2013's Annual PBR Corrections & Clarifications Guide
(*Pediatrics Board Review, 3rd Edition*)

A FEW WORDS OF THANKS

Every year I like to go through all PBR error submission and send corrections to PBR members before the initial certification exam. It's a very time consuming task (takes several full days), but I believe it's worth it. In 2012, several test-takers said the guide helped them review and correctly answer several questions that came up on the exam.

So, I'd like to first say **thank you** to EVERYONE who submitted suggested corrections or requests clarifications from within the PBR. I REALLY, REALLY appreciate it. It's a tremendous help to the entire PBR "Crew."

I'm especially thankful to those of you who provided me with spelling or typographical errors, and to those of you who provided a page number, a clear question and a reference. It **really** streamlined the lookup and verification process!

Also, a **MASSIVE** thanks to the following people who volunteered to do the initial lookups for many of the topics/questions:

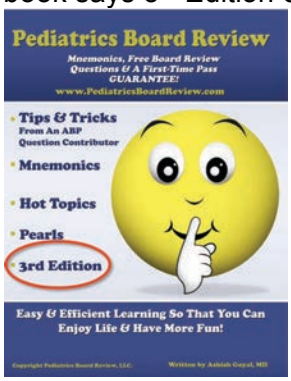
GSO, WN, CTM, SS, JL, MK, and SW

Without their help, it would not have been possible for me to get these corrections out to you prior to the 2013 pediatric board exam. I've sent this guide to them first as a way of saying thanks, but I'm hopeful that you are also reading this at a time when it will still be of help to you for your board exam.

Best of luck on your boards!
- Ashish

SOME NOTES/DISCLAIMERS

- The page numbers in this guide refer to the **3rd Edition of the Pediatrics Board Review**. The cover of the book says 3rd Edition on it.



- The **web links** in this document have NOT been updated yet. At the time of the publishing of this guide, approximately 10% of the links are no longer functional. A separate guide **may** be released prior to the 2013 boards with updated links.
- The **PBR Facebook CREW**! is a private, members-only area for those of you who have paid for a PBR product. **YOUR REQUESTS TO JOIN WILL BE REJECTED** if you have only signed up to get free info from PBR (GI & DERM study guides, emails about new PBR web article, tools, etc). We cross-check all requests to join before clicking the approval button in order to keep it a spam-free and intimate area.
- **PBR's 4th Edition will have many other changes**. The ones included here are only the ones that relate to questions, comments and corrections submitted by PBR members. Other changes will include my own updates to the introductory sections, topics, image links, etc. There may even be a new chapter or two.
- **Future error submissions**, corrections, requests for clarifications, etc. should be sent to me through the following page:

http://www.pediatricsboardreview.com/**ERROR**

HERE WE GO!

QUESTION/ERROR

Another mneumonic for remembering what protein is in breast milk versus what is in fomula: Breastmilk is WHEY better, and formula comes in a Case (or if you speak Yoda..."Case-in formula comes")

MY THOUGHTS

Uhh… I LOVE IT!!!!!! Thank you SC for submitting this!!!!

It made it into the next edition ☺

PBR MODIFICATION – Page 137

BREAST MILK

Breast milk contains arachidonic acid, DHA, whey, casein, colostrum, hind milk, etc. It's a lot to remember, so memorize the following and move on!

* ARACHIDONIC ACID (AA) & DOCOSAHEXAENOIC ACID (DHA): Help with neurologic development. Greatest in COLOSTRUM. Not as much in mature milk.

* WHEY: The primary protein in breast MILK.

* CASEIN: The primary protein in FORMULA.

* COLOSTRUM: The milk produced at the end of pregnancy and early after delivery. Only small amounts are expressed in the first few days until the more mature milk finally comes in.

- Yellow color is from carotene.
- Stimulates passage of meconium.
- High in PROTEIN (immunoglobulins, especially IgA).

* HIND MILK: Last bit of milk expressed during breast-feeding. It is highest in CALORIES and FAT.

* FROZEN BREAST MILK: Good for 3-6 months. Once thawed, use within 48 hours.

* CONTRAINDICATIONS TO BREAST-FEEDING: Mother with herpes simplex virus (HSV), HIV, tuberculosis (TB), on chemotherapy, on HYPERthyroid medications, on metronidazole, on sulfa drugs or on Tetracycline. Breast-feeding is also usually contraindicated if the baby has an **INBORN ERROR OF METABOLISM**. An inverted nipple may be a contraindication depending on the degree of inversion. Breast shells may be needed.

PEARL: Candidiasis, mastitis and fibrocystic disease are NOT contraindications.

PEARL: Breastfeeding is NOT a contraindication for Hepatitis B. For mothers who are CMV **carriers** (not recent converters), they may also breastfeed.

MNEMONICS:

- COLOSTRUM: Although it is supplied to babies very EARLY in life, it has tremendous LONG-TERM protective benefits/ingredients (AA, DHA, IG's/IgA aka protein).
- MATURE MILK is the regular, everyday milk that provides the regular, everyday ingredients to a baby (fat, lactose, "energy," etc.).

- HIND MILK: HIND milk has a high FAT and CALORIC content, like the unusually oversized be**HIND** of an appropriately overweight/fat new mom. (Sorry for the un-PC mnemonic. Hopefully it helps).
- WHEY: Breast milk is WHEY better, and FORMULA comes IN a CASE (CASE-IN). (Thanks to PBR member "**SC**" for submitting this one!)

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QUESTION/ERROR

FYI - On page 235 of PBR, it says an RBC lives 120 days or 3 months. Can you correct this? 120 days is 4 months, not 3 months. The Mnemonic doesn't make sense. It should be 120 days or 4 months.

MY THOUGHTS

I guess I need a refresher on basic multiplication and division. Thank you very much for the catch!

PBR MODIFICATION – Page 235 & 246

(DOUBLE TAKE) CELL LIFE SPANS

The red blood cell (RBC) has a life span of about 120 days (about 4 months). Platelets live for about 10 days.

MNEMONIC: Imagine an EGG carton (12) that has RBCs in it. Or, if you are worried that will make you think of 12 days instead of 120 days, then imagine a CAR (4) with the FOUR balloon-like RED BLOOD CELLS on the car instead of four wheels to remind you that RBCs live for 4 months.

MNEMONIC: Imagine going BOWLING (10) at a place that uses 10 PLATES instead of pins.

MNEMONIC: Bigger cell (RBC) = Bigger lifespan!

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QUESTION/ERROR

You can't answer question 7 (at least easily) on page 12, as the link to the image is wrong. There is no photo to look at.

MY THOUGHTS

Hmm… I'll have to go through all the links and will try to send out a modified file before the exam as well… BUT, this one seems to work for me.

It may have been temporarily down, or it could be a browser issue. For example, Safari acts up sometimes and give a bunch of random code and gibberish.

PBR MODIFICATION – None

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QUESTION/ERROR
Vacinnations: page 289 Flumist according CDC approved for healthy (no Asthma) two year old and above right ? Not 4 y.o ?

MY THOUGHTS
You're right! THANKS!

PBR MODIFICATION – Page 289

INFLUENZA VACCINATION
The CDC has made the influenza vaccination recommendations very easy. Give the "flu vaccine" to everyone over 6 months of age, every year. High-risk patients should get priority. The INHALED INFLUENZA (FLU) VACCINE (brand name Flumist®) can be given to anyone over the age of TWO, who is healthy and NOT PREGNANT, not IMMUNOCOMPROMISED and not an ASTHMATIC. It's delivered via a nasal spray.

PEARL: If you have a question about injectable flu vaccination, the answer will likely be to VACCINATE. For the inhaled vaccination, you will need to look at the age and comorbidities.

PEARL: Remember, influenzA is the virus. H. flu is the Gram negative organism and is spelled influenzAE (HIB vaccine).

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QUESTION/ERROR
Complement is misspelled several times as Compliment in the study guide

MY THOUGHTS
Wow… thank you CL!

Embarrassing to admit that I never actually realized that before.

PBR MODIFICATIONS – TONS of places

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QUESTION/ERROR
Hepatitis B: 2,4,6 months timing is ok right...if you looks at the schedules from CDC or catch up schedules: Time for the 3rd or last HEP B should be more than 6 months and 16 weeks from the 1st Hep B (not from the 2nd Hep B) as you referred ?

MY THOUGHTS
I think your question is somewhat unclear… but I ***think*** you are asking the following questions:

1. AGE for the 3rd Hep B shot should be at least 6 months of age. Correct?
2. 3rd Hep B shot should be at least 16 weeks from the 1st Hep B shot. Correct?

For #1, correct. As stated in the PBR "**Dose #3 must NOT be given before the age of 24 weeks (6 months).**"

For #2, correct. PBR says you have to wait 4 months from shot #2, but it's actually 4 months from shot #1. You only need to wait TWO months between shots #2 and #3.

THANKS to you and WN, DS and one more person.

PBR MODIFICATION – Page 290

HEPATITIS B VACCINE

Everyone gets the Hepatitis B vaccine. Start at birth and requires 3 doses.

PEARL: The last dose SHOULD NOT be given prior to 6 months of age. The IDEAL regimen for you to remember **is 0, 1 and then 6 months**. Here are a few reasons why:

- Dose #2 consideration: Time interval should be at least one month between dose #1 and dose #2.
- Dose #3 considerations: Time interval should be at least **four** months between dose #1 and dose #3. Time interval should be at least **two** months between dose #2 and dose #3. Does #3 must NOT be given before the age of 24 weeks (6 months).
- SUMMARY: This means 0, 2 and 6 months is allowed. 0, 1 and 6 is allowed. 2, 4 and 6 is allowed because four months have passed between doses #1 and #3, and 2 months have passed between doses #2 and #3. Also, 0, 2, 4 and 6 IS ALLOWED because 4 months passed between the 2-month shot and the 6-month shot. The 4-month shot was likely a combination vaccine and just went to waste (and is allowed by the CDC to do so). Four shots are sometimes seen in premie babies getting shot #1 before they weigh 2 KG.

PEARLS: If mom's Hepatitis B status is UNKNOWN, give the Hepatitis B **vaccine within 12 hours of birth**. Figure out mom's status, and if she is HBsAg POSITIVE, give the immunoglobulin (HBIG) no later than the 7th day of life. If the status is already KNOWN to be positive, give BOTH the vaccine and the HBIG at birth.

MNEMONIC: There are 3 intervals to remember. Between #1 and #2 is FOUR weeks. Between #2 and #3 is double that (EIGHT weeks). Between **#1** and #3 is double that (SIXTEEN weeks).

