Pediatrics Notes - contributed by anonymous

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Immunization Timetable

Birth: Hepatitis B (HBV)

2 Mo: Diphtheria, pertussis, tetanus (DTP), oral poliovirus (OPV), Hib Conjugate (hemophilus

influenza, type B), HBV

4 Mo: DTP, OPV, Hib Cong

6 Mo: DTP, Hib Conj, HBV

12 Mo: MMR, PPD

15-18 Mo: Diptheria, tetanus, acellular pertussis (DtaP), OPV, Hib Conj

4-6 Yr: DtaP, OPV, MMR

14-16 Yr: Tetanus-adult dose, diptheria-reduced dose, every 10yrs (Td)

For Children behind in immunizations (over 1yr old):

If < 7yo:

DTP, OPV at first visit and then 2, 4, and 10mo later

MMR at first visit if >12-15mo, or when becomes 12-15 mo

Hib at first visit if >15mo and < 5yo (if <15mo, Hib conj has a special catch-up schedule)

DTaP and MMR at 4-6yo and Td at 14-16yo; repeat Td every 10yrs.

If >7vo:

Td, OPV, MR at first visit (2nd MMR at any time at least 1 mo after 1st).

Td and OPV #2 10mo later

Td at 14-16yo; Repeat Td every 10 years.

Note: Live virus vaccines (OPV, MMR) are contraindicated in immunocompromised children, in children who have immunocompromised people in their household, and in pregnancy.

Rules of Thumb for Growth

Weight:

Neonates lose 5-10% of birth weight in 1st few days of life

Birth weight regained by 7-10d, x2 by 4-5 mo, x3 by 1yo and x4 by 2yo

Daily gain is 20-30gm/d for 1st 3-4 mo, 15-20gm/d for rest of 1st yr; 5lbs/yr from 2yo to puberty

Avg. weight: 3.5kg at birth, 10kg at 1yo, 20kg at 5yo, 30kg at 10yo

Height:

Avg. length: 20in at birth, 30in at 1yo, 3ft at 3yo, 40in at 4yr (x2 birth length)

Avg. gain: 2-3in/yr from 4yo to puberty

Head Circumference (H/C):

Avg. H/C at birth 34cm, at 2yo 48cm

Avg. gain: 2cm/mo for 1st 3mo (40cm by 3mo), 1cm/mo for next 3mo (43cm by 6mo), 0.5cm/mo for

rest of 1st yr (46cm by 1yo); 10cm over rest of life

Tanner Stages of Pubertal Development

Breast development.

Stage B1: prepubertal, elevation of the papilla only.

Stage B2: breast buds visible or palpable with enlargement of the areola.

Stage B3: further enlargement of the breast and areola with no separation of their contours

Stage B4: projection of areola and papilla to form a secondary mound over the rest of the breast.

Stage B5: mature breast with projection of papilla only.

Male genital development

Stage G1: prepubertal.

Stage G2: enlargement of testis to more than 2.5 cm, appearance of scrotal reddening, and

increase in rugations.

Stage G3: increase in length and to a lesser extent breadth of penis with further growth of testis.

Stage G4: further increase in size of penis and testes and darkening of scrotal skin.

Stages G5 and G6: adult genitalia.

Pubic hair growth among boys

Stage P1: preadolescent, no pubic hair.

Stage P2: sparse growth of slightly pigmented, slightly curved pubic hair mainly at the base of the penis.

Stage P3: thicker curlier hair spread laterally.

Stage P4: adult-type hair that does not yet spread to medial thighs.

Stage P5 adult-type hair spread to medial thighs.

Appearance of pubic and labial hair among girls

Stage PH1: prepubertal, no pubic hair.

Stage PH2: sparse growth of long, straight, or slightly curly minimally pigmented hair, mainly on labia.

Stage PH3: considerably darker and coarser hair spreading over mons pubis.

Stage PH4: thick adult-type hair that does not yet spread to the medial surface of the thighs.

Stage PH5: hair is adult type; distributed in the classic inverse triangle.

Females:

Thelarche (breast buds) 1st sign of puberty at about 11yo, followed by adrenarche (pubic hair).

About 1yr after thelarche at Tanner 3, peak growth spurt occurs.

About 6mo after growth spurt at Tanner 4, menarche occurs.

Breast development from Tanner 2-5 takes 4yrs on avg. and pubic hair development takes 2.5yrs.

Males:

Testicular growth at about 11.5yrs is 1st puberty sign, marking Tanner 2.

