test). Look for dystonic posturing of hands while they do this.
 - Proximal weakness: up steps, Gower's sign, wheelbarrows, play ball, push-ups. Gower's: lie on back – tell them to get up as quick as they can when you say 'go'. Muscular dystrophy will roll onto front then climb up legs
 - Touch toes – look for scoliosis
 - Handedness
 - Formal strength testing (grade 5 down to 0): Pull a toy, push me away, crazy glue (pretend to stick their finger to their nose and then try and pull it away), resistance, squeeze fingers
 - Pronator sign
 - Remember:
 - Proximal weakness: myopathy
 - Distal weakness: neuropathy (except myotonic dystrophy)

Reflexes

- Must be relaxed, be patient
- Hit your hand, not the child
- Test ankle jerk on the sole
- Swing with gravity, don't bash
- Use distraction (look over there...) and reinforcement: clenched teeth (chewing sticky lolly)
- Check for clonus
- Primitive reflexes (go at various ages): Moro, ATNR (atonic neck reflex) – turn head suddenly → extend arm on that side, Babinski

Sensation

- Difficult
- Test from out of range
- Test with a broken spatula: show them sharp and dull and then always use sharp
- Touch, pain, vibration, proprioception

Cerebellar

- Gait: Walk along a line on the floor – should be able to do it well by 6
- Romberg
- Finger nose: reach for toys (make sure they stretch)
- Foot tapping
- Rapid alternating movement
- Hands outstretched with eyes closed, look for drift

Head Size *

Microcephaly

- Head circumference below the 3rd centile with abnormally slow head growth
- Incidence: 1/1000, recurrence in siblings 1/50
- Causes:
 - Familiar: not associated with developmental delay
 - Autosomal recessive condition, associated with severe learning delay
 - Congenital infection: Rubella, CMV, Toxoplasmosis, Varicella Zoster, Listeria, Syphilis
 - Brain insult: eg perinatal hypoxia, neonatal meningitis. Likely to be accompanied by cerebral palsy, seizures, visual impairment, etc
 - Fetal Alcohol Syndrome

Large Head

- Megalencephaly = oversized brain
- Hydrocephalus: dilated cerebrum:
 - ↑CSF volume associated with ventricular dilatation and ↑intraventricular pressure

- Due to:
 - Aqueduct obstruction: injury, infection or genes
 - Arnold Chiari Malformation (downward displacement and elongation of hind brain, with herniation into the cervical canal)
 - Acquired causes: meningeal adhesions, mass lesions, etc
- Chronic Subdural Haematomas
- Hydrocephaly: no cerebrum
- Benign familial anatomic megalencephaly or macrocephaly
- Metabolic Megalencephaly: late manifestation of many cerebral degenerative disorders (eg lysosomal storage diseases)
- Neurofibromatosis
- Cerebral Tumour

Headaches

- Epidemiology: 12% of adolescents missed one day or more of school in the preceding month
- History:
 - Pain characteristics: how bad, do they vary from one to the next, throbbing (migraine)/tight (tension)
 - Is it acute or chronic, recurrent or progressive, etc
 - Auras: visual, unilateral slowly spreading tingling/numbness/weakness
 - Photo & phonophobic
 - Look pale/unwell →?migraine
 - Late afternoon →? hypoglycaemic
 - Stress: relationship to headaches to school and holidays
 - Relieving factors: Sleep, medication
 - Past Medical History: head injury. To assess severity ask: did he lose consciousness, did he go to hospital *and* stay overnight, have any stitches or need imaging
 - Family history:
 - What kinds of headaches do the rest of the family get (don't talk about migraine – different meanings to different people)? There is a family history in 80% of migraine sufferers
 - Serious neurological disorders, strokes
- Exam:
 - General: Well/unwell, growth (if big head then measure parents), dysmorphic features, skin (stigmata)
 - General neuro exam: observe walk, cranial nerves including diplopia, arm and leg strength
 - BLOOD PRESSURE
 - ↑ICP: ↓venous pulsations in retinal veins, papilloedema, ↓visual acuity, 3rd and 4th nerve palsy
 - Focal neurological signs: especially cerebellar (common site of lesion in kids 2 – 10)
 - Cranial bruit to check for AVM: common finding. Interested in asymmetry, or if it can be eliminated by compressing the ipsilateral carotid artery
 - Check sinuses, teeth, TMJ

Differential

Migraine

- 70% of chronic headache
- Migraine Definition:
 - Recurrent paroxysmal headaches with pain free intervals with normal health, plus two of:
 - Unilateral pain, nausea, visual or other aura, family history in parents or siblings
- Presentation:
 - 25% start with aura:
 - abnormalities of vision: flashing lights, coloured lines, blurred vision or visual loss
 - Perioral tingling or sensory loss in one limb (described as a “heaviness” but power normal)
 - Aura is stereotyped – changes between people, but for one person will always be the same
 - Lasts 5 – 60 minutes – if longer, or a different aura, then red flag
 - 75% without Aura:
 - *Gradual* onset of throbbing headache

- +/- nausea/vomiting/photo-phono-phobia. Key differential from chronic daily/tension headache: in migraine they have headache but also feel awful, with chronic daily/tension headache it's just the headache
- Resolve after 2 – 24 hours or on waking from sleep
- Neurologically normal between attacks
- Associated with migraines:
 - Motion sickness – patient and family
 - 'Ice-cream' headaches – like shooting pain into head when biting an ice-block
 - Benign Paroxysmal Vertigo of Infancy (not the same as adult BPPV): 2 – 3 minute episodes of unsteadiness, queasiness, nystagmus
 - Cyclical vomiting
 - Abdominal migraine

Acute Tension Headaches

- Occurs equally in boys and girls
- Presentation: Pain all the time, pressure over the whole head (“vicelike”, “pressure”, “squeezing”), no associated symptoms, no neuro signs
- Management: try and identify the cause of stress: school, family break-up, abuse

Chronic Daily Headache

- 10% of chronic headache.
- Doesn't have to be daily – but very regular
- Naming subtleties: Chronic Tension/Stress Headache if you can identify the cause, Chronic Daily Headache if you can't (or they have forgotten the precipitant and are now stressed about having headaches)
- Particularly in teenage girls. Rare before adolescence – children normally somatise rather than tense up
- Presentation: Pain all the time, whole head, no other symptoms, without well defined onset and ending, less impairment of function, don't look unwell (cf migraine), no neurological signs
- May co-exist with migraine: “do you have more than one type of headache”
- Try and identify cause of stress – stress can cause both migraines and Chronic Daily Headache so doesn't help with diagnosis
- Long term prognosis: 95% resolve in 6 months

Brain Tumours

- Headache is first symptom in 50%
- Vast majority diagnosed within 4 months of first symptoms (be beware slow growing frontal tumours)
- Pain is caused by focal distortion of meninges & blood vessels → dull, constant, focal pain
- ↑ICP → initially early morning headache, becoming constant. Effortless vomiting without nausea often relieves headache. Suspect if increasing severity, or wake in the morning or at night with a headache
- Key differential: severity and frequency of headache increases with time
- Location:
 - Infratentorial: 50%. Associated with ataxia/cranial nerve signs
 - Supratentorial: 50%. Associated with pyramidal signs/seizures

Other

- Benign intra-cranial hypertension: ↑ICP of unknown cause. Small or normal sized ventricles and no space occupying lesions. Complication of some viral illnesses. Steroids may be used.
- Sinusitis: Chronic sinusitis doesn't cause headaches. Very common to have x-ray evidence of asymptomatic sinusitis so don't xray head
- Whiplash Headache: Due to muscle contraction to limit neck movement. ?History of trauma. Can be longstanding. Occipital not frontal. If pain persists, check neck again – if Xray normal then CT. Confounded by medicolegal implications
- Drugs: eg daily use of analgesics
- Non-specific: whole head pressure, not obviously anything else. Don't force into a diagnostic group. The vast majority will improve. Ensure review

Management

- First, make a diagnosis
- Reassure: most parents seek help to check its not serious (eg brain tumour)

- Education: Migraines are familial, due to ischaemia and vasodilation (which stretches pain fibres in blood vessels)
- Symptom diary: check for food association with migraine (fairly rare). Chocolate, cheese, red wine, oranges and orange food colouring (aspartame) – no other evidence re food. Don't try restrictive diet. Instead keep diary of headaches, with food eaten in the preceding 6 hours and look for associations.
- Avoid triggers
- ?Psychologist referral:
 - ↓Stress, get to the bottom of stress problems, relaxation, coping
 - It will be life long – learning skills to cope better than life long medication
- Medication:
 - Analgesia. Issue is how quickly you get it in, not which one you choose. Paracetamol: need a big dose and right at the start to make a difference (otherwise ↓gastric motility and fail to stop spread). Can add maxalon if older → ↑absorption of pain killer. Also consider naprosyn.
 - Propranolol: tested in RCT, but not if asthmatic
 - Pizotifen (Sandomigran): less substantial evidence, weight gain
 - Epilim: Some RCT evidence, weight gain
 - Amitriptyline
 - Riboflavin: from health food shops, growing evidence of effectiveness
 - Ergotamine: contraindicated if complex migraine (focal neurological signs)
- Refer when:
 - Physical signs → neurologist
 - Concerning social history
 - Diagnosis not clear and reassurance has failed
 - Diagnosis has been made but the child fails to respond to treatment
 - Use of CT should be very rare (exceptionally low yield) – main use is to reassure parents when you've tried reassurance and then symptoms have persisted

Seizures and their Differentials

Seizure Diagnosis

- Classification:
 - Either partial or generalised
 - And one of:
 - **Acute symptomatic**: any person in that situation would seize eg hypoglycaemia, heatstroke, meningitis, hyponatraemia. Seizure will stop when cause goes away (unless scarring – when it becomes a 'remote symptomatic' seizure)
 - Single
 - Benign Febrile Convulsion
 - **Epilepsy**: Repeated unprovoked seizures
 - If Epilepsy then:
 - Localised: Idiopathic, symptomatic or cryptogenic
 - Generalised: Idiopathic, symptomatic or cryptogenic
 - Unclassified
- Seizures:
 - A symptom – not a diagnosis or a disease
 - Hyper-synchronous excessive discharge of CNS neurons associated with a clinical sign
- Diagnostic process:
 - Is it a seizure or not – *a very important question* – don't skip past it. If recurrent, get the family to video it
 - What type of seizures – is it generalised or localised, etc
 - Is it a single seizure, acute symptomatic, afebrile or epilepsy
 - If epilepsy, what syndrome is it (this step is critical to treatment and prognosis, but often ignored in practice)
 - Brain tumours cause 1 – 2 % of all seizures in children, and 4 – 6% of partial seizures
- Sorting out type of seizure:
 - When do the seizures occur
 - Does patient know they're going to have a seizure
 - What can the patient recall

- Detailed description from observers:
 - Are they aware – will they respond
 - Are their automatisms
 - Is there dystonic posturing
 - How long did it last
- After the seizure: are they confused, can they speak, any post-ictal Todd's
- EEG:
 - Should be done on all children with afebrile seizures before starting medication
 - Need to do when sleep deprived and not on medication
 - 2 – 4 % of normal children have an abnormal EEG
 - 55% of epileptic children have an abnormal EEG
 - Background:
 - Melody
 - Looking at frequency, morphology, location, reactivity, symmetry, etc
 - Changes with age and state
 - Abnormal = dysrhythmia (focal or generalised)
 - Paroxysmal events:
 - Noises
 - Benign variants: associated with age and state
 - Epileptiform: inter-ictal and ictal
- Neuro-imaging:
 - Indicated for:
 - Neurological deficit/asymmetry
 - Neurocutaneous syndrome
 - Developmental regression
 - Partial seizures
 - Infantile spasms or myoclonic seizures in 1st year of life
 - Persisting unclassifiable seizures
 - Brain seizures cause 1 – 2% of all seizures in children and 4 – 6 % of partial seizures
 - Not indicated for:
 - Idiopathic generalised epilepsy
 - Benign childhood epilepsy with centrotemporal spikes (Rolandic)
 - Simple Febrile Seizures
 - CT:
 - Initial scanning technique for exclusion of tumour
 - Show calcification
 - Available and easier to perform
 - MRI:
 - Preferred imaging technique
 - Sensitive to migrational abnormalities or very small lesions
 - PET/SPECT scan: localise lesion on the basis of metabolism. Only if considering epilepsy surgery

Seizure Types

- Generalised: bilaterally symmetrical without local onset
 - Tonic-clonic (Grand mal) seizures: Tonic phase: 10 – 20 secs – extension phase then tremour begins – repetitive relaxation of tonic contraction. Clonic phase: usually 30 seconds, random movements, tongue often bitten
 - Absence (Petit Mal) Seizures: Characteristic type of absence attack. Childhood or adolescent onset, associated with 3/sec spike and wave on the EEG. Blank stare and unresponsive for 5 – 15 seconds. No post-ictal confusion or sleepiness. May also have automatisms and mild clonic motion (usually eyelids at 3 Hz). May be induced by hyperventilation. 80% have no further seizures after 20 years old. Can also have atypical absence seizures. Treat with ethosuximide or sodium valproate. Don't use carbamazepine or phenytoin.
 - Atonic: complete, sudden loss of tone – completely collapse, may injure themselves
 - Tonic: sustained contraction, maybe with fine tremour
 - Myoclonic: Sudden, very brief jerk but still generalised
 - Clonic: rhythmic jerking
 - Infantile spasms:

- Sudden bilateral symmetrical jerk, extensor or flexor. Can be subtle, come in clusters
- Usually around 3 – 6 months, boys > girls
- Grow out of the spasms
- Bad prognosis: cerebral palsy, retardation, etc
- Medical emergency: try to urgently get them under control
- Partial: Begin locally
 - In simple partial seizures consciousness is preserved.
 - Complex partial seizures are focal seizures in which consciousness is altered (eg blank unresponsiveness followed by automatisms, eg lip smacking, other semipurposeful activity) – usually temporal lobe but may be frontal. Can go on for minutes. Aware it is coming (cf absence which is sudden)
 - Partial seizure secondarily generalised: they have an awareness first
 - Localising it:
 - Preceding aura: olfactory, visceral, auditory, visual, déjà vu
 - Dystonic posturing: contraction of agonist and antagonist muscles
 - Post-ictal Todd's Syndrome: if they have one area of weakness after a seizures (ie one hand weaker than the other) then it started locally
- Automatic behaviour usually seen in complex partial seizures: but can be in absence (petit mal) seizures. Eg Oral or manual automatisms
- Seizure location:
 - Frontal: focal tonic or clonic motor activity, posturing, prominent motor automatisms but no orofacial or experiential automatisms
 - Central: focal clonic seizures with preservation of awareness
 - Temporal: experiential, gustatory or olfactory hallucination. Motion arrest, automatisms
 - Parietal: exclusively somatosensory manifestations
 - Posterior: polymodal sensory, visual, auditory or somatosensory hallucinations

Epilepsy in Childhood

- Definition: Two or more unprovoked seizures
- Incidence: 0.5 – 1 %
- Lifetime prevalence of a seizure is 5%
- Management of children with new onset epilepsy:
 - Should be referred to a paediatrician so the specific syndrome can be managed
 - If a catastrophic syndrome, or have not responded to two medications, then to a paediatric neurologist
- Aetiology:
 - Idiopathic: seizures are the only symptom, normal kids, no structural abnormality, often family history, EEG normal, generally benign and good prognosis. Cause is assumed to be genetic – usually a channelopathy. Usually easily controlled with medication, and the child usually outgrows the condition.
 - Symptomatic: An underlying cause is known (eg previous hypoxic ischaemic encephalopathy), and there are usually other signs of a problem
 - Cryptogenic (Presumed Symptomatic): There are other problems besides seizures, eg retardation, focal signs, etc, but can't find a cause
 - Symptomatic and Cryptogenic: abnormal children, abnormal background on EEG, prognosis not so good and often seizures difficult to control
- Common epilepsy syndromes in childhood

	Idiopathic	Symptomatic/cryptogenic
Generalised	Childhood absence epilepsy Juvenile absence epilepsy Juvenile myoclonic epilepsy	Infantile spasms
Partial	Benign epilepsy with centro-temporal spikes Early onset childhood occipital epilepsy Late onset childhood occipital epilepsy	Mesial temporal lobe epilepsy with hippocampal sclerosis

- Benign focal (Rolandic) Epilepsy of Childhood:

- = Benign Childhood Epilepsy with Centrotemporal Spikes
- Commonest focal seizures in children
- Onset 3 – 10 years
- 80% focal, especially mouth and face. 50% only have fits in sleep
- EEG diagnostic. Prognosis excellent
- Childhood absence epilepsy:
 - Onset 4 – 10 years – often confused with daydreaming
 - Frequent daily absences
 - Neurologically normal
 - 90% outgrow the absence seizures, but 30% will have GTCS in adolescence
 - Usually easily treated
- Psychosocial aspects: More important than drugs. Peoples attitudes will do far more damage than a few seizures
 - Education
 - Counselling
 - Normalising
 - Respite and support for families
 - Refer to the NZ Epilepsy Association
- Seizure precautions: when driving, swimming, bathing (have showers instead), scuba diving, cycling (always wear a helmet), etc. But lead a normal life!!!
- Seizures more likely if: sleep deprived, unwell (particularly if febrile), stressed, missed medications
- Pharmacology:
 - Aim:
 - Seizure free with no side effects
 - Start low and go slow
 - Never stop abruptly
 - Start after 2 or more seizures
 - Stop after 2 years seizure free
 - Common drugs:
 - Carbamazepine
 - Valproic acid: Great for generalised seizure types. Side effects: weight gain, alopecia, neural tube defects, nausea, tremor, NOT rash. Serious side effects: Hepatotoxicity (< 2 years), thrombocytopenia, platelet dysfunction
 - Less common:
 - Clobazam
 - Ethosuximide: Absence only. Not so good for juvenile absence.
 - Phenytoin
 - Phenobarbitone
 - Rarer:
 - Vigabatrin
 - Lamotigine
 - Things to be concerned about on AEDs:
 - RASH!!
 - Behavioural problems
 - Anything
 - When to do levels:
 - Hardly ever
 - Phenytoin
 - Status
 - ?Compliance
 - Mentally impaired who can't describe toxic symptoms
- First aid advice on seizures:
 - Stay calm
 - Ensure safety
 - Watch and time seizure so they can describe it accurately
 - If it hasn't stopped in 5 minutes, call an ambulance
 - After seizure, put in the recovery position
 - If had a previous prolonged seizure, plan for use of rectal diazepam

- Sudden Unexplained Death in Epilepsy Patients (SUDEP): 1:900 – but the vast majority in symptomatic epilepsy. Risk of death in idiopathic syndromes is the same as for children without epilepsy.
- Status Epilepticus:
 - Continuous or intermittent fitting > 30 mins
 - Damages brain through lack of substrate (ATP). In kids with seizures > 1 hour, 30% will have new neuro deficit
 - Management:
 - Always consider what's causing the seizure
 - ABCDEFG – does airway need protecting. ALWAYS measure glucose and blood pressure (acute hypertensive encephalopathy)
 - Think ahead – “what will I do if this doesn't work” – saves time.
 - Benzodiazepams:
 - All have similar onsets. Good approach: 2 doses of rectal diazepam
 - Lorazepam – less respiratory depression, longer effect, not usually available IV
 - Diazepam – used rectally and IV. Lasts 20 minutes, then can seize again.
 - Midazolam – can give IM and intra-nasally
 - Main mistake is too many repeated doses → apnoea
 - Followed by other anticonvulsants if still seizing. Consider:
 - Paraldehyde
 - Phenytoin. Can't be mixed with dextrose, has to be given over 30 minutes, can cause thrombophlebitis (always flush well)
 - Phenobarbitone (can be infused faster)
 - Magnesium
 - Final option: general anaesthesia – but won't know if they still seizing – have to trust the anaesthetic drugs

Benign Febrile Convulsion

- Types of seizure occurring with fever:
 - Benign febrile convulsion
 - Epilepsy: eg first epileptic seizure unmasked by fever
 - Acute symptomatic seizure: meningitis, etc
- Benign Febrile Convulsions:
 - Frequency: 2 – 5% of all children
 - Age: 6 months – 5 years
 - Temperature: usually > 38.5 C
 - Boys: Girls = 1.4:1
 - Family history common (polygenic inheritance)
 - Unrelated to prenatal and perinatal brain damage
 - Have seizures with fever *only*
 - Treatment:
 - Stop seizure: rectal diazepam (0.5 mg/kg) at home or in ambulance
 - Find the cause of the fever: seizure won't hurt them but meningitis might!
 - DON'T use anticonvulsants – decrease recurrences but potentially significant side effects to treat something that is benign
 - Prevention: Avoid over heating. Paracetamol/ibuprofen or tepid sponging don't ↓ risk of seizure
 - Types:
 - Simple (75%): generalised, brief (< 15 minutes), does not recur in 24 hours
 - Complex (25%): Focal and/or prolonged and/or recurrence within 24 hours
 - Recurrence: 30% will have a recurrence, 70% of these within the next year
 - Risk of subsequent epilepsy:
 - Increased risk if neurologically abnormal prior, family history of epilepsy or first seizure is complex
 - Otherwise very slight increase in risk only
 - Neurological sequelae:
 - No impact on behaviour, growth, IQ
 - Prolonged febrile seizures associated with hippocampal sclerosis (temporal lobe). Controversial. (eg which direction is causation)

- Parental education:
 - Seizure does not cause brain damage (if less than 30 – 45 minutes)
 - 30% chance of another seizure
 - Next time: stay calm, clear airway, recovery position when stopped (low tone), time the seizure
 - Call ambulance at 5 minutes:
 - Most seizures stop by 5 minutes – so those that haven't are more likely to go on for longer
 - Want to be at hospital and have it stopped by 45 minutes

Anoxic Seizures

- White breath-holding attacks, Pallid syncope, Reflex Anoxic Seizures:
 - Vaso-vagal events due to stimulus: eg anger, pain, vomiting, etc. Stimulus minor compared with result [Big sympathetic drive → parasympathetic overcompensation????]
 - Occur in children from 2 – 10. Grow out of it
 - Reflex *bradycardia* or brief asystole or peripheral vasodilation
 - Symptoms: pallor, ocular revulsion, don't breath, extensor posture, a few symmetrical clonic movements, spontaneous resolution and then fine
 - Can't distinguish from arrhythmia ⇒ do ECG
- Blue breath-holding attacks:
 - 1 – 5 years
 - Follow a stimulus, eg crying. Deliberate breath holding
 - Get worked up, don't breath in, run out of breath and don't breath in (actually stop breathing)
 - Cyanosis with retained heart rate
 - May lose consciousness and have some clonic movements
 - Blow on face to start breathing
- Both white and blue breath holders are often iron deficient → do dietary history and Hb test
- Don't treat them. Ensure parent isn't being manipulated by breath-holding by child

Convulsive Syncope

- Syncope → short tonic-clonic seizure
- Presyncopal (get history of preceding light-headedness) → collapse (with very pale face) → briefly stiff → few jerks → rapid recovery (eg 5 – 10 minutes, not post ictal)
- Due to a lower threshold to hypoxia
- ECG a wise precaution to exclude arrhythmia

Syncope of Cardiac Origin

- Causes:
 - Prolonged QT: provoked by emotion/exercise and sleep. May be family history
 - WPW
 - Valvular disorders – eg syncope on exercise if aortic stenosis

Pseudoseizures

- Most common in adolescence, neuro-developmentally impaired children and children with epilepsy
- Clues: not responding to therapy and episodes not stereotypical (vary from one to the next)
- Movements tends to be semi-purposeful, suggestible (eg ↑ vigour if limbs held), not symmetrical. No post-ictal phase.
- If normal IQ then often serious underlying stressors – consider abuse.

Day Dreaming

- Confused with absence seizures (5% of childhood epilepsy, often multiple times a day, at home and school, very sudden onset. Hyperventilation provokes an absence seizure. EEG will always show spike and wave → diagnostic)
- Common in children with learning disability (whereas most kids with absence seizures have normal IQ)
- Last a long time. Often bored

Parasomnias

- Night Terrors:
 - 18 months – 5 years
 - Persist into adolescence in 30%
 - Partial arousal from the first cycle of stage IV sleep

- Always occurs 60 – 90 minutes after going to bed and only once per night
- No memory of events
- Somnambulism: Non-REM events in older children. Simple activity with semi-purposeful movement (eg going to toilet) and no memory of event
- Other seizures at night:
 - Frontal lobe seizures – abnormal behaviours, maybe rhythmical movements or abnormal posturing of a limb, get up and about, not rouseable. Will have them at other times of the night and maybe multiple times per night
 - Rowlandic seizure: will wake first, drooling, hemiclonic, speech arrest, aware

Tics

- Brief, sudden involuntary stereotyped purposeless movement involving the muscles of the face, extremities and trunk
- Worsens with anxiety
- May be able to be controlled for a short while
- Go away in sleep
- If florid, look like repeated myoclonic seizures
- Chronic Motor Tics:
 - Goes on for more than a year
 - Life long tendency
- Chronic Multiple Tics:
 - = Gilles de la Tourette Syndrome
 - Onset usually 2 – 15 years
 - Boys > girls
 - Tendency for life
 - Multiple motor and vocal tics which wax and wane
 - Wide spectrum from minor → severe

Cerebral Palsy

- = A persistent disorder of posture or movement caused by a non-progressive, non-hereditary lesion of the immature brain, acquired either in utero or later at a time of rapid development of the CNS (up to several years after birth)
- May be accompanied by other impairments, eg retardation, vision defects or epilepsy
- Though lesion is static, the clinical features may develop for several years as brain function matures (may give appearance of being progressive – clinical signs have to wait until that part of the brain ‘kicks in’)
- Incidence:
 - Stable at about 2/1000.
 - 80/1000 for very preterm babies
- Stages of brain development:
 - Up to 20 weeks → major brain malformations:
 - Lissencephaly (brain without cortex)
 - Microgyria: lots of little indentations
 - Migration defects: islands of grey matter in the middle of white matter
 - 26 – 32 weeks: neurons climb glial fibrils: intense growth – prone to ischaemia. If born then, prone to germinal matrix bleeds. But ischaemia more important → Periventricular Leukomalacia (PVL)
 - Myelination starts at about 30 weeks, but most is after birth. Damage only becomes obvious as myelination complete (conscious control of arm at 4 – 5 months, leg at 9 months)
- Causes:
 - Anything that damages neurons: ischaemia, hypoglycaemia, infection, trauma, toxins
 - Only 10-30% attributed to “intrapartum asphyxia”. Normal PO₂ in utero is 15 – 25 mmHg ⇒ well adjusted to hypoxia
 - For many it’s due to an unknown earlier adverse event
 - Significant proportion preterm (43%)
 - Intrauterine Growth Retardation → ↑risk by 5 times
- Exam findings:

- Hyperactive reflexes
- Abnormal movements of chorea, athetosis, dystonia
- Abnormal absence or persistence of infantile reflexes
- Differential:
 - Metabolic disorder
 - CNS degenerative diseases
 - Cerebellar dysgenesis or spinocerebella degeneration

Classification

- **Hemiplegia:**
 - 0.79/1000
 - Congenital:
 - Spastic paralysis of arm and leg on same side. Full term: arm usually weaker than the leg. Preterm: leg weaker than the arm. Distal parts worse than proximal. Growth of affected parts reduced
 - Face not involved
 - Epilepsy common – correlates with degree of mental retardation (but IQ often normal)
 - Mechanism: vascular (ie stroke in utero). If preterm, usually periventricular rather than cortical and leg weaker than arm
 - Acquired:
 - Following infection, trauma, CVA, status epilepticus, etc
 - Most in first 3 years of life
 - Flaccid with facial involvement, spastic later
- **Diplegia:**
 - 0.9/1000
 - Spastic:
 - Problem in preterm 28 – 32 weeks
 - Follow bilateral periventricular injury, especially with hydrocephalus complicating, or injury to basal ganglia or parasagittal cortex
 - Stiff lower limbs (may be floppy as neonates): flexion of hips and knees, scissoring (internal rotation and adduction), weak trunk and eventual contractures. May dislocate hips
 - Upper limbs variably affected (if worse then more global)
 - Hyperreflexia and spasticity with variable wasting
 - Epilepsy uncommon, intellect may be retained (69%). Head growth mirrors intellect
- **Quadriplegia:**
 - Global cerebral insult: massive haemorrhage, shock, obstructed umbilical chord
 - Upper limbs often worse than lower, generalised spasticity and wasting.
 - Severe mental retardation, cranial nerve palsies, aspiration, etc
- **Athetoid:**
 - Extrapyramidal injury, especially perinatal insults (including kernicterus – unconjugated hyperbilirubinaemia)
 - Appears after 5 months: involuntary movements and posturing, poor trunk control, hypotonia or normal, normal reflexes
 - Impaired speech, drooling, facial grimacing, often deaf (especially high tone)
 - IQ often normal but difficulties communicating. Epilepsy in 25%
- **Ataxic:**
 - Cerebellar symptoms predominate
 - Cerebellum abnormal on imaging
 - Presents at 1 – 2 years, but floppy and docile from the start
 - Ataxia, intention tremor, late walking, high tone deafness, normal IQ in 50%
 - Can be familial disease
- Management:
 - Team approach: physio, OT, orthopaedic surgeon, etc
 - Prevent contractures and encourage normal developmental stages (?botox injections)
 - Treat epilepsy
 - Rule out deafness, check special senses
 - Encourage communication
 - Prevent malnutrition

- Encourage mobility and upright posture (frees up hands for 'learning' activities)
- Support for child and family
- Manage constipation, incontinence

Muscular Dystrophy

- Not common. Most common is Duchenne: 1 in 3,500
- X linked recessive, females usually asymptomatic. 1/3 new mutations
- Caused by failure to make dystrophin (in muscle cell membrane)
- Present with muscle weakness that is slowly progressive. Gower's sign: proximal weakness → climb up their legs with hands to stand up
- Often mild intellectual handicap (IQ 85)
- Wheel chair bound by 12. ↓Respiratory function and ↑scoliosis → terminal bronchopneumonia
- Diagnosis: genetic tests, CK markedly elevated, myogenic pattern on EMG
- Treatment: supportive only

Acute Weakness in Childhood

- Consider:
 - Guillian Barre (LP shows elevated protein but normal cells), post-infectious (eg mycoplasma), often sensory involvement, treat with Ig
 - Transverse Myelitis: post-infectious, distinct spinal level, responds to steroids

Neural Tube Defects

- A neural tube defect – failure of closure of the neural tube (4 weeks gestation – often already happened by the time a woman knows she's pregnant)
- At lower ends leads to spina bifida and at upper end anencephaly or encephalocele
- Rate varies on population. High in Irish, Welsh, Scottish (3%) and those from poor backgrounds (poor nutrition, ↓folate, etc)
- Multifactorial causes:
 - Genetic
 - Environmental (eg diet)
 - Drug associations (eg antiepileptics)
- Any midline lesion of the skin overlying the CNS from the nose to the sacrum may indicate a lesion below the skin (same embryological origin) – eg hair, pigmentation, etc