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QUESTION/ERROR

Multiple review and pearl pat about "BARKY cough" like on page 246 or other places: pick Croup and then most commonly from H. flu ???...is it most commonly from PIV (parainfluenza virus) ??

MY THOUGHTS
The 3rd edition does not have the word “bark” or “croup” on it. In other areas, I consistently state that Parainfluenza is the most common cause of croup and its barky cough.

Thanks WN for the review

PBR MODIFICATION - NONE

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QUESTION/ERROR
Can you give us more pertinent information on research, statistics and quality control?

MY THOUGHTS
The items that I feel are most high-yield for safety and research/stats is already in the book. Ethics is not covered and is 1% of the exam.

In general, my advice would be to avoid getting hung up on percentages from the content specifications. That *can* be a recipe for disaster because you start to look things up that are not high-yield. For example, the ABP Content Outline will mention that 1% of your exam is on ethics, and then give you a list of about 50 bullet points of potentially testable information. In reality, only a small percentage of those topics are probably frequently tested.

Your time will be much better spent learning the VERY high-yield material already in the PBR. I truly believe that if you know the PBR inside and out, and you have average testing skills... you'll pass without any problem.

PBR MODIFICATION - NONE… but I may consider adding an ethics section to the 4th edition. **Would anyone like to help me with this? It would be paid help.**

==========

QUESTION/ERROR
…check the rabies prophylaxis guidelines on the CDC website--what the book says is not current.

MY THOUGHTS
You’re right! It’s now RIG + 4 doses instead of 5.

Thanks WN for helping on this.

PBR MODIFICATION – Page 172 (top) and page 266

(DOUBLE TAKE) RABIES VIRUS

Rabies is caused by a VIRAL infection in which the virus is transmitted through bites, scratches and contact with mucous membranes of infected animals, such as BATS, dogs, foxes, **RACCOONS** (most common in US), skunks, and WOODCHUCKS. If the history suggests a possible exposure (wild/aggressive animal), treat with standard wound care **PLUS** Human Rabies Immunoglobulin (HRIG) **PLUS** the 4 vaccine doses. If the animal is a pet, observation of the patient and animal is allowed without giving HRIG.

PEARL: Rabbits, rats and squirrels (rodents) are **NOT** associated with rabies.

PEARL: **For the boards, if the word "BAT" is mentioned (alive, escaped, whatever!), treat as an exposure!**

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QUESTION/ERROR

…I don't think Bacterial Vaginosis is indicative of sexual abuse as you stated in your book. It is seen more often in kids who have been sexually abused/sexually active but is not an STD and shouldn't prompt and automatic referral. You should, however, test for CT, GC and Trich if you have a prepubertal kid with BV simply due to the association of higher rates in kids who have been abused. Not sure that was clear in your book.

MY THOUGHTS

Thanks for the question. The topic is included in the **Gynecology & Some STDs** section of PBR. I did not state that it is an STD and I did not include it in the SEXUAL ABUSE IN GIRLS topic… BUT, I can definitely see how the placement within the book might confuse someone. I've kept it in the same place but modified it some.

Thanks to GSO for helping with this lookup.

PBR MODIFICATION – p. 72

BACTERIAL VAGINOSIS (aka GARDNERELLA)

Bacterial vaginosis (Gardnerella vaginalis), has a Homogenous, smelly/fishy odor. Discharge can be white, yellow or **gray.** Associated with anything that changes the usual flora, including IUD's and antibiotic use. Look for CLUE CELLS. The pH is Alkaline and > 4.5. FYI… While Gardnerella is the most common organism associated with Bacterial vaginosis, it is not the only causative organism. Also, while it is associated with sexual intercourse, concerns for sexual abuse should be taken on a case-by-case basis.

MNEMONIC: "the GARDNER found a missing CLUE in the GARDEN. It was a dead and smelly FISH!"

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QUESTION/ERROR

page 41. Under "premature adrenarche in girls", there's a PEARL that says "In 8 year old or greater...". This is confusing, because after 8 y/o, it's no longer premature adrenarche, correct? Or, does this pearl refer to adrenarche that precedes thelarche?

MY THOUGHTS

You're right! It should say "or younger."

PBR MODIFICATION – p. 41

PREMATURE ADRENARCHE IN GIRLS

Premature adrenarche is defined as having hair development without breast development, or other signs of puberty prior to the **age of 8**. Get bone age films! If they are within 1 year of the chronological age, it's ok to observe at this age!

PEARL: In patients less than 8 years of age with hair development without breast development, look out for extra androgens in the form of exogenous androgens (oral? topical?), ANDROGEN-SECRETING TUMOR, CAH, or EARLY ADRENAL PUBERTY. ADRENAL glands are typically responsible for the ANDROGENS that result in ADRENArche. Remember that LH and FSH are gonadoTROPS and are from the pituitary, not released from the adrenals or gonads. However, elevation in LH and FSH **can** result in GONADAL androgen production. Ovaries ALSO PRODUCE TESTOSTERONE. If that's difficult to remember, "think of it this way: In CAH, excess progesterone results in excess androgens being formed. So, maybe the same holds true for excess progesterone floating around from ovarian production!"

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QUESTION/ERROR

discovered an error in Stats section page 335 , for specificity....It should read a confirmatory test should have a high "specificity"

MY THOUGHTS

You're right. On the second line of the SPECIFICITY section I wrote sensitivity.

Thanks to GSO for the lookup.

PBR MODIFICATION – p. 335

SPECIFICITY = TN/(TN+FP)

A high specificity [TN/(TN+FP)] is needed for CONFIRMATORY tests. These are tests that are done to RULE IN (aka CONFIRM) a disease. So, a confirmatory test should

have a high **specificity**, which will mean that a positive result rules in the disease in that patient.

MNEMONIC: sp**IN** & sn**OUT**. sp**IN** should remind you that **SP**ecificity (**SP**in) is related to ruling **IN** a disease.

==========

QUESTION/ERROR
Hypoglycemia treatment was confusing and there are some errors - doesn't make sense - 3 cc/kg, then end up giving 5 cc/kg. You should review it.

Also…

On page 66 the hypoglycemia stuff is inconsistent. You say 3 cc/kg, but then in your mnemonic you end up giving a formula that end up giving 5 cc/kg? Which dose is correct?

MY THOUGHTS
You're right. It's confusing and contradictory. When my ER friend told me the mnemonic, I loved it. I'll have to yell at him.

The recommendations are a little all over the place, but I'd recommend the 3 cc/KG of D10 for neonates and young children. If profound hypoglycemia, then also start a dextrose drip. For adolescents, I feel 1 ampule of D50 should be used. One "amp" of D50 generally means giving the entire container of D-glucose 50%. As an FYI… it usually comes in a 50 ml container. Since percent solutions refer to grams/100ml, D50 refers to 50 grams of dextrose per 100 ml water. So a 50 ml "amp" of D50 yields 25 grams of dextrose.

Thanks to GSO for help w/ the lookup.

PBR MODIFICATION – p. 66

HYPOGLYCEMIA
Hypoglycemia is generally considered to be a glucose level < 60. Give 15 grams of fast acting carbohydrates (corn syrup, crackers, juice, sugar, soda) and recheck in 15 min if minimally symptomatic. If still < 60, repeat carbohydrate bolus. If more emergent, give a dextrose bolus via IV and consider a drip. For neonates and young children, give 3 ml/kg of D10. For adolescents, simply give 1 ampule of D50 (25 grams of dextrose).
PEARL: Percent solutions refer to grams/100 ml. For example, D50 refers to 50 grams of dextrose per 100 ml water. So a 50 ml "ampule" of D50 contains 25 grams of dextrose.

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QUESTION/ERROR

Pg 180 - B2 def - PEARL - mispelling -
should be B2 and not B1

MY THOUGHTS
p. 179 in the 3rd edition and you're right! Thanks so much for pointing it out.

And thanks CTM too.

PBR MODIFICATION – p. 179

RIBOFLAVIN (B2) DEFICIENCY

Riboflavin (Vitamin B2) deficiency can result in anemia, angular stomatitis, cheilosis, glossitis ("tongue = riboFLAVor") and seborrheic dermatitis.

PEARL: Phototherapy can result in decreased riboflavin (**B2**) levels. Keep this in mind with premature hyperbilirubinemia children.

MNEMONIC: Imagine this drawing represents a premature baby with protective glasses on (for phototherapy). The left lens have "2" written on them to remind you of the B2 deficiency. There is a rash at the scalp (seborrheic dermatitis), and there are steep angles at the edges of the lips to remind you of angular stomatitis. He's bleeding from the angles of his mouth to help you remember of the possible anemia.

==========

QUESTION/ERROR
On page 39, "Mammography is needed until patients"
Should be : Mammography is NOT needed until...

Do you mean a mammogram is NOT needed with teens because of dense breast tissue? That's why we do the u/s right?

MY THOUGHTS
Thanks for catching this!

And thanks to GSO too.

PBR MODIFICATION – p. 39

BREAST MASSES – FIBROADENOMAS AND FIBROCYSTIC DISEASE

Breast masses in children are usually benign and due to fibrocystic disease or fibroadenomas. Check the masses at end of menstrual periods. Mammography is NOT needed until patients are much older since their breast tissue is dense. To evaluate, use ULTRASOUND. Excisional biopsy is almost never indicated (only if aspirate is bloody).

* **FIBROCYSTIC DISEASE** is the most common breast disease and is usually bilateral and tender. OCPs may help.

* **FIBROADENOMAS** are unilateral and ESTROGEN dependent (bigger with OCPs/pregnancy). Refer to gynecology if there is bloody aspirate or if it persists beyond after 3 cycles

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QUESTION/ERROR

On page 103 of the 3rd edition it states that TOF has "widely split S2, a single S1 and possible a click." On the table on page 105 it states that TOF has a "Single S2 because PV component absent."
So which is it for TOF: a widely split S2 OR a single S2???