Pubic hair appears next with penile enlargement within 1yr of testicular enlargement.

Progression to Tanner 5 over 3yrs, with growth spurt in Tanner 4-5.

Infant Diet Guide

1st 4-6mo of life:

Breast milk or formula; water (helps with constipation)

Supplement with vitamin D (if breast fed), fluoride (if not in water)

Premies: multivitamin, Fe, +/- folate and fluoride

Require 100-120kcal/kg/d

After 4-6mo:

Start solids (preferably after 6mo) and decrease breast/bottle feeds to <32oz/d; may need to increase water (higher renal solute load)

Start 1 new single ingredient food at a time, new foods only q3-4d. Cereal (rice, oatmeal, barley);

Fruit (banana); Vegetables (orange before green); juices (0.5 adult juice, 0.5 water).

DO NOT give eggs, citrus, desserts, or whole cow's milk (can lead to allergies)

Decrease breast/bottle feeds as increase solids.

Decrease breast/bottle feeds to <24oz/d when teething and DO NOT leave bottle in crib ("milk-bottle caries).

APGAR Scoring

Heart Rate:

0 (absent); 1 (<100/min); 2 (>100/min)

Respiratory Effort:

0 (absent); 1 (slow, weak cry); 2 (good, vigorous cry)

Muscle Tone:

0 (limp); 1 (some extremity flexion); 2 (arms/legs well flexed)

Reflex Irritability:

0 (none); 1 (some motion); 2 (cry, withdrawal)

Color:

0 (blue, pale); 1 (blue extremities, pink body); 2 (pink all over)

Notes:

Insert nasal catheter and observe reflex irritability.

APGAR is taken at 1 and 5 min, and again at 10 and 20 min in compromised infant.

Record lost points (eg. 1min APGAR=9, -1 for color)

Teeth Eruption

U: Upper, L: Lower Central Incisors

Primary: U 5-7mo, L 6-8mo Permanent: U 7-8yr, L 6-7yr

Lateral Incisors

Primary: U 8-10mo, L 6-8mo Permanent: U 8-9yr, L 7-8yr

Cuspids:

Primary: U 16-18mo, L 14-18mo Permanent: U 11-12yr, L 9-10yr

First Bicuspids:

Permanent: U 10-11yr, L 10-12yr

Second Bicuspids:

Permanent: U 10-12yr, L 11-13yr

1st Molars:

Primary: U 12-16mo, L 10-14mo Permanent: U 6-7yr, L 6-7yr

2nd Molars:

Primary: U 22-26mo, L 18-22mo Permanent: U 12-13yr, L 12-13yr

3rd Molars (wisdom):

Permanent: U 17-22yr, L 17-22yr

Developmental Milestones

G: Gross Motor, F: Fine Motor, L: Language, S: Social

Age: 1mo

G: Raises head from prone

F: Tight grasp, follows to midline

L: Alerts to sound (startle)

S: Regards face

Age: 2mo

G: Lifts head 45deg from prone; lifts chest

F: Follows past mid-line

L: Social smile

S: Recognizes parent

Age: 3mo

G: Support on fore arms; steady head control

F: Holds hands open at rest, follows 180deg

L: Coos, vocalizes

S: Reaches for familiar people/objects; anticipates feeds

Age: 4-5mo

G: Rolls front to back, begins to sit when propped, supports on wrists & shifts weight

F: Hands to midline, grasps rattle, touches cube on table

L: Orients to voice at 5mo; orients to bell laterally, says "ah-goo", razzes

S: Enjoys looking at environment

Age: 6-8mo

- G: Sits alone, rolls both ways, bears weight
- F: Reaches with either hand, transfers hand to hand, raking, grasp
- L: Babbles, laughs; at 7mo, orients to bell diagonally; at 8mo, "dada/mama" indiscriminately
- S: Stranger anxiety

Age: 9-11mo

- G: Creeps, crawls, pulls to stand, cruises, pivots when sitting
- F: Pincer grasp, probes/pokes with forefinger, holds bottle
- L: Understands "no" & name, waves bye-bye; at 10mo, "dada/mama" indiscriminately; at 11mo, one word
- S: Plays pat-a-cake, peek-a-boo; explores environment

Age: 12-14mo

- G: Walks alone
- F: Throws objects, lets go of toys, mature pincer grasp, points
- L: Follows 1-step command with gesture, 2 words; at 14mo, 3 words
- S: Imitates actions, comes when called