Types

- **Myelomeningocele:**
 - Most common: 90% of spina bifida, failure of caudal closure of neural tube → failure of closure of skin and absence/leaking of the dura/meninges.
 - Lumbar sacral (25%), lumbar or thoracolumbar (50%) or thoracic/cervical (11%).
 - Spinal cord opened out flat. Variable neuro deficit below lesion. Possible tethering
 - Leads to:
 - Paraplegia: paralysis of knee and hip extensors with retained flexion. Talipes (club foot) – equinovarus is commonest
 - Variable loss of sensation
 - Autonomic problems: faecal incontinence, dribbling urinary incontinence on lifting baby or spastic urethral sphincter (→ urinary retention), spastic bladder (→ reflux, hydronephros)
 - Open lesion → risk of ascending infection
 - Hydrocephalus:
 - Very common (Arnold-Chiari malformation). Dislocation of cerebella tonsils and medulla into cervical canal, aqueduct stenosis (?primary lesion or tethering).
 - Signs of chronic hydrocephalus and ↑ICP: bulging fontanelles, rapid head growth, poor feeding, separation of sutures, 'sun-setting eyes' (looking down), drowsiness, venous congestion of skull.
 - If acute: vomiting, bradycardia, hypertension. Rare in infants as the fontanelle takes the pressure
 - Aside: Two types: Obstructive hydrocephalus (eg aqueduct stenosis) and communicating hydrocephalus (eg blockage of arachnoid granulations due to blood, puss, etc)

- Hydrocephalus is a prognostic factor: none → do well. Present at birth → intellectually impaired
- Management:
 - Caesar preferred
 - Interdisciplinary team
 - Close back to prevent infection
 - Drain hydrocephalus (ventriculoperitoneal shunt)
 - Bladder and bowel management
 - Review motor and sensory function, prevent contractures and aid mobility
 - Etc
- **Spina Bifida Occulta:** Range from failure of formation of dorsal spine (cord intact) to abnormal cord contents
- **Diastematomyelia:** bone or cartilage spur into the cord → progressive loss of spinothalamic function (pain and temperature) with growth (slices as spine elongates). Not common. Leads to regression of acquired skills.
- **Lipoma:** fatty mass → pressure effects. May involve cord. Don't just excise it!
- **Tethered cord:** complication of many types. Cord fixed lower down and gets stretched as spinal column grows → loss of power, sensation and autonomic function (ie sphincter function, weakness in toes and forefoot, saddle anaesthesia). Initially in lower myotomes.
- **Dorsal Dermal Sinus:** Epithelium lined tube from skin (lumbar/sacral) to dura or into spinal canal. Risk of meningitis (coliform) and tethered cord. MRI to confirm.
- **Meningocele:**
 - Rarer. Swollen lesion on back, full of CSF, brilliant translumination. Little neurological deficit, risk of tethering
 - Cranial meningocele: occurs on skull and contains CSF
- **Encephalocele:** occurs on skull and contains brain. Prognosis more guarded
- **Anencephaly:** Failure of cephalic closure of neural tube → absence of cranium. Frequent polyhydramnios. Most live births die within 24 hours. Also occurs in other syndromes (⇒ always do karyotype)

Prevention

- Folic acid levels in pregnant women only half the recommended
- Recurrence after one affected child is 3 – 5% (?inborn error of folate metabolism)
- Low dose folate prophylaxis highly effective – but 50% pregnancies unplanned
- Adequate dietary intake hard (5 portions of broccoli a day!)
- Can detect with antenatal ultrasound or ↑maternal or amniotic fluid alpha-fetoprotein (higher concentration in CSF than amniotic fluid, so if CSF leaking it will be raised in amniocentesis)

Eye disorders in Children

Assessment

- Routine eye checks for infants:
 - Fixing and following: ophthalmology referral if not doing this by 4 months
 - Pupillary red reflexes: view from about 50 cm. Leukocornea (white pupil) ⇒ ?retinoblastoma. Other irregularities ⇒ ?congenital cataract
 - Ocular alignment: symmetrical corneal light reflex (don't have to be exactly central). Strabismus (misalignment of visual axis) → amblyopia. May be intermittent. Test with cover test. Accommodative Esotropia = convergent strabismus related to accommodation
 - Eye movements: if not following then test vestibulo-ocular reflexes using dolls eye
 - Adnexa Oculi: Eyelids. Check for Congenital Naso-Lacrimal Duct Obstruction (tears, puss or mucus discharged by pushing on lacrimal duct) due to incomplete canalisation. Most resolve by age 1 (⇒ usually managed conservatively by twice daily lacrimal sac massage)
 - Globes and cornea: of equal size
- Serious disorders in the neonate (→ urgent referral):
 - Congenital Glaucoma: photophobia, corneal haze/opacity, corneal enlargement or asymmetry
 - Ophthalmia Neonatorum: conjunctivitis with infection and inflammation of the conjunctiva in first month of life. Urgent microbiology and iv antibiotics for chlamydia and/or N Gonorrhoeae
- Vision: fixation (test independently and together), pictures, symbol matching, E

- Alignment: inspection, alternating cover test
- Squint inspection:
 - Corneal reflection when looking at bright light source. Should be in the centre of the pupil on both sides. Cover good eye and see if corneal reflection shifts over the pupil of the bad eye
 - Check for equal sclera on either side of iris. Wide bridge of nose may give pseudo squint
 - Can have squint without amblyopia as long as brain alternates which eye it looks through. If preference for one eye, then amblyopia

Red Eye in Children

- *Conjunctivitis*
 - Common in newborns – may be serious
 - Bacterial: rapid onset, usually spills from one eye to the other. Puss.
 - Neonatal often Neisseria gonorrhoea (prevented with silver nitrate drops in new born if high risk). Marked lid oedema, profuse creamy discharge, early corneal ulceration. Can lead to perforation of orbit. Urgent referral. If systemic spread then septic arthritis. Treatment: *B Penicillin 25 mg/kg/12hr iv + 3 hourly 0.5% chloramphenicol drops for 7 days*. Treat parents.
 - 3 – 5 days post delivery: Chlamydia. Can progress to rhinitis and pneumonitis. Diagnosis requires special chlamydia swab. Treatment: *Erythromycin 10mg/kg/6hr po for 21 days* to eliminate lung organisms + *1% tetracycline drops (unavailable!)*. Treat mother and partner – oral erythromycin if pregnant or breastfeeding
 - Acute causes often Staph aureus, S pneumoniae, H influenzae or S pyogenes. Treatment: drops up to hourly (eg chloramphenicol)
 - Chronic: usually toxins or immune (eg Kawasaki, Erythema Multiforme, Reiter’s Syndrome)
 - Viral: acute onset, often bilateral, minimal pain, thin watery discharge, photophobia. Adenovirus, Herpes Simplex, measles, etc. Generally clears spontaneously.
 - If Herpes suspected (eg eyelid vesicles), start 4 hourly acyclovir and immediate referral. Dendritic ulceration with neovascularisation. Chronic inflammation and scarring. May lead to small white vesicles around the eye. Viewed with fluorescein drops under cobalt light (stains where there is no epithelium). Branching pattern ⇒ Herpes Simplex Virus. Never give steroids: → worse infection → permanent damage. Neonatally, 60% of infections have no maternal history
 - Adenovirus types 8 (epidemic) and 3 and 7 (sporadic). Conjunctivitis with pre-auricular lymph node hyperplasia. Over about a week get small white spots (WBC accumulations) just below the surface of the cornea
 - Allergic: history of atopy and itchy eyes. If mild then use astringent, topical anti-histamine or cromoglycate
- *Subconjunctival haemorrhage*: common after blunt trauma (eg birth), coughing (eg whooping cough) and vomiting.
- *Corneal abrasions*: trauma or infection (esp HSV)
- Iritis/Uveitis: uncommon in children. May have no pain but strabismus or visual loss. Cornea red near iris (unlike conjunctivitis). Look for white cells in anterior chamber.

Amblyopia

- = “Lazy eye”
- Affects 2 to 3 per 100 children. Can only occur in childhood while visual pathway still developing
- Usually unilateral: maybe bilateral if bad astigmatism or hypermetropia. If unilateral no effect on reading/writing. Treat as insurance against problems in good eye
- Affects central vision: peripheral vision OK
- Three major causes:
 - Squint: Most common cause: misaligned or crossed eyes. The crossed eye is ‘turned off’ to avoid double vision
 - Unequal focus (refractive error). One eye is more near/far sighted or astigmatic
 - Visual obstruction: eg Cataract
 - Also caused by ocular motor defects
- Treatment: force the use of the weak eye by covering the good one (for weeks or months), plus correcting refractive errors with glasses

Refractive errors

- Myopia

- Hypermetropia: if equal and severe then squint due to accommodation
- Stigmatism
- Anisometropia: difference between two eyes (especially if one normal and other long sighted) – accommodation just makes normal eye go out of focus

Other

- Congenital cataract
 - Can be autosomal dominant
 - Check for red reflex within 6 weeks
 - May be uni or bilateral, part of a syndrome or isolated
- Congenital epiphora (tearing)
 - Watery eye. Common – lacrimal system not fully developed. 5% have symptoms of naso-lacrimal duct blockage at 6 weeks
 - Nose is not wet
 - Spontaneous resolution the norm – 90% at 1 year, fewer after that. Conservative treatment until 12 months, then prodding. Massage of naso-lacrimal sac +/- antibiotics (stagnation of tear drainage)
 - Refer ASAP with nasolacrimal sac swelling (substantial risk of orbital cellulitis)
- Perinatal eye infections
- Retinoblastoma
- Retinopathy of prematurity
 - Very premature babies (low risk if over 30 weeks or 1200 g)
 - Spectrum from severe to norm
 - Problem with vascularisation → retinal detachment over time
- Congenital Glaucoma: rare. One cause of red watery eye. Wet eye and wet nose.

Ear Testing

- Voice Testing
- Tuning fork tests:
 - Rinne Test: 512 Hz fork beside the ear. If conductive loss then bone conduction is better than air conduction. If sensorineural, air conduction best
 - Weber Test: Tuning fork on top of the head. Louder in affected ear if conductive loss, softer in affected ear if sensory loss
- Pure Tone Audiometry:
 - Can establish severity of hearing impairment and whether sensorineural or conductive
 - Measures thresholds across a range of frequencies. Threshold = lowest intensity that can be detected
 - Usually only test in range of conversational speech (250 Hz to 8 KHz)
 - Normal hearing is 0 – 20 dB (zero is based on population surveys)
 - Harder if child aged 3 – 5: need to play games etc
- Auditory Brainstem Response (ABR):
 - Detects evoked potentials in the brainstem in response to sound
 - Used for neonatal testing (reliable from full term), in older kids where behavioural responses are unclear and for testing the auditory nerve (eg acoustic neuroma – but MRI is gold standard, CT with contrast poorer)
- Tympanometry:
 - Measures compliance of middle ear
 - Normal is -100 to 100 daPa
 - Type A: normal (peak compliance over 0 daPa). If peak is low ?scarring or adhesions
 - Type B: Flat curve (ie not compliant at any pressure).
 - Low volume type B: wax impaction or middle ear infection
 - High volume type B: perforation or grommet
 - Type C: Peak shifted to the left. Eustachian tube obstruction
- Otoacoustic emissions:
 - Test for cochlear function, eg in neonatal screening
 - Also for tinnitus: is it cochlear or non-cochlear
- Paediatric testing:
 - 0 – 3 months: referred from neonatal high-risk register. Need to correct (eg hearing aid implants) by 9 – 10 months otherwise speech impairment

- 6 – 12 months: distraction testing – looking for head turning, etc
- 1 – 2½ years: in a room with speakers

Hearing Loss

- See Hearing, page 26 for developmental delay resulting from hearing loss

Congenital Sensorineural Deafness

- Irreversible
- Pathology: problems with nerve or cochlear
- Profound hearing loss at birth: 2 per 1,000
- Most often detected by parents (ie believe them!)
- Aetiology: genetic or acquired, etc:
 - Idiopathic 60%
 - Genetic: most are spontaneous mutations rather than family history
 - Low birth weight
 - Infection (fairly rare now), eg Rubella, also toxoplasmosis, CMV, syphilis
 - Maternal drugs: eg aminoglycosides, alcohol
 - Lots of others, eg hypoxia, high bilirubin

IDDM – Type 1 (Juvenile Onset Diabetes)

- A chronic, progressive autoimmune process in genetically susceptible people, triggered by environmental factors
- Eventually cannot survive without insulin treatment. Ketoacidosis will develop unless insulin given (if any endogenous insulin then no ketones)
- Incidence up to 20 yrs: 10 – 15/100,000
- Prevalence: 0.25 – 3 % (10 – 15% of all diabetics)
- Peak age of incidence is 12 – but can present at any age (even after 40). Surges in presentation at 3- 4, starting school. ?Viral exposure
- 85 – 90% have no family history, but family history confers ↑ risk
- Siblings of Type 1 children have 3 – 4% risk of getting diabetes. If antibody positive then significantly ↑ risk, if antibody negative then no risk in next 5 years
- Kids presenting with mild hyperglycaemia: don't know if they will become IDDM or are MODY (Maturity Onset Diabetes Of The Young – ie Type 2). So when start insulin replacement back titrate (after stabilised) – type 1 may have honeymoon period until no endogenous insulin
- Currently being investigated for prevention in high risk individuals (ie have antibodies but not frank disease):
 - Cow's milk avoidance until 6 months of age
 - Early oral insulin therapy → autoimmune modulation
 - Nicotinamide (vitamin B) supplementation – now disproved
- Treatment goals: stable blood sugar, prevent/monitor complications, promote normal growth and development, maintenance of normal weight

Management of Hyperglycaemia

- Acute presentation: hyperglycaemia (polyuria when glucose > 10 mmol/l, thirst, polydypsia), tiredness, weight loss. Also cramps, blurred vision, superficial infections.
- Ketoacidosis (now rarer):
 - Also has nausea, vomiting, and drowsiness
 - BSL > 14 mmol/L, metabolic acidosis with pH < 7.3 or HCO₃ < 15 mmol/L and moderate to severe ketonuria
 - Degree of acidosis doesn't correlate with glucose
- Examination: check for degree of dehydration, LOC, temperature (often hypothermic), precipitating causes (eg infection)
- Investigations:
 - Blood sugar level
 - U & Es (watch for high K – although they will overall be K depleted)
 - Urine: ketones and culture
 - Capillary or venous blood gas for pH (unless oxygenation concerns)
 - Antibodies if newly diagnosed:
 - If negative suggests type 2
 - Islet Cell Antibodies: risk of IDDM ↑ with ↑ level of ICA. Frequency in newly diagnosed IDDM is 65 – 85%. Frequency in population is < 0.5%
 - GAD (glutamic acid decarboxylase) antibodies: mildly specific antigenic enzyme released from islet cells when destroyed. Can test for these in prodromal stage
 - Insulin autoantibodies
 - Also thyroid function tests and antiendomysial IgA if newly diagnosed
 - 24 hour urine and measure C-peptide: a by-product of insulin production (have they any endogenous insulin – as long as replacement insulin hasn't → islet cell atrophy)
- Management:
 - If haemodynamic instability then 20 ml/kg bolus normal saline
 - Commence careful IV then oral rehydration
 - Insulin infusion: 0.1 units/kg/hour (a big dose, often insulin resistant). Glucose will ↓ quickly. Ketones take longer to be metabolised so acidosis may persist. Adjust dextrose (not insulin) to stop glucose falling to far
 - K⁺ unless anuric or level > 5.5 mmol/L

- Watch for pseudo-hyponatraemia (measurement error due to ↑ glucose). Na will probably be normal, but will be reported as low. Expect measured Na to ↑ as glucose ↓
- Watch for cerebral oedema (and nurse head up):
 - Severe morbidity/mortality
 - Warning signs: headache, sustained bradycardia, age inappropriate incontinence, altered sensation
 - Risk factors: age < 5, 1st episode of DKA
 - If suspected then urgent 20 % mannitol 1g/kg – treat now, investigate later

On going management of Type 1 Diabetes

- Initial insulin regime:
 - Testing: initially 3 – 4 times daily, including overnight (checking not hypo)
 - Insulin doses for smaller kids:
 - Starting dose 1 unit/kg/day – if not ketoacidotic on presentation then 0.75 u/kg/day
 - Given as 2/3 intermediate acting, 1/3 short acting, 2/3 in morning, 1/3 in evening
 - Diet – essentially a healthy diet
- Regular food:
 - ↓ refined carbohydrates, prefer foods with low glycaemic index
 - Carbohydrate counting: regime where they judge how much carbohydrate is eaten each time and adjust insulin accordingly (pretty complex approach), and use basal bolus (multiple daily injections) of long acting at bedtime then fast acting each time you eat.
- Management of intercurrent illness (eg gastroenteritis)
 - Check BSL regularly (2 – 4 hourly) and ketones (every urine)
 - Insulin: must keep it going but in smaller amounts – eg regular amounts of short acting (eg 10% of total daily dose every 3 – 4 hours, OR give half morning long acting and top up with short acting)
 - Add in extra oral glucose with rehydration
 - Recently: low dose glucagon therapy as long as glycogen stores not exhausted (high dose → vomiting)
- Unexplained hypoglycaemia:
 - Test for associated autoimmune disorders: Coeliac disease, Addison's, (also thyroid disease – but less impact on glucose levels than the first two)
 - Alcohol → ↓ gluconeogenesis → hypoglycaemia
- In adolescents: ↑ risk of eating disorders and suicide (chronic disease + available method)

On going management of Type 2 Diabetes

- Lifestyle – diet and exercise
- Drugs: metformin (safe in kids) then straight to insulin. Sulphonureas → weight gain
- Monitoring:
 - Glucose testing
 - HbA1C. Any reduction worthwhile. Target is ≤ 7.0. Not all willing or able to achieve this
- Screen for risks factors (may have had it for a while at presentation):
 - Cholesterol & lipids
 - Microvascular complications: eyes, kidneys. Fundoscopy

Long Term Complications

- Microvascular disease:
 - Due to thickened walls and laying down of advanced end glycosylation products
 - Eye disease: mainly retinopathy. After 30 years 80% have background retinopathy and 7 – 8% are blind. Also ↑ Sorbitol → cataracts.
 - Nephropathy
 - Neuropathy: peripheral and autonomic
- Macrovascular disease:
 - Coronary heart disease. Male diabetics have 2 times risk, females 4 times risk. Very high risk if other risks present (eg ↑BP, lipids, smoking etc)
 - Accelerated atherosclerosis (but lesions look the same)
- Kidney disease:
 - Onset of diabetes leads to:
 - Functional changes: ↑GFR, reversible albuminuria

- Structural changes: GBM thickening, mesangial expansion
- Nephropathy has two phases:
 - Normal blood pressure, creatinine, and urines but microalbuminuria.
 - Overt neuropathy: proteinuria, hypertension, ↑creatinine and ↓GRF
- Glomerular damage:
 - Nodular glomerulosclerosis. Acellular hyaline material (Kimmelstiel-Wilson Lesion): BM proliferates (ie collagen expansion of mesangial matrix) → sclerosed and fibrotic due to fibroblast infiltration → chronic renal failure. Earliest sign is microalbuminuria, due to pores getting bigger
 - Diffuse glomerulo-sclerosis: glomerular loop obstruction → necrosis (seen in hypertension or any end-stage renal disease)
- After 30 years, 30 – 40% have nephropathy. Unlikely if hasn't developed after 30 years (?some protective factors)
- Papillary necrosis: least blood supply → susceptible to ischaemia
- Also pyelonephritis and reflux lead to kidney damage
- Investigations for Microalbuminuria:
 - Normal level < 20 mg/24 hours. Microalbuminuria = 30 – 300 mg/24 hours. Dipsticks detect > 150 g/l (ie insensitive)
 - Microalbuminuria hard to test (needs 24 hr urine). So use albumin : creatinine ratio. Normal < 2.8 in men, < 4.5 in females in random test
 - If abnormal result then patients qualify for statins with cholesterol > 6 (normal threshold > 9)
- Immune deficiency: White cells affected when glucose > 14 mmol/l
- Neuropathy:
 - Glycosylation of nerve
 - Demyelination of nerve due to sorbitol accumulation in Schwann cells → slowed conduction
 - Peripheral sensory AND motor neuropathy (eg foot deformity, fallen arches)
 - Autonomic neuropathy leads to bladder problems, impotence, gastroenteropathy
- → Diabetic foot
- Special management in surgery, pregnancy and in intercurrent illness

Proteinuria

- Definition: > 150 mg protein/day (same cut off as adults)
- Normally protein is lost from tubular cells. Pathological if:
 - Filtered protein from glomerulus
 - ↑Loss from tubular cells
- Categories:
 - Gross proteinuria: > 1 gm/day (→ nephrotic syndrome if severe)
 - Acute low grade
 - Chronic low grade
- Diagnosis:
 - Dipstick: measures concentration of protein, so if urine is concentrated → ↑protein concentration as well
 - 24 hour urine: problem if not continent
- Nephrotic syndrome:
 - = Proteinuria + oedema + ↓albumin in blood (hypoproteinaemia)
 - Oedema is due to ↓colloid osmotic pressure → ↑aldosterone → ↑Na → ↑H₂O retention → this leaks out as well
 - Caused by leaky glomeruli

Causes

- Minimal Change Disease:
 - = No change under light microscope
 - Passing up to 8 – 10 gm per day → gross oedema
 - 3 rare complications:
 - Hypoperfusion: classically the gut → abdominal pain
 - Loose Ig's as well → ↑risk of bacterial infection (eg pneumococcal)
 - Thrombosis (eg renal vein)
 - Usually grow out of it (eg over 6 months, although may persist until an adult). Unpleasant but not usually life-threatening
 - Treatment: steroids but side-effects
 - 10 – 20% have other causes which may → chronic renal failure
- Acute low-grade proteinuria:
 - No long term significance
 - Can be:
 - Exercise induced in some teenagers/adults
 - Urinary tract infection
 - Postural proteinuria (↑when standing up)
 - Have to demonstrate that it's gone (ie that its not chronic)
- Persistent/chronic low grade proteinuria
 - Always have some. Exclude exercise and postural
 - Significant finding: ↑risk of renal disease, eg in adult

Haematuria

- Definition: > 5 RBC in high powered microscope field
- Categories:
 - Gross/Macroscopic:
 - UTI (most common cause by far)
 - Trauma: visceral damage easier in kids
 - Post-streptococcal glomerulonephritis
 - Stones
 - Wilm's tumour
 - Bleeding disorder (eg haemophilia)
 - Red food colourings (eg beetroot)

- IgA nephropathy: IgA deposits on glomeruli
- Acute microscopic:
 - Infection
 - Plus above list (which are more likely to be microscopic than macroscopic, and may be intermittent/chronic)

Acute Renal Failure

- = Acute renal 'success'. If kidney didn't shut down after ATN, would continue filtering at 1-2 l/hr with no reabsorption → very rapid dehydration
- Causes:
 - Ischaemia
 - Obstruction (eg congenital malformation)
 - Sepsis: toxins killing tubular cells + hypoperfusion
 - Drugs: at glomeruli or tubular cells
- Complications:
 - Fluid overload: fluid restrict to insensible losses (breath, stools, skin = 400 mls/m² of body surface) plus urine and vomit
 - Hyperkalaemia:
 - No symptoms, so have to monitor
 - Treatment: salbutamol or insulin + glucose → shifts K into ICF
 - Calcium resonium: chelates K
 - Encourage anabolism with ↑ calories → ↑ cell creation
 - Frusemide if any urine output
 - CaCl or Ca Gluconate: prevents arrhythmia
 - Dialysis
 - Uraemia: vomiting, encephalopathy → dialysis
 - Hypertension: due either to ↑ aldosterone release or fluid overload

Chronic Renal Failure

- Incidence: 1 – 2 kids per million per year
- Disaster for families → very demanding treatment
- Causes:
 - Obstruction/congenital
 - Dysplasia (never developed normally)
 - Severe reflux
 - Glomerulonephritis
- Problems:
 - Nutrition (need ↑↑ calories → ?NG tube)
 - ↓ Linear growth: due to ↓ nutrition, ↑ PO₄ → secondary hyperparathyroidism, ↓ vitamin D
 - Anaemia: due to ↓ erythropoietin
 - Na/H₂O/K balance (may loose or retain too much)
 - Hypertension: angiotensin, overload, drugs
 - Ca balance
 - Development (always tired)
- Treatment: peritoneal dialysis

Ureter

- Congenital abnormalities:
 - Double/bifid ureters
 - Megaureter
 - Hydroureter
 - Usually present with UTIs
 - May have abnormalities elsewhere
- Ureteritis:
 - Associated with generalised UTI
 - May be caused by stones lodging the ureter
 - Rarely caused by Tb

Urinary Tract Infections

Epidemiology

- UTI is common:
 - 5% of febrile infants aged 2 months to 2 years have a UTI
 - Males usually have them in their first year, for girls it's on going
 - By age 7, 8% of girls and 2% of boys will have had at least one episode
- Caused by E coli in over 80% of cases. Others are associated with complicated UTIs or long term antibiotic therapy (eg Candida)
- Of 1000 kids with UTI:
 - 400 have vesico-ureteric reflux, 100 have renal scars, 10 will develop premature hypertension (eg in older childhood or pregnancy), end stage renal failure in 1
 - 10 – 20 will have obstruction due to urethral valves, VU or PUJ obstruction
 - Greatest risk usually kids < 4 and especially in first year of life
- Risk factors for UTI:
 - Previous infection
 - Normal anatomy but functional problem: e.g. vesico-urethral reflux (in child, sibling or parent)
 - Structural abnormality: e.g. urethral stenosis/stricture (more common in boys – congenital, trauma or inflammation)
 - Vulvovaginitis from poor perineal hygiene
 - Incomplete or infrequent voiding
 - In first year of life, uncircumcised male is 10 times that of circumcised
 - Sexual abuse: only 2% of patients investigated for sexual abuse have UTI as a symptom. UTI without other indications (lesions, bleeding, bruising) is very unlikely to be sexual abuse
 - Antibiotics: disrupt normal peri-urethral flora → predispose to infection
 - Constipation a risk factor: ask about this
 - Indwelling catheter
- Risk factors for VUR (prevalence 1 – 2 %):
 - Children with UTI (30 – 40%)
 - Siblings affected (30 – 50%)
 - Antenatal dilation of the urinary tract (8 – 22%)
 - No evidence that prophylaxis → ↓renal scars (controversial)
 - VUR is a risk factor for acute pyelonephritis and renal scarring (→ 5 – 10% risk of hypertension, proteinuria, progressive renal failure)
 - Management of VUR is controversial: Medical vs surgical. Renal scarring and function comparable. Reduction in pyelonephritis with surgery.
- Renal Scars:
 - Outcome already determined by renal status at the time of original presentation – persistent defects likely to be from pre-existing lesions.
 - Scarring independent of grade and persistence of VUR and recurrent UTI
 - If bilateral scars with hypertension and reduced renal function then increased risk of fetal loss and accelerated maternal renal disease

Diagnosis & Treatment

- Always have appendicitis as differential diagnosis: can have white cells in urine with appendicitis where appendix is in the pelvis (or elsewhere)
- Symptoms are highly variable:
 - 0 – 2: Fever/hypothermia (?sepsis), lethargy, poor feeding, diarrhoea, vomiting, abdominal distension, failure to thrive
 - 2 – 5: fever, rigours, vomiting, diarrhoea, colic, abdominal pain, some dysuria, offensive urine, haematuria, weak urine stream
 - 5 – 12: fever, rigours, abdominal pain (⇒ upper tract infection), dysuria, frequency, urgency, incontinence, haematuria
 - If systemic illness then ↑ likelihood of pyelonephritis as well as cystitis (fever a good indicator of pyelonephritis). If under one, can have pyelonephritis without systemic signs → if UTI under age 1 then presume Pyelonephritis
- Diagnosis:
 - Urine bag:
 - Wash genitalia before application
 - Test with urine dipstick. If positive, obtain definitive sample with catheter or supra-pubic aspiration (SPA)
 - Do not routinely send bag specimens for culture. Boys have 93% false positive
 - Positive predictive value of leukocytes and/or nitrites only about 35%, with NNT+ive of 2.9 and NNT-ive of 105
 - Catheter:
 - For children who can't void on request and where the bladder is in the pelvis (SPA won't work)
 - Uncomfortable. Discard first few mls
 - Growth > 10E6/litre suggest infection
 - Supra-pubic aspirate:
 - If child too young to obtain an MSU
 - Gold standard: any growth suggests infection (but beware contamination with skin commensals)
 - Preferable to catheter (purer sample) and risks are few (haematuria OK afterwards). However, if oligouric then may not get sample and need to do catheter
 - MSU: discard first few mls
- Investigations:
 - Dipstick: Under-rated
 - Nitrites (produced by an enzyme in *most* infectious bacteria which breaks nitrates down to nitrites) ⇒ presumptive diagnosis. But more frequent voiding in small children reduces time for nitrite production so false negative.
 - Leukocyte esterase is sensitive, not specific
 - If no leukocytes, nitrates, protein or blood then no infection. Ie high negative predictive value.
 - Urine Microscopy:
 - Some RBC and WBCs are normal
 - Look for casts, crystals, bacteria. Absence of bacteria not significant (treat empirically)
 - Culture:
 - Bacteruria ⇒ 10E5 colony forming units (cfu) per ml of urine. However, this was set using morning samples in young women via catheterisation ⇒ not much value.
 - In kids, a much smaller number may be significant, especially if:
 - In a boy
 - Obtained by catheter. In a supra-pubic aspirate any growth is important
 - Most UTIs are caused by a single bug. If multiple organisms then contaminated sample. Bugs can grow in transit → send to lab straight away or refrigerate
 - Antibiotic sensitivity: if multi-resistant then usually from Asia where antibiotics are freely available
- Exam:
 - Often normal, other than fever
 - Do **blood pressure**, search for loin, abdominal and supra-pubic tenderness, inspect spine and external genitalia, and brief neuro exam of the lower limbs. Check and plot growth
- Management:

- Admitted for IV antibiotics if:
 - Neonate or immunocompromised
 - Shocked
 - Vomiting frequently (ie oral antibiotics won't stay down)
- Hospital treatment:
 - Bloods: FBC, blood cultures, electrolytes and Cr. If toxic, consider LP and glucose
 - Antibiotics: *Amoxycillin 50 mg/kg/6hr (max 2g)* (for enterococcus) and *gentamicin 2.5 mg/kg/8hr* (if older than 1 week and normal renal function) to cover everything else.
 - Discharge on oral antibiotics to take total treatment to 10 – 14 days. Then prophylaxis until follow-up
 - Repeat urines to check it's cleared
 - Follow-up:
 - Aim is to identify those with underlying abnormality (obstruction, VUR, scarred or dysplastic kidneys) and identify those at greater risk of recurrent UTI
 - US within, say, 12 hours: checking for obstruction and kidney size. Poor sensitivity for reflux
 - If < 2 years then MCU (Micturycysto-urethrogram, for reflux → risk of scarring) + delayed DMSA scan (eg after 6 months, look for filling defects → renal scarring)
 - If > 2 years then delayed DMSA
 - If don't want to do DMSA scan then annual BP check and pubertal US to check kidney's have grown.
 - If reflux, then prophylactic antibiotics until out of nappies and 6 months since last UTI
- Oral Antibiotic treatment (equivalent to IVs for pyelonephritis):
 - Don't give antibiotics unless a definitive urine specimen has been obtained
 - Antibiotics standard treatment:
 - *Cotrimoxazole 200/40mg in 5 ml, 0.5 ml/kg bd 5 days* (= trimethoprim + sulphamethoxazole – less concern about allergy in kids). 15% resistance. Good in kids as comes in syrup. Don't use in adults due to risk of Stevens-Johnstone Syndrome.
 - Augmentin 15 mg/kg tds po (max 500 mg) for 5 days
 - Prophylaxis in children with recurrent infection is common – but duration, drug and dose all remain variable. *Cotrimoxazole 200/40mg in 5 ml, 0.25 ml/kg po od*
 - Repeat urines at conclusion of antibiotics to check it's cleared
 - Referral to paediatrician:
 - Boys: always refer for confirmed UTI, especially if circumcised
 - Girls: At least repeat urines after first UTI to check cleared. Refer after second UTI

Urinary Incontinence

Daytime incontinence

- History:
 - Previously continent?
 - Frequency, volume, urgency, pain, colour, continuous dribble (are nappies *never* dry - nearly always pathological)?
 - Infection history:
 - Associated symptoms
 - Past infections, kidney complications
 - Constipation (→ urinary retention due to pressure → infection). Need to fix bowels first
 - Family History
- Exam:
 - Palpable/distended bladder
 - Kidneys: palpable, tender?
 - Boy: examine penis carefully: balanitis (inflamed foreskin), constricted urethra
 - Girl: effusion of the perineum, can labia be parted
 - Signs of occult spina bifida (eg skin or vascular lesions over sacrum)
 - Are legs neurologically normal
 - **Blood pressure:** whenever risk of kidney disease
 - Screen for infection
 - Not PR

- Investigations:
 - Urine microscopy
 - Paediatric US referral
 - Further tests:
 - Bladder volume scanning
 - Paediatric MCU
 - Cystoscopy
 - Urodynamics
- If repeat infection:
 - ?Genitourinary malformation: do US or MCU to check for reflux
 - Infection leads to temporary scarring, which predisposes to infection. Break the cycle with prophylactic antibiotics

Bed Wetting/Enuresis

- Very common: 12% at age 6, 4% at age 14
- History:
 - Just at night time, or day as well (pathology more likely – must fix this first)
 - Is it primary or secondary:
 - Primary: have never been dry, most common, usually no associated pathology. No daytime problems. Pass large volume without waking. Ask about proportion of dry nights, getting worse or better?
 - Secondary: were dry, now wets (regression) → pathology common. Detailed history of when it began, pattern since then (↑ or ↓), symptoms of infection (dysuria, frequency), diabetes (weight loss, thirst), physical abuse. Can be induced by stress (eg starting boarding school, family disruption)
 - How much wetting: big patch, small patch. How often in the night (if several times then will take longer to come right)
 - Urinary symptoms: polyuria, dysuria, frequency
 - ?Constipation or soiling → need to fix this first
 - Family history (if one parent wet the bed, 40% of children will wet, if both parents then 80%). This is key information – normalises it for parents and child → ↓anxiety
 - Parents management style: punitive (unhelpful but common) or supportive (ignore wet pants, praise for waking to pass urine, not common but more helpful)
 - Previous treatment experiences
 - Expectations of parents and child
 - General developmental screen, including faecal continence, bladder training
 - Social history: how much extra support will the child or parent need to manage the treatment
- Exam:
 - End of bed: note weight loss, hydration
 - Growth
 - Lumbosacral area (midline defects → ?spina bifida), perianal sensation and neurological exam of legs
 - Abdominal palpation: kidneys, distended bladder, constipation
 - External genitalia
 - Blood pressure
- Investigations:
 - If primary then tests usually reveal nothing
 - MSU: blood, protein, glucose, casts, bacteria, urine analysis
 - May be: blood sugar (diabetes) and electrolytes (renal failure)
- Treatment:
 - **Reassurance:** a nuisance, but normal and curable. Not silly or on purpose. Primary enuresis is NOT a psychological problem, a personality disorder or ADD, but one of delayed maturation. However, stress will make a tendency to bedwetting worse
 - Parental intolerance will worsen it and ↓self-esteem
 - Avoid covert rewards (eg getting into parent's bed when their bed is wet)
 - No night nappy, leave lights on in toilet, normal fluids before bed
 - Convenient hygienic care of bed (eg waterproof under-blanket)
 - **Keep a diary** (good for any symptom):

- Day, time of bed, hourly check till parents go to bed, size of wet patch
- Helps keep accurate record and has therapeutic value (gives feedback, is something to do, etc)
- Don't treat until age 6 or 7 – but do treat then otherwise psychological sequelae as they head into teens
- Four options:
 - Encouragement (rewards). See Behaviour Management, page 14
 - Systematic Waking: wake half an hour before normal wetting time, and shift toileting time closer to bedtime/morning by half an hour a week
 - **Pad Alarms:** Good ones best. Not funded. Parents need to be instructed on how to get maximum value from them. Explain and demonstrate to child. Hard work for parents as they must get up (take turns, may need extra support if solo parent). Must wake child properly (eg cold flannel on face). Relapse → immediate resumption of pad and alarm. Relapse reduced by over-training (once consistently dry, push fluids at bedtime, will recommence wetting but overcome it quickly)
 - Bladder training exercises
- Which options:
 - Wets once or twice a week: Rewards for 4 weeks then pad and bell
 - Wets at the same time each night: systematic awakening
 - Wets many times through the night with small patches: bladder retraining and alarm
 - Wetting more than twice a week at unpredictable times: bell and pad
- If not improvement after two lots of 4 weeks then ?anatomical problem
- Not medication: Nasal ADH/vasopressin (specialist only) treats symptoms but doesn't change behaviour. Maybe useful for short term protection (eg school camps, etc)

Testes

Undescended testis

- = Cryptorchidism
- Descent complete in 96% at birth, in 99% at 3 months
- Premature will have ↑rate of undescended testis (5% at 1 year)
- Two types:
 - Arrest of descent: at internal or external ring, or at scrotal neck
 - Ectopic: outside of the line of descent
- May present with a hernia
- Surgical correction at about 12 months
- Sequelae of non-descent:
 - 20 times risk of malignancy
 - ?Impact on fertility (due to ?higher temperature impairs spermatogenesis)
 - If don't bring them down they may end up over the pubic ramus → very uncomfortable sex!

No testis

- If bilateral undescended testis and hypospadias → ambiguous genitalia → urgent referral
- Torsion in utero → no testis
- No testis = anorchia. Maybe no kidney on that side ⇒ check

Retractile Testis

- Normally in scrotum but retracts upwards during examination
- Testis normal size
- Follow-up 2 yearly
- Surgery unnecessary. Will drop into scrotum at puberty

Hydrocele

- Fluid collection between the layers of the tunica vaginalis secondary to trauma, infection or idiopathic. Implies a patent process vaginalis
- May be bigger in the evening than in the morning
- Transilluminates well, is non-tender and non-reducible
- Can get above it at the inguinal ring (as opposed to a hernia)

- Herniotomy if not resolved by age 2. 50% disappear in first year. Remove tunica vaginalis → removes potential space
- Predisposes to hernia

Acute Scrotum

- Must examine the genitalia of every boy who presents with acute lower abdominal pain (may not localise to testis)
- In descending order of frequency, causes of an acute scrotum are:
 - Torsion of the appendix testis
 - Testicular torsion
 - Idiopathic scrotal oedema. Symmetric swelling, no testicular tenderness. May include penis, inguinal and perineal regions. Exclude torsion
 - Rarely, epididymo-orchiditis
- Management of torsion:
 - High probability: short duration and negative urinalysis → surgery
 - Low probability: longer duration and positive urinalysis → ?Doppler US for ↓blood flow

Torsion of Appendix Testis

- Most commonly caused by Hydatid of Morgagni (Mullerian duct remnant) at top of testis
- Peak incidence at 10 – 12 years. Oestrogen stimulates enlargement of the remnants → predisposes to torsion
- Symptoms range from minimal inflammation to florid, swollen hemi-scrotum indistinguishable from testicular torsion
- Urgent surgical referral

Testicular Torsion

- Testes are covered by tunica vaginalis – has parietal and visceral surface (like lungs in pleura)
- Testis rotates on its chord within parietal tunica vaginalis
- Once torsion has occurred in one, more likely in another
- < 6 hours will probably not cause infarct
- Two peaks for incidence:
 - Neonatal: Testis usually dead by diagnosis. May not operate (will atrophy). May ‘pex’ contralateral side to prevent torsion
 - Age 13 – 15: History and presentation variable. Surgical emergency. If testis viable, untwist and fix. Fix contra-lateral side
- Need to remove a torqued testis, otherwise he will develop autoantibodies for spermatozoa → infertility of other testis

Epididymo-Orchitis

- Very rare in children. Two peaks
 - Newborn, with underlying urinary tract anomaly. Do US and MCU. MSU to rule out infection
 - In 13+ due to reflux up the vas → infection/inflammation
- Mumps orchitis does not occur in pre-pubertal boys

Penis

Smegma

- Yellowish coloured secretion-desquamation which occurs normally and keeps the foreskin separate from the glans
- May appear like a dermoid cyst underneath the skin
- Is normal, and will eventually extrude spontaneously

Retraction of the foreskin

- By age 4 most boys foreskins will be able to be retracted
- May have intermittent pain during separation of the adhesions and the foreskin may be red or swollen for a day or two

Phimosis

- Irretractable, scarred foreskin. May balloon on urination
- If mild, application of Betnovate ointment to the tight portion of the foreskin (retract loose bit to access it) is effective
- If ongoing problems → circumcision
- Paraphimosis: foreskin stuck behind glans → swollen. Always put foreskin back after catheterisation

Balanitis

- Infection of the foreskin, may remain distal or involve whole penile shaft
- Can be secondary to phimosis
- Treat with topical bactrim or oral antibiotics

Hypospadias

- Combination of dorsal hood, proximal urethral opening and chordae (central penile tilt)
- Presentation varies from mild to severe peno-scrotal type with ambiguous genitalia (check for testis)
- Correct at 9 – 12 months
- ↑UTIs
- ↑Infertility as the opening moves closer to the base of the penis

Ambiguous Genitalia *

- Relative complexity of male differentiation → vulnerable to wide variety of incomplete masculinisation
- History and exam:
 - Exposure to progesterone, testosterone, phenytoin, aminoglutethamide
 - Previous neonatal deaths
 - Phallic size, position of urethral orifice, fused labia, descended gonads
- Don't rely on appearances whenever babies have:
 - Bilateral cryptorchidism, even if there is a phallus
 - Unilateral cryptorchidism with hypospadias
 - Peno-scrotal or perineoscrotal hypospadias
- Causes:
 - Androgen resistance (extreme form: testicular feminisation)
 - Early in boys development, sertoli cells release anti-mullerian peptide → stops formation of the fallopian tubes, uterus and the upper 1/3 of the vagina
 - Testosterone and dihydrotestosterone from Leydig cells responsible for the rest of male genitalia. If a problem in this pathway → Girl with short vagina.
 - Present in puberty with primary amenorrhoea
 - Adrenogenital Syndrome:
 - Incidence: 1 in 14,000
 - Congenital adrenal hyperplasia: masculinised females
 - ↑Androgenic hormones because of ↓ 21-hydroxylase, 11-hydroxylase or 3-B-hydroxysteroid dehydrogenase
 - Can't produce cortisol → adrenal hyperplasia → overproduction of cortisol precursors → ↑androgens
 - Presentation: vomiting, dehydration and ambiguous genitalia. Hyponatraemia (with paradoxically high urine Na) and hyperkalaemia is common → may present with circulatory collapse in early life or hyponatraemic seizures (misdiagnosed as febrile convulsions)
 - Boys may present with precocious puberty or have ambiguous genitalia (reduced androgens in 17-hydroxylase deficiency)

- For Abdominal exam, see Examination, page 7

Abdominal Radiology

- Should always be gas in the:
 - Stomach. If not, then ask why. ?Oesophageal atresia without fistula to the bronchus. ?To sick to swallow
 - Rectum
 - RLQ (Caecum)
- Gas bubbles. If only:
 - 1: pyloric stenosis. Do US to confirm (not barium meal)
 - 2: Duodenal atresia – “double bubble trouble”. Associated with Down’s. Ante-natally: polyhydramnios (can’t swallow)
 - 3: Jejunal atresia
 - Lots of bubbles but no normal caecal gas: Ileal atresia (Colonic atresia very rare)
- Other signs:
 - Gas on both sides of bowel wall → wall stands out as opaque line → Rigler’s sign
 - Malrotation: wandering small bowel below duodenum with barium meal. If corkscrew then ?malrotation with volvulus
 - Pneumatosis Intestinalis: gas bubbles in intestinal wall (‘string of pearls’): if also in portal venous system (eg bubbles in liver) then necrotising enterocolitis

Congenital abnormalities

Tongue Tie

- Short lingual fraenum
- Rarely interferes with eating or speech
- Generally requires no treatment

Oesophageal Atresia

- Happens early in embryonic life:
 - Lots else happening then too – look for associations as well
 - Cardia, Renal, Anus, Vertebral, Oesophagus, Trachea: CRAVET
- Symptoms: dribbles all the time
- Usually distal oesophagus attached to trachea (fistula) → air in stomach
- Urgent neonatal repair

Pyloric Stenosis

- = Hypertrophic pyloric stenosis
- 4:1 boys to girls. Males 1/200 – 1/400
- Family history: in 15% of siblings or previous generation
- Pathophysiology: circular muscle hypertrophy → progressive narrowing of pyloric stenosis
- Presentation: 3 – 6 weeks, initial spilling → progressive dysfunction → several days of non-bilious high volume projectile vomiting with or between feeds. Dryish nappies (from dehydration)
- Exam: peristaltic waves of exaggerated gastric peristalsis + palpable lump in RUQ (= pyloric tumour) when hips flexed and relaxed (eg immediately after a feed)
- Differential:
 - Gastro-oesophageal reflux – but baby well and growing
 - Exclude infection: UTI, meningitis, gastroenteritis, chest infection
- Investigations: usually clinical diagnosis. Check electrolytes and blood gases for hypochloraemic hypokalaemic alkalosis
- Treatment:
 - Rehydration: IV saline + KCL + glucose
 - Surgery: pyloromyotomy

Duodenal Atresia

- Present in first 24 hours with green vomiting after feed
- Xray shows double-bubble sign: gas in stomach
- Associations: 1/3rd have Down syndrome, 10% of Downs have duodenal atresia

Small Bowel Atresia

- Due to loss of blood supply to that part of the gut in utero. Infarcts and heals (as opposed to after birth when bacteria → gangrene)
- Bowel distal to the obstruction may be malformed

Malrotation

- In 80% of cases, diagnosed in first month of life. Usually presents after 2 –3 days with bilious vomiting
- Exclude: strangulated hernias, bowel obstruction secondary to adhesions, intussusception and sepsis
- Investigations: barium meal → duodenal jejunal junction hasn't ascended to level of pylorus and is not to the left of the midline
- Pathogenesis: Dates from time when the midgut is in the umbilical chord. Failure of fusion (syngosis) of the small bowel mesentery to the posterior abdominal wall → narrow “universal mesentery” with the superior mesenteric artery supplying the whole mid-gut → torsion leads to mid-gut ischaemia
- Surgical emergency

Meckel's Diverticulum

- Most frequent congenital abnormality of the gut (2% of autopsied adults). Due to persistence of omphalomesenteric duct
- Illness of 2's: incidence 2%, 2 feet from ileocaecal valve, symptomatic from 2 years onwards
- Wide mouthed diverticulum (approx 5 cm), on antimesenteric border of the ileum, usually within 100 cm of ileocaecal valve. 30% of the time ectopic tissue is opposite the diverticular
- 50% have normal ileal mucosa, rest have duodenal, pancreatic, colonic or gastric (not subject to feedback → ulcers) mucosa
- Symptoms:
 - 40% of GI bleeds
 - Maroon not melaena
 - Intermittent
 - ↓HB but no shock
 - 30% present with intussusception or band, 20% with diverticulitis like appendicitis, 5% with umbilical mass
 - Meckel's diverticulitis: blocked, inflamed → enlarged → burst
- Rarely symptomatic after age 5, but may →
 - Haemorrhage, before age 10, due to peptic ulceration of surrounding ileal mucosa,
 - Inflammation, may mimic acute appendicitis
 - Obstruction in kids/teenagers: intussusception into lumen of bowel, or twist on fibrous remnant of the omphalomesenteric duct extending from bowel to abdominal wall (remnant of the yoke sac)
- Diagnosis and treatment:
 - Pertechnetate scan: looking for hot spot
 - Diagnostic laparoscopy
 - Treatment with laparotomy and end to end anastomosis

Meconium Ileus

- Tenacious meconium won't shift, gets colonised and ↑gas
- ⇒ Cystic fibrosis. See page 65

Hirschsprung's Disease (Aganglionic Megacolon)

- = Aganglionic Megacolon
- 1st described by Hirschsprung in 1886
- Incidence: 1 in 3 – 5,000 live births. Boys four times girls. Familial tendency
- Aetiology: ?arrest in migration of ganglion cells from neural crest down GI tract
- Pathology:

- Absent GI ganglion cells (co-ordinate motility of bowel – Meissner’s and Auerbach’s plexuses). Always includes internal anal sphincter and spreads proximally a variable length:
 - 50% to recto-sigmoid junction, through to
 - 10% including total colon
- Affected colon can’t relax → greatly dilated proximal segment
- Macroscopic: affected segment may look normal, proximal segment damaged mucosa, possible perforation, thickened wall
- Microscopic: absence of normal ganglion cells (visible with acetyl cholinesterase stain) and hypertrophy of nerve fibres (non-specific – can occur with other conditions)
- Clinical presentation:
 - Age of presentation unrelated to length of affected segment. Eg someone with their total colon affected can present after years
 - 3 groups of kids:
 - Neonate: acute lower GI obstruction, abdominal distension, bilious vomiting, maybe fulminant diarrhoea, maybe perforation. Clue: no gas in rectum on x-ray
 - Infancy: constipation, abdominal distension, possibly precipitated by change in bacterial flora on introducing other foods after exclusive breast-feeding (may → massive diarrhoea)
 - Older: severe constipation, chronic abdominal distension, scybala (hard mass of faecal matter), failure to thrive, never soil pants
- Diagnosis:
 - Barium enema: narrow rectum, enlarged proximally. Usually rectum lumen twice the diameter of the colon. Don’t do an enema if risk of perforation (barium in the peritoneum → serious peritonitis)
 - Rectal manometry: inflate balloon in rectum, distal rectum and internal anal sphincter should normally relax, this won’t
 - Rectal biopsy: But finding ganglion cells is hard even in a normal specimen. Histochemistry – test for ↑ Ach (preganglionic nerve cells looking for ganglion cells)
- Treatment: Two stage surgery:
 - Stage 1: Disobstruct: ostomy in lowest portion of bowel with ganglia
 - Stage 2: Later, bring ganglionated bowel to anus

Imperforate anus

- In girls, often attaches into the posterior wall of the vagina
- Adequacy of the levator sling depends on how high the lesion is. If low, only a superficial problem. If high (growth arrested above levator sling), then reconstruction is more difficult
- Look for other developmental abnormality: eg GU, vertebral, etc

Other

- Gastroschisis: paraumbilical defect with evisceration (extrusion of viscera) of abdominal contents. Incidence 1.6/10,000. No covering. Usually only bowel hanging out. Usually no other defect. Corrective surgery has good outcome. Delivery in tertiary centre
- Exomphalos: Herniation of abdominal contents (may include liver) into the base of the umbilical chord. Covered with peritoneum. Incidence 4.3/10,000. Other abnormalities often present

Acquired Causes of Obstruction

- In addition to congenital causes above (malrotation → volvulus, atresia, etc):
 - Hernias
 - Adhesions (always ask about previous surgery)
 - Intussusception
 - Volvulus

Intussusception

- Peak incidence 3 months – 2 years
- Usually ileocolic
- Causes:
 - 95% idiopathic
 - 5% mechanical: intestinal polyp, Meckel’s diverticulum, lymphosarcoma (> 6 years old)
- Symptoms:
 - Often URTI 10 days before (→ adenovirus in Payer’s patch)

- Initial: severe abdominal colic every 15 – 30 minutes, very well in between
- Later: red-current jelly stools, prostration, pallor (shock)
- Exam: sausage shaped mass → clinical diagnosis
- Treatment:
 - Hydrostatic reduction: blow air or barium up anus at 50% diastolic pressure. Not if small bowel obstruction or peritonitis (barium in peritoneum is nasty)
 - Surgery

GI Bleeding

- Causes:
 - Serious, life threatening bleeding rare: varices, stress ulcer, etc
 - Serious disease more common (the bleeding itself is not life-threatening):
 - Necrotising Enterocolitis (NEC)
 - Intussusception
 - Inflammatory bowel disease
 - Familial polyposis
 - Other differentials: Meckel's Diverticulum, A-V malformation, Anal fissure
- History:
 - Is it really blood? Is baby vomiting mum's blood (swallowed in delivery or cracked nipple), is it a UTI not rectal?
 - Do they have clotting problem? (Did they get vitamin K?)
 - How much, how fast

Hernias

Inguinal Hernia

- 4:1 male to female. 1% of boys
- Virtually all indirect. A widely patent proximal process vaginalis allows bowel (and ovary in girls) to enter the inguinal canal
- Presentation: intermittent swelling overlying the external inguinal ring that has been noticed by a parent
- 50% right, 25% left, 25% bilateral
- Do not resolve spontaneously
- If < 1 more likely to present with strangulation
- Incarcerated (bowel loop stuck through):
 - Peak incidence in first year – main cause of obstruction. High index of suspicion in any child with abdominal distress
 - If neglected will strangulate – testes will die first due to ↓venous return
- Management: Should be repaired ASAP.
 - 98% of acute or strangulated hernias can be reduced by taxis: manipulating it back in. Then fix electively (ie within a week)
 - If signs of ischaemic gut or peritonitis → surgery
- Complications:
 - Girls: fallopian tube and ovaries may be within the hernia. May tort. Care with surgery. Can completely close the internal ring.
 - Boys: damage to vas or testicular atrophy if surgery while acute

Umbilical hernia

- Rarely cause problems, even if large
- Repair at age 3 if haven't resolved by then

Congenital Diaphragmatic Hernia

- 1:5,000 live births. 1:2,000 total births (⇒ lots of still births)
- Diaphragm should close just before mid gut comes back from umbilicus. In this case, returning gut enters chest. Compromises ipsilateral lung development (more common on left) → mediastinal shift and lung hypoplasia
- Symptoms:
 - Early respiratory distress/cyanosis

- Scaphoid abdomen
- Bowel sounds in chest
- Dextrocardia (diaphragmatic hernia most common cause)
- Treatment: don't bag the child → bagging also blows up stomach and guts → compromises lung expansion further. Ventilate. Surgery
- Complications: pulmonary hypertension in severe cases
- Overall survival of 40 – 60%

Common Childhood Presenting Chronic Symptoms

My Child Won't Eat

- Key issue: do they have normal growth:
 - Normal growth:
 - 1st year: go from 3.5 → 9 or 10 Kg
 - 2nd year: from 9 or 10 kg to 12.5 or 13 kg. I.e Growth slows markedly
 - Normal intake for first year:
 - 100 cal/kg/day
 - 150 mls fluid/kg/day
 - Breast milk has 67 cal/100 mls → 100 mls breast milk at 150 mls/kg gives 100 cal/kg
 - If normal growth – what are parent's perceptions of amount the child does and should eat. If perceptions not right then → stress, unhelpful dynamics around food (especially for strong willed child) → parents give them lots of milk so they at least get something → iron deficient
 - If not normal growth consider disease, congenital syndromes, are they being offered enough (eg maternal depression/anorexia). See Failure to Thrive (FTT), page 39

Reflux

- Symptoms: poor growth, vomiting, cry (especially after food), cough
- But:
 - All babies have some reflux
 - All babies cry – parents may not realise how much is normal! Average baby peaks at 4 hours per day at 6 weeks, then declines. Is an association between crying and maternal depression
- Can measure pH via NG tube over 24 hours, or scope them (only in Auckland). But most babies with presentation of reflux don't have oesophagitis.
- If neuromuscular problems (eg Cerebral Palsy) then more likely to have problems with severe reflux oesophagitis
- Treatment:
 - Antacids, ranitidine, omeprazole
 - Crying decreases from 6 weeks – is this a treatment effect or normal development
 - Ensure good support: wider family, Plunket, etc
- See The Crying Baby, page 39 for Colic

Abdominal Pain

- 'Functional' pain (no organic cause) is 'benign':
 - Parents didn't know until child said
 - Distractible from it
 - Central pain (point to umbilicus)
 - No sleep disturbance
 - No associated symptoms
 - Intermittent
- Organic causes mimicking functional pain:
 - Constipation (parents may not be aware that child has problem with constipation)
 - Abdominal migraine: migraine in 3 – 8 year old often presents as abdominal pain. Intermittent, goes pale, last an hour or two, not distractible. As they get older may develop into normal migraine. Check family history
 - Always examine genitalia in a boy with acute abdominal pain
- Other causes: appendicitis, intussusception, UTI, testicular torsion, volvulus secondary to malrotation, Meckel's diverticulitis, renal colic, pyelonephritis, acute glomerulonephritis, drug ingestion, reflux oesophagitis

- Other causes are rare without associated symptoms (eg coeliac, Crohn's)

Diarrhoea

- Is growth normal:
 - Yes ⇒ no significant malabsorption:
 - Low grade infection, eg Giardia, Cryptosporidium
 - Diet, eg too much juice → overload sucrose absorption → osmotic diarrhoea
 - 'Toddlers diarrhoea': 18 – 24 months, sloppy poos 3 – 4 times a day. ?Variation of normal. Gets less messy/tiresome when toilet trained
 - No:
 - Chronic infection: giardia, Cryptosporidium, parasites/worms
 - Immunosuppressed: any infection (eg Rotavirus, campylobacter etc) may become chronic
 - Coeliac (T cell mediated response to Gliaden fraction): bloating, miserable, diarrhoea, signs of malabsorption
 - Cow's milk protein intolerance
 - IBD: uncommon < 10 years. Abdominal pain, diarrhoea, blood in stool
 - CF: pancreatic insufficiency
 - Metabolic problems: disaccharidase deficiencies, other enzyme deficiencies
 - Constipation → ?overflow diarrhoea
 - Labs:
 - FBC (Hb, MCV, neutropenia)
 - LFTS (ALP, Alb)
 - Immunoglobulins – IgA deficiency
 - Vitamin levels
 - Sweat test
 - Coeliac serology: TTG and antiendomysial antibodies
 - Stool: microscopy (ova, cysts, fat droplets), elastase, reducing substances
 - AXR for overflow
 - Management:
 - Team approach
 - Caloric requirement: 100% of expected weight for age, 130 – 150% of actual weight
 - Vitamin and micro-nutrient requirements
 - Education and support groups

Encopresis/Constipation

- Definitions vary: mainly long term constipation/soiling pants, but may also include inappropriate toileting behaviour (eg going on the lounge floor!)
- Constipation is common
- The main issue is that hardness of the stool, not the frequency
- History:
 - Need information from both parent and child. Parent is unlikely to know about an older child's toileting habits. Perhaps ask child while you're doing the exam – that way parents are off to one side. "I'm going to ask you some really silly questions about your poos..."
 - Soiling (HPC): duration, frequency, severity, ever been continent
 - Associated constipation, withholding, absence of warning (likely), pain (eg fissure), associated wetting
 - Toileting behaviour: avoidance, motions in toilet rare
 - Associated behaviours: hiding soiled underpants (common), scared of toilets at school, more serious conduct disorder (rare)
 - Parent's management style: what's been tried, punitive (unhelpful but common), supportive (ignore soiled pants, praise for toileting, not common but more helpful)
 - General developmental milestones
- Exam:
 - Inspection of perineum: situation of anus, dilated anus
 - Inspection of lumbo-sacral area
 - Neurological exam of the legs: (spina bifida), test ankle jerk (S1-2, anal reflex is S2-5)
 - Abdominal palpation: for palpable faeces
 - PR usually not necessary

- Differentials:
 - Fissure: usually secondary to constipation → vicious cycle
 - Drugs: morphine, codeine, leukaemia drugs
 - Hypothyroidism (NB: associated with Downs)
 - Rare causes: Spina bifida occulta, Hirschsprung's (ask about delayed passage of meconium), anal stenosis (often anal opening is more anterior)
- Pathogenesis:
 - Vicious cycle: chronic dilation of rectum, sigmoid and descending colon → ↓sensation of fullness → go less often → faeces dry out more → hard → don't completely evacuate → ↑distension → ↓strength → overflow diarrhoea (with no awareness)
 - Constipation is common post-gastroenteritis or after surgery
 - Can more rarely be due to food allergy
- Management:
 - Explain normal anatomy and function of the rectum. Use a diaphragm.
 - Explain process: withholding stool → dilated rectum → loss of normal sensation → no warning its coming → he's not being naughty and will take a while to come right (ie stick with treatment)
 - Test transit time by eating a pile of whole kernel corn and seeing how long it takes to come out the other end. The ideal is < 24 hours
 - Structured toileting programme: diary and reward system for sitting (take a book if they're bored) not for clean pants. Toilet for 10 minutes after each meal. Use timer
 - Fibre and adequate fluids to keep stools soft
 - Treatment of severe constipation:
 - Use enemas to completely empty bowel – get visiting paediatric nurse to do it – easier on Mum and Dad
 - Laxatives every day to empty bowel (eg lactulose, magnesium sulphate) + regular toileting. Coloxyl drops (a stimulant) may → colic in kids. Lubricants (eg paraffin oil) are good but not very palatable
 - Continue for weeks/months until rectum normal size again
 - Frequent visits for support of parents and encouragement of child