MY THOUGHTS

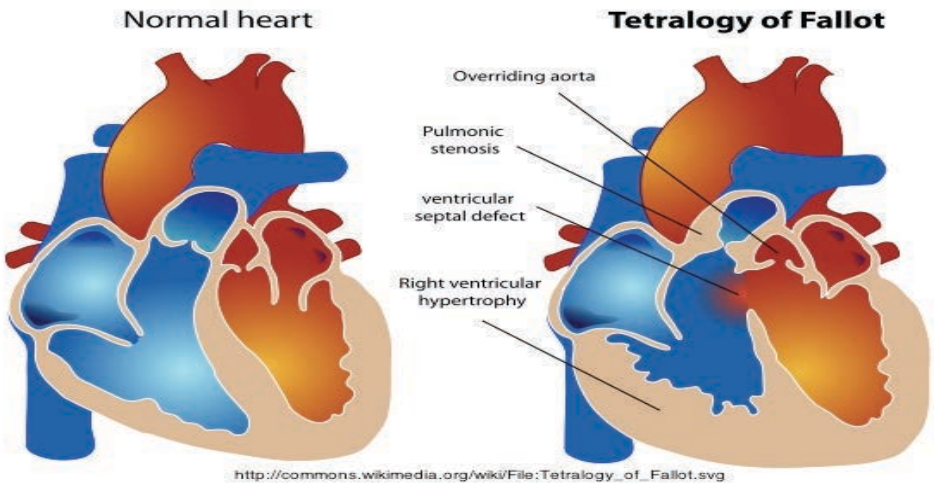
Great question, complicated answer. S2 is made up of A2/P2. In Tetralogy of Fallot, there actually is a wide split of S2... BUT, the P2 component is usually so soft that it's EXTREMELY difficult to hear. Since it's RARELY audible, you'll only hear a single S2. I'll update the book for the next edition. Thanks for asking.

PBR MODIFICATION – p. 103 and table on 105

TETRALOGY OF FALLOT (TOF)

Tetralogy of Fallot is the most common cardiac cause for cyanosis in children (of any age). The four primary findings include PULMONARY STENOSIS (PS), RVH, OVERRIDING AORTA and a VSD. EKG shows RAD consistent with RVH. Exam reveals a **PS murmur** (harsh LUSB murmur), a widely split s2, a single S1 and possibly a click. Note that the P2 component is SO soft that it simply sound like a single S2. There is a RIGHT **to LEFT** shunt at the VSD because blood cannot flow to the lungs due to the PS. Therefore there is NO pulmonary hypertension or evidence of congestion on the CXR (look for a **clear CXR**). That also means giving **PGE** will result in a PDA shunting blood from the LEFT to the RIGHT (from the Aorta to the PA, with no concerns for directional change from pulmonary hypertension). **CXR** findings include a lack of vascular congestion, BOOT shaped heart and RVH.

* **MNEMONIC**: "you have to **PROV**e it is a TET!" = **P**S, **R**VH, **O**verriding aorta, **V**SD!



* **TET SPELLS:** Episodes of cyanosis (precise mechanism is uncertain) possibly occurring at times of relative anemia or increased pulmonary vascular resistance. An acute increase in R **to LEFT** shunting occurs which results in cyanosis, possible syncope or even death. Older children squat, younger children's knees are forced to the chest, in order to increase systemic vascular resistance and decrease/reverse the flow of the shunt. Other possible treatments include **morphine**, phenylephrine, propranolol and volume.

CHD	Murmur	Shunt	Notes
TOF	Pulmonary stenosis	R → L at VSD	Shunt at VSD, more as PS worsens. Single S2 because P2 is soft/inaudible. No increase in pulm vasculature on X-ray.
TGA/TOGA	NONE	R → L at ASD or VSD.	Cyanosis within hours. Single S2, Egg shaped. Pulm congestion from LV → PA
Tricuspid Atresia	Possible systolic. Possible PS sounding murmur	R → L at ASD L → R at a VSD and PDA	Blood goes from RA to LA to LV to RV to PA (with a possible PS murmur) to lungs to LA/LV to Aorta to the Periphery
Truncus	Possible systolic	R → L	PA & Ao are joined. Connected to RV & LV
TAPVR		L → R at anomalous vein R → L at ASD	Snowman (to me looks like a balloon)

==========

QUESTION/ERROR
Pg 126 – SSSS. Line 6/7 ... Obtain a BIOPSY to prove that it is SSSS and NOT SJS or TEN. You have to add NOT

MY THOUGHTS
Thanks to you and to GSO!

PBR MODIFICATION – p. 126

BULLOUS IMPETIGO/STAPH SCALDED SKIN SYNDROME (SSSS)
Bullous impetigo, or Staph Scalded Skin Syndrome (SSSS), is a spectrum of the same disease.

* **IMPETIGO**: Look for honey colored, crusting lesions and bullae. Non-bullous impetigo and look similar but without vesicle/bullae (more oozing/crusting)

- **IMAGE**: http://bit.ly/rmVh8u

*** SSSS**: A very painful and red rash in which large, thin blisters are the result of an exotoxin. There is "**sheet-like"** skin loss/separation. Looks very superficial compared to impetigo. Obtain a BIOPSY to prove that it is SSSS and NOT Stevens Johnson Syndrome (SJS) or Toxic Epidermal Necrolysis (TEN), both of which have deeper/dermal involvement.

- **IMAGES**: http://emedicine.medscape.com/article/788199-media

PEARL: Lesions are **NOT** in eye or mouth, but may be **around** the eyes and mouth (as opposed to SJS/TEN, which may be IN the eyes and mouth).

==========

QUESTION/ERROR
you say that the treatment for CAH is combination of hydrocortisone and fludrocortisone, i.e. coverage for both glucorticoid and mineralocorticoid effects.

The 5th edition of Laughing your way to passing the pediatric boards states that hydrocortisone alone is treatment enough as it will have mineralocorticoid effect in high enough doses.

So... now confusion sets in (especially at 4 AM on a PICU call).

Any idea which one is the correct choice if both are given?

I guess that is why you say not to use other resources, lol

MY THOUGHTS
I agree… you should stop looking at other resources ☺

I stick to my recommendation of using both forms of steroids. **UpToDate** has comprehensive reviews of this and concurs.

PBR MODIFICATION - NONE

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QUESTION/ERROR
On page 33 (or 62?), the diagram associate with "21- hydroxylase related congenital adrenal hyperplasia," I believe that 18- hydroxyprogesterone converts to aldosterone via the enzyme 18-oxidase and not 11-hydroxylase. Otherwise it would mean that 11-hydroxylase deficiency is salt wasting, which is not the case.

MY THOUGHTS
Thanks for catching the typo!

And thanks to CTM for looking it up.

PBR MODIFICATION – p. 62

21-HYDROXYLASE DEFICIENCY RELATED CONGENITAL ADRENAL HYPERPLASIA

PROGESTERONE → **17-HYDROXYPROGESTERONE -- -- -- > ADRENAL ANDROGENS**

⊗↓ (21-hydroxylase) ⊗ ↓ (21-hydroxylase)

DEOXYCORTICOSTERONE 11-DEOXYCORTISOL

↓ ↓ (11-hydroxylase)

CORTICOSTERONE CORTISOL ⊗

↓

18-HYDROXYPROGESTERONE

↓ (18-hydroxylase)

ALDOSTERONE ⊗

==========

QUESTION/ERROR

On page 41, under treatment options for PID inpatient, one of the alternatives you mention is Augmentin (Ampicillin/Sulbactam). I think you meant Unasyn instead of Augmentin.

MY THOUGHTS

Absolutely correct. I wrote the right generic components but wrong trade name. Thanks!

And thanks GSO for the reference.

PBR MODIFICATION – p. 71

PELVIC INFLAMMATORY DISEASE (PID)

Pelvic inflammatory disease (PID) can result in **potentially serious outcomes** (sepsis, infertility) so it should be **treated on the basis of your clinical diagnosis.** If there is no improvement on appropriate outpatient therapy and cultures are negative, obtain an ultrasound to evaluate for a tuboovarian abscess. Also obtain an RPR to look for Syphilis and consider getting HSV PCR (not HSV titers since they are nonspecific). Treatment options depend on the setting:

* **OUTPATIENT**: Ceftriaxone (or Cefoxitin) x 1 + (Doxycyline BID x 14 days or AZITHROmycin x1) +/- Metronidazole (optional)

* **INPATIENT**/IV: Cefotetan (or Cefoxitin) + Doxycycline. Alternatives: Clindamycin + Gentamicin, or Unasyn (Ampicillin/Sulbactam) + Doxycycline. Inpatient therapy is reserved for patients who are pregnant, have intractable nausea and vomiting, high fever, or for those who failed outpatient therapy.

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QUESTION/ERROR
Is 37 wks is preterm or term .

MY THOUGHTS
37 weeks, believe it or not, is preterm. Term starts on or after the 38th week. Anything before the 38th week is preterm. This is defined by AAP and ACOG.

Thanks to CTM for looking this up. Annoying topic that confuses many… meaning you'll hopefully never have to make that distinction on the boards.

PBR MODIFICATION - None

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QUESTION/ERROR
On page 235 of PBR....there is the word "Marfan" under cord compression. Is that there by mistake?

MY THOUGHTS
You're right. Thanks to you and to GSO. I deleted Marfan.

PBR MODIFICATION – p. 235

CORD COMPRESSION
Treat cord compression with IV steroids +/- radiation therapy

==========

QUESTION/ERROR
on pg 307 gaucher dx you mention short stature. I came across board study question in which the answer was gaucher but the mentioned nothing of short stature. It was a 5 yo with recent onset of symptoms. Is short stature a requirement?

MY THOUGHTS
You're right. It should say "can have."

Thanks GSO for the help.

PBR MODIFICATION – p.307

GAUCHER DISEASE (aka GAUCHERS DISEASE)

Patients with Gaucher disease (aka Gauchers disease) often have **hepatomegaly**, thrombocytopenia, easy bruisability, **osteosclerosis and lytic lesions with bone pain**. They may also have short stature.

MNEMONIC: Rename it OUCHers disease since patient complain of bone pain.

MNEMONIC: Rename it GROUCH-ers disease. Imagine some GROUCHY old lady complaining of bone pain and easy bruising. She's so dramatic about it all that she starts wrapping her arms and legs in TOILET PAPER (TP = Thrombocytopenia) to show you how much pain she has!

==========

QUESTION/ERROR

What is the normal age for the first ophtho exam? I don't think it's in your book.

MY THOUGHTS

Great question. I'll add something to the book. You get a gold star!

According to the AAP, you should do routine eye exams whenever you see the child and s/he lets you. In terms of more **formal** vision screening, "Formal vision screening evaluations should begin at 3 years of age."

PBR MODIFICATION – New topic added to p. 206

VISION SCREENING

The AAP recommends that all infants be examined by 6 months of age for fixation preference, ocular alignment and the presence of any eye disease (by pediatricians). Formal vision screening evaluations should begin at 3 years of age.

==========

QUESTION/ERROR

What's the normal age for first dental exam?