Age: 15-17mo

- G: Crawls upstairs, walks backwards, stoops to recover object
- F: 2-block tower, scribbles, finds hidden toy, inserts raisin into bottle
- L: 1-step command without gesture, points to 1-2 body parts, 4-6 words, immature jargoning
- S: Indicates wants without crying

Age: 18-20mo

- G: Runs, kicks/throws ball, sits in chair, walks upstairs with one hand held
- F: 3-4 block tower, turns 2-3 pages at a time, spoon feeds self, dumps raisin from bottle
- L: 7-20 words, mature jargoning
- S: Copies parents at household tasks, plays with other children

Age: 21-23mo

- G: Goes upstairs, squats in play
- F: 5-block tower, drinks from cup
- L: Points to 3 body parts
- S: Asks to have good and go to toilet

Age: 24mo

- G: Up & downstairs, jumps
- F: Turns pages one at a time, remove shoes, pants, imitates stroke (writing)
- L: 50 words, 2-word sentences, uses pronouns, points to 5 body parts, follows 2-step commands
- S: parallel play, asks questions

Age: 30mo

- G: Jumps with both feet off floor, throws ball overhand
- F: Unbuttons, holds pencil in adult fashion
- L: Repeats 2 digits, understands concept of "1"
- S: Tells 1st & last name when asked, gets self a drink

Age: 3yr

- G: Pedals tricycle, alternates feet going up steps, stands briefly on one foot
- F: 9-block tower, imitates bridge or 3 cubes, draws a circle
- L: 3-word sentences; uses plurals, past tense, 250 words, understands concept of "2"
- S: Group play, shares toys, takes turns; knows full name, age, and sex

Age: 4yr

G: Hops, skips, rides tricycle, climbs ladder, alternates feet going down stairs

F: 10-cube tower, catches ball, can cut & paste, draws cross, person with face, arms, body and legs

L: Knows 3-4 colors, counts to 10, says song or poem from memory

S: Tells "tall tales", dresses with supervision

Age: 5yr

G: Walks on tiptoes, jumps over obstacle

F: Ties shoes, uses a knife to spread, and draws triangle, prints 1st name

L: Knows colors, defines 1 word, and identifies coins

S: Plays competitive games, abides by rules, sexual curiosity, dresses alone

Age: 6yr

G: Rides bicycle

F: Draws person with 6 body parts

L: Knows right from left

Age: 8yr

F: Draws diamond

L: Tells time, reads for pleasure

S: Sense of humor, home chores

Developmental Delay

DQ=Developmental score/Chronological age x 100 If <80 (1 standard deviation) do work-up

Most common causes of developmental delay:

- 1. Mental Retardation
- 2. Autism
- 3. Psychosocial
- 4. Hearing loss
- 5. Expressive/receptive language disorder
- 6. Selective mutism

Primitive Reflexes

Reflex: Duration

Palmar (hand) grasp: Birth to 4mo Plantar (foot) grasp: Birth to 9mo Automatic stepping: Birth to 2mo

Moro: Birth to 3-6mo

(tested by supporting the baby's head in supine position, then suddenly dropping it 2-3in while

supporting)

Head in space: 1-2mo/permanent

(hold baby upright in air and tilt slightly to side; observe if it can hold head vertically (reflex present)

Asymmetric tonic neck (ATNR): Birth-1mo to 4mo

(Turning head to one side causes ipsilateral arm to extend and contralateral arm to flex)

Symmetric tonic neck (STNR): 5-6mo to 8mo

(Extending the head causes hips and knees to flex, while flexing causes hips and knees to extend)

Landau: 3mo to 12-24mo

(Hold the trunk in prone position and while in the air, look for arm, leg and head extension)

Parachute-downwards: 3-4mo/permanent Parachute-sideways: 6mo/permanent Parachute-forwards: 7-8mo/permanent Parachute-backwards: 9-10mo/permanent

(Parachute reflexes are protective to sudden disruptions of balance; leg extension for downwards,

arms out for others. The baby must have good head control (around 4mo old) to test.