Gastroenteritis

Differential of acute vomiting/diarrhoea

- Enteric infection:
 - Virus: rotavirus (45% of acute gastro), also enteric adenovirus, caliciviruses, astroviruses
 - Bacteria: Campylobacter, Salmonella (more common spring/summer), also Yersinia, enterohaemorrhagic E coli, shigella
 - Protozoa: giardia, cryptosporidia, also microsporidia, amoeba
 - Food poisoning: (had anything different to eat from the rest of the family?) Staphylococcus enterotoxin, bacillus cereus, Campylobacter, salmonella, E coli, Norwalk virus
- Systemic infection: if sicker than history suggests then UTI, Pneumonia, otitis media, meningitis, sepsis (including meningococcaemia)
- Surgical conditions: Appendicitis, intussusception, bowel obstruction, Hirschsprung's enterocolitis, pyloric stenosis, incarcerated inguinal hernia, testicular torsion
- Other disorders:
 - Diabetic ketoacidosis
 - Antibiotic associated diarrhoea
 - Haemolytic uraemic syndrome (renal failure, haemolytic anaemia and thrombocytopenia, eg due to E Coli verocytotoxin, also drugs, SLE, etc)
 - Poisoning

Warning Signs

- Seek urgent advice if any of:
 - Vomiting bile or blood
 - Severe abdominal pain
 - Toxic appearance (ie more than just gastro): lethargy, poor perfusion, hypo/hyper ventilation, cyanosis
 - Abdominal signs: distension, tenderness, guarding, mass, hepatomegaly

- Failure to thrive
- Neonate

Diagnostic Clues

- Sudden onset of fever, vomiting and watery diarrhoea: viral gastroenteritis
- Cramping abdominal pain and frequent bloody, mucousy stools: bacterial gastroenteritis. If an infant and severe pain or pallor, consider intussusception
- Colicky pain, RIF pain, bile stained vomiting and distension → surgical case
- Season: Rotavirus during winter epidemics, giardia and cryptosporidia during the spring and campylobacter in the summer

History

- Vomiting: bile, blood, coffee grounds, volume, frequency, total duration
- Diarrhoea: nature, colour, consistency, blood, mucus, frequency, volume, total duration
- Amount and type of recent food and fluid intake
- Urinary output
- Other symptoms:
 - Fever
 - Abdominal, groin or scrotal pain
 - Urinary symptoms
 - Respiratory symptoms
 - Recent illness
- Other:
 - Antibiotics and other drugs
 - Infectious contacts
 - Possible contaminated food ingestion, including shellfish
 - Overseas travel in the last 2 months
 - Immunisation
 - Other medical conditions, GI, diabetes, heart or renal

Management

- Principles:
 - Dehydration is the most important complication. In infants it can appear in several hours
 - See Assessing fluid loss, page 154 for assessment of dehydration and rehydration
- Investigations:
 - Stool microbiology: Only if:
 - Blood in the stool
 - Recent overseas travel
 - Suspected epidemic or food poisoning
 - Child in an institution
 - Chronic diarrhoea (> 3 weeks)
 - Biochemistry: Na, K, Cr +/- glucose +/- ABG if severe, < 3 months, or on IV therapy
 - Other: urines, blood, and CSF culture, CXR, AXR, LFT etc if indicated
- Management:
 - Ambulatory if diagnosis not in doubt, family able to cope, have transport, no dehydration and good fluid intake
 - Admission if: diagnosis in doubt, < 3 months, high risk, dehydration, failure to improve, pre-existing condition (get sicker quicker: eg Ileostomy, short gut, cyanotic heart disease, renal failure, diabetes, etc)
 - IV Rehydration if: shocked, severely dehydrated, failed trial of oral therapy
- Treatment principles:
 - For a non-dehydrated child:
 - Small, frequent sips of Gastrolyte (doesn't fix diarrhoea) – not for bloody dysentery (dehydration not the biggest concern). 5 – 7 ml/kg/hr
 - ½ strength formula feeds
 - Fruit juice diluted 1:4
 - Maintain nutrition: Get back to solids within 6 – 12 hours if possible: banana, apple, rice, potato, noodles, toast and vegemite
 - Breast-feeding is continued

- Do **not** use anti-emetics nor anti-diarrhoeal agents
- For a dehydrated child, see page 155

Infectious agents

- Bacterial enteroinvasive:
 - Campylobacter Jejuni
 - Poultry
 - Haemorrhagic colitis
 - Erythromycin (used to be ciprofloxacin but it's been put in chicken feed → ↑resistance)
 - Escheria Coli
 - Enterotoxigenic subspecies
 - Travellers diarrhoea
 - Salmonella/Shigella: Poultry
- Bacterial enterotoxins:
 - Vibrio Cholerae
 - Staph Aureus: Food poisoning (eg cream buns)
 - Clostridium Botulinum
 - Some subtypes of E Coli
- Protozoa
 - Giardia
 - Trophozoites reside mainly in duodenum, also small bowel
 - Cysts in faeces
 - Small bowel diarrhoea → diarrhoea during night (large bowel 'sleeps')
 - Entamoeba histolytica: causes amoebic dysentery (colitis)
- Cryptosporidium
 - Common in kids
 - Is chronic in immunocompromised
 - No effective antibiotic treatment

Lactose Intolerance

- Small bowel injury → temporary lactose intolerance
- Most common in bottle feed babies < 6 months. Uncommon in breast-feed babies.
- Clues are consistent fluid stools, or their restarting with reintroduction of milk feeds, excess flatus, perianal excoriation
- Testing: Collect 5 drops of stool from a plastic lined nappy, mix with 10 drops of water and add a Clinitest tablet. Colour reaction of > ¾% indicates sugar is present
- Change to a lactose free formula for 3 – 4 weeks, then introduce the old feed over 2 – 3 days

Nutritional Deficiencies in Childhood

Iron Deficiency

- Commonest deficiency in NZ and worldwide
- Marker of poor diet generally
- Associated with:
 - Inadequate iron intake:
 - Homogenised cows milk
 - Late introduction of iron-rich foods
 - Prolonged sole breast feeding (> 6 months)
 - Intrauterine growth retardation and placental insufficiency (especially rapid catch up growth)
 - Excess losses: chronic gut losses (eg infestation, food intolerance) and skin loss in severe ectopic eczema
- Sources of iron:
 - Poor sources:
 - Spinach: poorly absorbed
 - Cows milk: poor source and may lead to gut bleeding
 - Breast milk: only sufficient to 4 – 6 months, but ↑ absorption once food is introduced
 - Good sources:
 - Meat: haem iron well absorbed. Especially dark red meat (eg liver)

- Pulses: lentils, peas, baked beans and soya beans (not green beans), but ↑ gas
- Dark fish, shell fish and spices
- Breast milk and vitamin C ↑ absorption, Cows milk and tea ↓ absorption (NB some Polynesians call tea milo – so ask what sort of tea)
- Anaemia:
 - At birth, Hb = 170, several weeks later = 105
 - Clinical effects: tired, lethargic, irritability, slow cell mediated immunity, pica (eat anything) which may → lead poisoning (small RBCs and anaemia)
 - Diagnosis:
 - Look for pale **earlobes**
 - Blood tests, iron studies etc. MCV < 71 in child over 3 months
 - Ferritin low (but high if infection – test CRP as well and ignore ferritin if raised)
 - Serum iron – altered in presence of infection. Zinc Protoporphyrin is a new, sensitive test (Zn substituted for Fe in haem).
 - Reticulocyte count useful test of response to treatment. Should respond within a week
 - Treatment:
 - Find and fix cause: if diet then → dietician.
 - Ferrous gluconate elixir: 50 mg/kg/day (= 6mg/kg/day elemental iron) in 2 – 3 doses with fruit juice until MCV normal

Rickets

- Usually a lack of Vitamin D. With fear of sunburn, it is likely to increase
- At risk:
 - Pigmented people with low dietary vitamin D intake and low sunlight exposure. Breast milk is not a very rich source of vitamin D
 - Preterm infants with low Vitamin D intake
 - Fat malabsorption
 - Other rarer causes: anticonvulsant therapy, chronic renal disease, Ca or phosphate deficiency
- Diagnosis:
 - Clinical: broad wrists, tender joints, avoidance of weight bearing, bowed legs if weight bearing, bent pelvis (→ obstructed labour later in life), Rickety Rosary (swelling of costochondral junctions)
 - Lab: ↑ALP, ↓PO₄, Ca usually normal
 - Xray: Widened metaphysis and splaying of softened bones, generalised osteopenia
- Treatment:
 - 1-α cholecalciferol: 0.05 – 0.1 mcg/kg/day until ALP normal
 - Surgery to bones not usually necessary, even when very bent

Other deficiencies

- Breast Milk is short of:
 - Vitamin K (fat soluble). Deficiency → haemorrhagic disease of the new born in first few weeks/months
 - Vitamin B12: if mother is vegan → CNS symptoms (fits, abnormal movements, mental retardation) + macrocytic anaemia
- Chronic malabsorption or prolonged TPN → Zn deficiency → Acrodermatitis Enteropathica (rash, especially around buttocks) and immunodeficiency
- Vitamin A deficiency: from fat malabsorption or ↓ intake → night blindness and ↑ risk of complications from eg measles
- Folic Acid: Deficiency during pregnancy → ↑ risk of neural tube defects

Food Allergy

- Commonest in first years of life (gut less good at keeping allergens out)
- 5 – 6% of children cf 1 – 2 % of adults
- Commonest allergens: milk, eggs, peanuts, soy, wheat
- Mechanisms include:
 - IgE mediated – rapid onset, due to mast cell activation and histamine release
 - Delayed hyper-sensitivity
- Scenarios:

- Urticaria/angio-oedema:
 - Rapid onset after contact with oral mucosa
 - Chronic urticaria rarely due to foods (except ?food colourings)
 - Confirmed by skin testing
 - IgE mediated
 - Risk of future anaphylaxis (systemic reaction distant from contact point).
 - Risk factors for fatal outcome: Asthma, peanut/nut allergy
 - Treatment: Adrenaline 0.01 ml/kg of 1:1000 im (10 mcg/kg) [relatively safe im, compared with iv, which risks tachycardia and arrhythmia so would only want to do it in cardiogenic shock]
- Atopic dermatitis: takes days to weeks following food exposure. Strong association between severe asthma and food allergy (60%)
- GI symptoms may be at any point in the gut: oesophagus (reflux), stomach (vomiting), small bowel (colic, diarrhoea, malabsorption) to large bowel (diarrhoea, gas, bloody stools, mucus, constipation)
- Respiratory symptoms: much less common with foods. Oral allergy syndrome: pollen allergic individuals may get oral tingling/swelling after eating some fruits/vegetables

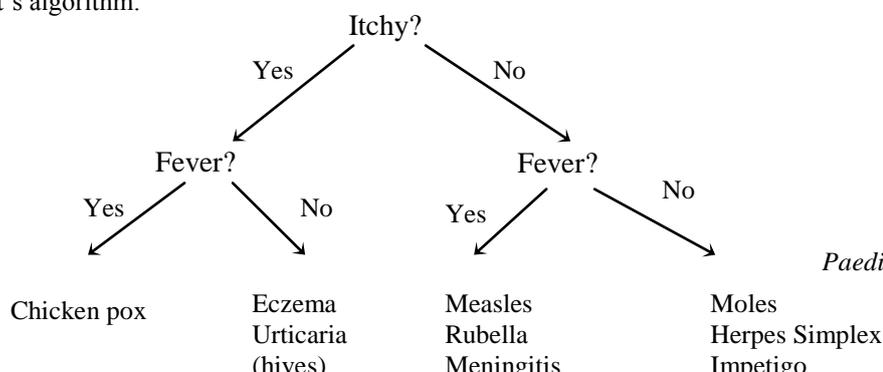
- References:
 - Mainly Dr Lisa Judd's notes in GP, Paediatrics and Musculo-skeletal
 - Prof Delahunt's Pathology notes
 - Dr Stanley's Paediatric eczema notes

Dermatology Glossary

- Annular lesion - ring shaped
- Erythema: dilation of blood vessels – colour goes away if pressed (blanching)
- Macule: an alteration in colour (e.g. macular erythema)
- Papule: a small lump, less than 0.5 cm in diameter
- Nodule: lump bigger than 0.5 cm
- Erythematous-squamous: red and scaly
- Plaque: elevated (maybe only very slightly) area of skin > 2 cm. Altered texture
- Vesicles and bullae = fluid within or beneath epidermis (blister). Vesicles < 0.5 cm, bullae > 0.5 cm. Can have both. E.g. vesicular-bullae eruption from a plant allergy
- Pustule: accumulation of pus (can be just inflammatory not infectious, e.g. psoriasis)
- Cellulitis: inflammation of deep dermis and subcutaneous tissue
- Ulcer: loss of dermis and epidermis
- Scale: at edge of inflammatory lesion, can be fine, large, dark, silvery (psoriasis)
- Scar: fibrous tissue due to healing. Atrophic scar is thin and wrinkled. Hypertrophic scar is elevated
- Poikiloderma: cutaneous pigmentation, atrophy and telangiectasia
- Comedo – pl. comedones: a plug of keratin and sebum in a dilated pilosebaceous orifice. Closed comedo = blackhead, open comedo = whitehead
- Cyst: any closed cavity with a membranous lining containing fluid
- Petechiae – pl., petechiae: a haemorrhagic spot 1 – 2 mm diameter
- Purpura: haemorrhagic spot > 2 mm. Pressing down doesn't blanch – red cells are extravascular → ?vessel damage. If purpura are palpable ⇒ vasculitis
- Ecchymoses: bruises – larger extravasations of blood
- Telangiectases: permanently dilated small vessels
- Guttate: a profusion of small macules or plaques
- Serpiginous: a linear eruption which is S shaped or snake like (e.g. larva migrans – a worm)
- Dermatitis: usually means eczema

Diagnosis

- Where is it:
 - Psoriasis: likes scalp and extensor elbows/knees
 - Atopic eczema: likes flexor elbows and knees
 - Nose & cheeks: lupus, especially if it leaves a pigment behind
- Does it itch?
 - Atopic eczema (if it doesn't itch its not eczema)
 - Chicken pox
 - Urticaria/allergic reactions
 - Contact dermatitis
 - Scabies
 - Insect bites
 - Fungal infections
 - Dermatitis herpetiformis
 - Pityriasis Rosea
- Idiot's algorithm:



Skin Infections

Bacterial infections of skin and soft tissue

Impetigo (School Sores)

- = Superficial infection involving the epidermis
- Most common in children during summer months
- Non-bullous impetigo:
 - = Streptococcal impetigo
 - Vesicles on erythematous base → pustules (highly contagious) → yellow-brown scabs (CRUSTY), associated with regional lymphadenopathy
 - Ecthyma is deeper version – cut out edge
 - Commonly result of skin break such as insect bites or chicken pox. Especially if overcrowding and warmer climates
 - Goes for limbs and face
 - Fever uncommon. Check lymph nodes
 - Caused by *Streptococcus Pyogenes* with or without co-infection with *Staphylococcus Aureus* (can → Scalded Skin Syndrome, see page 135)
 - Commonest cause of post-strep glomerulonephritis
- Bullous impetigo:
 - Due to *Staph aureus* of phage II (usually type 71)
 - Usually younger children
 - Lesions: begin as vesicles – turn into flaccid bullae in response to toxins. Following rupture of the bullae, a moist red surface remains and varnish like crust appears
- Neonatal Impetigo: *Staph Aureus*. Can spread to deeper tissues, umbilicus, bone and joints. If only one site, antiseptic bath once a day. If > 1 site then systemic antibiotics
- Treatment:
 - To relieve symptoms, stop new lesions, prevent complications (e.g. cellulitis, acute glomerulonephritis), and stop spread to others
 - Flucloxacillin, dicloxacillin, a cephalosporin, erythromycin or clindamycin are all effective
 - If MRSA: usually susceptible to co-trimoxazole (although not so good against *S Pyogenes*). Resistance to fusidic acid is also growing
 - Resistance is growing to topical agents (e.g. Mupirocin)

Scalded Skin Syndrome

- Due to staph aureus toxin (may be distant site)
- Skin peels off with little pressure – skin looks abnormal – damage from within
- Commonest in infancy
- Treatment: flucloxacillin plus burn treatment (including fluid balance)

Folliculitis

- Pyoderma located within the hair follicle
- Usually caused by *S aureus*
- Responds well to topical antibacterial measures

Furuncle

- = A 'boil'
- A deep inflammatory nodule
- In skin areas subject to friction and perspiration and containing hair follicles
- Often drain spontaneously, especially with moist heat

- If recurrent, then ?nasal carriage of *S aureus*. Treat with topical intranasal mupirocin or systemic rifampicin
- May progress to a carbuncle: more extensive involving subcutaneous fat. If surrounding cellulitis or if on face then need iv antibiotics

Cellulitis and Erysipelas

- Infection of subcutaneous layer by *Strep Pyogenes*
- Symptoms: inflammation, warmth, erythema, pain, fever
- Can → sepsis, bullae and small abscesses
- Also erythema around anus with puss and blood in stool
- May desquamate
- Impaired lymphatic drainage predisposes to recurrent cellulitis (e.g. pelvic, joint, breast surgery)
- Erysipelas is a distinctive superficial cellulitis, primarily involves dermis. Raised and well demarcated. Prominent lymphatic involvement. May → chills, fever and malaise
- Treatment: *S Pyogenes* still very susceptible to penicillin

Diabetic Foot infections

- Due to neuropathy, ischaemia, and infection
- Causes: often *S aureus*, also coagulase negative staphylococci and streptococci
- Often nasal carriage of *S aureus*
- Treatment: anti-staphylococcal agents. IV treatment if deep tissues or bone involvement

Deep Tissue Infections

- Necrotising Fasciitis: See *Streptococcus Pyogenes* (Group A, β Haemolytic), page 75
- Superficial necrotising cellulitis or streptococcal gangrene (rare)
- Gas Gangrene (*Clostridium myonecrosis*): rapidly progressive and life threatening infection of muscle due to *Clostridium Perfringens*

Scarlet Fever

- See *Streptococcus Pyogenes* (Group A, β Haemolytic), page 75

Lymphadenitis

- May require drainage. Distinguish from lymphadenopathy
- Usually *Staph aureus*, also TB
- See Cervical Lumps, page 67

Toxic Shock Syndrome

- See *Streptococcus Pyogenes* (Group A, β Haemolytic), page 75
- Desquamation a week later characteristic

Dog Bites

- Clean carefully (may need local anaesthetic)
- Treat with broad-spectrum antibiotic. Amoxicillin/clavulanate. NNT = 14. So limit to high risk of infection only. Consider anaerobe cover (eg metronidazole)
- Screen for post-traumatic stress disorder afterwards
- Report the dog

Lyme Disease

- Tick borne spirochete (*Borrelia burgdorferi*)
- Gives erythema migrans, headache, fever, myalgia, fatigue
- Leads later to widespread systemic manifestations
- Discovered in Connecticut, USA. No in NZ

Fungal Infections/Dermatophytosis

- = Tinea
- Fungal infections of animal (zoophilic) origin: These include “ringworm” (which causes a scaling macule – not a ring – and there is no worm!). Usually in children, for example from cows, dogs, cats or mice

Clinical Description

- Fungal infections usually itch. Have a raised scaling margin that extends outwards
- There are several classical presentations:
 - Tinea Cruris: in the groin. Mainly affects men. Sharp margin. On thighs or buttocks may get follicular pustules. If feet involvement as well then systemic treatment, otherwise topical
 - Tinea Capitis: Scalp. Invasion of hair shaft. Causes alopecia. Usually M Canis. Usually children aged 3 – 7, nearly always cervical occipital lymphadenopathy. Needs systemic treatment (topical doesn't penetrate hair shaft).
 - Athlete's Foot/Tinea pedis: on the feet (usually lateral toe clefts – compared with eczema which in medial toe clefts). Increased sweating predisposes to fungal infection. It can be spread to the sole with a powdery scale. To hands by itching, where it presents with a dry, hot rash on one palm, with well defined lesions with a scaling edge
 - Tinea Corpus: on the trunk. Presents with an erythema and itching, and a well defined, scaling edge. May not itch
 - Tinea manuum: Hand. Almost always a pre-existing foot infection.
 - Fungal infection of the nail (Onychomycosis): occur mainly in adults, usually in their toenails (fingernails uncommon, ?psoriasis), and especially following trauma. The nails become thickened, yellow, and crumble, usually asymmetrically. The changes occur distally, and move back to the nail fold (compared with psoriasis, which is symmetrical and moves distally from the nail fold)
 - Tinea Incognito: Fungal infection treated with steroids. Stops inflammation but fungus slowly spreads → follicular pustules etc.
- Tinea Versicolor:
 - Infection due to a commensal yeast *Malassezia Furfur* (= *pityrosporum ovale*. Not a fungus). In young adults, causes hypo- or hyper-pigmented macules with powdery scale, on upper trunk, upper arms and neck. Slightly itchy
 - Differential diagnoses:
 - Vitiligo: but pure white lesion (amelanotic), no scaling
 - Pityriasis alba: Usually children and on the face. Tinea Versicolor rare in children
 - Treatment: Imidazole cream, sporanox, selsun shampoo

Diagnosis

- Consider in any patient where isolated, itching, dry and scaling lesions occur for no reason (e.g. no history of eczema). Fungal lesions are usually asymmetric. Clippings or scrapings can be sent for culture

Pathogenesis

- Common: *Microsporum Canis* (from cats, fluoresce under Wood's light), *Trichophyton rubrum*, and *Trichophyton mentagrophytes*
- Less common: *Trichophyton tonsurans*, *Epidermophyton floccosum*, *Trichophyton erinacei*
- Fungi consist of thread-like hyphae that invade keratin (yeasts do not have hyphae). Vegetative spores (conidia) develop in culture. When immune response is impaired, superficial infections may invade deeper tissues

Management

- Topical Treatment: imidazole preparations, such as clotrimazole and miconazole. Dusting preparations are also available. Terbinafine is available as a cream
- Systemic Treatment: Diagnosis should be confirmed before commencing treatment. Terbinafine (250mg, once daily PO) for 2 to 6 weeks for skin infections and 3 months for fingernail infections, 6 months for toe nail infections. (Pregnancy and lactation are relative contraindications). Can take itraconazole 1 week per month for 3 months (200 mg bd) → ↓ side effects. Takes 12 – 18 months to grow a new nail. Given length of treatment, confirm with nail scraping for culture first.

Viral Infections

Molluscum Contagiosum

- Viral infection with pox virus
- Small solid papules with umbilication in middle. Stay fairly localised
- If you squeeze them then virus released (ie infective)
- Histology: acanthosis and molluscum bodies
- Disappear in under 9 – 12 months. Treat if severe

Verrucae (Warts)

- Papova virus: **P**apillary lesion + **p**olyoma (lots of them) + **v**acuolation of cells containing the virus
- Locations:
 - Verruca vulgaris
 - Verruca plana: flat, eg on face
 - Verruca plantaris: on feet, can be painful
 - Verruca palmaris: on hands, can be painful
 - Condyloma accuminatum: Genital. Rarely premalignant
- Histology:
 - Hyperkeratosis/parakeratosis
 - Acanthosis
 - Nuclear and cytoplasmic inclusions
 - Perinuclear vacuolation

Other Viral Illness

- See Varicella Zoster, page 79
- See Herpes Simplex Virus (HSV), page 78

Other Infections

Paronychia

- Loss of cuticle (due to eczema, wet work, etc) allows growth of organisms beneath the proximal nail fold → inflammation and nail dystrophy. Acute usually staph, chronic usually candida
- Differential:
 - Onychomycosis
 - Lupus, psoriasis, chilblains
- Treatment: avoid wet work, treat eczema, drying agent, systemic antibiotic if bacterial

Pitted Keratolysis

- Small craters in the sole of the foot. Asymptomatic. Foot odour
- Various attributed to *Corynebacteria*, *Dermatophilus*, *Micrococcus*
- Treatment: keep feet dry, topical erythromycin, systemic tetracyclines

Pityriasis Rosea

- Usually 10 – 35. Starts with herald patch (larger than later lesions). After 5- 15 days general eruption begins. Oval, dull pink, with marginal scale. Itch varies. On trunk, rarely on face
- Was thought to be viral, but erythromycin effective
- Fades after 3 – 6 weeks
- Differential: eczema, psoriasis, seborrheic dermatitis, tinea versicolor

Candidiasis

- Yeast infection
- Common in infants – either mouth (esp inside cheeks) and in nappy area, maybe on hands if sucked. More common in damp areas. Need to treat Mum's nipple as well.
- Lesions whitish with satellite lesions characteristic
- Also with oral/inhaled steroids or broad spectrum antibiotics
- Systemic spread in immuno-compromised is nasty
- Treatment: nystatin or miconazole

Scabies

- Irritation from hypersensitivity after 4 weeks of scabies mite burrowing
- Papular vesicular lesions

*Headlice **

- The insect: 2 – 3 mm long, breeds all year round. They live in the scalp and suck blood for food 5 or 6 times a day. They are only transmitted through close head contact. They don't come off with swimming or washing. The eggs are a similar colour to scalp skin. The empty egg shells, known as nits, are white
- Life cycle: female lice lay about 7 – 10 eggs each night, these hatch in 9 days. A louse will live for 40 days

- Where to find them: around the hairline at the back of the neck, behind the ears, on the crown
- Treat if you find a live insect or an egg within 1 cm of the scalp (hair grows 1 cm a month, so more than 1 cm from head means they're dead)
- Use special shampoo from the chemist. Leave on scalp for 5 – 10 minutes. Don't use too much water. Repeat a week later
- Don't need to wash bedclothes: lice only lay eggs on hair. Instead check kid's heads once a week
- Prevention: regular hair brushing, don't share brushes, keep clothes separate, contact tracing

Eczema

- = Dermatitis
- Formal definition: pattern of inflammatory response of the skin which can be defined histologically by the presence of a predominantly lymphohistiocytic infiltrate around the upper dermal blood vessels, associated with spongiosis (= oedema between keratinocytes) and varying degrees of acanthosis.
- Clinical features include itching, redness, weeping, scaling and clustered papulovesicles
- Endogenous forms:
 - Atopic
 - Seborrheic
 - Discoid
 - Juvenile plantar dermatosis
 - Pompholyx
 - Pityriasis Alba
- Exogenous forms:
 - Asteatotic
 - Irritant contact dermatitis
 - Allergic contact dermatitis

Atopic Eczema

Symptoms

- Onset usually 2 – 6 months
- Acutely:
 - Itchy
 - Redness, swelling, usually ill-defined border
 - Papules, vesicle, extremely large blisters, may look weepy
 - Exudates and crusting
 - Scaling
 - Can be papular
- Chronic:
 - Less vascular and exudative
 - More scaly, pigmented and thickened
 - Fissuring
 - More likely to be lichenified (epidermal thickening with exaggeration of skin markings) and develop painful fissures
 - If dark skin: post inflammation change in pigmentation
 - Pitting of nails if involved with ridging of nails
- In babies:
 - Common onset in first few weeks
 - Quite weepy/blistery
 - Around face (spares eyes and base of nose) and trunk. If extensor distribution think of contact sensitivity (eg house dust mite)
 - Can be due to antigens in breast milk
 - The itch that rashes: itchy skin is scratched and an eruption occurs – don't see rash where child can't reach
- Children, and older:
 - Bends of elbows, behind knees
 - More leathery
 - Between big toe and 2nd toe (compared with tinea between 4 and 5)
- Associated with asthma and hay fever

- Associated with food allergy – commonly cows milk but this is overstated
- Atopic skin has lower threshold to irritation (eg soaps) and is more prone to staph infection
- Prognosis: ½ have cleared by 12, few persist after age 30
- Increased tendency to: dry skin, urticaria, pityriasis alba, keratosis pilaris, irritant contact dermatitis, etc

Pathogenesis

- Genetic predisposition
- ?Imbalance of Th1 and Th2 cells in the thymus in favour of Th2
- ?Early childhood infections → preferential induction of Th1 type cytokines and prevent atopic sensitisation. ↓Infections → greater risk of atopy
- Inversely proportional to the number of older siblings (marker of exposure to infection)
- Atopy does not equal allergy:
 - Level of IgE, which may be elevated, doesn't correlate to severity
 - Up to 50% of children with eczema do not have +ive skin prick tests (especially if mild eczema and no asthma)
 - Skin prick tests for histamine release (type 1 reaction) may be positive but the person may have not react when exposed to that allergen
 - Rast test looks for antigen specific IgE
 - Type 1: normally asthma, rhinitis, urticaria, not usually eczema
 - Patch testing (Type 4) may be relevant to childhood eczema
 - Only 50% with severe eczema develop reactions when challenged with particular foods – most are delayed reactions

Management

- Investigations
 - Patch testing
 - Is there infection? (Yellow crusts, weepy, failure to respond to treatment) → systemic antibiotics
- Prevention:
 - Don't itch
 - Avoid aggravators:
 - Light cotton clothes, no scratchy woollens
 - Avoid excess humidity/dryness
 - Avoid local or systemic aggravators
 - Care with soaps, perfumes, solvents etc
 - Baths not shower, not too hot, pat not rub dry
 - Reduce stress
 - Control dry skin: Emollients – aqueous cream, white soft paraffin
- Medical:
 - Topical corticosteroids:
 - Reduce inflammation but doesn't treat cause
 - Use weakest possible – 1% hydrocortisone OK for most
 - At night use in conjunction with wet dressings (containing emollient)
 - Not for too long otherwise skin atrophy, striae and rebound afterwards, wrinkling, ↑vascular markings, also dynamite to viral/bacterial infections. Even worse with systemic steroids
 - Lotion for scalp, ointment for dry areas (may cause folliculitis), cream
 - Strength:
 - Face and flexures: mild only
 - Scalp, palms and soles: can tolerate very potent steroids (eg betamethasone dipropionate)
 - Body and limbs: potent for short periods (a week or two), mild to moderate as maintenance
 - Systemic steroids for severe eczema, for a short time only
 - Tar compounds: esp. at night to prevent itching
 - Antihistamines: stop itching (more in kids and for sedative effect) and urticaria
 - Antibiotics for infection
 - For severe eczema: phototherapy, azathioprine, cyclosporin

Seborrhoeic Dermatitis

- Scalp, eyebrows and nasal labial folds. Cradle cap in babies whose scalp was clear at birth
- Red, greasy scale, sharply circumscribed

- In kids = another presentation of atopic. Treat the same. Differential: Infantile psoriasis
- In adults = allergy to yeast (*Pityrosporum ovale*) which arrive with grease gland activation at puberty
- Differential:
 - Psoriasis. But doesn't often affect the face
 - Discoid, and other forms of eczema
 - Pityriasis rosea (usually on trunk and not on the face)
 - Fungal infection: annular, scaling isn't greasy

Contact Dermatitis

- May be irritant or allergic or both. May co-exist with endogenous forms (eg atopic)
- Differentiate from endogenous on the basis of history, distribution and maybe allergy testing, not morphology

Contact Irritant Dermatitis

- Irritant: a substance which induces dermatitis in anyone if applied in sufficient concentration for long enough → penetrates skin and produces cellular damage
- Individuals vary in their threshold
- Heat and ↑ or ↓ hydration impair barrier function → more susceptible
- Cumulative effect of different irritants
- Irritants include: acids, alkalis, solvents, soaps, detergents, enzymes, abrasives
- Diagnosis:
 - Exposure to irritants for what length of time and frequency
 - Are sites consistent with exposure
 - Does it improve after exposure stops
 - Can contact allergy be excluded (eg have they had it since childhood ⇒ more likely to be allergy)
- Management:
 - Steroid creams, emollients
 - Reduce exposure, remove occlusion (ie sweat inside gloves → over hydration), other work

Contact Allergic Dermatitis

- Type 4 cell mediated immune reaction
- Often takes repeated exposure, so no previous symptoms may not be significant (same for type 1 reactions). Eg may have worn rubber gloves for years
- Once sensitised, further exposure to even minuscule amounts → reaction after a day or two. Takes 24 – 72 hours, compared to type 1 which takes 15 – 20 minutes
- Will involve primary sites, and maybe distant sites (eg eyes, genitals)
- Photoallergy = need exposure to allergen + UV light to cause rash. Eg sunscreens
- Common allergens: nickel (eg pierced ears), rubber additives, plants, chromate in cement, hairdressing chemicals, perfumes, ...
- Rubber glove allergy can be:
 - Type 1 due to rubber
 - Type 4 due to rubber additives
 - Contact dermatitis due to sweaty hands - ↑ risk of type 1 or 4 reaction (mediated by Langerhans cells) due to ↓ barrier function
- Diagnosis:
 - Exposure to possible allergens
 - Sites consistent with exposure, goes away when exposure stops. NB some sites resistant (scalp, soles)
 - Patch testing
- Management:
 - Steroids, emollients, etc
 - Avoid exposure

Other Eczema Related Conditions

Discoid Eczema

- Descriptive term: round or oval, well circumscribed, red, scaly, +/- vesicular (→ weepy)
- In kids: often atopic
- In Adults: often cause unknown

- Differential:
 - Ringworm: tends to be annular (worse at the edge). May have alopecia or pustules. Take scraping for culture.
 - Superficial BCC: doesn't usually itch, often a shiny surface, dots of pigment
 - Psoriasis: silvery scale, not weepy

Juvenile Plantar Dermatitis

- Fissured dermatitis of the plantar surface of the forefoot – red, glazed, cracked, symmetrical, toe clefts normal
- In children 3 – 14 years
- Usually atopic
- Treatment difficult: Urea creams, moisturisers, steroid creams. Has usually resolved by teens.