MY THOUGHTS

Looks like the recommendations have changed.

6 months after the eruption of the first tooth, or by 12 months of age (whichever comes first).

Thanks WN.

PBR MODIFICATION – p. 380

FIRST DENTAL EXAM

The first visit to the dentist should be either 6 months after the eruption of the first tooth, or by 12 months of age (whichever comes first).

==========

QUESTION/ERROR

How many parts per million of fluoride are recommended by the CDC or AAP before we have to think about supplementation?

MY THOUGHTS

Actually… this is my own question to myself. Thanks to WN for the lookup.

PBR MODIFICATION – NEW PBR TOPIC

FLUORIDE SUPPLEMENTATION

If the local water supply contains less than 0.6 parts per million of fluoride, oral supplementation is recommended.

==========

QUESTION/ERROR

Kernicterus in biliary atresia in ur book pg 185. I thought its only increase direct bili. Please clarify

MY THOUGHTS

Technically my current explanation is correct, but I should expand.

Thanks SO MUCH to CTM for the extremely thorough explanation and lookup on this topic!

PBR MODIFICATION – p.185

BILIARY ATRESIA

In cases of biliary atresia, look for an elevation in the direct bilirubin (aka conjugated bilirubin) in a neonate. If found, obtain an abdominal ultrasound followed by a HIDA scan. If left untreated, can result in liver failure. Until the liver fails, conjugation of bilirubin continues. KERNICTERUS only occurs once the liver fails and indirect bilirubin (aka unconjugated bilirubin) can no longer be conjugated (only unconjugated bilirubin can crossing the blood-brain barrier).

==========

QUESTION/ERROR
Is double bubble sign in antral web? Yes or no

MY THOUGHTS
No. As mentioned in the DOUBLE BUBBLE topic, that sign is associated with duodenal obstruction. The antrum is part of the stomach.

Thanks GSO for looking it up.

PBR MODIFICATION - None

==========

QUESTION/ERROR
clarify what's the best test to dx PWS on pg 223

MY THOUGHTS
As mentioned under PEARLS and MNEMONICS sections, "The gene location is probably not important for the test, but the fact that it is KNOWN (15q11-13) means that this disorder can be diagnosed by FISH."

So the answer to your question is FISH. For review, here's the topic:

PRADER-WILLI SYNDROME (aka PRADER WILLI SYNDROME)
Prader-Willi syndrome (aka Prader Willi syndrome) patients can have hypotonia (floppy baby), mild retardation, almond shaped eyes (often with mild strabismus), small hands, a HUGE appetite, obesity and small testicles/penis in boys.

* **PEARLS:** Like Angelman's, it can also be found in BOYS or GIRLS. This disorder occurs due to MATERNAL DISOMY with MATERNAL IMPRINTING. That means Dad's genes have been deleted and the patient has two of mom's copies of this particular gene. Symptoms are much milder in females. The gene location is probably not important for the test, but the fact that it is KNOWN (15q11-13) means that this disorder can be diagnosed by FISH.

* **IMAGE**: http://www.aafp.org/afp/2005/0901/afp20050901p827-f1.jpg

* **IMAGE**: http://www.nature.com/ng/journal/v40/n6/images/ng.158-F1.jpg

* **MNEMONIC:** Imagine a FAT Will Smith with TINY HANDS shoving tons of ALMONDS in his mouth. He's wearing a t-shirt that says, "MOMMY'S LITTLE FATTY."

- **KEY:** ALMONDS represent the shape of the eyes and the t-shirt represents maternal imprinting.

* **MNEMONIC:** Sorry, this is a good mnemonic, but not politically correct. FISH + WILLY = _____ WILLY! Imagine a HUGE, OBESE and DUMB FISH/WHALE named WILLY with such a SMALL PENIS that you can hardly see it. It's so DUMB that it tried to jump

over a dock, but ended up landing on it instead. Now he's stuck on the dock, HUNGRY and just FLOPPING around.

* **MNEMONIC IMAGE**: http://www.imdb.com/media/rm2549784064/tt0106965

KEY: FISH represents the mode of diagnosis, HUGE/OBESE represents obesity, DUMB represents mental retardation, HUNGRY represents the insatiable appetite and FLOPPING represents the hypotonia. In this mnemonic, you could also make Willy's eyes almond shaped and imagine that he has small hands/fins.

PBR MODIFICATION - NONE

==========

QUESTION/ERROR
According to the CDC it can be either 0,1,6 or 0,2,6

MY THOUGHTS
Either is fine. You do need a 6 mo interval between dose #1 and #3.

Thanks SS for looking it up!

PBR MODIFICATION – p.290

HUMAN PAPILLOMA VIRUS VACCINE (HPV)
The HPV vaccine requires 3 doses starting at 11 years of age. Dose #1 and dose #3 should be separated by 6 months. Dose #2 can be given at 1-2 months after dose #1. Therefore, 0-1-6 OR 0-2-6 month schedules are acceptable. It's recommended for **males** and females and can be administered as early as 9 years of age.

==========

QUESTION/ERROR
Meningitis vaccine at 11 then booster at 16 (yes or no).

MY THOUGHTS
Yes ☺

PBR MODIFICATION – Slightly clarified recap below from p.290

MENINGOCOCCAL VACCINE (aka MENINGOCOCCUS VACCINE)
The meningococcal vaccine is recommended at 11 years of age for ALL kids, NOT just those starting at college and living in a dorm. Requires a booster. If started on time at 11 years of age, the booster is given at 16 years of age. If given later than 11 years of age, the booster can be given approximately 3 years later. For unimmunized freshman **living in a dorm**, give one shot and no booster.

PEARL: Minimum age is 2 years. Children < 11 year of age may receive the vaccine if they are at high risk (asplenic, **complement deficiency**, generally immunocompromised or traveling to an endemic area).

PEARL: If a CLOSE INTIMATE contact of your patient has meningitis, give your patient prophylaxis with **rifampin** or ceftriaxone within 24 hrs of diagnosis **regardless of the immunization status**. Close "intimate" contact includes nursery school, but **not** elementary school classmates or healthcare providers.

PEARL: Subtype B is Bad for Babies and has no vaccine. Most meningococcal meningitis cases in babies is due to this subtype.

==========

QUESTION/ERROR
pg 291 VZV exposure. Immunoglobulin (pl clarify who need this

MY THOUGHTS
It's for people at high risk for complications. Pregnant, immunocompromised and some infants.

Thanks to GSO for the reference!

PBR MODIFICATION – p.291

POSTEXPOSURE PROPHYLAXIS
The following discusses postexposure proplylaxis for N. meningitidis, H. influenzae B, pertussis, Hepatitis A, Hepatitis B, Varicella and measles.

* RIFAMPIN PROPHYLAXIS: It's used for both **N. meningitidis** (aka meningococcus) and **H. flu** prophylaxis. Once prophylaxis is started, reddish-orange urine and tears may be noted.

- N. MENINGITIDIS: Only 1 case of exposure is required to start prophylaxis in contacts.
- H. INFLUENZAE B: TWO documented cases are required. If the immunization status is unknown, also give the Hib vaccination. If you are presented a question about the first child to be diagnosed the H. flu (the index case), then be sure to also immunize his/her household members.

* PERTUSSIS: Give a macrolide.

- **PEARL**: Give azithromycin for kids < 6 weeks of age due to concern for pyloric stenosis. For those > 6 weeks of age, give erythromycin. If the patient is unimmunized, also give the vaccine.

* HEPATITIS A: Given Hepatitis A immunoglobulin to the unimmunized **family members** only.

* HEPATITIS B NEEDLE STICK:

- IMMUNIZED: If the patient's immunity status is known to be up to date by titers, no prophylaxis is needed.
- UNCERTAIN IMMUNIZATION STATUS: CHECK for antibody. If negative, give the immunoglobulin (HBIG) **and also start** the full vaccination series. If the antibody is positive, do nothing.
- UNIMMUNIZED: Give the immunoglobulin (HBIG) **and also start** the full vaccination series.
- **PEARL/SHORTCUT:** CHECK FOR IMMUNITY. Then either do nothing, or give **both** the immunoglobulin (HBIG) and the Hepatitis B dose #1 of 3.

* VARICELLA (VZV): Give the immunoglobulin to individuals who are at "high risk" for complications, including those who are pregnant, immunocompromised and some infants.

* MEASLES: For an exposure less than 72 hours ago, give the vaccine to unimmunized patients who are **at least 6 months old**. For exposures greater than 72 hours ago, give the immunoglobulin (MIG) and then DO NOT GIVE THE MMR for at least 5 months after that.

==========

QUESTION/ERROR
pg 135. Mid parental height . Is it +or - (5 cm or 5 inches).

MY THOUGHTS
The adult height is predicted to be "within 5 cm" or "within **2 inches**" of the mid-parental height.

Thanks to CTM for the support on this one.

Please note that my formulas are easier to remember and result in the same result when compared to formulas that specifically try to force you to remember which parent's height the 5 inches (or 13 cm) should be added to or subtracted from.

PBR MODIFICATION – p.135 clarified

NEWBORNS LENGTH
Here are some great shortcuts to help you calculate the expected length for newborns:

* 1-year-old: 1.5x the birth length

* 4-year-old: 2x the birth length

* 13-year-old: 3x the birth length

* **Mid-parental height**: Expect the adult height to be within 5 cm (or within 2 inches) of the mid-parental height. Can be shorter OR taller. The mid-parental height is calculated as follows:

  - Boys = (Mom's Height + Dad Height **+** 5 inches)/2
  - Girls = (Mom's Height + Dad Height **-** 5 inches)/2
  - NOTE: If using centimeters, replace "5 inches" with "13 cm" in the formulas above.

* **PEARL**: 50th Percentile for LENGTH at birth is 50 cm.

* **PEARL/EXAMPLE:** The most average baby you can find would be 50 cm in length at birth and by 1 year of age s/he would be 75 cm. By 4 years old, s/he would be about 100 cm (2 x 50), and by 13 s/he would be 150 cm (3 x 50). 150 cm / 2.5 = 60 inches = 5 feet.

==========

QUESTION/ERROR

page 365, HSP: it says not to treat with steroid monotherapy; however, a few lines down it says, "...for severe sxs ...or for a hospitalized patient, give steroids with or without other steroids."

So are there times to treat with steroid monotherapy?

MY THOUGHTS

It looks steroid use as monotherapy should be fine. In patients without renal disease, but sure to also give NSAIDS.