Fluids & Dehydration: Assessment of Hydration Status

- 1. Parent estimate of intake: fluid type (water, formula, juice, etc...) and amount retained
- 2. Parent estimate of output: urine, stool, vomit, drainage, insensible losses (# of diaper changes now vs. usual, consistency of stool)
- 3. Physical signs (% Dehydrated)

Skin Turgor: good (5%), good/slight tenting (10%), tented (15%)

Mucous Membranes: moist (5%), tacky (10%), dry (15%)

Eyes: normal (5%), deep set (10%), sunken (15%) **Fontanelle**: flat (5%), soft (15%), sunken (15%)

Mental Status: consolable (5%), irritable (10%), lethargic/coma (15%)

Cardiovascular: normal (5%), tachy, normal BP (10%), decreased BP, poor capillary refill (15%) **Urine/tears**: concentrated urine, poor tear production (5%), oliguria, poor tear production (10%),

oliguria/anuria, no tears (15%)

Note: dehydration estimated from % wt loss from premorbid state

Initial Bolus

Give a bolus of isotonic (NS or Lac Ringer's) to rapidly re-expand the intravascular volume.

Assess response (Urine output, capillary refill, vital signs, mental status, etc...).

Repeat bolus if necessary.

10cc/kg over 1hr for mild dehydration (5%)

20cc/kg over 1hr for moderate dehydration (10%)

30-50cc/kg over 1hr for severe dehydration/shock (15%)

Rehydration

- Add fluid deficit to maintenance; give half of total volume in 1st 8hr and the rest over 16hr.
- Fluid Deficit (cc) = est. % dehydration x wt. (gm); ignore initial bolus in calculation
- Maintenance Fluid
 - Based on wt:
 - 100cc/kg (4cc/kg/hr) for each of the 1st 10kg
 - 50cc/kg (2cc/kg/hr) for each of 2nd 10kg
 - 20cc/kg (1cc/kg/hr) for each additional kg

Based on surface area (wt>10kg):

1500-1800cc/m2/d x surface area (m2)

<20 kg/m2 = [(3.6 x wt)+9]/100

>20 kg/m2 = [(2.5 x wt) + 9]/100

Require 3mEq Na/100cc, 2mEq Cl/100cc, 2mEq K/100cc

(D51/4NS in small children and D51/2NS in older children with 20mEg KCl after 1st void.)

Replace ongoing losses (stool, NG tube, etc...) cc for cc.

Note: above schema is only a guideline; continually reassess and adjust management to needs.

Diarrhea

Mechanisms

- 1. Osmotic: ingestion of poorly absorbable solutes.
- Dx: diarrhea ceases when osmolytes are removed from intestine.
- Increased stool osmolality (>280 mosm)
- eg. intestinal disaccharidase deficiency, monosaccharide malabsorption, cathartics (phos and magnesium salts)
- 2. Secretory: pathological stimulation of normal Na, K, Cl, HCO3 secretion.
- Dx: diarrhea continues with fasting.
- Stool is isosmotic to plasma (280 mosm)
- eg. toxin producing bacteria (E.coli, V. choerae, staph), hormones (gastrin, histamine, prostaglandins, VIP), drugs (phenophthalein, ricinoleic acid)
- 3. Malabsorptive:
- Inflammatory conditions (Crohn's, Sprue), drugs (Digitalis, colchicine, Abx), digestive enzyme deficiency (pancreatic insufficiency), obstruction
- 4. <u>Exudative</u>: damage to intestinal mucosa resulting in discharge of mucus, serum proteins, and blood into bowel lumen
- egs: infection (amebiasis, shigellosis, staph), inflammation (UC, pseudomembranous colitis), ischemia, drugs(Abx, heavy metal poisoning)
- 5. Increased intestinal motility

Bacterial diarrhea

Si & Sxs: Bloody stool

Dx: Wright stain of stool for poly's; if <3 months, consider bacteremia

Causes

- 1. Yersinia (pseudo-appendicitis, erythema nodosum)
- 2. Campylobacter
- 3. Salmonella (cystitis)
- 4. Shigella (generalized seizures due to toxin, hi temp, hyponatremia)
- 5. E. coli

Tx: Give Pedialyte; feed through diarrhea; DO NOT give Anti-diarrheals (may cause toxic megacolon and retention of toxins)

Causes of Chronic Childhood Diarrhea

Neonates

Infections: previous infections decrease enterocytes Protein intolerance: causes damage to enterocytes

Short bowel syndrome

NEC

Congenital absorptive defect

Congenital disaccharidase defect

CF

<2 yrs

Infections

Protein intolerance

Giardiasis

Celiac disease

Sucrase/isomaltase deficiency

Irritable bowel syndrome

CF

>2 yrs

Infections

IBD

Giardia

Celiac disease

Irritable bowel syndrome

CF

Meningitis

Presentation

- -Displays meningeal irritation signs by 18 mo old if meningitis.
- -If > 2-3yo meningeal irritation absence (can kiss knees) suggests no meningitis Si/Sxs
- -Hypo/Hyperthermia
- -irritability
- -lethargy
- -poor feeding
- -Brudzinski's sign-neck flexion causes hip flexion
- -Kernig's sign- cannot flex hips with thighs at 90 degrees
- -bulging fontanelle
- -rising sun sign (from hydrocephalus)
- -coma/obtundation