Pompholyx (dyshidrotic eczema)

- Not related to atopic eczema
- Vesicles +/- bullae on palms, soles, sides of fingers or toes
- Erythema or scaling absent. If present then just a vesicular eczema
- Heals with desquamation
- Differential: fungal infection
- Treatment: ?steroids

Pityriasis Alba

- Round/oval, hypopigmented, fine lamellar scaling, from 5 – 20 mm, commonly on face
- Usually age 3 – 16
- Associated with atopy – but may be independent
- No treatment – goes away by itself

Asteatotic Eczema

- Related to dry skin
- Usually on legs, usually diuretics, excessive washing or hypothyroidism
- Superficial fissures create a crazy paving pattern
- Treatment: soap substitute, moisturiser +/- topical steroid

Intertrigo

- Generic term for inflammatory dermatosis in skin folds (eg submammary or genitocrural)
- May be a form of atopic or seborrheic dermatitis
- Can be secondarily infected with candida or staph
- Treatment:
 - Reduce friction, avoid tight clothing
 - Mild steroid, antibacterial or anti-candida cream

Angular Cheilitis

- Affects the fold of skin at the corner of the mouth. Especially in denture wearers
- May be a form of intertrigo, can be associated with atopic or seborrheic dermatitis
- May be infected with candida or staph, may be folate deficiency, a frequent complication of *Roaccutane* treatment

Nappy Rash

Napkin Dermatitis

- Irritant contact dermatitis caused by prolonged contact with wet nappies
- Sparing the flexures
- Bacterial conversion of urine to ammonia →alkaline irritant
- Treatment: frequent changing, carefully washing, protective cream

Differential: Candidiasis

- Frequently superimposed on nappy rash
- Flexures involved + satellite lesions or superficial pustules on a background of erythema
- Treatment: antifungal cream (nystatin)

Acne

- Inflammatory disease occurring in and around the sebaceous glands, generally affecting the face, also the chest and back. Characterised by papules and pustules, or by cyst and other more specific lesions. Deeper lesions are associated with scarring: hypertrophic, keloidal or depressed
- Differential:
 - Rosacea
 - Perioral dermatitis
 - Acneiform drug eruptions

Pathogenesis

- Four factors:
 - Increased sebum production by the sebaceous glands (normally produced to maintain epidermal hydration)
 - Cornification (→blockage) of the pilosebaceous duct: abnormal keratinisation and desquamation of follicular epithelium combine with increased amounts of sebum production to obstruct the duct.
 - Bacterial proliferation - abnormal colonisation of the follicle duct by *Propionibacterium acnes*. But severity not proportional to number of bacteria
 - Inflammation
- If the obstruction is closer to the skin surface it will form open comedo and oxidation of the fatty material causes discoloration (blackhead). A closed comedo (white head) occurs when the duct is blocked at a deeper level
- Acne is dependent on:
 - Genetic factors (high concordance in monozygotic twins)
 - Hormonal factors: androgens → sebum production
 - Environmental factors: aggravated by humidity, some cosmetics and oils (block pilosebaceous orifice)
 - Diet rarely implicated
- Usually starts in adolescence and resolves by mid 20s (starts earlier in females and is more persistent)

Management

- *Reassurance*: Treat as a physical and psychological disorder. Undermines patient's self-confidence, especially in the adolescents. Myths of poor diet and hygiene make patients feel responsible and/or guilty - reassured that they are not the cause
- General advice:
 - Avoid humid conditions
 - Avoid occlusive creams and sunscreens
 - Only use moisturisers if the skin is dry
- *Topical agents*. For mild to moderate acne:
 - Comedolytics: most effective option is Tretinoin. Normalises desquamation of the follicular epithelium promoting drainage of pre-existing comedones. This increases penetration of antimicrobial agents
 - Antibiotics such as benzoyl peroxide and erythromycin gel reduce bacterial numbers and inflammation
- Oral agents. Are generally used for severe or persistent acne in addition to topical agents:
 - Antibiotics such as tetracycline, doxycycline, trimethoprim and erythromycin suppress inflammation by inhibiting neutrophil chemotaxis and production of bacterial lipases and proteases. For a minimum of six months with an 80-90% improvement expected after this time. Often recur. SE of Minocycline: vertigo, discolouration of teeth, *grey skin pigmentation*
 - Oestrogens. They have a direct effect on sebaceous gland activity. They are combined with progesterone in an oral contraceptive, which may counteract the effects of the oestrogen
 - Antiandrogens (in a female only) such as cyproterone acetate and spironolactone act peripherally to inhibit androgen stimulation of sebaceous glands and hair follicles. They are useful in mature presenting acne
 - Isotretinoin (Roaccutane)
 - A synthetic Vitamin A derivative that inhibits sebaceous gland activity, reduces *P. acnes* cell numbers, alters follicular keratinisation and is anti-inflammatory
 - At adequate doses permanently cures acne in 80% of cases after 4 – 6 months

- Highly teratogenic. Women need to be fully informed of the risks, need to have a negative pregnancy test before starting treatment, and need to be on reliable contraception throughout course (i.e. birth control pills and condoms) and one month after
- Causes liver damage and hyperlipidaemia: baseline bloods and then after one month
- Causes dry lips and maybe nasal mucosa (→ epistaxis), skin and eyes, angular cheilitis
- < 10% will get aching muscles, depression, hair loss, headaches

Rosacea

- Cardinal signs in order of importance:
 - Erythema
 - Telangiectasia
 - Papules
 - Swelling
 - Tiny pustules
- On cheeks, chin, forehead, nose and neck, sun exposed sites. Flushing may precede other signs
- Many theories
- May be associated with rhinophyma (bullous swelling of the nose)
- Minor ocular involvement in 50%: especially conjunctivitis, may be blepharitis, etc
- Treatment:
 - Systemic or topical antibiotics (as per acne)
 - Retinoids
 - Metronidazole

Perioral Dermatitis

- Mainly young women
- Cause a mystery. ?Steroids implicated
- Starts in nasolabial fold and spreads to involve the perioral area. Minute papules and pustules on an erythematous base with some scaling
- Treatment: Systemic tetracyclines or erythromycin until rash gone then for another couple of weeks

Other Skin Lesions

Erythema Multiforme

- Confusion/overlap between Erythema Multiforme (EM), Stevens Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) [Later two at the severe end of the spectrum]
- Varying degrees of mucosal involvement and rash
- Typical lesion: target lesion – dull red macule or maculopapule 1 – 2 cm across, erythematous rim with cyanotic or purpuric centre. May be blistering. Typically affects acral areas (dorsal hands, feet, palms, soles, forearms, legs). Usually crop over a couple of days and fade after a couple of weeks.
- Trunk only in extensive reactions. Also if severe: erosions, haemorrhagic crusting, lesions uncomfortable (not usually painful). May affect cornea. May get systemic upset (fever, anaemia, etc)
- Histology: vacuolar degeneration of lower epidermis
- Provoking factors:
 - HSV – major cause. Rash worst at periphery (+/- oral mucosa). Will get it with subsequent outbreaks as well. History: Do you get cold sores?
 - Mycoplasma (<1% of EM)
 - Drug reactions – more likely if severe outbreak. Not typical targets (eg red blotches), on trunk as well as acral, may be blistered. Implicated drugs: anticonvulsants, sulphonamides, NSAIDs, allopurinol. Stop drugs if at all possible. Treat like a burn. Steroids controversial
 - Idiopathic

Erythema Nodosum

- Lesions: 2 – 4 cm, erythematous, tender, especially on shins but also on thighs or forearms. A little raised. Look like purplish bruises
- Number from 2 – 50 (usually 5 – 6), erupt over 10 days and subside over 3 – 6 weeks
- Regress with bruise like yellow/green colour changes
- Systemic signs: fever, generalised aching and malaise
- Due to deposition of immune complexes in and around venules in the deep dermis
- Causes:

- Kids: Streptococcal infection
- Sarcoidosis (rare in kids)
- TB
- Cat scratch disease
- Yersinia
- Some drugs
- Differential:
 - Nodular vasculitis (tend to ulcerated, don't heal with bruise like changes)
 - Meningococcal or gonococcal septicaemia (smaller lesions, often purpura, ill patient)

Erythema Toxicum Neonatorum

- Up to 50% of full term infants (less if preterm), occur up to 4th day
- Erythematous macules, wheals, papules and pustules – few to several hundred
- Face, buttocks, torso, proximal limbs, not palms or soles
- Usually resolve in several days
- Cause unknown
- Differential: HSV, Staph spots

Urticaria

- = Hives or welts. Intensely itchy.
- Relationship to allergy and atopy:
 - More likely in atopy
 - 50% related to allergy – type 1 only ⇒ exposure 15 – 30 minutes prior to onset and last < 24 hours ⇒ careful history
 - Allergy likely to be all over, and no further outbreak for weeks/months
 - Most chronic urticaria is non allergic
 - Some foods/drugs may cause urticaria without immune involvement (ie histamine release without IgE involvement)
- Common causes:
 - Idiopathic – common
 - IgE mediated:
 - Food: peanuts, strawberries, milk, eggs
 - Animal dander: horses, cats
 - Physical: pressure, cold, heat
 - Complement mediated: hereditary angioedema and blood transfusion reactions
 - Mast cell releasing agents: opiates, penicillins
 - Prostaglandin inhibitors: Aspirin, NSAIDs
 - Infections: cause of 80% of acute childhood urticaria (eg hepatitis)
 - Serum sickness: type 3 reaction. Drugs, especially penicillin. Fever, raised ESR, starts within 5 – 20 days of exposure and lasts 5 – 28 days.

Papular Urticaria

- Hypersensitivity to an insect
- Itchy, urticarial weal → firm itchy papule
- Usually gone in a day to two, may persist for months
- Grouped in clusters, and develop crops at irregular intervals
- Treatment: try insect repellent
- If dark skin, may be post-inflammatory hypopigmentation

Alopecia Areata

- Circumscribed areas of hair loss but skin normal.
- Presentations:
 - Often scalp – with a few bald areas 1 – 3 cm
 - Loss of all scalp hair is alopecia totalis
 - Loss of hair at all sites is alopecia universalis
- Not a diagnosis
- Autosomal, autoimmune dominant disorder with variable penetrance
- Duration < 1 year in 50 %, relapse common. Kids get it worse
- Associated with Atopy, Downs, Hashimoto's Disease, Pernicious Anaemia, Addison's Disease, Vitiligo

- Treatments include local steroids, topical minoxidil (antihypertensive), etc
- Differential diagnosis: all produce circumscribed hair loss, but skin itself is abnormal
 - Fungal infections
 - Anything causing scarring (eg skin cancer)

Keratosis Pilaris

- Common. More common in atopics
- Small whitish plugs of keratin obstruct the follicle mouth. Usually extensor surfaces. Feels like sandpaper
- Variable perifollicular erythema
- Facial involvement usually resolves in teens. Elsewhere can persist until middle age
- Autosomal dominant with variable penetrance
- Differential: Acne (shouldn't feel like sandpaper)
- Treatment: mild steroids, urea creams, salicylic acid creams, etc

Granuloma Annulare

- Ring of smooth, firm, skin coloured or slightly purplish papules from 1 – 5 cm. No scaling (cf ring worm which is) or blistering (\Rightarrow epidermis fine)
- Enlarge centrifugally, with beaded rim gradually flattening until it disappears without trace within 2 years
- Dorsal surfaces of feet, hands and fingers are the commonest sites
- Lymphohistiocytic granulomata
- Mainly children and young adults
- Can treat with intra-lesional steroids

Lichen Planus

- Occurs in 30 – 60 year olds. Insidious onset, can be explosive, localised or generalised. In 80% resolves in 18 months
- Clinically: flat topped papules, discrete or coalescing. White lines on papules = Wickham's Striae. Can also get annular, hypertrophic, atrophic or even bullous forms. Should linear lesions characteristic. Itch variable. Rash resolves with hyperpigmentation. Can be painful on lips or genitals.
- Looks like everything else. Differential:
 - Plane warts
 - Eczema
 - Drug reaction: gold, quinine, thiazides, etc
 - Treatment: Acitretin, steroids, miscellaneous

Tuberous Sclerosis

- Disorder of haematoma formation: especially in eye, brain, skin, kidney and heart
- Skin lesion:
 - Angiofibromas: appear from 3 – 10, firm, discrete red/brown telangiectatic papules, 1 – 10 mm, cheeks and chin
 - Periungual fibromas: smooth skin coloured excrescences emerging from the nail folds
 - Shagreen patch: skin coloured plaque in lumbosacral region
 - Oval white macules (Ash-leaf-macules) seen under Woods light. But also similar lesions common in normal kids
- Classically (but not invariably) seen with epilepsy and mental retardation ('zits, fits and nit-twits')
- Autosomal dominant with variable penetrance, 50% are new mutations
- Prevalence ?1/10,000

Neurofibromatosis

- Look like intradermal naevi but soft
- Type 1: commonest, 1/3000, Autosomal dominant, 30% new mutations
- Type 2 (1:50,000): 2 or more of:
 - 6 or more café-au-lait macules over 5 mm in pre-pubertal patients
 - 2 or more neurofibromas
 - Freckling in axillary or inguinal regions
 - Optic glioma
 - Others

- May lead to short stature, skeletal overgrowth, tibial bowing, thinning, fracture, macrocephaly, kyphoscoliosis, intellectual handicap, endocrine problems (precocious puberty, acromegaly, Addison's), neuro tumours (optic nerve glioma, astrocytomas), etc
- NF2: characterised by bilateral acoustic neuromas
- See Other Congenital Skeletal abnormalities, page 90

Ichthyoses

- All genetic
- Ichthyosis vulgaris: common, usually mild. Entire skin is scaly. Controlled with moisturisers
- Rare sorts: Collodion Baby, Bullous and non-bullous ichthyosiform erythroderma, lamellar ichthyosis, X-linked ichthyosis, Harlequin fetus

Erythroderma

- Inflammatory skin disease involving 90% or more of the body surface. Don't call it Exfoliative Dermatitis – meaning is unclear
- May have sudden onset over weeks or days. Scaling varies in degrees. Itch varies
- Well unwell, feel hot or cold even though temperature normal. Hypoalbuminaemia and oedema common
- Fatal in 20 – 40% due to pneumonia, septicaemia, cardiac failure
- Cause:
 - Eczema: 40%
 - Psoriasis: 25%
 - Lymphoma, leukaemia: 15%
 - Drug reaction: 10%
 - Unknown: 10% (usually elderly)
- History usually helpful, histology usually unhelpful
- Management: monitor fluid balance, rest, nutrition (shedding lots of protein), Moisturiser, careful use of steroids, methotrexate, etc.

Epidermolysis Bullosa

- All rare
- Variety of inherited forms. An acquired form exists
- Can be localised or generalised
- Types:
 - Generalised simple autosomal dominant epidermolysis bullosa
 - Junctional EB
 - Autosomal Recessive Dystrophic EB
 - Autosomal dominant dystrophic EB

Incontinentia Pigmenti

- X-linked dominant, usually lethal in males
- Presents within first 2 months
- Tense bullae on limbs then red nodules or plaques on limbs and trunk
- Pigmentation ranges in colour from blue-grey to brown

Epidemiology of Genetic Disorders

- 9,000 known genetic diseases
- 5% have genetic diseases before 25
- 60% during lifetime (includes diabetes, heart disease, cancer)

DNA

- There is frequent mutation in rapidly dividing cells: but repair mechanisms 'mop up'
- If there is a sustained mutation in:
 - Essential gene → lethal
 - Non-coding gene → no effect
 - Non-essential gene → human variability/disease

Chromosome Disorders

- Polyploidy: duplication of whole sets of chromosomes (eg triploidy: $n = 69$). Non-survivable → fetal wastage
- Anuploidy:
 - One missing or additional chromosome
 - Trisomy 21: 47XY + 21. See Down Syndrome, page 27
 - Trisomy 13: next most common trisomy
 - Turner's Syndrome: 45, XO
 - Puffy feet, poor toe nails, redundant skin behind head/neck, kidney and cardiac malformation
 - Later: short, infertile, normal mental ability (unless 2nd X ring chromosome → mental disability)
 - The 10% that survive to term are the good end of the spectrum
 - Differential: Noonan's Syndrome – similar symptoms but karyotype is normal
 - Klinefelter Syndrome: 47, XXY
 - 1/3 present in childhood with learning difficulty
 - 1/3 present in adolescent: failure of puberty due to no testosterone (ie hypogonadism)
 - 1/3 present in adulthood due to infertility
- Chromosome abnormalities:
 - Lead to multiple gene errors/deletions. Suggested by dysmorphisms, multiple congenital abnormalities, developmental problems.
 - Deletions, insertions, etc. Will be different in each child → variable presentation. Eg deletion in 5p: Cri du Chat syndrome, cat like cry
 - Fragile X Syndrome: commonest cause of mental disability in males. 1:1,000. Dynamic mutation – changes over generations. Not dysmorphic – post pubertal changes + non-specific cognitive delay. Widely tested for as a result → low yield.
- In testing for mosaics, may need to test skin, not blood, as abnormal cells don't reproduce so well so get weeded out in tissues (eg blood) with high turnover

Other Disorders

- Agenesis: complete absence of an organ
- Aplasia: absence of an organ with the persistence of an undeveloped rudiment
- Anencephaly: congenital absence of cranial vault – with cerebral hemispheres completely missing

Patterns of inheritance:

- Autosomal Dominant
 - =Single gene abnormalities expressed in heterozygotes
 - M = F, 50% risk of passing it to kids
 - 1 abnormal gene causes disease
 - Eg Huntington Disease, Marfan Syndrome, Achondroplasia (disturbance of epiphyseal chondroblastic bone formation)
 - But:
 - Variable expression, variable age of onset
 - Non-penetrance happens

- Gonadal mosaicism (esp if old paternal age) → somatic genes normal, mutation in gonads
- Autosomal Recessive:
 - = Only symptomatic when both alleles at a locus on homologous chromosomes are defective
 - Must have mutations in both genes ⇒ both parent's carriers
 - Shows up early (no normal genes)
 - Eg cystic fibrosis, phenylketonuria
- X-linked Recessive:
 - Only affects males
 - Impact early (no normal gene)
 - Females are carriers (random X inactivation should mean that 50% of cells are abnormal. But they're usually in the minority. Still may have some traits)
 - Eg haemophilia, Duchenne muscular dystrophy (wasting muscle disease – most dystrophies are X linked)
- X-linked Dominant: eg Fragile X
- Multifactorial:
 - Common diseases
 - Genetic predisposition plus environmental influence
 - Eg Cleft lip/palate
- Others: mitochondrial, tumour predisposition

Non-Mendelian Genetics

Genetic Imprinting

- = Differential expression of genetic material depending on whether it has been inherited from male or female parent
- ⇒ Parent of origin of mutation matters for many genes
- Affected genes are usually highly conserved (ie the same genes appear in mice and humans – conserved through evolution)
- Myotonic dystrophy:
 - Autosomal dominant
 - Progressive weakness from 3rd decade
 - Unstable triplet repeat on 19 (upper limit of normal is 50 repeats)
 - Most unstable when its from mum (ie parental imprinting)
 - As number of repeats increases goes from normal → premutation carrier → affected
- Fragile X Syndrome:
 - Abnormal if triplet repeat > 200
 - Only expands when passed from mother to son, not to daughter
- Huntington's:
 - Unstable triplet repeat syndrome
 - If father passes it on then greater risk of ↑ number of repeats

Uniparental Disomy (UPD)

- = Presence of a cell line containing 2 chromosomes both inherited from only one parent
- Has been demonstrated in cystic fibrosis, haemophilia (ie got both mutated genes from the one parent)
- Prader-Willi Syndrome:
 - Floppy baby, low birth weight, retarded, ↑↑appetite → obesity, short stature
 - Caused by deletion on father's Chr 15, or have both normal Chr 15 from mum (ie no 15 from dad)
 - Angelman Syndrome caused by maternal deletion of the same chromosome → low birth weight, unusual cry, stiff legged gait, tremour and seizures

Mitochondrial Disorder

- Mitochondria:
 - Generate ATP for energy using the respiratory chain
 - Contain their own DNA: circular double stranded DNA
 - All come from mother
 - Have higher mutation rate
- Heteroplasmic: up to 200 mitochondria per cell, up to 20 different DNAs per cell
- Usually involves all tissues, very variable expression
- Difficult to test for and hard to treat

Genetic Testing

- Types of test:
 - Screening tests: done on normal population to identify those at risk (not diagnostic)
 - Diagnostic tests to confirm the presence of disease
- Guthrie Card – for screening all neonates:
 - Second test needed in about 1 in 100 babies (usually due to poor sample)

Disease	Treatment	Risk
Biotinidase Deficiency	Take vitamin H (biotin)	1:50,000
Congenital Adrenal Hyperplasia	Steroids	1:20,000
Cystic Fibrosis		1:3,000
Galactosaemia	Diet	1:120,000
Hypothyroidism	Thyroid replacement	1:4,500
Maple Syrup Urine Disease	Diet	1:250,000
Phenylketonuria (PKU)	Diet	1:15,000

- Indications for neonatal genetic testing: Physical, growth or developmental disorders:
 - Still birth
 - Multiple congenital abnormalities
 - Small for age
 - Facial dysmorphism: not “a constellation of features outside the normal range”, but a particular constellation of features and the specificity of the combination. Dysmorphism is only a small part of the diagnosis. The developmental trajectory is critical.
 - Significant mental retardation
 - Post-natal growth retardation
 - Microcephaly
- Principles of testing
 - Need to request specific test, so need to think of the question you want to ask
 - Tests are getting more sensitive due to higher resolution (eg now pick up micro-deletions) – sometimes karyotypes and DNA test suggest abnormalities that turn out to be benign variants. Genetic tests are *not* definitive
 - You’re not just testing an individual, you’re testing a family. Need to take them all with you. Testing can cause psychological damage. Go carefully.

Types of testing

- Karyotype: need live blood, use Lithium heparin tube. High resolution Karyotype harder on amniocentesis blood
- Telomere testing: telomeres are gene rich – but too small for small errors to be picked up in karyotypes, so do telomere tests
- Chromosome painting (Spectral karyotyping) – not available in NZ

Childhood Cancer

- Epidemiology:
 - 10% of childhood deaths, most common cause of death after accidents ⇒ have high index of suspicion
 - 160 < age 18 diagnosed in NZ annually
 - Bimodal onset: age 2 – 6 (embryonal and leukaemia) and 12 – 18 (lymphoma and bone)
 - Median age 5
 - Survival has improved dramatically over last 40 years – mainly due to improved management than to new agents
- Differences compared with adults:
 - Adult cancers are epithelial: lung, colon, breast, prostate, pancreas...
 - Childhood cancers arise in developing organs: marrow, lymph glands, kidney, brain, bone,...
 - Childhood cancers rarely inherited
 - Childhood leukaemia and solid tumours respond better to chemo than adult versions

- Distribution:

	% of cancers
ALL	23
CNS	21
Neuroblastoma (→ chemo)	7
Non-Hodgkin's Lymphoma	6
Wilm's (Kidney)	6
Hodgkin's Lymphoma	5
AML	4
Melanoma	2

- Signs and Symptoms:

- Often non specific
- Adult symptoms rare, eg epistaxis, dysphagia, non healing lesion, rectal bleeding, change in bowel habit
- Para-neoplastic syndromes are rare

- Headaches warranting investigation. Headaches are common, but watch out for:

- Recurrent morning headaches
- One that awakens the child
- Intense and incapacitating
- Headaches that change in quality, frequency and pattern (eg getting more frequent)
- Focal signs or ataxia
- MRI more sensitive than CT

- Lymphadenopathy:

- Common finding in cervical, axillary and inguinal chains. Usually < 1 cm
- Most enlarged nodes are due to infection
- Suspicious if found in mediastinum, posterior auricular, epitrochlear and supraclavicular

- Bone and Joint Pain:

- Early symptoms rarely include pain – except in bone (bone cancer and malignancy)
- Usually no pathognomic signs on Xray → need biopsy

- Pancytopenia:

- Common finding in ALL and AML
- Need neutrophil count specifically. ↑Lymphocytes may mask ↓neutrophils.
- From 6 months to puberty, anaemia is 110 g/L. 50% of leukaemia presents with Hb < 75 g/L
- Involvement of two or more lines → bone marrow evaluation

- Leukocytosis: Common in AML and ALL. But count may get up to 50,000 with septicaemia and some viruses, also in Down syndrome and post-natal

- Presenting signs of cancer:

- Recurrent bone pain, paleness, weight loss: leukaemia
- Morning headache with vomiting: brain tumour (usually a migraine)
- Lump in neck not responsive to antibiotics: Lymphoma
- White dot in new born eye: Retinoblastoma
- Proptosis (bulging eye): Leukaemia, neuroblastoma
- Swollen face and neck: lymphoma, leukaemia (compression of veins)
- Abdominal mass: Wilm's, neuroblastoma, liver & spleen enlargement in leukaemia
- Cough, stridor, haemoptasis, Horner's: Mediastinal tumour

- Diagnosis: tumour markers (only in neuroblastoma: catecholamine), imaging, bone scan, biopsy

- Bone Marrow Transplant:

- Allows megatherapy – removes side-effect limit on dose where a dose-response exists
- Types: Autologous, allogenic, syngenic
- Source: Peripheral blood stem cells, marrow, cord blood
- Indications for autologous transplant: High dose therapy for solid tumours
- Indications for allogenic transplant: High risk ALL, relapsed ALL, relapsed AML, non-malignant (eg SCID)
- Risks include veno-occlusive disease: liver disease involving partial or complete occlusion of branches of hepatic veins
- Conditioning regime – an individualised cocktail to prepare the body and reduce the risk of graft vs host disease
- Graft Vs Host:

- Causes severe side effects
- Prophylaxis includes T cell depletion, cyclosporin, methotrexate, methylpred
- Acute: within 100 days of transplant – affects skin (rash to bullae to necrolysis), liver and gut
- Chronic: Will usually have had acute
- Side effects of treatment:
 - Radiotherapy:
 - Toxicity depends on dose and dose rate.
 - Extent of learning disability depends on age and dose
 - Gonadal function: ovaries more resistant than testis. Sertoli cells more sensitive than Leydig cells (ie azospermia before no testosterone)
 - Anthracyclines: myocyte damage dependent on cumulative dose, age and concurrent RT
 - Cisplatin: hearing and kidney impairment
 - Alkylating Agents: Sterility esp in males, renal tubulopathy, secondary malignant neoplasms (myelodysplastic syndrome)
- Late Effects:
 - 1 in 900 people aged 16 – 34 are survivors of child cancer. 50 – 60% have at least 1 major chronic problem
 - Therapy may disrupt normal growth and development:
 - Skeletal growth: radiotherapy and steroids
 - Cardiac growth: anthracyclines → cardiomyopathy, sudden death
 - Lungs: alkylating agents, radiotherapy → pulmonary fibrosis
 - Endocrine: radiotherapy → growth failure, hypopituitarism
 - Fertility: radiotherapy, alkylating agents → menstrual disorders, ovarian failure, azospermia
 - Renal → glomerular impairment, tubulopathy
 - CNS: radiotherapy, intra-thecal chemotherapy → cognitive, seizures, vision
 - Cognitive function: radiotherapy
 - Psychological → PTSD (especially adolescent tumours and mothers), other anxiety disorders and depression
 - Secondary malignant neoplasms (1 – 5%): depends on type of treatment – solid tumours, leukaemia, breast cancer

Leukaemia (ALL and AML) and Lymphoma

- History:
 - May be minimal symptoms in ALL
 - AML more aggressive so more unwell, but less common
 - Pallor, bruising most common
 - Bone pain, limp, pauciarticular arthritis
 - Recurrent infections uncommon
- Exam:
 - Maybe few clinical signs
 - Pallor, bruising
 - Gum hypertrophy
 - Lymphadenopathy – rapid progression in lymphoma
 - Hepato-splenomegaly
 - Testes – infiltrate in ALL
 - CNS – papilloedema in CNS disease
 - ALL: risk factors – males worse than females, older worse than younger, Philadelphia translocation (t9:22) bad

Solid Tumours

- History:
 - Depends on location
 - Asymptomatic mass with increase in size
 - Localised bone pain or progressive swelling over bone
 - Abdominal distension, may be subtle
 - Flank mass +/- pain
 - Pelvic masses: constipation, urinary incontinence, pain
- Exam:

- Masses usually non-tender, very firm, may or may not be fixed
- Neuroblastoma → lymphadenopathy + hepato-splenomegaly

CNS Tumours

- History:
 - Depends on location
 - Headache
 - Seizure (especially focal)
 - Posterior fossa (brain stem and cerebellum): ataxia, visual disturbance
- Exam:
 - Papilloedema
 - Ataxia for cerebellum
 - Cranial nerve signs
 - Cord lesions very uncommon except for metastases

Emergency & Surgical Management

Child's body weight

- Two formulas:
 - Under 9 years: $kg = 2 * age + 9$
 - 9 and over: $kg = 3 * age$
- or
 - $(age + 4) * 2$

Assessing Fluid State

Assessing Vital Signs

- Rate is always subservient to quality:
 - Thready pulse: eg palpable at neck and groin only
 - Respiration: more important than rate are grunting, flaring, subcostal retraction, use of accessory muscles (in neonate → bobbing of head)
- Blood volume:

Neonate	90 ml/kg
Child	80 ml/kg
Adult	70 ml/kg
- Urine output:
 - In nappies: 2 ml/kg/hr
 - Toilet trained: 1 ml/kg/hr
- Heat loss:
 - 70 kg person: surface to mass ratio is 0.02
 - 2 kg person: surface to mass ratio is 0.08
 - Rate of heat loss is proportional to $(body\ temp - room\ temp)$ to the power of 4. Best way to maintain body heat is therefore to heat the room.