PBR MODIFICATION – p.364-365

HENOCH SCHONLEIN PURPURA (HSP)

Henoch Schonlein purpura (HSP) is a vasculitis that can involve **multiple systems**, including the skin, joints, GI tract and kidneys. Classic findings include **PALPABLE** and **TENDER** purpura that **blanch.** These are most often found at the **lower extremities and buttocks**, but may be elsewhere. There may also be **peri**articular joint involvement (soft tissue only) at the knees or ankles, and a faint rash. Patients may initially present with colicky abdominal pain +/- blood in stool +/- **intussusception +/-** gallbladder hydrops. Skin findings are impressive, but the labs show a **normal platelet count**. Urinalysis will likely show hematuria +/- proteinuria which can range from mild to nephrotic range (**order a spot protein to creatinine ratio**). This may be diagnosed on clinical findings. Complement levels can be low. A **biopsy may be obtained if there is doubt** about the diagnosis. Biopsy would show IgA, IgG and C3 deposits. The disease

often resolves without intervention and the use of medications is debatable, meaning it's unlikely to be tested. For the boards, give NSAIDS for joint pain in the **absence of renal disease**, and for severe symptoms (can't eat) or for a hospitalized patient, give steroids with or without other therapies (i.e., cyclophosphamide, azathioprine, plasmapheresis, IVIG, etc.).

IMAGE: http://bit.ly/nsNXZo

==========

QUESTION/ERROR
Do you have more ENT topics to share with us?

MY THOUGHTS
Not at this time.

Again, there's more than enough info in the study guide and Q&A book to help you pass. Avoid letting your nerves and fears drive you into looking for other topics to study.

PBR MODIFICATION - None

==========

QUESTION/ERROR
Do you have more preventative medicine topics to share with us?

MY THOUGHTS
It's interspersed in the book. I may consider adding some more info to the 4th edition.

PBR MODIFICATION – None at this time

==========

QUESTION/ERROR
The PBR states that Serum Sickness is treated by anti-venom…I have never heard of this…can you explain anti-venom to what? Or expand on this concept? Thanks

MY THOUGHTS
Thanks for bringing my attention to this topic because there was an error in it, **and** it warrants further explanation :-)

PBR MODIFICATION – p.78

SERUM SICKNESS
Serum sickness results in rash, fever, polyarthritis (or polyarthralgias) and possibly nephritis within 1-2 weeks of exposure to certain non-human agents (**cefaclor**,

minocycline, amoxicillin, anti-venom). Treat by removing the inciting agent, antihistabmines for prutitis or rash, NSAIDS for fever, and glucocorticoids for high fever, severe rash or arthritis.

==========

QUESTION/ERROR

There's some vagueness between pages 37 and 38 regarding the basal growth rate in males (37 says 6-8cm and 38 says 5-6cm for both genders)

MY THOUGHTS

It should be 5-6 cm/year for SMR1 on page 37.

Thanks GSO for the lookup!

PBR MODIFICATION – p.37

SMR	Girls Delayed Puberty: 13 – 14 yo Precocious Puberty: 2' signs before 8 yo	Boys Delayed Puberty: 14 – 15 yo Precocious: 2' signs before 9 yo
1	**Basal growth at 5-6 cm/yr**, boyish chest (papilla elevation only), no hair	< 4 ml volume or 2.5 cm diameter of testicle, no hair, baby penis, **basal rate of 5-6 cm/yr**, no hair
2	**Accelerated growth at 7-8 cm/yr**, a breast bud is the 1st sign of puberty (palpable, areola enlarges), Hair only along the labia (coarse)	**>4 ml or >2.5 cm** (this is the 1st sign of puberty), hair at base of penis. Penis *may* start to enlarge (usually at SMR 3)
3	**PEAK ht velocity of 8-10 cm/yr**, elevation of breast contour, areola enlarges, curly hair at pubis, axillary hair begins, acne. This stage is similar to a boy's SMR 3 + 4 combined. "Imagine a girl sitting on a 3-LEGGED STOOL crying because she has hair in her armpit and now has acne!"	**Accelerated vertical (and penile) growth** >12 ml/3.5 cm, Gynecomastia in 50% boys 10-16 yo, resolve in 3 yrs), CURLY hair at pubis. "Think about the 3 Stooges. They all had funny pubertal voices, and the fat one had BOOBS/GYNECOMASTIA and was named CURLY."
4	**Mound on mound**, enlarged areola. Dense hair, none at the thigh. Menses usually occurs around SMR 3 or 4.	**PEAK height velocity at 10 cm/yr**, no thigh hair, develops **AX**illary hair, acne, and body odor. "Teenage boy with raging hormones is pissed about acne & hair so takes an **AX** to his **4 DOOR CAR** (SMR **4**) which explodes and burns his hair!"
5	Stop growing at about 16 yo, areola recesses to general contour of breast and the **breasts again look like tanner 3**	>4.5 cm penis, thigh hair, stop growing at ~17 or 18, +facial hair at sides, no more gynecomastia

==========

QUESTION/ERROR
p43, just wanted to clarify the decelerated growth rate in GH deficiency: is to the point of crossing lines or not crossing lines?

MY THOUGHTS
Yes. It crosses line for height.

PBR MODIFICATION – updated page 43 to clarify

GROWTH HORMONE DEFICIENCY
Short stature from **growth hormone deficiency** is rare, but findings will include a micropenis or clitoris and hypoglycemia (seizures may be the only clue that a patient has hypoglycemia). There is **DEcelerated growth rate** to the point of **crossing lines**. Diagnosed by seeing a lack of GH release following insulin or arginine stimulation.

==========

PBR FACEBOOK CREW POST
https://www.facebook.com/groups/pedsboardreview/permalink/315473435265271/https://www.facebook.com/groups/pedsboardreview/permalink/315473435265271/
On page 268 regarding Lyme Disease, it says it's hard to diagnose using labs and to check Lyme antibody titers. A friend who is a pedi ID doc told me this: "A patient with erythema migrans does not need lyme serology. He said to treat the patient. Erythema migrans is pathognomonic for the disease." Comments?

MY THOUGHTS
I agree! The PBR states that diagnosis is often difficult by labs and that the diagnosis is often clinical… Still, if it's not a slam dunk case, you should still know how to test for it on the boards ☺

Thanks GSO for the input and reference.

PBR MODIFICATION – pp.129, 265

(DOUBLE TAKE) ERYTHEMA CHRONICUM MIGRANS
Erythema chronicum migrans (aka erythema migrans) is caused by BORRELIA BURGDORFERI, the spirochete that causes LYME DISEASE. Look for a large, flat lesion **(> 5 cm) that is annular and has a red border. It is located at the tick bite site in about 75% of patients. Classis description is a "bullseye" lesion.** The rash shows up 1-2 wks after the bite. Titers may still be negative during this period. Borrelia is transmitted via the Ixodes deer tick. IF the patient has an acute arthritis, disseminated erythema migrans, a palsy (BELL'S PALSY) or neuropathy, then treat with ORAL

medication (**Doxycycline if >8 years old, or Penicillin or Amoxicillin if < 8 years old**). If the patient has **CARDITIS**, neuritis (encephalitis/meningitis) or **RECURRENT** arthritis, then treat with INTRAVENOUS medication **(PCN or Ceftriaxone)**. Arthritis is usually located at the large joints (especially the **knees**). Diagnosing using labs is often difficult. Obtain **Lyme antibody titers**. If positive, confirm with a Western blot. Lyme Disease is often a **clinical diagnosis** (for example, if you see erythema migrans – TREAT).

* **IMAGE**: (BULLSEYE LESION) http://web.princeton.edu/sites/ehs/bullseye.jpg

* **IMAGE**: (BELL'S PALSY) http://en.wikipedia.org/wiki/File:Bellspalsy.JPG

* **SIDE NOTES**

- BELL'S PALSY: Unilateral facial nerve paralysis (CN VII). Often idiopathic.
- The Jarisch-Herxheimer reaction results in fever, chills, hypotension, headache, myalgia, and exacerbation of skin lesions during antibiotic treatment of a bacterial disease (typically spirochetes). Due to large quantities of toxins released into the body. Classically associated with syphilis, but can also occur with Lyme disease. May only last a few hours

* **MNEMONICS:**

- From now on, think/say borreLIYME. "Don't ever throw a borLIYME to MY GRANy!" Or, "Don't ever borre-LIE to MY GRANy." Borrrelia = borreLIYME. MY GRANy = Migrans.
- Imagine that BULL'S EYES are made of 2 bright neon-green LIMES! This should remind of you of the classic description.
- Imagine squeezing LYME into a CAN = **C**arditis, **A**rthritis, and **N**euritis.

==========

QUESTION/ERROR

Correction needed? Page 345 of the PBR under First-Time seizure, mentions a "non-focal (partial)" seizure. I believe this should be "focal (partial)", as on page 346, it says: "partial seizure: It's focal in location".

MY THOUGHTS

Thanks for catching that! The error is actually the **opposite** of what you said. It should read as follows below…

Thanks for the input GSO.

PBR MODIFICATION – p.345

FIRST-TIME SEIZURE

If presented with a **non-focal (aka generalized)** first-time seizure in a child who is **greater than 1 year of age** and **otherwise healthy**, no further workup is indicated as long as the seizure was less than 5 minutes. Discharge from the ER with the above mentioned in this section.

PEARLS: After a single seizure, the **chance of future epilepsy is > 30%**. If the child has any neurologic issues (hyperreflexia, cognitive impairments, etc.), the chances are much higher. Diagnosing a seizure is better done with a good **history** rather an EEG.

==========

PBR FACEBOOK CREW POSTS

https://www.facebook.com/groups/pedsboardreview/permalink/318301274982487/

https://www.facebook.com/groups/pedsboardreview/permalink/332082590271022/

Page 366 of PBR says in CF you have a hypochloremic metabolic ALKALOSIS, then 5 lines below the acid-base issues in CF, it says you have a hypochloremic metabolic ACIDOSIS in the mnemonic section.

Also address in this PBR Facebook Crew Post and reference image –

MY THOUGHTS
Thanks guys… I *meant* alkalosis. Grr.

PBR MODIFICATION – p. 366-367

CYSTIC FIBROSIS (CF)

Cystic fibrosis (CF) is an autosomal recessive disorder that **will be on your test**. Children who are carriers of the cystic fibrosis gene are asymptomatic. It result in excessive loss of chloride through sweat and is associated with significant pulmonary, electrolyte and gastrointestinal problems. The The diagnosis is made through a sweat chloride test showing a level of greater than or equal to **60 mEq/L**. A list of the many associations with this disease are listed below by category.