Bacterial Meningitis

General

- -Meningoencephalitis with vascular involvement > pure meningitis in kids.
- -Most common entry into CNS is via bacteremia
- -3 mo-8 mo age of peak incidence

Etiology

Neonate <28 days

- -Foci of infection: gut, bladder, swallowing of vaginal/perineal flora intrapartum or in utero infection
- -Pathogens: GBS, gram negatives (E.coli, Klebsiella, Pseudomonas), Listeria Infant/Children 3 mo-5yrs
- -Pathogens=pneumococcus, meningococcus, H. flu
- -Children >3mo have decreased immunity, low nasopharyngeal IgA
- -Bacteremia can seed CNS, joints, heart, lungs
- -Most virulent meningitis but most easily treated

Labs(LP)

- -Glucose <50, protein >50, WBC >10. -Opening pressure >140-180 mm /H2O in BM or in a small crying child.
- -If bloody tap fluid does not decrease with flow, then has hemorrhagic CNS disease (HSV).
- -Pus=100 PMN's/ml.
- -Protein cause turbidity
- -Yellow csf indicates bilirubin

Sequelae of BM

- -Deafness/hearing loss (mild or total)due to CN VIII swelling; etiology: pneumococcus > meningiococcus>H.flu. (pmh). More H.flu in prevaccine population.
- -Seizures-tonic clonic sz can be presenting symptom of BM in 20-30% of pts. Leukocytes release cytokines which cause cerebral edema. Focal sz can be seen 3-4 days into MB due to localized dz (brain abscess, subdural effusion, empyema). Focal sz indicate poor prognosis.
- -Cranial nerve palsies-mostly CN VI, the longest intracranial course
- -increased ICP
- -Cerebral Palsy/Mental Retardation
- -Speech problems
- -Focal neural loss
- -Death- 5-10% even with abx (GM 20-25%, Strep 10-15%)
- -Hydrocephalus- increased CSF production in lateral ventricles. Communicating or noncommunicating. Tx with VP shunt (complications: obstruction with no long term mental sequelae to infection with Staph epidermidis with mental sequelae).

Aseptic Meningitis

- -Pathogens=80% Enteroviruses (Coxsackie, polio, echo), 7-8% Arbovirus
- -Peaks in summer
- -CSF- lympocytosis at 6-8 hours
- -Herpes rarely causes meningitis, usually encephalitis

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Antibiotics for Meningitis

Neonates <28 days

Pathogens

- 1.GBS-Amp and Gent. Many are pen/gent resistant, but amp (cell wall inhibitor) helps.
- 2. E. coli-Gent
- 3. Gram (-) (Klebsiella)-Gent
- 4. Listeria (tumbling gram (+) rod)-Amp

Infants 4-8 weeks

All pathogens for infants and kids should be considered and treated.

Abx=Amp and Cefotaxime

Infants >8 weeks

Encapsulated pathogens

- 1. Strep pneumo
- 2. Hib
- 3. N. meningitides

Abx=Amp (enterococcus and listeria) and Cefotax; rifampin for HIB or N. meningitides to decrease carrier state.

Chloramphenicol is no longer used; can cause bone marrow suppression and gray baby syndrome (vascular collapse) in neonates

always treat h. Flu or neisseria also with rifampin to decrease carrier state (treat contacts also)

If no improvement with abx think

- -not enough or wrong drugs
- -new pathogen (secondary infection)
- -abx not penetrating (ie. Abscess)
- -viral infection

Meningococcus: better prognosis if presenting as meningitis (pt survived seeding) rather than bacteremia; most rapidly fulminant but easiest treated.

Pneumonia

An inflammation of lung parenchyma

Viruses are the most common agents in infants

Mycoplasma pneumoniae most common in children

Bacterial PNA, bugs isolated from blood: strep pneumo, H. flu, Staph aureus in infants, debilitated pts, and adolescents with widely disseminated disease

Predisposition to bacterial pna: aspiration, immunodeficiency, congenital anolamies (TE fistula, cleft palate) abnormal mucus clearance (CF, ciliary dysfunction ie Kartagener's, bronchiectasis), CHF.