Assessing fluid loss

- Only reliable indicator is pulse. BP doesn't drop till severe dehydration (compared with adult where BP declines proportionately with losses)
- No physical signs until > 3% loss
- Most signs of dehydration are those of shock
- Recent (not chronic) change in body weight is the most accurate estimate of fluid loss – but is rarely available
- Dehydration in obese children is often under-estimated

	Mild	Moderate	Severe
Loss: total body weight	3 – 5%	6 – 9%	> 10%
Mental state	Thirsty, alert	Thirsty, lethargic	Drowsy, hypotonic
Skin turgor	↓	↓↓	↓↓↓
Mucus membranes	Dry	Very dry	Parched
Skin colour	Pale	Grey	Mottled
Urine	Oliguria	Oliguria	Marked oliguria
Blood pressure	Normal	+/- Normal	< 70 systolic
Peripheral temperature	Cool	Cool	Cold & clammy
Pulse	+/- ↑	↑	↑↑ & thready

- Assessing turgor: pinched edge of skin goes down slowly. Do centrally on abdomen, chest, thighs
- Also when severe: rapid, sighing respirations (Kussmaul breathing)
- Poor predictors of dehydration: Sunken eyes or anterior fontanelle, dry mucous membranes, absence of sweat or tears

Management of Non-Dehydrated Child

- If no or infrequent vomiting that is not interfering with fluid intake then 5 – 7 ml/kg/hour of:
 - Breast milk
 - ½ strength formula

- Fruit juice 1 part in 4 with water
- After 6 – 12 hours introduce: bananas, rice, potato, parsnips, pumpkin, dry biscuits/toast with vegemite

Management of Mild-Moderate Dehydration

- Admit or observe in a short stay facility for several hours
- Don't use homemade solutions – use Gastrolyte
- Orally, or by NG tube if necessary:
 - Replace calculated losses over 6 hours (don't worry about maintenance requirements). Hourly observations and reassess and reweigh after 6 hours
 - Give the remainder of the daily fluid maintenance over the next 18 hours
- Resume breast feeding as soon as rehydration is complete or sooner if this takes longer than 6-hours
- If after 4 – 6 hours the child remains dehydrated, then IV

Management of Severe Rehydration

- WEIGH THE CHILD to assess progress
- 3 stages:
 - Initial bolus if necessary. 10 - 20 ml/kg of Ringers Lactate or normal saline over 10 – 15 minutes, reassess and repeat if necessary
 - Replacement + maintenance
 - Maintenance only
- Rehydration of isotonic dehydration:
 - *Replacement*: Normal saline (or Ringer's Lactate or Hartmanns – more physiological)
 - *Maintenance*: 1/5th normal saline + 5% Dextrose + 20 mmol/l KCl [Barts] (gives a bit much Cl but the kidneys can sort that)
 - If initially shocked, do not add KCl until urine is passed. If they have ATN following shock (→ renal failure) don't want to overload K
- Timing:
 - Infuse replacement fluid over 24 hours with the first 24 hours of maintenance using *ongoing replacement*: ½ normal saline + 2.5% dextrose + 10 mmol KCL (in 500 ml)
 - Monitor electrolytes before, and during, up to 6 hourly
 - Once they are able to tolerate oral fluids, treat as for mild/moderate dehydration
- Theme and variations:
 - Diarrhoea:
 - Lost Na, HCO₃, Cl and K from GI mucosal cells – replace slowly
 - Resuscitation with bolus of crystalloids, eg Ringer's lactate, normal saline
 - Maintenance with: ½ normal saline + 2.5% dextrose + 20 mmol/L KCL
 - If persistent acidosis due to HCO₃ loss or lactic acidosis then add in HCO₃
 - Rehydration of **hypernatraemic** dehydration (eg serum Na > 150):
 - Often the result of administering hyper-osmolar fluids (eg sports drinks) with vomiting and diarrhoea → greater water loss due to water sucked into GI from circulation then vomited/passed
 - Will be more dehydrated than they appear due to fluid shifts from ICF → ECF
 - If shocked give 10 ml/kg boluses of normal saline until circulation restored
 - Calculate deficit
 - Calculate ongoing requirements **over 48 hours**
 - Give both **over 48 hours** – serum sodium should not fall faster than 0.5 mmol/hr
 - If oral rehydration, replacement is over 24 hours
 - Diabetic ketoacidosis:
 - If give insulin too fast, serum glucose will drop quickly → rapid change in ECF osmolality → cerebral oedema
 - If giving hypotonic solution then ↑ cerebral oedema – go slow
 - Rehydration of **hyponatraemic** dehydration (serum Na < 130):
 - Resulting from gut or renal losses, or excessive hypotonic fluid administration
 - Appear more dehydrated than they are as fluid shifts into the ICF. Can → cerebral oedema, seizures, etc
 - Never give 1/5th normal saline (except to keep vein open). Do serial Na measurements
 - If asymptomatic: As for rehydration of isotonic dehydration, over 24 hours. Fluid restrict to 50% of maintenance

- If symptomatic (seizures, coma) or if severe ($\text{Na} < 120$) then give 5 – 10 ml/kg or 3% hypertonic saline IV over 60 – 120 minutes in addition to the calculated fluid requirements
- Notes:
 - Be careful about measuring volume: never hang a bag straight into a child
 - If lung or brain disease (eg meningitis), SIADH is common \Rightarrow may need to fluid restrict (eg to 50% maintenance fluids). Check serum Na regularly
 - In a term baby, born water logged (ECF > ICF). Can pass 500 ml urine per day (7 ml/kg/hour). Handles water well but not used to passing a NaCl load
 - Enemas for constipation can \rightarrow dehydration
 - Na to K ratio in urine should be > 1. If < 1 then body frantically reabsorbing Na \Rightarrow not in balance

Monitoring adequacy of Fluid Replacement

- Monitor pulse, BP, respiratory rate and urine output (i.e. put in catheter):

	Per hour
Infants in Nappies	2 mls/kg
Kids & adults	1 mls/kg
Elderly	0.5 - 1 ml/kg

Replacement fluids

- Maintenance fluid: 4% dextrose + 0.18% saline + 20 mmol KCl/L at:

	Per hour	Per day
First 10 kg	4 mls/kg	100 mls/kg
Second 10 kgs	2 mls/kg	50 mls/kg
All subsequent kgs	1 ml/kg	25 mls/kg

- Losses (e.g. nasogastric tube, fever, diarrhoea) replaced with an equal volume of 0.45% NaCl + 20 mmol KCl/L. Give as boluses of 20 ml/kg over 15 – 30 mins. Losses decrease with renal failure

Paediatric Coma

- Assessment: Coma scales – main function is to assess progress
 - AVPU scales
 - Glasgow scale (but designed for adults)
 - Child Coma scale
- General observation:
 - Alert states:
 - Fully alert (what this mean depends on age of child)
 - Confused
 - Delirium: agitated and confused
 - Reduced alertness:
 - Lethargic: fails to maintain wakefulness without stimulation
 - Obtunded: drifts into sleep unless constantly woken
 - Stuporose: unconscious but withdraws to painful stimuli
 - Comatose: fails to respond. May be decorticate or decerebrate. At risk of airway failing
- Differential in children:
 - Hypoxic: respiratory or circulatory failure
 - Epileptic seizures
 - Trauma: intracranial haemorrhage, brain swelling
 - Infections: meningitis, encephalitis
 - Poisons
 - Metabolic: Renal, hepatic failure, Reye's syndrome, hypoglycaemia, diabetes, hypothermia, hypercapnea
 - Vascular lesions: bleeding, AV malformations, arterial or venous thrombosis
 - Hypertension
- Diagnosis:
 - Must be bilateral cortex or brainstem involvement

- Is it focal, multifocal or diffuse
- Is it getting better or worse
- Metabolic disturbances (including hypoxia and seizures) account for 90% of unconscious children
- Supratentorial mass lesions compressing the brain stem: 3rd nerve palsy and dilated pupil on same side – NOT 6th nerve palsy
- Subtentorial lesions affecting the brain stem directly: slow pulse, high BP, irregular breathing
- Management:
 - Stabilise vital functions: ABC then DEFG
 - Complete history: esp trauma, poisoning, previous diseases – diabetes, epilepsy
 - Exam: vital signs and progression, trauma, neck stiffness, CNS function, and:
 - Verbal responsiveness
 - Ocular responses: eye opening, papillary responses and spontaneous eye movement, ocular reflexes (eg Dolls eye)
 - Respiratory patterns: Cheyne Stokes (rate slows down, stops, restarts), irregular, apnoeas, stridor
 - Motor system: Motor responses, reflexes, tone, posture
 - Investigations:
 - Blood: gases, electrolytes, glucose, FBC, LFT, ammonia, calcium, lactate, clotting factors
 - Urine: poisons, sugar, organic acids, ketones
 - Chest Xray, consider skeletal survey
 - ECG
 - CT Scan
 - LP only when safe: risk factors – prolonged fits, focal neuro signs, purpuric rash, CGS < 13, dilated pupils, reduced Dolls Eye, abnormal posture, signs of herniation, coagulation disorder, papilloedema, hypertension

Resuscitation

- Summary:
 - A. and cervical spine
 - B.C.
 - Exsanguinating haemorrhage (if it's not bleeding, ignore it)
- Get help early
- **Airway and cervical spine** immobilisation: Look/listen/feel
 - Airway opening: Jaw thrust
 - Suction of foreign material *under direct vision*
 - Airway devices:
 - Oropharyngeal/nasopharyngeal airways, ET tube, surgical airways.
 - Oropharyngeal: Right size: should reach from midline of lips to angle of the jaw. Use tongue depressor to help insert oropharyngeal (cf adult)
- **Breathing:**
 - Monitor:
 - Work of breathing: rate, noises, recession, accessory use, grunting
 - Effectiveness of breathing: breath sounds, chest expansion, SpO₂
 - If inadequate commence assisted ventilation
 - Indications for intubation:
 - Inadequate O₂ via bag mask
 - Inability to protect airway (eg do they have gag reflex, muscle tone in jaw, etc)
 - Prolonged ventilation required, or control required (eg in transport)
 - Flail chest
 - Inhalational burn injury
 - Intubating
 - If using sedating drugs, must be confident you can completely manage ventilation, do surgical airway if necessary, etc
 - Pre-oxygenate if possible with high flow O₂
 - Need: working, correctly sized laryngoscope, suction, bag valve mask, syringe
 - Take collar off to intubate
 - Tube size = (age/4) + 4 (or size of kids little finger)
 - Must secure tube or it will slide out

- Auscultate the chest to check air entry and check end-tidal CO₂
- Identify and treat life-threatening problems:
 - Tension pneumothorax: ↓sounds on affected side, trachea shifts to good side → needle decompression in 2nd intercostal space, midclavicular line, then chest drain. Little harm if they don't have a pneumothorax.
 - Open pneumothorax: 3 sided sealed dressing then chest drain
 - Massive haemothorax: chest drain and cardiothoracic consult
 - Flail chest: intubate and ventilate. Rare in kids as ribs too spongy – but can get very severe injury without breaking ribs
 - Cardiac tamponade: Urgent cardiothoracic consult
- **Circulation:**
 - Assess: heart rate, pulse volume, central capillary refill < 2 secs (eg over sternum after 5 secs pressure), skin temperature
 - Identify and treat life threatening problems:
 - Shock
 - Stop uncontrolled haemorrhage
 - Stabilise pelvis
 - Initial management of shock:
 - O₂
 - Large IV line placement. If can't then inter-osseous needle. 1 cm medial and distal to tibial tuberosity. Have to squeeze in fluid
 - Crystalloid 20 mls/kg bolus. Reassess and repeat if needed. After that, warmed blood. After transfusion of > ½ blood volume then FFP.
 - If still unstable consider blood and urgent surgical opinion
 - Keep them warm
- **Disability** (ie simplified coma scale):
 - A: Alert
 - V: Responds to voice
 - P: Response to pain
 - U: unresponsive
 - Pupils and posture (decorticate/decerebrate)
- **Exposure:**
 - Uncover to inspect for injuries
 - Keep warm and minimise embarrassment
- **Glucose:** all severely injured children at risk of hypoglycaemia: check during primary survey
- Assessment:
 - Monitors: Pulse/BP/RR/SpO₂/Temperature + EtCO₂ if intubated
 - History taking: parents/ambulance crew/child, past medical history, medications, allergies, last meal
 - Blood tests: baseline FBC and U&Es, cross matching, glucose
 - X-rays: Trauma series – AP chest, AP pelvis, lateral C-spine. NB Soft bones are less likely to break despite strong force ⇒ ↑chance of internal organ damage in absence of breaks than in an adult (eg ribs)
 - Urinary catheterisation/naso-gastric tube placement
 - Analgesia: morphine, 0.1 – 0.2 mg/kg IV (*not* IM)
 - NG tube to empty stomach: kids graze all day so stomach never empty. Also, swallow lots of gas when in pain → tube lets air out → ↓risk of aspiration due to pressure in stomach and less pressure on thorax
- Then secondary survey: head to toe inspection

Paediatric CPR

- Respiratory distress/failure much more common cause of cardiac arrest than cardiac problems. Hypoxia and global ischaemia therefore often precede arrest (in adults it follows arrest), which results in asystole – not VF. Also caused by hypovolaemia, poisoning, drowning, etc
- Ventilation therefore more important than defibrillation. Kids have a higher metabolic rate and O₂ reserves consumed quicker

- Survival associated with duration of arrest (after 5 minutes it plummets), not more than one dose of adrenaline, and presence of VF
- Procedure:
 - Ensure your and patient's safety
 - Assess responsiveness. Don't shake a baby. If unresponsive, shout for help
 - Open airway: head tilt (not too much extension) and chin lift. Jaw thrust instead if cervical trauma. Check for obstruction
 - Assess breathing. If chest moves but no breath, recheck airway
 - Ventilate: 5 attempted breaths 1 – 1.5 seconds. In babies and infants, give through nose or nose and mouth. Slow breaths at low pressure better than fast/high pressure (↓gastric distension). Ventilate just sufficiently to make chest rise and fall
 - For no more than 10 secs, check circulation. Infants: brachial, femoral, axillary arteries or apex beat. If over 8, carotid best
 - If no circulation or less than 60 bpm, external chest compression. Over junction of middle and lower 3rd of sternum.
 - In neonates, use two fingers to depth of 1 – 1.5 cm. Rate of 100 bpm, ratio of compressions to ventilations is 5:1
 - Kids over 5, heel of one hand, depth approx. 2 – 3 cm
 - Larger kids, two handed compression, depth of 3 – 4 cm, rate of 80 – 100 bpm, and ratio of 15: 2
 - After one minute alert emergency services
 - Resume CPR: reassess circulation after 3 minutes. Give adrenaline. If iv access time consuming, then 18 gauge perpendicular into anterior surface of tibia, 1 – 3 cm below tibial tuberosity. Failing this, give 10 times iv dose down endotracheal tube. Repeat cycle and adrenaline
 - When defibrillator arrives, assess rhythm. Use paediatric paddles if < 10 kg. ONLY if rhythm is VF or VT deliver 3 shocks at 2, 2, then 4 joules per Kg. Perform CPR for one minute, reassess rhythm. Every 2nd loop give adrenaline
- Ventilation: Harder in kids – use two people to do bag-mask. Beware of barotrauma
- If hypovolaemia → 20 ml/kg saline or Ringers

Other Emergency Situations

Severe Anaphylaxis

- = Severe allergic reaction
- Problems:
 - Acute CV collapse: hypotension, myocardial ischaemia, arrhythmias
 - Lower airway: Bronchospasm → respiratory difficulty. Respiratory problems account for 70% of fatalities. Asthmatics at higher risk.
 - Upper airway: Laryngeal oedema (ie angioedema)
 - Also skin problems (urticaria, erythema, itch), nausea, vomiting, diarrhoea, anxiety, etc
- Pathogenesis:
 - Type 1 allergic reactions mediated by IgE antibodies
 - Previously sensitised → IgE antibodies against allergen → mast cell activation → massive mediator release (histamine, leukotrienes, prostaglandins, kinins)
 - Histamine leads to:
 - Smooth muscle contraction → bronchospasm
 - Vasodilation & ↑permeability (can lose 1½ L of blood volume straight away)
 - ↑HR and arrhythmias
 - ↑Noradrenaline
 - Itch & oedema
- Anaphylactoid reaction: activation of mast cells and release of mediators without IgE involvement. Only relevant to investigating cause – not to treatment
- Examples of allergens:
 - Drugs: 50% of fatalities. Includes penicillin, muscle relaxants (can be sensitised by exposure to similar drugs), aspirin, contrast media, blood products, streptokinase, preservatives (e.g. in adrenaline)
 - Foods: 25% of fatalities. Peanuts, milk, eggs, fish
 - Insect bites: 25% of fatalities

- Also latex, semen, blood products, physical stimuli (eg exercise, cold, heat)

Presentation

- Anaesthetics: If IV – then as fast as 1 minute, but normally 5 – 10 minutes. Food up to 30 minutes
- 1 in 2,500 surgical patients in Wellington. Death rate 4 – 6 %

Treatment

- Stop administration of antigen. Call 777
- Adrenaline:
 - If no current venous access then 0.5 ml 1:1000 IM. 0.01 mg/kg for kids
 - If venous access for adult then: 0.3 – 0.5 mls iv of 1:1,000 slowly, repeat until BP > 100. Start low (eg 10 µg) and titrate up
 - Can be nebulised for laryngeal oedema
 - If on TCAs then ↑sensitivity to adrenaline
 - α agonist → vasoconstriction – but not too much otherwise cardiac vasoconstriction
 - β agonist → bronchodilator
 - ↑Force of heart contraction
 - ↓Mediator release
 - T_{1/2} is short: common error is to give too little too infrequently
- Also:
 - Metaraminol (α agonist) to stop arrhythmias
 - ?Steroids: prevent late symptoms
 - Promethazine 25 mg slow iv or im (H1 antagonist) + H2 antagonist (e.g. ranitidine), or
 - Antihistamines: Phenergan 25 mg iv slowly for itch
- If bronchospasm alone:
 - Salbutamol: 5 – 20 µg/min
 - Hydrocortisone 200 mg iv
 - Aminophylline 5 mg/kg over 30 minutes
- Elevate legs → ↑venous return
- O₂ by mask: intubate if necessary
- 20 ml/kg bolus colloid rapidly
- If anaesthetic reaction, always investigate so next anaesthetic is safe. Should have skin tests, etc. Cross reactivity between muscle relaxants is not uncommon

Differential Diagnosis

- Measure serum **tryptase** (longer T_{1/2} than histamine) to confirm anaphylaxis
- Anaesthetic overdose: Tryptase raised in anaphylaxis, normal in overdose
- Respiratory: Pulmonary oedema/embolism, asthma, foreign body
- Heart: Pericardial tamponade, MI, arrhythmia, vasovagal faint
- Venous air embolism
- Septic shock
- Pneumothorax
- Transfusion reaction
- Hypoglycaemia, CVA, epilepsy

Prevention

- Avoid treatment with β-blockers – makes treatment of anaphylaxis difficult
- Carry and use adrenaline (eg Epi-pen)
- Medic alert bracelet
- Call an ambulance, don't 'wait and see'

Traumatic Injury

- Most common cause of death < 14 years (way out in front)
- Under 1 year: cause of death – congenital abnormalities > infection > trauma
- Trauma: poisoning > suffocation > MVA
- What makes kids different:
 - Large, poorly supported head. Always land head first
 - Thin skin → ↑evaporative skin losses and burn at a lower temperature
 - ↑Surface area: mass ration → ↑rate of heat loss

- Relatively large, poorly attached spleen
- Renal function, conserves water, secretes sodium
- Greenstick fractures
- Child abuse: differential diagnosis in all cases of trauma (do history and physical findings correlate)
- Dealing with children:
 - Never lie – say if it's going to hurt
 - Kid that is injured will almost always have been injured doing something they were told not to do – child will consider you part of the punishment
 - Parents will get mad at you because they feel guilty. Wear it – this is not the time to deal with it
 - Child will regress
- Frequency of visceral injury: spleen > liver > kidney
- Splenectomy. The younger the child the greater the risk of fatal post-splenectomy sepsis (adults have greater previous antigenic exposure so less susceptible). Leave it in if vital signs stable
- Kidney trauma: most common injury is contusion → mild haematuria
- Bladder: easily ruptured
- Closed head injury:
 - Full neuro exam
 - Level of consciousness: **A**wake, responds to **V**oice, to **P**ain, or is **U**nresponsive
 - Localising signs: can be very subtle, watch for changes
 - Pupils
 - Can rupture middle-meningeal artery without fracturing skull
 - Head injury almost never causes shock
- Pain management: early – consider regional blocks (eg femoral nerve block in fractured femur)

Car Crash

- Without seat belt, risk of death is ↑10 times. All children being held in the front seat die
- Assessment of severity:
 - Speed of crash
 - Was seatbelt on
 - Was child thrown from car
 - Was any other child killed

Burns

- > 50 % of burn admissions are children
- Full thickness burns don't hurt (nerves are dead)
- Partial thickness burns blister and heal
- Rule of 9's doesn't work – needs age adjustment
- Fluid resuscitation: Ringer's 4ml/kg/% of burn (half in 1st 8 hours) + maintenance (ie pour it in till they urinate)

Airway Obstruction

- Suspect in any airway distress with coughing, gagging, or stridor with rapid onset. May also be caused by infections (e.g. croup or epiglottitis). If infective cause then medical emergency
- Only intervene if child's attempts to clear the obstruction are clearly ineffective and there is inadequate respiration
- For infants (<1 year) and children, 5 back blows with the child's head below the level of the chest if possible
- Then 5 chest thrusts to sternum in supine position: sharp, vigorous and rate of 20 bpm
- Check mouth: grasp tongue and jaw and lift. Don't put finger into mouth unless foreign body is clearly visible
- Reassess airway. If not breathing, attempt to ventilate
- Repeat back slaps, chest thrusts, attempted ventilation. In children, alternate abdominal and chest thrusts

Asthma

- Arrest due to: bronchospasm (→ asphyxia), tension pneumothorax (often bilateral), β agonists → arrhythmias
- Arrest Prevention:

- Maximal O₂
- Nebulised salbutamol (beware overdose → tachycardia and VF/VT) or iv 5 µg/min up to 20 µg if necessary
- IV hydrocortisone
- Adrenaline
- IV sodium bicarbonate (acidosis prevents action of sympathonimetics)
- Intubation and IPPV: sedate with ketamine or benzodiazepines, paralyse with suxamethonium
- During arrest:
 - Consider assisted exhalation (bilateral manual squeeze over lower chest at end of inspiration)
 - Plus normal routine

Near Drowning

- Effective immediate resuscitation critical. Use standard CPR procedure
- Remove foreign bodies from airways, don't attempt to drain fluid
- Suspect spinal injury if diving or in surf
- Early tracheal intubation may be indicated. 100% O₂
- Recovery may occur even after long immersion times, especially in cold water
- In hospital, cerebral oedema may require hyperventilation and diuretics
- Remember, alcohol or epilepsy may be involved
- Avoid steroids, consider antibiotics

Hypothermia

- Signs: hypotension, bradycardia, J wave on ECG, SV arrhythmias, VF at 28 C, metabolic acidosis, loss of consciousness at 28 – 30 C, shivering replaced by rigidity at 33 C, pupils dilated
- Lengthens tolerance of arrest: don't discontinue till they've been warmed
- Arrest prevention:
 - Prevent further heat loss
 - Transport avoiding rough movement, which can precipitate VF
 - If core temperature < 34 C, can rewarm with oesophageal rewarming tubes, peritoneal lavage (warmed saline or gas). Warm trunk not peripheries. Reduce movement (risk of VF). Rewarm slowly – 0.5 degrees/hour (unless fit and sudden hypothermia)
- During arrest:
 - Take 30 – 45 secs to confirm cessation of ventilation and pulselessness
 - Don't assume death until resuscitation has failed in an adequately rewarmed patient
 - If < 30 C give maximum of 3 shocks until core temperature increases
 - Reduced responsiveness to defibrillation and drugs. Impaired drug metabolism → watch for toxicity
 - Monitor fluids during rewarming

Hyperthermia

- Heat Exhaustion: hypovolaemic shock due to fluid loss through sweating. Cool, restore volume, position supine with legs raised
- Heat stroke: failure of heat regulation through failure to sweat. Hot, flushed and dry. If temperature > 40 degrees → neurological disturbances. Rapid cooling, cool iv fluids
- Reduces tolerance time for arrest
- Cooling can be external or internal
- Watch electrolytes and fluid replacement following arrest
- Watch for tendency to cerebral oedema and multi-organ failure

Poisoning and Overdose

- Cause of significant proportion of arrests in 18 – 35 year olds
- Duration of arrest and dose of toxin determinants of survival
- Do general management
- Manifestations of poisoning:
 - Exaggerated therapeutic response (eg sedation with BZD)
 - Pharmacological effects (eg respiratory depression, convulsions)
 - Accidental: children, single poison
 - Intentional: adult, often multiple, taken in conjunction with alcohol
- Management:

- History:
 - Find out the drug if you can (has someone brought in the packet?): but only a few will change management (paracetamol, salicylates, lithium, paraquat, quinine, phenobarbitone, iron salts)
 - Very unreliable – especially the number of tablets taken
- Supportive treatment:
 - A,B,C: Respiratory depression is the most common cause of death.
 - Maintain airway, check blood gases. Check for hypotension (→raise legs except in heart failure, volume expanders). Monitor electrolytes
 - Maintain safety: close supervision, no access to drugs on ward, etc
- **Intensive support treatment:** IV fluids, ?NG tube, maintain vital functions, nursing care (eg suction, pressure sores, limb movement → ↓thrombosis)
- **Treat complications:** hypothermia, hyperthermia (salicylates and stimulants – sponge down, use fan), seizures (iv diazepam), arrhythmias (leave bradycardia, tachycardia – correct acidosis, try amiodarone), hypoglycaemia (salicylates, oral hypoglycaemics)
- Plus treatment specific to poison
- Mental health assessment
- Investigations:
 - Blood levels: sometimes useful (eg Li, aspirin, theophylline, carbamazepine). Waste of time for TCAs
 - Urine Screen: Rarely changes management, no quantitative information, really only for criminal cases (eg after MVA)
- Corrosives:
 - *Never* induce vomiting
 - Drink copious fluids
 - Soak eyes, skin, mucous membranes
 - Petroleum: beware of inhalation, cup of milk
- Eliminating poisons. If sure it's not petroleum products, caustics, corrosives or acids, then options may include:
 - Activated charcoal
 - Better than emesis
 - Charcoal powder, mixed with H₂O: needs to be within 60 minutes. Give SINGLE 50 g dose in an adult (1 g/kg)
 - Reduces GI absorption of paracetamol, aspirin, phenytoin, digoxin, TCAs, theophylline, carbamazepine.
 - Don't use for volatile hydrocarbons or corrosives or an unprotected airway
 - Good for unionised drugs. Does not bind with acids, alkalis, alcohols, lithium.
 - Generally safe but constipation, aspiration may be problems (protect airway)
 - Multiple doses only for drugs undergoing enterohepatic circulation or diffusing into the gut ⇒ drugs with a small V_d, low clearance, low protein binding and long T_{1/2}. Eg Theophylline, carbamazepine, quinine, phenobarbitone. NOT Paracetamol.
 - PH adjusted diuresis:
 - Alkaline diuresis: aspirin, phenobarbitone
 - Acid diuresis: amphetamine, methadone. Doubtful use and dangerous
 - Dialysis: Haemodialysis. Only useful if low V_d, small molecule and low protein binding (eg lithium, theophylline, salicylates, alcohol and barbiturates)
 - Whole bowel irrigation: 'Go Lightly' – Xray prep
 - Questionable effectiveness:
 - Emesis: Not effective? Ipecac – never if airway reflexes not intact. Causes emesis in 90% within 15 – 30 minutes
 - Gastric lavage: large bore catheter through mouth. 1ml/kg of body temperature water, recover, repeat. Little evidence of benefit and ↑ risk of aspiration. Contraindicated if acid, alkali, or petroleum

Common poisons

- Paracetamol
 - Walk in, conscious
 - RUQ pain
 - Conjugation pathway easily saturated. Of the remainder, 15% is metabolised to a metabolite that combines with glutathione. If glutathione is depleted, metabolite causes hepatic damage

- Toxic dose 140 mg/kg (textbook), 200 mg/kg (Starship), lower if chronic alcoholism, enzyme inducing drugs or fasting
- Measure plasma concentration. Treat if > 200 µg/ml, 100 if liver disease, anorexia, etc. Threshold declines for each hour after ingestion
- Absorbed quickly so gastric lavage or activated charcoal only effective within 45 minutes
- N-acetylcysteine (NAC) best antidote – saturates alternative pathway so all the paracetamol is metabolised through the main pathway. Normal dose is 10 - 15 mg/kg per 4 hours acutely, per 6 hours at home
- Monitor AST/ALT, PT (INR)
- Opioids:
 - Marked sedation, pinpoint pupils, ↓↓ respiration (differential: stroke)
 - Naloxone (but short T_{1/2} → will lapse back)
- Stimulants (cocaine type drugs): ↑BP, tachycardia, arrhythmia, dilated pupils [sympathetic effects], seizures
- Barbiturates → flumazenil (can cause seizures – so not if SSRIs/TcAs/antihistamines as well – which also cause seizures)
- Dibenzazepine antidepressants: Rapidly absorbed, high Vd, protein bound. Look for myocardial toxicity, hypotension, hyperreflexia, convulsions. Treatment supportive + naso-gastric tube and charcoal (up to 24 hours later). Monitor ECG, iv propranolol and NaHCO₃
- Benzodiazepines: Diazepam, temazepam → Post-OD “cerebellar syndrome” (dizziness, confusion, ataxia, nystagmus, bullous lesions). Supportive treatment
- Anticonvulsants: carbamazepine
- Bronchodilators: theophylline
- Cocaine → benzodiazepines
- Carbon Monoxide → 100% oxygen, treat cerebral oedema. Presentation: pink, headache, vomiting, tachycardia, seizures, arrest
- Cyanide → 100% O₂ + cobalt edetate
- Methanol and ethylene glycol poisoning → correct acidosis, ethanol
- Chelating agents for arsenic, copper, lead, iron, cyanide
- Aspirin: risk is pH balance