* **MNEMONIC**: If **sweat test >/= 60 meq = "6-tic fibrosis" =** diagnostic.

* PULMONARY SYMPTOMS: Diffuse cysts/fibrosis (hence the name), bronchiectasis, hemoptysis, pneumonias (Staphylococcus aureus, Pseudomonas, Haemophilus influenzae), chronic sinusitis, nasal polyps, pneumothorax, Burkholderia cepacia noted in sputum cultures, Pseudomonas pneumonias (and colonization).

- **PEARLS:** If Burkholderia cepacia is mentioned, go to the answers and pick cystic fibrosis!

* GASTROINTESTINAL SYMPTOMS: Pancreatitis, cholelithiasis, neonatal cholestasis, meconium ileus, rectal prolapse, chronic diarrhea, malabsorption with steatorrhea, fat soluble vitamin deficiencies (A, K, E, **D**), hypoproteinemia.

- **PEARLS:** Regarding the fat soluble vitamin deficiencies, look for problems with proprioception, ataxia, vision, coagulation, hypocalcemia,

* ACID-BASE ISSUES: Hypochloremic Metabolic Alkalosis. So a low chloride and high bicarbonate level.

* ELECTROLYTE ABNORMALITIES: Hyponatremia, Hypokalemia, Hypochloremia, Hypocalcemia. Sodium and chloride are lost in the sweat. Potassium can be lost in stool. Calcium can be low due to a vitamin D deficiency from fat malabsorption.

- **MNEMONIC**: All of the lytes are low, except for bicarbonate. This makes sense given the finding of hypochloremic metabolic alkalosis.

* ENDOCRINE SYMPTOMS: Diabetes mellitus, amenorrhea, failure to thrive

- **PEARLS:**
  - **Start VITAMIN E supplementation prior to age 5**
  - If a child presents with rectal prolapse or meconium illeus/plug, screen for cystic fibrosis! For meconium ileus, the boards could mention a “ground glass” on abdominal X-ray.
  - Patients can have cystic fibrosis exacerbations in which they produce quite a bit of mucus, sputum and have a difficult time breathing. Along with respiratory nebulizer treatments, treat with an **aminoglycoside** (possibly inhaled tobramycin) **and an anti-pseudomonal –cillin** drug, such as piperacillin.
  - PNEUMONIAS: **Staphylococcus infections** are more common early in life, followed by pseudomonas. Lifetime colonization is common with these two organisms as well as Haemophilus influenzae. The role of Streptococcus pneumoniae in cystic fibrosis children is debatable, but it is usually **not** a colonizer. If there is mention of a nodular pneumonia in a CF patient, choose Staph. If the boards mention lung abscesses, choose pseudomonas.
  - There is a strong association between Trisomy 21 (aka Down Syndrome) and cystic fibrosis.

- **HEMOPTYSIS**: This is not a common occurrence in children. If it happens, think CYSTIC FIBROSIS. Hemoptysis can also be seen in children with congenital heart disease (CHD) and chest trauma.
- **NASAL POLYPS:** 20-70% of cystic fibrosis patients eventually developing nasal polyps. In children, and on the pediatric boards, nasal polyps are very commonly associated with cystic fibrosis.
  - **PEARL**: If you are given the history of an asthmatic patient with nasal polyps, don't choose CF. Look instead for an answer related to a classic triad of **nasal polyps + asthma + aspirin (or NSAID) hypersensitivity**
  - **PEARL**: Nasal polyps are also associated with allergic rhinitis and sinusitis.

==========

PBR FACEBOOK CREW POST
https://www.facebook.com/groups/pedsboardreview/permalink/319600811519200/

Addition regarding vascular ring diag according to prep 2011 qest 53. PBR book page 368 vascular ring presumptive diag or may be the initial test is barium swallow, definitive diag is CT.

MY THOUGHTS
Hmm… I'm a little confused about what your question is. Regardless, I did NOT mention that it could be a presumptive diagnosis, nor did I mention a CT scan. For now, I'd leave things are they are for EXPIRATORY STRIDOR as below

PBR MODIFICATION – p. 368

EXPIRATORY STRIDOR
Expiratory stridor suggests obstruction in the lower trachea.

* **TRACHEO**MALACIA: Like laryngomalacia, this usually presents around 4-6 weeks of age, and **may also be worse when supine**. The differences are that it's **usually** an **expiratory stridor, or wheeze**, and of the location (trachea). This can sometimes this CAN also have an inspiratory stridor, but expiratory is much more common.

- **MNEMONICS: tracheOUTmalacia** = Stridor when breathing OUT. Also, note that both of the "–malacias" present around 4-6 weeks!

* **VASCULAR RING**: Look for a neonate having **trouble feeding and expiratory stridor since birth.**

- **PEARL**: **Diagnose** this with a **barium swallow**, not a scope!

==========

QUESTION/ERROR
Page 66 mistake? Dawn phenomenon cause hyperglycemia not hypo in the morning due to release of cortisol and GH.

MY THOUGHTS
You're right! The entire section should say HYPER instead of HYPO. As an added pearl…. I doubt the Somogyi effect will ever be tested again unless it's re-proven. Any topic that has controversy around it's validity will NOT be on the boards, so don't waste your brain space with it.

Thanks GSO for the lookup

PBR MODIFICATION – p.66

SOMOGYI EFFECT & DAWN PHENOMENA

The Somogyi effect (aka Somogyi phenomenon) and the Dawn phenomenon (aka Dawn effect) have been tested less in recent years. Both cause early morning HYPERglycemia.

*** SOMOGYI EFFECT:** This has recently been discredited and is unlikely to be tested. The pattern USED to include hypoglycemia around 2 – 3 AM (diaphoresis or nightmares in the middle of the night) followed by REBOUND HYPERGLYCEMIA at 6 – 8 AM. Treatment = DECREASED nighttime insulin

- **MNEMONIC:** Imagine "Mr. Miyagi from that karate movie is having trouble reading his insulin vial. What's the worst thing he could do? He gives himself TOO MUCH insulin every night (resulting in low glucose levels around 2 AM). Treatment? Get the old man some glasses!"

*** DAWN PHENOMENON:** Hyperglycemia occurs at 6 – 8 AM. Several theories, but most believe it's due to a PHYSIOLOGIC RELEASE OF GROWTH HORMONE or other hormones that can signal the liver to release glucose. Treat by decreasing nighttime carbohydrates, giving an early AM dose (by pump) or changing the injection site.

==========

QUESTION/ERROR
Update/correction needed on Inflammatory Acne: Page 119 of PBR says first line drug for severe cases is Tetracycline or Erythromycin. Tetracycline has been out of production for about 1 to 2 years now. You MIGHT find it in aquarium supplies at a pet store!

MY THOUGHTS
It's still available, but in limited areas. Thanks for the submission.

PBR MODIFICATION – p.119

INFLAMMATORY ACNE

Inflammatory acne is differentiated from comedonal acne by its RED BASE.

* **Minor cases**: If the acne is localized with small lesions, use a TOPICAL antimicrobial agent, such as Benzoyl peroxide, Clindamycin or Erythromycin. Retinoic acid topicals are also included in most regimens.

* **Severe cases**: If large, nodular or in multiple areas, use ORAL antibiotics. First line is Tetracycline, Doxycycline, or Erythromycin. Minocycline Minocycline is a second line agent. These antibiotics provide a bacteriocidal and an anti-inflammatory effec. May also try oral contraceptive pills (OCPs) in females for their anti-androgen effects. If all else fails, use ISOTRETINOIN.

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PBR FACEBOOK CREW POST

https://www.facebook.com/groups/pedsboardreview/permalink/306524402826841/

The derm chapter of the pbr review text states on p. 119 that benzoyl peroxide inactivates retinoids so one should be used in the night and the other in the morning. How does that explain the new combo products like epiduo that has both ingredients in one?

MY THOUGHTS

Tretinoin and Adapalene are both retinoids.... but from some BEIEF research I just did on this particular matter, Adapalene has a more stable structure and therefore does not breakdown in the presence of benzoyl peroxide.

Thanks RC for the question.

PBR MODIFICATION – p.119

COMEDONAL ACNE

Think of comedonal acne as an OBSTRUCTIVE process that creates white heads and black heads. Treat with a RETINOID keratinolytic agent. May also prescribe benzoyl peroxide.

PEARL: An answer with topical retinoic acid + benzoyl peroxide twice daily is probably WRONG. Benzoyl peroxide inactivates traditional retinoids (tretinoin), so one should be used at night, and the other in the morning (or at least with some time in between). Newer retinoids, like Adapalene and Tazarotene, are more stable and may be used at the same time.

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PBR FACEBOOK CREW POST
https://www.facebook.com/groups/pedsboardreview/permalink/322426321236649/
Not to nit pick, but Page 123 of PBR, about tuberous sclerous say one item may be Sebaceous Gland Hyperplasia. Should this be facial angiofibromas OR adenoma sebaceum instead? None of my other references mention Sebaceous Gland Hyperplasia with tuberous sclerous. I just want to know which way the ABP will "phrase this" in a test question.

MY THOUGHTS
It's okay… nit picking is good! Well, sometimes ☺

I'm still a little confused by the terminology, but "Angiofibroma" is the currently accepted term. So, I'd hope that older terms (adenoma sebaceum and sebaceous hyperplasia) will not be used.

Thanks GSO for the input, and CM for this (and other questions/input).

PBR MODIFICATION – p.123

TUBEROUS SCLEROSIS

Tuberous sclerosis is AUTOSOMAL DOMINANT. Look for at least **2** of the following features:

* ASH LEAF SPOTS: These are hypOpigmented lesions, which can be seen with a Woods Lamp. Need at least **3** on the body to help make the diagnosis.
    - **IMAGE**: http://bit.ly/nwloFE
    - **IMAGE**: http://www.nlm.nih.gov/medlineplus/ency/imagepages/2538.htm

* SHAGREEN PATCH (hypERpigmented plaque that can be rough/thick and papular)
    - **IMAGE**: http://bit.ly/n02Q8f
    - **IMAGE**: http://bit.ly/ojT5CF

* ANGIOFIBROMAS (aka ADENOMA SEBACEUM or SEBACEOUS HYPERPLASIA)
    - **PEARL**: Often misdiagnosed as acne. LOOK FOR SPARING OF THE FOREHEAD
    - **IMAGE**: http://en.wikipedia.org/wiki/File:TuberSclerosisCase-143.jpg

* PERIVENTRICULAR OR CORTICAL TUBERS: Usually associated with INFANTILE SPASMS or seizures

* CARDIAC RHABDOMYOMAS: Look for a kid with **arrhythmias**!