Pneumococcal pna: only signs may be fever + tachypnea. In adults the classic picture is shaking chills, pleuritic pain, prostration, rusty sputum and high mortality Involvement is characteristically lobar consolidation. Complications include empyema &

Involvement is characteristically lobar consolidation. Complications include empyema & septicemia. Sicklers are particularly susceptible to overwhelming pneumococcal infections. Tx with penicillins

Hemophilus influenza: also lobar pna. Distinguished from pneumococcal pna by more indolent course and inadequate response to penicillin. Abx include amox or amp PO but if PO inadequate may require IV amp.

20% of H. flu is resistant to amox/amp, use Augmentin or IV cephalosporin.

Staph pna: most common in first year, usually unilateral, abscess, tissue destruction, overwhelming septicemia. 2 typical features: pneumatocele, rapid progression. Empyema with thick purulent fluid is a hallmark. Tx is chest tube for drainage

Mycoplasma pna: #1 cause of adolescent pna (25-35%). Incubation period is 2-3 weeks. Sx of headache, malaise, fever, cough. WBC and diff usually normal, "cold agglutinins" may be + after 1st week of illness. CXR looks worse than the pt. Can be fulminant in SS dz pts. Abx = erythromycin.

Assoc with bullous myringitis

Chlamydia trachomatis: contracted from birth thru infected vagina, usually presents at 1-2 months with low grade fever, machine gun or "staccato cough". Increased eosinophils on differential. cxr will have diffuse interstitial infiltrate. 50% have proceeding conjunctivitis. Dx by giemsa stain of conjunctival inclusion bodies. Abx with erythromycin.

Bordetella pertussis: 2000-4000 cases per year in US. Is an encapsulated gram negative rod, infects ciliated epithelium of upper respiratory tract. Pertussis toxin increases adenylate cyclase, organism also synthesizes and exports adenylate cyclase. Dx with culture on Bordet-gengou medium (high % of blood) ID by agglutination with specific antiserum or by fluorescent Ab staining. Assoc with apnea

3 stages of clinical dz:

- 1) Catarrhal phase, lasts 2 weeks, coryza with increased mucus and secretions, sx similar to cold
- 2) Paroxysmal phase: usually lasts 1-2 weeks but up to 4 weeks, consists of paroxysms of hacking cough with copious mucus followed by inspiratory "whoop" air rushes past narrowed glottis.
- 3) Convalescent phase: can last for months.

Abx erythromycin, but does not work well after phase I. If used after phase I, will decrease # of organisms in the throat to prevent spreading.

Contraindications to giving pertussis vaccine: previous hx of serious reactions to vaccine (temp >105 or severe swelling at injection site) seizure disorder or other significant neurological disorder

Indication for admission: age <4-6 months, resp distress, toxic appearing, dehydration, vomiting if not taking PO's, social factors, immunocompromised patients, esp sickler's, CF patients

Indic for d/c home:

not hypoxic, good sats on room air, good PO intake, good UOP, negative bcx

Viral PNA

RSV, parainfluenza, influenza A & B

Otitis Media

Pathogens

- 1. Strep pneumo (30%)
- 2. Hib (22%)
- 3. Moraxella cararrhalis (7%)
- 4. Group A Strep (2%)
- 5. Staph aureus (1%)
- 6. Sterile (35%)

3 tiers of management

- 1. Amox or amp (amp is QID, causes more GI upset, is not absorbed as well as amox)
- 2. Pediazole (erythromycin EES + sulfisoxazole) /Bactrim (trimethoprim + sulfamethoxazole)
- 3. Augmentin-useful against beta-lactamase producing H. flu
- 4. Ceclor- 2nd gen cephalosporin, can cause serum sickness

Wait 48 hours then usually increase from tier 1 to tier 3.

Mastoiditis

-90% have OM too

Pathogens=Group A Strep, Pneumococcus, Staph, H. flu

Try Cefotaxime +/- Oxacillin (staph coverage)

Neoplastic disorders

- -Leukemia is the most common onc dz of childhood
- -CNS tumors-most common solid tumor in kids
- --Medulloblastoma-Midline cerebellar lesion causes gait/balance deficits. Highly malignant, tends to metastasize throughout CNS. Likely to cause obstruction of 4th ventricle and cause noncommunicating hydro and icreased ICP due to location and rapid growth
- --Astrocytoma-usually in cortex and cerebellum, causes fine motor deficits, usually slow growing, most common brain tumor in children