Paediatric Anaesthetics

Pre-operative assessment of child with a URTI

- Peri-operative risk variably increased
- Postpone high risk:
 - Neonates and infants
 - Existing upper airway/respiratory pathology (eg CF) - ↓ reserve – easy to tip over the edge
 - Systemic symptoms
 - Lower respiratory tract involvement
 - Surgical impact on respiratory function (eg upper abdo surgery)
- Complications usually manageable

Pre-operative assessment of child with a murmur

- Innocent murmurs often detected by anaesthetists
- Murmurs in up to 95%, but pathology in only 0.5%. May need referral for investigation
- 3 Common innocent murmurs:
 - Early systolic from ventricular outflow tracts (either pulmonary or aortic)
 - Continuous murmur from SVC
 - Grade 1 – 2
- Bad murmurs mimicking benign ones:
 - Severe hypertrophic obstructive cardiomyopathy
 - Critical aortic stenosis
 - These develop after birth – so may not have been picked up in post natal checks
- Postpone and refer if suspicious, esp if < 1 year
- ECG recommended if echo unavailable (can fax to a paediatric cardiologist for interpretation)
- SBE prophylaxis may be indicated

Risk Factors for Aspiration

- High risk for aspiration: Treat as full stomach
 - Full stomach
 - Regurgitation
 - Impaired protective reflexes
 - Airway obstruction (big negative pressure in thorax in order to suck air in past obstruction – but this also sucks contents out of stomach)
- Hazards of fasting:
 - Discomfort
 - Hypovolaemia. Guidelines are:
 - Clear fluids till 2 hours before
 - Breast milk till 4 hours before
 - Food till 6 hours before (no chewing gum)
 - Hypoglycaemia: only an issue for neonates

Assessment for Sedation

- Need to risk assess any child before any sort of sedation – its all too easy for something to go wrong (or more usually, for lots of little things to mount up)
- Always need to be confident you could ventilate, intubate and get IV access quickly if necessary

Pain Management in Children

- Myths:
 - Neonates don't experience pain
 - Neonates have no memory of pain (they retract from a needle the 2nd time)
 - Pain is not harmful (it leads to stress response → ↓healing, etc. ? Impact on the development of pain pathways)
 - It is dangerous to treat pain
- Management principles:
 - Mild to moderate pain relief is achieved through oral or rectal doses
 - Children hate needles, especially repeated IM injections
 - Using loading doses and regular maintenance doses to achieve therapeutic effect
 - Don't overdose with paracetamol (may → hepatotoxicity). Limit duration
 - Child-friendly environment and parental involvement important
- Available drugs:
 - Paracetamol (oral better than rectal). Only use aspirin where specially indicated (eg Rheumatic fever)
 - NSAIDs: Diclofenac, Ibuprofen, Naproxen
 - Codeine Phosphate (metabolised to morphine): constipation, plus dose related opioid side-effects – sedation, respiratory depression, nausea and vomiting
 - Morphine for serious pain (eg burns and fractures)
 - Pethidine less used in kids - ↑toxicity (including seizures)
 - Tramadol – not often used but less respiratory depression
 - Nitrous Oxide (always administered with O₂). OK for brief analgesia (eg fracture immobilisation). Ensure resuscitation equipment available. Mouth pieces preferred to masks

Consent in Children

- Issue is not whether to get consent – but how
- Inconsistency about when they are autonomous:
 - Guardianship Act: 16
 - Common Law (Gillick case 1985) and H&D code: Capacity to make decision
- Exceptions to age limits and parental consent
 - Emergencies
 - Blood transfusion when life saving (if under 20 years)
 - Compulsory treatment (eg Mental health Act, Tb)
 - Blood alcohol
 - Abortion and contraception (CSA Act 1977): at any age, and no requirement to inform parents
 - Child Abuse examination (CYFS Act 1989)

- When Guardianship invested in the Court or DG of Social Welfare
- Good practice to involve the parents wherever possible
- Consent and the UN Convention on the Rights of the Child
 - The best interests of the child are paramount (article 3)
 - Have the right to express their views and have them taken into account (article 12)
 - Privacy and Confidentiality (article 16)
 - Accessibility of information (article 17)
- Conflict over consent:
 - Maori issues: greater expression of autonomy collectively, and collective responsibility for Tamariki. Involve whanau
 - If a child says no – it's usually because they are frightened. Take a child's views seriously. Reduce fear by ensuring understanding. But best interests may be in conflict with their wishes
 - If parents say no, consider reasonable alternatives and legal (last option). CYPS Act, sections 14 & 67 – child in need of protection. Guardianship Act 1968 may place child under guardianship of the court
 - Allow time to work it through, plan ahead
 - Avoid rushing important decisions
 - Give information, check it is understood, opportunity to ask questions
 - Enlist supports, Maori/PI staff, translator, etc

Central elements in maltreatment

- Parent's strong negative and irrational engagement with the child, featuring a distorted perception of the child
- Parent's lack of ability to engage positively with the child
- Child is continually left in a state of worry or anxiety

General History

- History of injuries – how, who, when, where. Note details of different caregivers, change over times, etc. Clarify custody arrangements well
- Developmental history
- PMH, especially previous injuries (do you need notes from hospital, other GPs etc)
- Social history: supports, domestic violence, other stresses, previous CYFS referral
- What are parent's expectations of toddler behaviour, etc

Physical Abuse

- Non-accidental injury to a child or young person
- Includes: bruises, cuts, fractures, head injuries, injuries to internal organs, suffocation, poisoning, burns
- Risk factors:
 - Hard to parent child: eg handicapped or behaviourally difficult
 - Poor parenting skills/experience
 - Unrealistic expectations of the child
 - Poor mental health of the parents
 - Reduced social support
 - Alcohol or substance abuse
 - Domestic violence
 - History of child abuse in the abuser
 - Triggering event precipitating loss of control by the perpetrator
- Be suspicious when:
 - No history is given for the injury
 - The history changes
 - History is partial
 - Unbelievable explanation
 - Unreasonable delay in seeking help
 - Previous similar episodes
 - Parents affect or behaviour is abnormal
- Questions to include in history taking:
 - When, where and how did the injury occur
 - What was the child doing at the time
 - Who saw it
 - What is the child's developmental level
 - Is a scene examination necessary
- Patterns of injury suggesting non-accidental injury:
 - Fractures: multiple sites or different ages, rib fractures, any fracture in a child < 2: consult radiologist. Look for missing teeth
 - Head injuries: any child < 1, unexplained coma, retinal haemorrhages (from shaking). Usually closed head injury rather than a fracture
 - Bruises: on face or back, non-mobile baby, fingertip pattern bruises, other pattern bruises (strap, belt), yellowing ⇒ older than 18 hours. If suspicious, referral immediately to a paediatrician (who can arrange for evidential photos to be taken). Tell mum you need to refer so they can be checked for other injuries
 - Burns: Child will withdraw hand or foot before a burn is full thickness, pattern burns (eg held in hot bath, cigarette burns), burns on back
- Examination:
 - Normal general assessment: growth, consciousness, play and behaviour, language

- Carefully full survey: looking for bruises, tenderness, acute abdomen (eg splenic rupture), genital bleeding (leave full genital exam for an expert)
- Developmental assessment
- Systems Review – any other possible cause for the injuries
- Document everything carefully, use a body chart and measure lesions, ask for explanation of each injury
- Investigations:
 - FBC and coagulation
 - Referral for specific investigations: X-ray, ophthalmologist, ENT surgeon, CT
 - Consider urine toxicology
- Differential to physical abuse:
 - Bruising: Mongolian spots, coagulopathies, coin rubbing
 - Cigarette burns: bites or vesicles
 - Hot fluid burns may be non-intentional
 - Fractures: osteogenesis imperfecta, spiral fractures of the tibia in toddlers

Sexual Abuse

- Any act resulting in sexual exploitation of a child – whether consensual or not, including:
 - Non-contact abuse: exhibitionism, suggestive behaviours, exposure to pornography
 - Contact abuse: fondling, masturbation, oral sex, object or penis penetration
- Risk factors:
 - Family dysfunction
 - Female sex
 - Pre-adolescence
 - Previous victimisation: don't think they're worth it – won't say no
 - Non-biological parent
 - Developmental delay: don't understand, scarred to say no
- Alleged perpetrators are all ages. If < 10 years, are they acting out abuse to them. 60% are family members
- History taking:
 - Evidential interview is the job of the police and CYPFS – usually videoed
 - If child discloses to a doctor, record questions and answers carefully. Don't ask leading questions. Qualify notes with "the above history was taken in order to direct the exam and does not necessarily constitute a full or detailed history". If not acute, leave questions for police
- Presentation:
 - Behavioural indicators: non-specific so don't over interpret. They're the same for anything that's upset them, eg parents separating: sleep disturbance, change in appetite, regression, running away, fear (specific or generalise), anger, ↓concentration, sexualised behaviour
 - Adolescence: self-harm, suicidal ideation, alcohol/drug abuse, eating disorders, unprotected consensual sex, promiscuity, school failure, loss of peer group
 - Vaginal discharge in a pre-pubertal child is common:
 - Non-specific eg irritant/allergic
 - Infection: Gp A strep, shigella, Candida (uncommon once out of nappies)
 - Foreign bodies
 - Polyps
 - Systemic illness eg measles, chickenpox
 - Vulvar skin disease
 - Vaginal bleeding: accidental straddle injury, vaginitis, foreign body, precocious puberty
- Normal sexual development:
 - 0 – 2: genital exploration, masturbation (boys > girls), learning names
 - 3: talk about sexual differences, genital interest increases, masturbation common
 - 4: Play doctors and nurses, mothers and fathers, games involving undressing, exhibitionist activities, demand privacy for themselves, interested in others bodies
 - 5 – 6 years: familiar with and has less interest in sexual differences, likely to be more modest
 - Sexualised behaviour:
 - Masturbation is normal, but inappropriate if older and still public
 - Sexual play: if > 5 shouldn't be touching other genitals
- Physical findings in abuse:

- > 50 % of disclosures will have no physical findings
- Urgent forensic exam only if incident < 72 hours ago
- Perpetrator usually doesn't want to hurt the victim, otherwise won't have continued access ⇒ physical injuries less common
- Investigations:
 - Pre-pubertal: don't screen for STD's unless symptomatic. HIV testing in time if high risk
 - Adolescents: screen for STDs and ?pregnancy test
- Prognosis: 25% have no adverse psychological sequelae. The more invasive the abuse, the more severe the effects long term

Neglect

- = Act or omission that results in impaired physical functioning or development, or injury. Includes physical neglect, neglectful supervision, medical neglect, abandonment, refusal to assume parental responsibility
- Risk factors:
 - Poor attachment
 - Parental psychiatric illness
 - Maternal depression
 - Isolated unsupported parent
 - Poverty
- Presentation:
 - Often associated with physical and emotional abuse
 - In an infant: failure to thrive, frequent attendance at A&E, severe nappy rash, unexplained bruising, cold injury, developmental delay, attachment disorder
 - Pre-schoolers: short stature, unkempt and dirty, delayed language, very disorganised play (eg aggressive and impulsive, indiscriminate friendliness)
 - School children: short stature, poor hygiene (including teeth), unkempt, learning difficulties, ↓self esteem, disordered/few relationships, unusually patterns of defecation or urination

Emotional Abuse

- = Act or omission that impairs the psychological, social, intellectual or emotional development of a child or young person. Includes: Rejection, isolation, oppression, deprivation of affection, inappropriate criticism, threats or humiliation, exposure to violence, involvement in illegal or anti-social activities, negative impact of substance abuse or mental/emotional condition of parent or caregiver
- Risk factors:
 - Poor attachment
 - Parental psychiatric illness
 - Maternal depression
 - Isolated unsupported parent
 - Parental alcohol and/or drug addiction
 - Domestic violence
- Presentation:
 - Socio-emotional indicators: can't enjoy themselves, refuses to defend self, cheats, steals, bizarre or extreme behaviours, failure to accept responsibility for behaviour, low self-esteem, withdrawal, defiance, compulsivity, seeks love and acceptance outside the home, apathy
 - Cognitive indicators: learning problems, short attention span, hypervigilance, hyperactivity, developmental delay, lack of curiosity
 - Physical indicators: Failure to thrive, accident prone, self destructive behaviour, eating disorders, GI and bowel problems, poor posture, sleep disorders, ↓energy
- Differential: Munchausen's by proxy

Management of Abuse

- Paramount principle: The interests, safety and well being of the child should be the paramount concern (Section 6, Children, Young Persons and Their Families Act)
- Doctor's role is medical management, not the assessment of child abuse
- If child abuse is suspected:
 - Trust your instincts
 - Look for signs of abuse

- Document the facts
- Recognise and treat medical sequelae
- Prevent pregnancy
- Provide ongoing support, and watch for and help behavioural sequelae
- Contact CYPFS immediately and discuss your concerns. You cannot be guaranteed anonymity, but when reporting to CYPFS or the police you are protected from court action if acting in good faith
- Mother/other person can also contact CYPS [good approach if you consider this is really a custody issue. Alternatively advise the mother to get a lawyer]
- There is no legal requirement to contact CYPFS or to give a CYPFS social worker information if they contact you. There is likely to be an ethical obligation, and referral guidelines will exist and should be followed.
- Investigation and management is multi-disciplinary: should involve paediatrician, social worker, police, psychologist
- If a child discloses abuse:
 - Listen to the child but do **not** interview them
 - Well being of the child comes before the interests of any other person
 - Write down what the child says
 - Reassure them they've done the right thing
 - Tell them that they will get help – but don't make promises. Say it's got to stop but that you'll need to tell someone else who will help
 - Tell your manager/supervisor as soon as possible
 - Look after yourself: discuss the matter with someone you trust
 - If nothing seems to be happening, contact CYPFS again
 - Complete an ACC M45 form and forward to the Sensitive Claims Unit at ACC
- Care for mother: may be domestic violence, depression, addiction, etc
- Care for perpetrator: talk with intake social worker about help for them (eg violence prevention programmes)

Attention Deficit/Hyperactivity Disorder (ADHD)

- Background:
 - Estimates range from 2 – 5%
 - Boys > girls
 - 60% take some symptoms into adulthood (eg restless, disorganised, poor attention, impulse control)
 - Was first described 100 years ago – only recently received appropriate recognition
 - Could be better described as ‘behaviour inhibition disorder’
 - Is strongly genetic and is biological
- Diagnosis:
 - Behaviour:
 - Inattention: easily distracted, doesn’t finish tasks, works best with supervision, poor short-term memory. “How does he get on with daily tasks like dressing/eating breakfast/doing homework” “Do you ever have to stand over him to make sure he finishes”
 - Impulsiveness: acts without thinking, short fuse, aggressive, little self-control. “How often does he get into trouble for not thinking before he does something”
 - Overactivity: restless, fidgets. “How easy is it for him to sit still”
 - Insatiability: rarely satisfied, interrogates, over-intrudes in others space
 - Also poor co-ordination, disorganisation, fluctuation, and specific learning disabilities
 - Older child: low self esteem, mood swings, aggression, underachievement
 - Inappropriate for age and development
 - Pervasive across at least 2 settings
 - Onset < 7 years
 - Impairs social and academic functioning
 - Hard to diagnose pre-school – tantrums and ↓attention common. Issue is whether they mature on transition to school. Gap widens as they get older. A 6 year old should be able to complete tasks, concentrate, etc
 - Usually normal to high IQ
 - Diagnostic boundary is disputed – this falls on a continuum (like everything else!)
- Differential:
 - Learning disability → not coping at school, frustrated → acting out
 - Gifted child whose bored
 - Psychosocial stress: disruption at home, abuse
 - Anxiety
 - Psychiatric disorders (mood, anxiety or personality)
 - Problems with parenting – no boundaries or inconsistent boundaries
- Associated factors:
 - Lower socio-economic status: poverty, poor housing, unemployment, illness, family breakdown
 - Childhood depression/anxiety → ↓concentration
 - Auditory/visual perceptual difficulties → inattention, loose interest
 - Reading problems: visual sequencing, letter-word orientation → appears inattentive
- Assessment:
 - Onset of behaviours
 - Situation specific or pervasive
 - Other learning difficulties
 - Context: parents management style, life events, teacher, etc
 - Use parent questionnaire
 - What are child’s strengths – basis of self esteem
 - Get information from school: general behaviour, problems in specific situations (transitions between lessons, unstructured time eg playground, changes to routines eg outings, academic problems)
 - Thorough developmental history (ABFWIMPS), especially:
 - Head injury
 - Perinatal problems

- Attachment problems in first 2 years (eg PND, stresses, violence, drugs)
- Exam: dysmorphic features, tics (more common in ADHD and also side effect of ADHD medication), observation during interview
- Classifications:
 - Primary: early onset, feeding/sleeping problems from early on, overactive/unmanageable toddler, parents exhausted
 - Secondary:
 - Psychosocial causes: family disruption, demands of school, etc
 - Specific learning disability (→↑stress once school starts)
 - Mixed: an adolescent presenting with all of the above, plus ↓self-esteem
- Management:
 - Multidisciplinary assessment
 - Behaviour strategies:
 - Clear, firm, consistent guidelines
 - Check understanding of instructions
 - Anticipate problems and have planned responses ready → ↓parental stress and ↑consistency
 - Avoid triggers (eg crowds)
 - Predictable routines (eg at bedtime)
 - Managed use of time out, withdrawal of privileges
 - Encouragement: see Behaviour Management, page 14
 - At school:
 - Structured approach – plan day
 - Sit near teacher, between quieter kids
 - Brief, clear instructions
 - Supervision during transition times (coming in from breaks, etc)
 - At home:
 - Force leads to confrontation, resentment, broken relationships
 - Behavioural techniques work poorly – it’s a biological problem
 - Ignore all but the important misbehaviours. Have a few clear rules, with clear consequences, if broken act without argument. Don’t debate or escalate
 - Esteem: Encourage. Find something they are good at. Swimming, bike riding, cooking, judo and computers may be better than team sports. Encourage friendships – take a friend on outings
 - Diet: < 10% sensitive to synthetic food colouring
 - Many dodgy therapies: avoid unless proven
 - Stimulant medication:
 - → Concentrate for longer (stimulates inhibition) → complete tasks → less disruptive and ↑ self-esteem
 - First: education for child and parents.
 - “Have you heard about medication – what?”
 - Address myths: they’re addictive, they sedate the child, child at ↑risk of substance abuse later in life
 - Side effects: sleep disturbance, appetite suppression (small effect, if marked → growth suppression), moodiness, rebound, tics
 - First line options are Methylphenidate (Ritalin) or Dexamphetamine. Both require specialist endorsement. Introduce slowly. Short T_{1/2} ⇒ need to fine tune dose times. Eg give before school → Ok at school but difficult by the time they get home. Not in evening otherwise ↓sleep. Review – should have noticeable improvement, if not re-evaluate
 - Referral if:
 - Diagnosis/differential in doubt
 - Assistance with management of challenging behaviour
 - Assessment of role of family relationships in perpetuating the problems

Anxiety Disorders

- Fears are normal during childhood and adolescence:
 - Age 1 – 2: fear of separation from parents
 - Young child: scared of the dark, animals, storms, monsters
 - Age 7 – 8: begin to worry about their performance

- Adolescents: concern about being disliked, rejected, or criticised by their peers
- ⇒ Fears generally reflect developmental stage
- Anxiety disorder:
 - Fears become intense or pervasive and substantially impair functioning
 - Can follow chronic, fluctuating course
 - Not easy to recognise as young people often know that their fears are groundless and feel ashamed of what they think is a flaw in their character
- Anxiety disorders: Separation anxiety disorder, social phobia, generalised anxiety disorder, obsessive compulsive disorder, post-traumatic stress disorder

Separation Anxiety Disorder

- Child very anxious away from home or from their parents
- May present with:
 - Refusal to attend school – but school's not the problem, the separation is
 - Feeling physically ill in the morning. Monday's the worst day
 - Reluctance to sleep at friends places, school camps, etc
 - Worried that harm will befall their parents while they're away
 - Difficulty coping with parents going out
 - Difficulty going off to sleep, or needing company of a parent while they do
- History should include:
 - School: problems, bullying, fears, etc
 - Home: stressors, conflicts
 - Maternal depression, anxiety, adjustment disorder, etc
 - Parents may have some insight – but usually underestimate the severity of the maternal-child dependence and are very defensive
- Diagnosis: irrational fear of harm to parents or that they will be abandoned by them
- Differential for school non-attendance:
 - Truancy, conduct disorder: doesn't go to school – but doesn't stay at home either
 - Anxiety-based refusal
 - Major depression: lacks motivation
 - Other reasons: at home to help with work, etc
- Epidemiology: F > M. Peaks in early adolescence
- Course:
 - May be triggered by a worrying or traumatic incident. May be family history of anxiety problems
 - Eventually become isolated from friends and get behind at school. Feel embarrassed and different. ↓Self esteem. All makes returning to school more difficult
 - Prognosis depends on the young person, family strengths and severity
 - Increased risk of agoraphobia in adulthood
- Management:
 - Support for parents and child
 - Quick return to school before problem becomes entrenched, even if only for a small portion of the day
 - Education for child and parent. Facing the fear is initially distressing but reduces the anxiety, avoidance increases it
 - Parents need to be consistent in their commitment to return the child to school
 - Involve school teachers (eg meet at gate, etc). Problem is actual separation – once settled into the day problem is likely to reduce
 - Severe or chronic → referral. Support for parents if they're having difficulties. SES Behaviour Support Teams or Resource Teachers for Learning and Behaviour (RTLBs) for child.
 - No place for medication unless underlying conditions

Bullying

- = An act of aggression/harassment by a child/youth
- Starts mid-primary, peaks 3rd form, nearly gone by 7th form
- Typical bullying behaviour: boys hit, girls tease and exclude
- Teachers generally under-estimate bullying
- Characteristics of someone who is bullied:
 - Something different: high achiever, less physically attractive, etc

- Vulnerable: more anxious, cry easily, don't fight back
- Problem compounded for the bullied in that no one wants to be friends with a person who is bullied → ↑isolation. Standing up to a bully is pretty sophisticated behaviour in early teens – not developmentally consistent with wanting to identify with the peer group
- Long term outcomes worse for the bully than for the bullied

Depression

- Mood disorders are prevalent and serious disorders in children and adolescents. Leads to difficulties at school and in social relationships
- 1 year prevalence estimated as high as 10%
- Same diagnostic criteria as for adult – but diagnosis harder. More likely to present with separation anxiety, phobias, somatic complaints and behaviour issues. More likely to talk of profound boredom and feeling unloved and lonely than appetite and sleep change
- Most do recover, but recurrence is more common than in adults
- Clinical approach:
 - See the teen on their own
 - Observe: ↓energy, anxiety, anger, shame, variability in affect
 - Listen: the teen is more likely to talk if they feel they are being heard
 - Consider differentials: Depression, drug abuse, eating disorder, psychosis (actual or prodrome), medical
 - Suicide assessment
- Aetiological factors to consider:
 - Family context
 - Cultural context: are they comfortable about who they are in a cultural sense
 - Peer group: Have they friends, how do they support him/her?
 - School: bullying, what's hard at school, current stressors
 - Life events: losses, abuse
 - Psychological: negative ways of thinking, learned helplessness
- Treatment involves the child, parents and school. Aim is to shorten the episode. Treatment can include:
 - Education
 - Counselling: for milder depression, no remediable family factors, recent life events, if they want it
 - Family therapy
 - A range of individual therapy types – usually through referral
 - Medication: less evidence of effectiveness in adolescents. Consider discussion with a psychiatrist. Usually SSRIs
- Referral when:
 - Significant suicide risk
 - Possible psychosis
 - Abuse
 - Severe family discord
 - Failure to improve

Youth Suicide

- Epidemiology:
 - Second only to MVA as cause of death – but still uncommon.
 - 3 fold rise in last 30 years.
 - Females attempt, males succeed
 - Second highest rate for 15-24, Finland higher
- Postulated factors contributing to increase:
 - ↑Depression and substance abuse
 - Unemployment
 - ↑Isolation and alienation
- Key issue: identifying those at risk
- Risk factors:
 - Male gender

- Psychiatric illness: depression (most common association), alcohol or substance abuse, personality disorder, psychosis
- Previous suicide attempts
- Available means: firearms, toxic medications
- Social adversity: recent interpersonal loss, homelessness, school failure or drop-out, family or relationship problems, unemployment
- Recent exposure to suicide
- Most common presentations are over-dose, self-poisoning and lacerations
- Management:
 - Treat underlying psychiatric disorder (not TCAs – too lethal in overdose. Use SSRIs)
 - Reduce ongoing stress: counselling to reduce interpersonal conflict
 - Promote social supports
 - Liaise with specialist health services

Other Mental Health Issues

- Eating disorders
- Substance Abuse:
 - Drug and alcohol use prevalent
 - Often comorbidity
- Sexual maturation: sexual behaviours, orientation, attitudes to sex and relationships, awareness of socially defined roles. Knowledge about pregnancy and STIs doesn't automatically translate into behaviours
- Risk taking behaviour:
 - Adolescence is a time of experimentation, pushing boundaries
 - Contributing factors: ignorance, impulsiveness, cognitive immaturity (sense of omnipotence and poor comprehension of long term consequences), peer groups, drugs and alcohol

Definition of Adolescence

- Developmental period between childhood and adulthood
- Age of majority in NZ = 20 years
- WHO definitions:
 - Adolescents: 10 – 19
 - Youth: 15 – 24
 - 10 – 24: Young people

Demographics

- Young people are the only age group whose health status has not improved in the last 40 years
- Current issues:
 - Accidents and injuries
 - Mental health issues
 - Health risk behaviours: smoking, alcohol, drugs, sex
 - Chronic illness: eg obesity, asthma, diabetes, etc
- Access to health services for adolescents fragmented – fall between child and adult services

Adolescent Development

- Summary:
 - An age of transition
 - Experimentation and change: inherent risk taking
 - Behaviours reflect maturational tasks
 - May use maladaptive behaviours to achieve developmental goals (eg smoking to gain peer acceptance). Need to change them to adaptive behaviours
- Also see Cognitive Development, page 24
- Physiological:
 - Puberty: highly variable – generally from 9 – 14 years. Can take 2 – 5 years to complete
 - Gain 25 cm in height, 50% of ideal adult body weight
- Stages of adolescence:
 - Early: coming to terms with body/biological changes
 - Middle: establishing self among peers as a worthwhile individual
 - Late: vocational/education direction and one-to-one intimate relationships
- Developmental issues
 - Abstract reasoning
 - Preoccupied with their own thinking
 - Peer group membership and conformity important
 - Consolidation of self image and identity
- Psychosocial:
 - Who am I and where do I fit in
 - Identity: self, culture, ethnicity, sexuality
 - Autonomy vs relatedness/connectedness
 - Goals and future direction
- Developmental tasks of adolescence:

Tasks	Early: 10 – 13 years	Mid: 14 – 16 years	Late: 17 – 21 years
Psychological tasks			
Independence	Separates from parents: questions, tests	Separation creates anxieties, ambivalence as retreats to family	Comfortable away from home, able to return for counsel without shame
Body image	Adjust to dramatic changes in body Constant comparisons	Try on images to find real self (incl. Sexual identify), attempts to improve image	Satisfied with realistic body image
Sexual drives	Marked sexual	Sexual experimentation,	Beginnings of intimacy

	curiosity, masturbation	narcissistic sexual relationships	and caring
<i>Social Tasks</i>			
Relationships	Boys 'gangs', girls 'best friends'. Crushes on adults	Other sex friendships, dating, try on other philosophies and beliefs	Individual relationships more important than group, ↑ intensity of relationships
Career plans	Vague, unrealistic	↑Efforts but influenced by 'escape' from home, glamorousness of career	Hard decisions → occupational identity. Delayed by higher education
<i>Cognition</i>	Concrete, literal, limited abstraction	Formal operations; use abstractions (what if...), introspection, less literal	Mature abstractions, problem-solving & self-reflection
Moral Growth: values	Need to follow rules of peer group or family	Narcissistic: feels good or is what I want ⇒ right → impulsiveness	Idealism, rigid standards of right and wrong, intolerance

Talking with Adolescents

- Keys to effective intervention:
 - A positive relationship
 - Thorough assessment
 - Inclusive of family and young person
 - Plans made with the young person and family
- Building a trusting relationship: introductions
 - Friendly, confident welcome but still professional
 - Introduce yourself directly to the teen, 'And is this your mum?'
 - Clear introductions: yourself, your role, what you'll be doing and why
 - Clear boundaries: explain that you see young people alone and with family and why:
 - 'You're on your way to being an adult. Want to support that process. But your parents also still have a role'
 - Allows them both to say things they might not in front of the other
 - Outline confidentiality:
 - 'I want to talk about confidentiality. Do you know what that means?.... Want to keep your information private'
 - 'There are 3 things I can't keep a secret: if someone's harming you, if you're harming yourself or if you're harming someone else. I need to do something about it – but will tell you what I'm doing'
 - 'Will talk to my colleagues for review – to check I'm doing the best I can'
 - Consider what you put in notes (they get around). Use standardised abbreviations.
- If adolescent doesn't want you to tell parents (and you think it's in adolescents best interests for them to know):
 - Why doesn't teen want parents to know ('You seem worried about your parents knowing this. Can you tell me about that?')
 - Attempt to persuade the teen to tell her parents
 - Offer to tell them yourself
- Keys to building the relationship:
 - Be keen to get to know this young person now
 - Accepting atmosphere
 - Respect
 - Non-threatening explanations
 - Give adolescent some control – encourage normal independence
 - Reveal hidden agendas
 - Give them time to talk – hold off asking questions
 - Make plans with the young person and family
 - If they don't want to talk, probably anxious/frightened. "It seems you're pretty angry about being here. Did someone make you come?"