* RENAL ANGIOMYOLIPOMA

MANAGEMENT OF TUBEROUS SCLEROSIS: Most of the management has to do with seizures/infantile spasms and cardiac arrhythmias.

* **MNEMONICS:**

- Imagine a TUBULAR bazooka shooting out WHITE LEAVES. The leaves have DANCING (seizing) tics on them!
- ASH is typically GRAY/WHITE/HYPOPIGMENTED, whereas a "PATCH of GREEN" is typically DARKER/HYPERPIGMENTED.

* **MNEMONIC**: **ASHES** come from burned WOOD. Woods Lamp needed to see them.

==========

PBR FACEBOOK CREW POST
https://www.facebook.com/groups/pedsboardreview/permalink/323336917812256/

I've got a burning question about protein intake on page 136 of PBR. It states: "High protein intake can cause a high osmotic load. Osmotic load is also affected by chloride, phosphorus, potassium, sodium, but not calcium." Can anyone give me a formula to calculate the osmotic load or source to look up more info on this topic?

…does this need to be in the board review book? Is it important for board review? I would like to review this in more detail if it is. Any sources you can recommend if it is important for ABP?

MY THOUGHTS
Too detailed for the boards in my opinion… I can't guarantee that it won't be on the boards, but I can guarantee that your time would be better spent in other challenging PBR areas.

PBR MODIFICATION - None

==========

QUESTION/ERROR
Question regarding your book - Page 69 (four mg for Folic acid is recc to prevent NTDs)

The American Academy of Pediatrics endorses the US Public Health Service (USPHS) recommendation that all women capable of becoming pregnant consume 400 µg of folic acid daily to prevent neural tube defects (NTDs). Studies have demonstrated that periconceptional folic acid supplementation can prevent 50% or more of NTDs such as spina bifida and anencephaly. For women who have previously had an NTD-affected pregnancy, the Centers for Disease Control and Prevention (CDC) recommends increasing the intake of folic acid to 4000 µg per day beginning at least 1 month before conception and continuing through the first trimester. Implementation of these recommendations is essential for the primary prevention of these serious and disabling birth defects. Because fewer than 1 in 3 women consume the amount of folic acid recommended by the USPHS, the Academy notes that the prevention of NTDs depends on an urgent and effective campaign to close this prevention gap

please clarify

MY THOUGHTS
This is very much outside the scope of the exam. I'd expect a question something like this to be on an obstetrics board exam.

Thanks to GSO for helping with the following lookup:

All women should consume 0.4-0.8mg of folic acid daily if planning or capable of becoming pregnant. If any woman has had a prior NTD, they should consult their physician about whether they need to increase to the recommendation of 4000ug a day of folic acid.

http://www.cdc.gov/ncbddd/folicacid/recommendations.html

PBR MODIFICATION - None

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QUESTION/ERROR
page 104: Pulm Atresia - .error statement : "...blood goes from RA to the RV via a PFO"

Correction should be : should be blood goes from RV to the LV via VSD or it goes from RA through PFO to LA

MY THOUGHTS
Good catch! And it was worded poorly. Thanks to you and to GSO

PBR MODIFICATION – p.104

PULMONARY ATRESIA (aka PULMONARY VALVE ATRESIA)
Pulmonary atresia is very similar to tricuspid atresia. Since the pulmonary valve did not form, blood cannot flow from the right ventricle to the lung. This results in tricuspid regurgitation (systolic murmur). There's usually a right to left shunt, either through a VSD (RV to LV) or through a patent foramen ovale (PFO) being kept open from the high pressure in the right atrium (RA to LA). Cyanosis occurs early (minutes to hours), a systolic murmur may be heard, and the EKG will show **LVH**. Both a PDA **and** a right to left shunt is needed, otherwise this is fatal. To get oxygen into the pulmonary circulation, treat with prostaglandin to sustain the PDA. Diagnose by echocardiogram.
PEARL: It is unlikely that you would be asked to differentiate between tricuspid and pulmonary atresia.

==========

QUESTION/ERROR
Page 84. Please clarify hyper Ig M syndrome.? B and T cell deficiency or B cell deficiency.

MY THOUGHTS
It has components of both! The problem is within the T-cells since they don't send the appropriate signals to the B-cells to initiate the IgM to IgG class switch. Since the switch itself is a B-cell function, it acts very much like a B-cell problem and is thus listed under the B-cell deficiencies

PBR MODIFICATION – NONE, but I thought I'd include the topic here as a recap

HYPER-IGM SYNDROME (aka HyperIgM Syndrome)
In hyper-IGM syndrome (aka HyperIgM Syndrome), the IgM to IgG class switch does not occur due to a missing signal to B-cells from T-cells. There is LYMPHO**cytosis and NEUTROPENIA.** Patients have frequent otitis media and sinopulmonary infections. They also get diarrhea and opportunistic infections, including PCP. Usually starts around 6 months of age. This is a **T-cell abnormality**, so there is an increased risk of lymphoma/cancer. For treatment, give IVI**G** to make up for the missing immunoglobulins and Trimethoprim-Sulfamethoxazole (aka Bactrim). Also give PCP prophylaxis

MNEMONIC:

Bactrim is used for **P**C**P** proplhylaxis

MNEMONIC: Think of the LYMPHOcytosis as a compensatory mechanism in which there are high numbers of LYMPHOcytes circulating and releasing elevated levels of IgM to "make up" for the lack of IgG, IgE and IgA. Also, think of the neutropenia as being a relative "lack of neutrophils" on the WBC resulting from an excess of lymphocytes.

PEARL: Infections are similar to agammaglobulinemia, but the CBC looks different. Also, these patients can get PCP so suspect this diagnosis in any **HIV negative patient diagnosed with PCP**.

==========

QUESTION/ERROR
Pg 291 how many vaccines at 12 months?

MY THOUGHTS
Unless someone out there has any other thoughts on the usual vaccines a 12 month old gets, I think I'd stick to what I have written in the book:

** 12 MONTHS: 6 vaccines are given (MMR #1, VZV #1, Hepatitis A #1, Hib #4 and the 2 boosters of PREVNAR #4 of 4, and DTaP #4 of 5).*

Thanks to CTM for some thoughts on DTAP and when it could be given. It was pointed out to me that depending on when DTAP#3 was given, perhaps DTAP#4 would not yet be due. For these types of questions (i.e. "How many shots at X number of months?), I

wouldn't expect it to be too tricky. Unless they specifically give you caveats, they are just asking about the usual # of shots for a child in a compliant family. In this case, I'd assume the child got his/her 6 month shots 6 months ago and that included DTAP#3, so now would be a good time DTAP#4. Having said all of that, the CDC's chart now lists DTAP#4 at 15-mo of age but says under the chart that 12 mo is also okay.
http://www.cdc.gov/vaccines/schedules/hcp/imz/child-adolescent.html

PBR MODIFICATION – None

==========

QUESTION/ERROR
Pg 345 seizure free interval before return to driving?

MY THOUGHTS
It varies from state to state, but the minimum would be 3 months.

Thanks to WN for having my back and confirming.

Topic Recap:

EPILEPSY & SEIZURE PRECAUTIONS & EDUCATION
Seizure precautions and education should be given to the parents of children with epilepsy. During a seizure, parents should put the child on the floor on his/her side, time the seizure, and **call 911 if it lasts more than 5 min**. When epileptic children are on wheels (bicycle), they should wear a helmet. When near water, an adult should be watching. Let parents know that recurrent seizures rarely cause death or long-term brain damage, unless a patient goes into **status epilepticus** (continuous seizure activity for > 30 minutes). For teenagers, they need to have a seizure-free interval of at least 3 months before they can return to driving (some say 6 or even 12). If a patient has regular epilepsy (not related to any other neurologic disorder), medications can be stopped after 2 years of a seizure-free interval to see how the child does off of medication.

PBR MODIFICATION - NONE

==========

QUESTION/ERROR
The notes say, for SCID, "Look for a low Absolute Lymphocyte Count (ALC) that is well below 40." Primary Immunodeficiency Diseases: Definition, Diagnosis, and Management says "Absolute lymphocyte counts are often less than 1000, but normal lymphocyte counts do not exclude SCID," and another article, "Diagnosis of severe combined immunodeficiency," says "an absolute lymphocyte count of less than 2.8 × 109/litre is 2 SD below the mean. When infants with infection have a count lower than this, it is highly likely that they have SCID." Where does this very low criterion (ALC < 40) come from?

MY THOUGHTS
It's been a few years, so I could be wrong… but I think the reference was from a lecture. Since I can't find anything to support my number, I've modified the text. I think the key here would be to know that you need to look for a low ALC. Hopefully they would give you an age-appropriate reference range, or possibly just state that there is a lymphopenia.

Keep in mind that lymphocyte counts are **higher in infancy** than in adulthood, so what looks normal for a 5 yo would actually be LOW for an infant. Here's a chart of normal CBC with Diff values from the American ACADEMY of Pediatrics to demonstrate the point:

Lymphocyte counts are higher in infancy than in adulthood

WBC and Diff

Age	WBC (x $10^3/mm^3$)	Segs	Bands	Lymphs	Monos	Eosinophils	Basophils	Atypical Lymphs	No. of NRBCs
0-3 d	9.0-35.0	32-62	10-18	19-29	5-7	0-2	0-1	0-8	0-2
1-2 wk	5.0-20.0	14-34	6-14	36-45	6-10	0-2	0-1	0-8	0
1-6 mo	6.0-17.5	13-33	4-12	41-71	4-7	0-3	0-1	0-8	0
7 mo to 2 y	6.0-17.0	15-35	5-11	45-76	3-6	0-3	0-1	0-8	0
2-5 y	5.5-15.5	23-45	5-11	35-65	3-6	0-3	0-1	0-8	0
5-8 y	5.0-14.5	32-54	5-11	28-48	3-6	0-3	0-1	0-8	0
13-18 y	4.5-13.0	34-64	5-11	25-45	3-6	0-3	0-1	0-8	0
Adults	4.5-11.0	35-66	5-11	24-44	3-6	0-3	0-1	0-8	0
Segs = segmented neutrophils.									
Bands = band neutrophils.									
Lymphs = lymphocytes.									
Monos = monocytes.									