Hodgkin's Disease

- -Lymphonas are third in frequency of childhood tumors
- -Accounts for 40% of new cases of childhood lymphona
- -usually presents with enlarged NONTENDER cervical node
- -Dx by biopsy
- -Reed-Sternberg cells are NOT pathognomonic
- -4 types=nodular sclerosing, mixed cellularity, lymphocytic predominace, lymphocyte depleted
- -Staging
- Stage 1-one lymph node or one extralymphatic organ involved
- Stage 2-more extensive dz on 1 side of diaphragm
- Stage 3-both sides of diaphragm
- Stage 4-diffuse widespread dz with extralymphatic sites
- "A"- no systemic symptoms
- "B"- systemic symptoms (fever, night sweats, wt loss of >10% normal body wt in 6 mo)

Neuroblastoma

- -embryonal tumor of neural crest origin
- -most common in the first years of life
- -50% of pts are <age 2
- -tumor usually in adrenal gland
- -also frequent in thoracic and cervical sympathetic ganglia
- -70% secrete catecholamines
- -catecholamine metabolites VMA, HVA, TM are diagnostic markers

Wilms

- -Nephroblastoma
- -Most common abdominal neoplasm in children
- -associated with sporadic aniridia, hemihypertrophy and GU malformations

Bone tumors

- -skeletal malignancies are 4th in frequency in adolescents
- -uncommon in younger kids
- -Osteosarcoma-frequently seen in adolescent growth spurt, usually distal femur, proximal tibia, or proximal humerus
- -Ewing sarcoma-usually age 10-20, can present with pain, swelling, occasionally fever. Radiation is primary therapy.

Pediatric Emergencies

Fever in the neonate (8 weeks)=ROS

Fever=temp > 100.4 or 38 C

Labs= CBC c diff, BCX, UA, UCX, CSF protein, glucose, cell count, CX

If pt rules out then dx is viral syndrome.

6-7% of neonatal swu's are positive. Of these, 3-4 look sick, 2-3 have an identifiable source of infection and 1-2 would have been missed.

The 1-2 that would have been missed is the reason we are so aggressive.

Symptoms to look for: changes in mental status, changes in feeding, hypthermia (esp in immediate neonatal period)

Fever + SS disease = ROS

Sicklers can be functionally asplenic as early as 6 months (when fetal hgb drops) see Howell Jolly bodies (nuclear remnants normally removed by spleen) in blood smear.

Asplenics are susceptible to encapsulated organisms (h.flu, meningococcus, pneumococcus) Sicklers with influenza, viral syndromes have 500 times the risk of pneumococcal infection so CX's and ABX are necessary

3 clinical scenarios:

- 1)"sick" at any age gets admitted for iv abx
- 2) well appearing and < 4yrs admitted for abx and cx's
- 3) well appearing and >4 yrs may be treated as an outpt with po ampicillin and blood cx's

SS dz + positive CXR = r/o chest crisis

A positive CXR in a sickler may be ateletasis, infiltrate or pulmonary infarction

In children the cxr appearance will lag weeks behind clinical improvement

Chest Crisis: pulmonary infection causes hypoxemia and acidosis which makes RBC's sickle and leads to vasoocclusion which leads to more hypoxemia and acidosis (viscious cycle)

This creates an environment for pulmonary infection to thrive

TX: fluids and O2 to prevent sickling, PRBC if needed, abx (cefotax or cefurox for encapsulated bugs)

Fever + painful limp = r/o septic arthritis

Fever with a painful hip = r/o septic hip

Most likely dx is toxic synovitis (aka transient synovitis, an inflammatory response 7-10 days post URI. Tx'd with antiinflammatories)

Septic hips must be ruled out because it can lead to necrosis of the femoral head

Hip joint tap done by ortho in the OR

Mnemonic for ddx of painful limp:

STARTS HOTT

Septic arthritis

Toxic transient synovitis

Acute rheumatic fever

Rheumatoid arthritis

Trauma

Sickle cell dz

Henoch Schonlein Purpura

Osteomyelitis

TB

Tumor

Other causes: Legg-Calve-Perthes (idiopathic avascular necrosis)

Slipped capital femoral epiphysis (head of femur falls posterior and inferior off femur, usually seen in overweight pubertal boys)

Fever + Seizure = r/o meningitis

Labs: CSF protein, glucose, cell ct, gram stain and cx

Be aware of cushings triad from increased ICP: hypertension, bradycardia and variable respirations

Fever + Petechiae = ros in adults think mening in children think H. flu type B tx with 3rd gen cephalosporin

Cyanosis of newborn (dusky)