- **Communication:**
 - Use language that is understood (no medical or adult jargon). *Check understanding*
 - Listen
 - Move from less sensitive to more sensitive topics
 - Move from third person approach to the personal
- **Set clear boundaries:** It is appropriate to identify what is and is not acceptable behaviour (eg creating risk of harm to themselves or others). Middle adolescents still require the security of clear boundaries. However, try not to be judgemental
- **Beware:**
 - Transference: person projects their feelings about someone else (eg parents) onto you
 - Counter-transference: You transfer feelings appropriate to someone else (eg your own kids) onto the adolescent (eg act as though you were their parent)
 - Objectivity: understand the most likely reason they won't talk is that they're frightened

HEADSS Risk Assessment

- Gives an overview of this individual's risk and resiliency
- If you don't ask they won't tell you
- Do ask, even if you think you know the answer
- **Home:**
 - Where do you live and whom do you live with?
 - Who do you get on with, who would you talk to if you had a problem?
 - What's good about home? What's not so good?
 - Who makes the rules and what happens if they're broken?
 - Is there ever any violence at home?
- **Education/Employment:**
 - What do you enjoy most about school?
 - What subjects do you like?
 - How are you getting on at school?
 - Do you get into any trouble?
 - How do you get on with your teachers/friends
- **Activities:**
 - What do you do after school/in the weekends?
 - What do your mates do? (Get an idea of peer relationships)
 - What did you do last weekend that you enjoyed?
- **Drugs:**
 - 'I check with all young people – not picking on you. Remember it's confidential. You don't have to answer if you don't want to'
 - Lot's of people your age smoke/take drugs/drink. Is it like that at your school?
 - What do you think about that?
 - What have your friends tried? What about you?
 - If no, make it positive 'that's fantastic - how come you don't and lots of others do?'
- **Sexuality:**
 - Most young people have become interested in sex at your age. Have you had sex education at school? What was it about – body changes, infection, preventing pregnancy, relationships?
 - Do you talk with anyone in your family about sex
 - Have you had a sexual relationship with anyone?
 - Do you have sexual feelings to boys or girls? Ever had sexual experiences with someone your own sex? If this is confusing or frightening for them then need to talk further.
 - Want to help you stay healthy...ask about safe sex
 - If not active – encourage them. But also check they can get condoms, etc: 'if you ever were to, where would you go for information or contraceptives (tie them down to specifics)'
- **Suicide risk and Depression:**
 - 'Everyone has good days and bad days. Do you ever have really good days? Really bad days?'
 - 'Often adolescents see me because they've been feeling down. How have you been?'
 - 'Have you been happy with the way things are going? How would you rate yourself over the last couple of weeks if 1 was foul and 10 was brilliant?'
 - 'Do you ever feel like you want to end it all?'
 - 'Do you have a plan to hurt/kill yourself?'

- ‘How do you plan to?’
- Determining the degree of risk:
 - Well adjusted
 - Vulnerable
 - Experimenter
 - Troubled
 - Out of control
- See Youth Suicide, page 174

Physical Exam

- Use chaperones
- Be thorough but assure privacy and modesty (work around clothing)
- Talk and explain (especially about growing bodies)
- Pubertal ratings: Get them to self report genital development off an Adelaide chart – don’t examine yourself unless specific problem

Puberty

- Physiology:
 - Pre-puberty: Inhibition of GnRH pulse generator by higher centres
 - Puberty: increasing frequency and amplitude of pulsatile GnRH secretion, initially at night, with FSH (→ follicles or Sertoli cells) and LH (→ hormone production) secretion in response
 - Also involvement of adrenal glands → androgens → secondary sex characteristics (eg pubic hair but not ↑ testicular size)
- Terminology:
 - Gonadarche: onset of gonadal function
 - Thelarche: onset of breast development
 - Adrenarche / Pubarche: Onset of development of sexual (pubic/axillary) hair
 - Menarche: Onset of menstruation
 - Spermarche: Onset of production spermatozoa
- Clinical signs:
 - Measured in Tanner stages (1 = no development, 5 = adult)
 - Girls: breast development first (ovaries enlarge first but can’t see them)
 - Boys: Testicular enlargement (use orchidometer)
 - Pubic hair development initially related to adrenal androgens and may be discordant with other changes
- What’s normal:
 - Girls: traditionally < 8 years or > 13 years abnormal. But ↑ number of girls have breast development at 7. Menarche relatively unchanged at 12 (ie earlier onset, but endpoint relatively unchanged). Getting earlier by 3-4 months per decade (but psycho-social development unchanged)
 - Boys: < 9 or > 14 abnormal. No strong evidence of it getting younger

Normal variants

- Mini-puberty in neonatal period
 - Usually neonate – but up to 4 months
 - Due to hormones in utero and underdeveloped CNS inhibitory mechanisms
 - Breast development +/- milk (Witches milk - completely normal)
 - Withdrawal uterine bleeding (following endometrial development in utero)
 - Estrogenic effects on genitalia
- Premature Thelarche
 - Isolated early breast development
 - Tanner 2 or 3 maximum
 - No advancement in bone age
 - Follow-up to ensure it is isolated not progressive (ie that it’s a normal variant)
- Premature Adrenarche:
 - Isolated early pubic hair development +/- acne +/- BO
 - Caused by adrenal androgens
 - No advancement in bone age and normal menarche/spermarche

- Need follow-up (eg to exclude adrenal tumour)
- ?Association with future hyperandrogenism (eg Polycystic Ovary Syndrome)
- Gynaecomastia:
 - Breast development up to stage 3 during male puberty (75% of males)
 - Usually in early puberty – resolves in about 2 years
 - Reassurance, occasionally surgery
 - Pathological:
 - In rare instances: Klinefelter’s syndrome, gonadal failure
 - Outside of puberty (eg oestrogen producing tumour)
- Key sign indicating normal: normal bone age/no growth spurt

Precocious Puberty

- Definition arbitrary
- Consequences:
 - Short stature
 - Psychosocial (out of sync with peers)
- Clinical signs: Old bone age and growth spurt (in addition to eg breast development)
- Gonadotrophin Dependent:
 - = Central/complete. Hypothalamic or pituitary cause and → balanced development
 - Girls:
 - Normal progression through puberty (ie variant of normal?)
 - Rapid progression suggests pathology
 - Boys:
 - Normal progression of puberty
 - Less common than girls, more likely to be pathology
 - Causes:
 - Idiopathic (95% in girls)
 - Hypothalamic hamartoma: developmental anomaly
 - Tumours (eg of hypothalamus or pituitary)
 - Other CNS conditions (eg hydrocephalus, spina bifida)
- Gonadotrophin Independent:
 - = Peripheral/Incomplete. Peripheral cause and not all characteristics of normal puberty
 - Girls: rapid progression or virilisation
 - Boys: Testes remain small, rapid progression
 - Causes:
 - Hormone ingestion
 - Congenital Adrenal Hyperplasia (ie adrenal androgens)
 - Tumours: adrenal, gonadal or hCG secreting
 - Autonomous hormone production (rare)
- Investigations:
 - Bone age from hand x-ray
 - Measure hormones
 - GnRH stimulation test
 - Imaging
- Treatment:
 - GnRH agonist for central precocious puberty via depot. If GnRH is not pulsatile it switches off FSH and LH
 - Girls: progesterone delays menarche

Delayed Puberty

- Hypogonadotropic: Hypothalamic/pituitary causes:
 - Constitutional delay (check for bone age)
 - Exercise/nutrition (eg anorexia)
 - Generalised pituitary failure (eg post surgery/radiotherapy for CNS tumour)
 - Rare isolated deficiencies
- Hypergonadotropic: Gonadal failure
 - Chromosomal: eg XO, XXY
 - Infections (eg mumps, especially during puberty)

- Autoimmune
- Surgery, radiotherapy, chemotherapy
- Galactosaemia
- Other:
 - Structural (eg normal puberty but no menarche)
 - Intersex disorders: chromosomal sex \neq phenotypic sex
- Pubertal arrest: always pathological (eg pituitary tumour)
- Investigation and treatment:
 - Gonadotrophins +/- GnRH stimulation test
 - Hormone replacement
 - Fertility issues (eg with gonadal failure)

Chronic illness and disability in Adolescents

- See also Effect of Chronic Disease on Development, page 28
- Between 1- 20 % of young people have a chronic or disabling condition
- US prevalence:
 - Asthma: 50/1,000
 - Mental retardation: 25/1,000
 - Epilepsy: 4.1/1,000
 - Diabetes mellitus: 4.1/1,000
 - Down syndrome: 1.1/1,000
 - Cystic fibrosis: 0.2/1,000
- Survival rates are improving
- Developmental impacts:
 - Primary: effects of the disease
 - Secondary: effects of delayed development
 - Tertiary: effects of treatment
- Similar risk taking behaviours to healthy adolescents
- Impacts on development:
 - Most don't have major problems, and consider their illness less severe than doctors do
 - Process of separation from parents may be slowed if dependent on parents for care or limited opportunities to socialise with peers
 - Learning disorders → embarrassment, failure, frustration, ↓self worth, performance anxiety, learned helplessness
 - Slowed sexual development or physical deformity → problems with sexual identity
 - Normal developmental issues such as struggle for independence, concrete thinking, narcissism (what feels good is right) and sense of omnipotence (future is a long way off) → non-compliance with treatment
- Mental health in those with chronic illness:
 - Highly variable
 - Vulnerable periods: early years, transitions, severe illness
 - Important variables: onset, disruption to early attachments, premorbid function, family stress, mental health of parents, experience of failure or victimisation, repeat hospitalisation, life expectancy
- Resilience:
 - Focus on the young person not just the disability/illness
 - Focus on building strengths, achieving successes
 - Competence in self-care/management of illness/disability
 - Access to age appropriate coping strategies
 - Opportunities for responsibility/required helpfulness
 - Family relationships
 - Peer relationships
 - School attendance

Sexual Health

- References: Wellington Sexual Health Service, 4th and 5th year Handout, 2000

Sexual History Taking

- Purpose of sexual history is to determine:
 - Whether or not there has been a risk of exposure to an STI including HIV
 - If it is an appropriate time to take tests (window period – genital tests are not taken unless at least 14 days has elapsed from unprotected sex, unless symptomatic. For blood tests wait 3 months)
 - Who else had been at risk and may need testing/treating
- Approach:
 - ‘Going to ask personal questions – want to be able to offer right tests and care’
 - Ask why they’ve come
 - Use patient’s language
 - Don’t make assumptions about anyone
 - Lot’s of reassurance: STD’s are common, confidentiality, support relationship issues – let them decide, continue at a later date
 - Not interested in their orientation but what they do
- Questions:
 - Are you sexually active?
 - How many partners have you had in the last 6 months – male or female?
 - Alcohol and drug history
 - Do you suspect that you may be at risk from HIV or other STD?
 - Need to ask about sexual abuse – won’t volunteer it: Ever had sex when you didn’t want to, ever been sexually assaulted?

Exam

Female

- Inspection: pubic lice, genital warts, ulcers, blisters, scabies
- Palpation: inguinal lymph nodes
- Vaginal examination with speculum
- Bimanual examination of pelvis

Male

- Examination of external genitalia
- Palpation of inguinal nodes
- Palpation of scrotal sac and testes

Sexually Transmitted Diseases (STDs)

- Types and incubation:
 - Chlamydia (7 – 21 days) ⇒ don’t test till 14 days after contact (unless symptomatic)
 - Gonorrhoea (range 1 – 14 days, commonly 2 – 5 days)
 - Trichomonas (3 – 21 days)
 - Herpes Simplex Virus (2 days onwards – maybe years)
 - Human Papilloma Virus (2 – 4 months, up to a year, vertical transmission possible)
 - Human Immunodeficiency Virus (HIV) – (seroconversion illness 2 – 6 weeks after exposure, HIV antibodies almost always present after 3 months. Mean time to developing AIDS defining illness 9 – 12 years)
 - Hepatitis B (1 – 6 months)
 - Syphilis (9 – 90 days)
 - Non-specific urethritis
 - Pubic Lice (eggs – 2 weeks to mature, larvae – 1 week to mature)
 - Scabies (3 – 30 days, 6 weeks for itch to develop)
 - Hepatitis C and A may be sexually transmitted
- Not necessarily sexually transmitted:
 - Normal anatomical variants
 - Commensals → bacterial vaginosis
 - Dermatoses
 - Candidacies (commensals)
 - Molluscum contagiosum (3 weeks – months)
 - Urinary tract infections

- Prostatitis
- Vulval disorders

Tests for STD's

- Urethral swab or first pass urine for chlamydia
- Anal or throat swab for gonorrhoea if appropriate
- Female:
 - Cervical sample for gonorrhoea, chlamydia (endocervical cells needed)
 - Cervical smear
 - High vaginal swab for bacterial vaginosis, candida, trichomonas
- Male: Urethral swab for gonorrhoea
- Blood tests:
 - Hepatitis B (Ag and Ab) and C (Ab)
 - Syphilis: VDRL, TPHA
 - HIV Ab if appropriate with counselling and consent. Always attend for results

Vaginal Discharge

- Cervical secretions in women not on the pill, and which change during the cycle, are part of normal discharge. Mucus is clear or clear/white. Some inflammatory cells are normal in the latter half of a cycle
- Desquamating vaginal cells with healthy lactobacilli are major part of normal discharge – pH < 4.5
- Key history questions:
 - Colour
 - Odour
 - Itch
- Differential:
 - Thrush (Candidiasis): white curds, very itchy, not smelly
 - Trichomoniasis: grey/green discharge, fishy smell, moderate itch
 - Bacterial Vaginosis: green, fishy, itchy
 - Chlamydia: asymptomatic or discharge
 - Atrophic vaginitis: brown, spotty discharge (from bruising), pain, no itch. Treatment: oestrogen cream or HRT

	Bacterial Vaginosis	Trichomoniasis	Candidiasis
Prominent symptoms	Discharge odour	Discharge, vulval irritation	Itch
Classical signs	No vulvitis or vaginitis	Vulvitis, vaginitis, strawberry cervix	Vulvitis, vaginitis – fissured and sore
Classical discharge	Greyish-white, thin, may be frothy	Green/yellow, watery, pools in posterior fornix, may be frothy	White, flocculent, thrush plaques
Risk factors			Pregnancy, antibiotics, steroids, diabetes
Vaginal pH	pH > 4.5 (often 5.0 – 6.0)	pH > 4.5 (often 6.0 – 7.0)	pH < 4.5 (often 3.0)
KOH test (amine/Whiff test)	Positive	Weakly positive	Negative
Wet mount preparation	Clue cells present (vaginal cells covered by anaerobes & Gardnerella vaginalis). Replacement of lactobacilli with small coccobacilli (Gardnerella) or motile curved rods (Mobilunus). Few pus cells	Trichomonads (motile flagellate), pus cells	Yeast cells (blastospores)
Gram stained smear	Clue cells: G-ive curved rods. G variable coccobacilli.	Pus cells: acridine orange stain	

Notes	Also called Gardnerella. Multiplication of anaerobic bacteria and gardnerella. Associated drop in lactobacilli ↑Risk of prem delivery	Possibly commonest STI worldwide – mainly 3 rd world. A Protozoa. Doesn't culture well. Can cause PID, prem delivery. Exclude gonorrhoea	Most common cause of discharge Candida albicans often normally present. Poor immunological control → recurrent candidiasis
Treatment	Anti-anaerobe: oral metronidazole	Oral: doxycycline (remember 7 day rule)	Clotrimazole pessary

Neisseria Gonorrhoeae

- Description: G –ive diplococci
- Symptoms:
 - Male: 80% symptomatic. Discharge & dysuria (razor blade pain). 30% also have chlamydia
 - Female: only 20% symptomatic – can have vaginal discharge or pelvic pain. Pick up with opportunistic/selective screening if under 25, multiple partners, changed partner in last 6 months, IUCD, etc
 - Rectal and pharyngeal: often asymptomatic
- Diagnosis: gram stain microscopy if symptomatic or contact, or culture on chocolate agar
- Advice: no sex until minimum of 3 days since treatment completed
- Treatment:
 - Amoxicillin 3 gm and Probenecid 1 gm stat (no longer standard due to ↑ penicillin resistant), or
 - Ciprofloxacin 500 mgs (a quinolone) stat if penicillin allergy or if resistant. Specialist endorsement required. If resistant to that then Ceftriaxone (common in Auckland).
 - Azithromycin will cover gonorrhoea if it is being used to treat concurrent chlamydia
 - Resistance possible
- Contact tracing required. Treat partners
- Test for cure at 14 days (legal requirement)
- Complications: See Pelvic Inflammatory Disease (PID), page 185

Chlamydia Trachomatis

- Description: obligate intracellular bacteria, STIs are types D – K. Highest in 20 – 24 year age group
- Symptoms:
 - Urethritis, unexplained cystitis, mucopurulent cervicitis, pelvic pain, irregular bleeding
 - 80% of females and 50% of males have no symptoms. Suspect and test if sexual contacts have it, if patients asks for STI tests, patients under 25 with new/multiple partners
 - Up to 30% associated with concurrent N Gonorrhoea infection
- Diagnosis:
 - Female: swab from affected area, including from endocervix. Rotate 6 – 10 times. Urine test alone not sufficient. Most common site of single infection is cervix (ie urine is clear)
 - Male: urine test
 - New PCR test easier sampling (urine test)
 - Opportunistic detection has been shown to reduce rates of PID and ectopic pregnancy
- Advice:
 - Abstain until treated – if not use condoms
 - Contact trace
- Treatment:
 - Without test results: Doxycycline 100mgs bd for 7 days (remember 7 day rule for patients on OC)
 - Known positive **and** partners: Azithromycin 1 g stat orally – directly observed treatment
 - In pregnancy: erythromycin ethylsuccinate 800mg qid for 7 days – must be treated to prevent amnionitis and premature rupture of membranes
 - In PID: Doxycycline/erythromycin for 14 days and ornidazole 500 mgs bd for 7 days, plus consider gonorrhoea in which case penicillin/ciprofloxacin in addition
 - Test of cure in 3 weeks if non-compliance or re-infection suspected. Urine test is adequate for males and females
 - Test high risk patients only for cure
 - If reinfection, then ?untreated partner

- Complications:
 - Neonatal: conjunctivitis, pneumonitis 2 – 4 weeks later
 - See Pelvic Inflammatory Disease (PID), page 185

Herpes Simplex Virus (Type 2)

- See Herpes Simplex Virus (HSV), page 78

Pelvic Inflammatory Disease (PID)

- ~ Tubulo-peritoneal Disease
- Cause: ascending infection of vagina and cervix to endometrium, fallopian tubes and other structures:
 - Chlamydia – often chronic
 - Gonorrhoea – often acute
 - Can also be anaerobes (e.g. after instrumentation of the uterus or long standing PID)
- Symptoms: Acute pain, but 30% asymptomatic, dyspareunia (pain on sex)
- Risk factors:
 - Young age (< 25)
 - ↑Sexual activity, multiple partners, multiple infections
 - Postpartum infections
 - IUCDs in first several weeks after insertion
 - Decreased rates with condoms, diaphragms, spermicides (bacteria can use sperm as vector), tubal ligation, OC pill
- Diagnosis:
 - Difficult to make clinically: there are multiple causes of abdominal pain
 - Cervical motion tenderness (also occurs with ectopic pregnancy)
 - Purulent cervical/vaginal discharge
 - Oral temperature > 38 C
 - Irregular bleeding and break through bleeding on the OC pill
 - Ultrasound of no help. Test for other STIs. May require laproscopy
- Treatment: Antibiotics must cover anaerobes, chlamydia and gonorrhoea. E.g. Doxycycline 100 mg bd for 10 – 14 days plus an anti-anaerobe such as metronidazole or ornidazole
- Sequelae:
 - Often repeat episodes due to:
 - Continued at risk behaviour
 - Partner is not treated
 - Past infection compromises cilia of the fallopian tubes making another infection more likely
 - Infertility risk after 1 infection is 11%, but up to 54% after 3 infections
 - Other sequelae: ectopic pregnancy, adhesions, chronic pelvic pain

*Reiter's Syndrome**

- Triad of arthritis (big joints – hot, red swollen, bilateral), urethritis and conjunctivitis
- 10:1 are males, usually 25 – 35 years
- Often (not always) caused by chlamydia (an immunological reaction, HLA B27+ more susceptible)
- Treatment: treat residual infection, if any

Genital Warts

- Can get anal warts without anal intercourse
- External warts usually benign (types 6 & 11 – not oncogenic)
- Treatment:
 - Destructive: Condyline, liquid nitrogen – high recurrence rate
 - Imiquimod – topical cream, up-regulates immune system, expensive (\$150 per month), 19% recurrence, requires treatment for 8 – 12 weeks
- A vaccine is at stage 3 trials

Non-Sexually Transmitted Genital Skin Lesions

- Not all skin lesions on the genitals and surrounding areas are due to STDs
- Normal anatomical variants:
 - Pearly penile papules: small papillae around the corona of the penis
 - Sebaceous cysts of the penis, labia minora and scrotum
 - Normal papillae in the vaginal vestibule: can be mistaken for warts

- Dermatoses:
 - Contact dermatitis: soaps, deodorants, etc
 - Psoriasis: especially head and corona of the penis. Red, scaly plaques. Not itchy. Look for it elsewhere
 - Reiter's Syndrome: urethritis, conjunctivitis, arthritis in addition to skin lesion
 - Lichen Planus: itchy plaques on the penis
- Infections (not necessarily sexually acquired):
 - Seborrhoeic dermatitis: a fungus, red, sharply defined area covered with honey coloured scales
 - Candidiasis: red, irritating, itchy rash. Treat with Clotrimazole (Canesten)
 - Dermatophyte infections (tinea) are common. Characteristic spreading edge, itchy
 - Folliculitis: small pustule around a hair follicle
 - Scabies: red, itchy nodules – may not resolve despite treatment. Treat with malathion 0.5%
 - Erythrasma: scaly, flat, brown, pigmented rash, not itchy. Caused by corynebacterium
 - Molluscum contagiosum: may be sexually acquired. Small, pearly umbilicated lesions on the thigh and buttocks

Contraception

- Ideal contraceptive is 100% effective, only desirable side-effects, readily reversible, and able to be used un-supervised
- Reference: OHCS + numerous pamphlets
- For a younger person wanting to start on the pill:
 - Discuss the possibility of coercive sex, especially if under 15
 - Discuss the emotional and physical consequences of sex
 - Ask about prior contraceptive use
 - Ask whether they want to become pregnant – establish a context for motherhood in terms of the next 5 years and their life goals
 - Find out their thoughts about birth control (many myths: birth defects, ↓ fertility)
 - Inform about all methods
- Risk assessment questions:
 - Current sexual history
 - Past problems with weight gain
 - Acne
 - Headaches/migraines
 - Dysmenorrhoea/irregular menses
 - Nausea/abdominal pain
 - Diabetes
 - Smoking
 - Personal or family history of DVT
 - Hypertension or IHD

Natural Family Planning

- No intercourse from 6 days before to 2 days after ovulation – free and no drugs
- Monitor fertility by:
 - Checking cervical mucus – clear and stretchy when fertile
 - Temperature ↑ 0.3 C after ovulation (affected by fevers, drugs, drink)
- Success if regular cycles, dedication and self-control
- Peak effectiveness is 2% - usually 10 - 20 % (pregnancies per woman years)

Barrier Methods

- Low health risk, need high motivation, some STD protection
- Condoms, Caps +/- spermicide, female condom (Femidom)
- Don't use oil-based lubricant or anti-thrush cream with condom
- Spermicide gives extra protection

IUCD

- Eg Novagard
- Very effective (failure rate 1-2 per 1000 woman years)
- Inhibit implantation and may impair sperm migration
- Need replacing every 3 – 5 years

- Best in older, parous women in stable relationships
- Contraindications: Pregnancy, high risk for STD, undiagnosed vaginal bleeding, very heavy periods
- Complications:
 - Can be expelled from a nulliparous or distorted (eg fibroids) uterus
 - Ectopic pregnancy more likely (1 in 2000)
 - Associated with PID following insertion or STD
- If she becomes pregnant then must take the IUCD out (little risk of inducing miscarriage). If it's left in then ↑risk of chorioamnionitis, miscarriage or pre-term labour
- Mirena – carries levonorgestrel (a progesterone) → ↓risk of implantation and lighter periods (Good for menorrhagia). Lasts 3 years. 20% experience reversible amenorrhoea. Expensive. Can use with oestrogen only HRT (no ↑risk of endometrial hyperplasia) and avoid progesterone side effects

Combined Oral Contraceptive

- Initial exam should include:
 - Weight
 - Blood pressure
 - Cervical screen if appropriate
- = Oestrogen (usually ethinylestradiol) + progestogen:
 - Oestrogens:
 - Ethinylestradiol (most common), mestranol (which is converted to estradiol. 50 µg mestranol = 35 µg estradiol)
 - High dose = 50 µg estradiol. Low dose is ≤ 30 µg oestrogen. Adverse effects are dose related ⇒ give lowest dose that gives good cycle control.
 - Progestins:
 - Estranes: norethindrone, ethynosiol acetate, norethindrone acetate (latter two converted to the former)
 - Gonanes: norgestimate, levonorgestrel, desogestrel (gonanes have a longer half-life)
- About 4 * physiological dose of oestrogen
- Triphasic pill: mimic's body's fluctuation in oestrogen but → ↑break through bleeding
- Action: G type mucus + ↓GnRH (→ no ↑FSH or LH surge). 'Puts the ovary to sleep'
- Take for 3 weeks, then pill free for a week → withdrawal bleed
- Benefits of CoC: 99% effective, reversible, lighter periods, ↓PMS, ↓ovarian and endometrial carcinoma (but slightly ↑risk of breast cancer), ↓endometriosis
- Problems: compliance
- Contraindications:
 - > 35 and smoker (death 8 times more common in smokers – but still as safe as childbirth)
 - Any disorder predisposing to venous or arterial problems, eg ↑ lipids, APC resistance
 - Many cardiovascular problems (except mild hypertension)
 - Liver disease
 - OCP and Migraine – risks of stroke:
 - If uncomplicated migraine then OK (small increase in risk only)
 - Must not smoke
 - Migraine with aura or for > 72 hours (status migrainosus) or requiring ergotamine. Pill → 4 times risk of ischaemic stroke.
 - Contraindicated if complicated migraine (neuro signs/symptoms, hemiplegia or confusion during headache phase)
 - Contra-indicated in any woman with migraine if > 1 other risk factor for stroke (lipids, BP, diabetes, etc)
 - If OCP, smoker, and complicated migraines then 30 fold ↑ in stroke
 - Pregnancy
 - Undiagnosed uterine bleeding
 - Gross obesity or immobility (stop before major surgery)
- Special precautions: Family history of DVT, ↑BP or breast cancer; epilepsy, diabetes, illnesses causing diarrhoea (eg Crohn's)
- Side-effects: (usually worse when starting the pill), intra-menstrual bleeding, breast tenderness, nausea, ↑ or ↓ weight, mood changes, headaches
- Risks:
 - MI. Risk ↑ sharply over 40 – 1:2500 for non-smokers, 1:500 for smokers.

- Older progesterones: breakthrough bleeding, acne, headache, ↑weight → lead to search for new progesterones (ie 3rd generation progesterones like gestodene eg in Mercilon, etc)
- Nausea: due to oestrogen. Take with meal or with a snack at bedtime
- ↑Blood pressure (rare – but monitor 6 monthly)
- DVT Risk (Source: Medsafe flier)
 - 35/100,000 on the pill per year develop a clot, one dies ⇒ two deaths per year in NZ
 - Risk increases 3-4 times over population risk on 2nd generation, 6 – 8 times on 3rd generation. Increased risk of Diane 35 and Estelle 35 of 4 times over 2nd generation.
 - No ↑risk with PoP
- Drugs interfering with the pill: liver enzyme inducers (eg anticonvulsants, rifampicin). Consider higher dose pill
- Monitoring: 6 monthly-BP check. Check weight and breasts etc if > 35. Up to date with smears?
- Starting the pill:
 - On day 1 of cycle, or day of TOP, 3 weeks post-partum or 2 weeks after mobilisation after major surgery. Contraceptive cover immediate
 - Breakthrough bleeding is very common – especially in the first 3 months. Can add 20 µg estradiol every 24 hours, 12 hours after the usual pill, for one week.
- Missed pill: 12 hours late OK, after that the seven day rule (also if diarrhoea) – take 7 active pills before unprotected sex (eg if pill free days coming then skip them and go straight onto the next pack)
- Stopping:
 - 66% menstruate within 6 weeks, 98% by 6 months
 - At menopause: Stop at 50 with > 1 years amenorrhoea. CoC masks menopause, so stop at 50 and use non-hormonal method. Little evidence that it's not safe to continue to menopause

Progesterone Only Pill (PoP)

- = Mini-pill
- → Cervical mucus hostile to sperm (G Type mucus) + prevent ovulation in some + ↓tubal motility. Effectively a barrier method. Woman may still ovulate. Small risk of follicular cyst (one that doesn't pop) → pain with full bladder or rectum
- Worst side effect: erratic bleeding. Some women have amenorrhoea. Less risk of weight gain, acne, depression, breast tenderness, headache
- Less effective than CoC (0.3 – 4% failure) – age and compliance dependent
- OK where CoC contra-indicated and in breast-feeding mums (full breast feeding alone is protective for 3 months).
- Contraindications: History of ectopic pregnancy, breast cancer, liver disease or enzyme inducing drugs
- Must be taken same time each day (+/- 3 hours).
- Starting on the PoP: Alternative precautions for 7 days
- If pill missed then at risk for 2 days. Safe again after 2 days of restarting the pill
- Depot progestogen:
 - Safe, simple and effective (failure rate 0.4 – 1.2 %). Suppress ovulation, G type mucus, ↓ motility and implantation
 - Eg Depot-provera – deep IM 12 weekly, given during first 5 days of cycle, 5 days post partum if bottle feeding, 6 weeks if breast feeding.
 - Contraindications: pregnancy, abnormal undiagnosed vaginal bleeding, acute liver/cardiac disease
 - Advantages: no oestrogen, ↓PMS, secret, no compliance problems, good with GI disease, Ok with breast feeding, etc... Particularly good around major surgery, epileptics, after vasectomy and bowel disease
 - Problems: irregular bleeding – usually become amenorrhoea, also weight gain and acne. May also ↑depression and ↓libedo. Median delays of 10 months return to ovulation on stopping (fat soluble → very slow metabolism)

Sterilisation

- Reversal is only 50% successful ⇒ see it as irreversible
- Tubal ligation has 1% failure (1:200) – 10 times worse than vasectomy and same as IUCDs
- Vasectomy – easier than tubal ligation, but takes up to 3 months before stored sperm used up. Need to be tested and have 2 sperm-free ejaculates. Has been discussion of ↑risk of prostate cancer – best evidence says no association.

Emergency Contraception

- Ask why: unprotected intercourse, condom broke, etc. If no condom, then check why. If indicated: 'Are you worried about infection?' and 'Was it OK with you that it all happened the way it did' [checking for non-consensual intercourse]
- Ask:
 - How long ago was sex?
 - LMP
 - Regular partner (→ ↓risk of STD)
 - Medications
 - Previously had an ECP – any side effects. Sometimes nausea +/- vomiting with Progesterone only ECP
 - Other conditions. Old Oestrogen + Progesterone ECPs required history of DVT and focal migraine
- If sex < 72 hours ago prescribe:
 - Norelgestromin 2+2/Antinaus 5mg (12 hours apart, few side effects) or
 - 2/Microval 25+25 (can have while breast feeding, may reduce breast milk for ~ 1 week)
- Discuss:
 - How to take it
 - Pregnancy test in three weeks
 - Ongoing contraception, other advice
- Emergency IUCD: inserted within 120 hours of unprotected intercourse. Screen for STDs. Prophylactic cover if suspected

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