---really blue lips--polycythemia
sepsis/infxs
metabolic/hypoglycemic
pulm stenosis
cardiac lesion
neuro - lose stim to breathe

*check vitals, O2 sat, ABG on RA and O2, EKG and CXR, consider PG (for tiny babies)then echo last

Tet of fallot-- if pulm atresia, need PG to keep open ductus arteriosus (blue tet)

Tricuspid atresia-- need EKG to show all vent large except RV, dec pulm bloodflow, gen VSD

Truncus arteriosus-- AV cushion defect so always VSD, pulm blood flow sounds cont murmur in blue baby, echo find pulm art off trunk, tx will close VSD and remove pulm art so trunk will be Aorta Transpositoion of great arteries--2 circuits in parallel, no shunting and ductal dependent; CXR ball on string= parallel great vessels c cardiomegaly, gen male and big baby, need surgical swap of vessels

Total anomalous venous return-- may get obstructed vein thru organs (below diaphragm) and lead to pulm edema, tachypnea and *small heart with big Right heart

Hyperbilirubinemia

Indirect ddx--hemolytic anemias, Gilbert's, Crigler-Najjar

Hemolysis=spherocytosis, pyruvate kinase or G6PD deficiency, hemoglobinopathies (sickle cell, thalassemia)

Gilbert=UDP glucuronosyl transferase defect. Jaundice with mild illnesses, fasting, physical stress

Crigler-Najjar=type 1 is complete absence of UDP glucuonosyl transferase, type 2 limited activity so phenobarbital will reduce bili but will not have any effect in type 2.

Direct hyperbili ddx

- *Viral--Hepatitis, EBV, CMV, HSV
- *Metabolic--Wilson's, Alpha-1-antitrypsin deficiency, CF
- *Biliary Tract d/o--Cholelithiasis, Cholecystitis, Choledochal cyst, Sclerosing cholangitis
- *Autoimmune Liver Disease--Type 1 (anti-smooth muscle Ab), Type 2 (anti-liver-kidney-microsomal Ab)
- *Heptotoxins--Acetaminophen, anticonvulsants, anesthetics, antituberculous agents, chemotherapeutic agents, antibiotics, OCP. Others include Etoh, insecticides, organophosphates *Vascular causes--Budd-Chiari, veno-occlusive disease

Hepatitis A-high AST, ALT and conjugated bili

Wilson's disease-AR d/o of copper metabolism results in accumulation in the liver, CNS, kidney, cornea and other organs. CNS manifestations may present with or without hepatic sx. Kayser-fleischer rings (golden brown discoloration in the sone of the descemet membrane of the cornea is virtually always present when neuro or psych sx develop. Can have coombs-negative hemolytic anemia due to oxidative injury to rbc by excess copper.

<u>W/U for indirect</u>: CBC, retic, smear, haptoglobin, direct and indirect coombs, hemoglobin electrophoresis, red cell enzyme assay, test for spherocytosis

<u>W/U for direct hyperbili:</u> LFT's, coags, albumin, glucose, cholesterol, ammonia, abdominal U/S, Hepatitis panel, Ceruloplasmin/24 hr urine copper, ANA, ASMA, anti-liver-kidney-microsomal Ab, serum alpha-i-antitrypsin level and phenotype, liver biopsy

the most sensitive test for synthetic liver function is the PT

worrisome signs

- *onset of hepatic encephalopathy
- *Vit K resistant prolongation of PT
- *Cerebral edema
- *Bili >18
- *Rising serum bili with decreasing ALT/AST
- *Rising serum creatinine
- *Hypoglycemia
- *Sepsis
- *Ascites
- *pH < 7.3 in acetaminophen overdose

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Cystic Fibrosis

Mutation on chromosome 17, autosomal recessive Most common lethal genetic mutation among caucasians, carrier rate 5%

Disorder of exocrine gland function that results in abnl mucus production Characterized by:

- -Recurrent pulmonary infections
- --Infants usually with staph
- --Children with pseudomonas

Any child with a second episode of pna should have a sweat chloride

Malabsorption from pancreatic exocrine insufficiency

FTT

Increased electrolyte conc in sweat

Meconium ileus

Endocrine insufficiency (DM)

Cor pulmonale

Clubbing

Nasal polyps

Infertility

Hepatic and biliary cirrhosis

Bleeding from decreased vitamin K absorption and decreased coag production from liver cirrhosis

Pneumothorax: mucous plugging causes ball valve phenom with air trapping

rectal prolapse