PBR MODIFICATION – p. 81

SEVERE COMBINED IMMUNODEFICIENCY (SCID)

Severe combined immunodeficiency (SCID), is a **T AND B CELL DEFICIENCY** (hence the word COMBINED). The deficiency in T and B LYMPHOcytes results in severe **LYMPHopenia. Look for a low Absolute Lymphocyte Count (ALC)**. These patients

do NOT have **LYMPH**adenopathy, but they DO have a small thymus. The presence of a thymus means a bone marrow transplant **is** a viable treatment option. These patients get **all kinds of infections** (viral, bacterial, fungal and opportunistic). Usually present in the **first 3-6 months with otitis media (OM), thrush, diarrhea and dermatitis.** There is **complete absence of T-cell function** (on flourometric analysis of T, B, and NK cells). **This condition usually presents closer to 3 months**. In contrast, a pure B-cell deficiency presents around 6 months of age. When looking at LYMPHOcyte counts, keep in mind that normal infants can have lymphocyte counts in the 40s while a normal 5 year old can have counts in the 20s. Therefore, if you see a LYMPHOcyte count of 30 in an infant, it's LOW!

* **Possible presentations: PCP**, a viral pneumonitis that doesn't resolve or recurrent candidiasis that seems to be refractory to treatment. For PCP patients, always consider HIV in the differential too.

* LIVE VACCINES: Do not give SCID patients live vaccines!

* CURE: Bone Marrow Transplant (BMT)

==========

QUESTION/ERROR

For concussions in sports, is the grading system still valid in time to return to play 1 week.2 week/1 month or now the 24 hour progressive 5 day return to play as listed on AAN?

MY THOUGHTS

The guidelines changed THIS year. That means there will likely be NO concussion questions, or there will be OLD concussion questions. The gist of the NEW recommendations seems to be that the "return to play" timeline should not be set in stone and should be taken on a case by case basis. Also, concussion evaluation should be done by a Licensed Healthcare Provider (LHCP).

For this year's exam, I'd probably answer based on the current PBR recommendations. Be familiar with the topic modification below in case they have miraculously created and approved a question with newer recommendations

Here is a good snippet from the AAN:

"The updated guideline recommends athletes with suspected concussion be immediately taken out of the game and not returned until assessed by a licensed health care professional trained in concussion, return to play slowly and only after all acute symptoms are gone. Athletes of high school age and younger with a concussion should be managed more conservatively in regard to return to play, as evidence shows that they take longer to recover than college athletes."

PBR MODIFICATION FOR 2014 EXAM – p.174

POST CONCUSSION TREATMENT

"Concussion" is a clinical diagnosis and my include symptoms such as confusion, amnesia, loss of consciousness, balance/coordination abnormalities, sound/light sensitivity, headache, fogginess, etc. Remove any athlete suspected of a concussion immediately from play. Player needs to be evaluated by a licensed healthcare provider **trained** in concussion diagnosis **and** management, and should not return to plan until all acute symptoms have resolved. There is NO set timeline for recovery or return to play. There is weak evidence that a step-by-step plan of return to activity might be helpful. Any activity that makes symptoms worse or puts the athlete at risk for another concussion should NOT be a part of the management plan while any concussion symptoms are still present.

* IMAGING: Obtain if there's loss of consciousness (LOC), post-traumatic amnesia, persistently altered mentation, focal neurological deficits, evidence of skull fracture, or signs of clinical deterioration.

* **PEARLS**: Football, rugby, soccer and basketball players are at highest risk. No specific type of helmet has been proven to be better than another. **Symptom checklists**, the Standardized Assessment of Concussion (SAC), neuropsychological testing and the Balance Error Scoring System may be helpful tools in diagnosing and managing concussions but should **not** be used alone for making a diagnosis. High school and younger athletes should be treated more conservatively than adults.

==========

QUESTION/ERROR

On pg 217 Is retinitis pigmentosa x linked R?

MY THOUGHTS

Yes… and no… and yes ☺

Too many ways to inherit it, so you won't be asked a specific pattern. See modification below.

Thanks WN for the help.

PBR MODIFICATION – p. 217

RETINITIS PIGMENTOSA

Retinitis pigmentosa is a retinal dystrophy that eventually leas to blindness.

PEARL: There are multiple inheritance patterns (mostly dominant) so you will not be asked to identify a single inheritance pattern. BUT, look for a family history.

==========

QUESTION/ERROR
You can't answer question 7 (at least easily) on page 12, as the link to the image is wrong. There is no photo to look at.

MY THOUGHTS
You're right… Dead link.

Thanks for clicking GSO.

PBR MODIFICATION – Q&A Book Page 12, Question #7

You are shown an image of a newborn. He has what looks like a thin, transparent film on his body. Eyelashes are missing and eyelids seem inverted (IMAGE: http://bit.ly/nhplsa). What is the diagnosis?

a. Lamellar ichthyosis
b. Harlequin ichthyosis
c. Lichen striatus
d. Ichthyosis vulgaris

==========

QUESTION/ERROR
Question 8, page ----. Cannot see contrast study. Link does not work. Don't know if xray is needed to pick correct answer.

MY THOUGHTS
Yup… dead link. I think you meant page 18. New link below.

PBR MODIFICATION

A newborn is noted to have a distended abdomen and bilious emesis. It has been 24 hours and meconium has not been passed. A contrast study (similar to the one here http://bitly.com/18gQR8M) shows contrast throughout the colon. Which of the following is the most likely diagnosis?

a. Malrotation
b. Duodenal atresia
c. Microcolon

d. Intussusception

==========

QUESTION/ERROR
Page 250 - about macrolides, says to avoid in children less than 6 "years" of age... Clinical literature I've seen says this should be 3 months. Or should it be 6 "months" for the ABP exam?

MY THOUGHTS
You're right. Typo on my part! Thanks!

And thanks to GSO.

PBR MODIFICATION – p.250

ANTIBIOTIC AGE PEARLS
AGE LIMITATIONS: When choosing an antibiotic (especially tetracycline, doxycycline, fluoroquinolones and macrolides), ALWAYS look at the age of the patient.

* TETRACYCLINE & DOXYCYCLINE: May be given **after the age of 8**.

- **PEARL**: If you diagnose a 5 year old child with Rocky Mountain Spotted Fever, go ahead and GIVE doxycycline. That is the first-line therapy regardless of age.

* FLUOROQUINOLONES: Give after **18 years** of age due to possible tendonitis and tendon rupture. Avoid choosing this as an answer for your adolescents with STDs.

* MACROLIDES (ERYTHROMYCIN): Avoid in children less than 6 months of age due to an association with pyloric stenosis. Give azithromycin instead.

==========

QUESTION/ERROR
Check out the excellent article below. To make it brief: Azithromycin is for >6 MONTHS OLD, BUT the current guideline still recommends it for infant <1 MONTH OLD (instead of Erythromycin) for Pertussis Tx, to avoid the risk of Hypertrophic Pyloric stenosis.

Reference article: http://bitly.com/1ayAg62

So, correction needed? on page 260 of PBR, it will need a correction for the treatment of Bordetella Pertussis then. It only lists erythromycin for treatment.

PBR Facebook Crew comment:
https://www.facebook.com/groups/pedsboardreview/permalink/333431203469494/

Per PREP: AZITHROMYCIN (NOT Erythromycin) is a preferred Tx. If they put these two in the choices. Choose Azithromycin.

MY THOUGHTS

This is a tough one, but hopefully not one you'd have to choose between.

Here's a summary of the CDC guidelines:

"*For treatment of pertussis in persons older than 1 month, the preferred agents are the macrolide drugs erythromycin, clarithromycin, and azithromycin. For younger infants, azithromycin is preferred, and erythromycin and clarithromycin are not recommended. An alternative to macrolides in persons older than 2 months is trimethoprim-sulfamethoxazole (TMP-SMZ).*"

Thanks GSO for the help.

If it was me, I'd basically choose AZITHROMYCIN for any child < 6 mo of age.

PBR MODIFICATION – p.260

BORDETELLA PERTUSSIS (aka WHOOPING COUGH)
Bordetella pertussis (aka whooping cough) patients are described as having bursts, or "paroxysms," of coughing. They cough so much they can't breath, and then they inspire deeply causing a WHOOP! Patients may have a HIGH WBC of > 20,000. Diagnose with a nasopharyngeal swab. Treat with **ERYTHROMYCIN.** Use **AZITHROMYCIN** for children < 1 month of age. All contacts (**even if immunized**) need to be given erythromycin for prophylaxis since immunity of the vaccination wanes. Hence the need for Tdap in teens now (more coming up later).
PEARLS: Consider this diagnosis in anyone with a **chronic cough**. Antibiotic treatment shortens the **early** stage in which the patient is infectious and has URI-type symptoms (catarrhal stage). It dose NOT decrease the "whooping," or paroxysmal stage. Erythromycin, clarithromycin and azithromycin are all acceptable agents. If < 6 months of age, if given erythromycin **and** azithromycin as options, choose azithromycin.
MNEMONIC: If it ends in –ELLA, it's probably a Gram negative organism! Brucella, Shigella and Salmonella fit better elsewhere in this chapter.

==========

QUESTION/ERROR
PBR Facebook Crew Post -
https://www.facebook.com/groups/pedsboardreview/permalink/330123663800248/

I'm a little confused. Page 275 under OM, says do not give a macrolide for beta lactamase because they don't work well. Page 284 under Empiric Treatment for acute lymphadenopathy says to use a beta lactamase drug like augmentint first, then 2nd choice is cefazolin, clindamycin, and "erythromycin". Erythromycin is a macrolide. Am I missing the point here?

MY THOUGHTS
I think you're confusing 2 different disease processes. On page 275, the book is discussing OTITIS MEDIA where there IS a high amount of resistance for the marcolides. On page 284, the condition being discussed is empiric treatment for ACUTE LYMPHADENOPATHY in the head and neck. I state that first line is Augmentin and the other abx could also be considered.

Thanks GSO for the crosscheck.

PBR MODIFICATION – none… topic revisted

EMPIRIC TREATMENT
Empiric treatment for acute lymphadenopathy in the head and neck area should aim to cover beta-lactamase producers. AMOXICILLIN-CLAVULANATE! Other appropriate options include cefazolin, clindamycin and erythromycin.

==========

QUESTION/ERROR
PBR Facebook Crew Post –
https://www.facebook.com/groups/pedsboardreview/permalink/330133283799286/

Page 240 of PBR says that staph aureus is the most common cause of osteo in sickle cell patients. I've seen one other recent book (circa 2010) and one review article in Pediatrics from 1998 that says Samonella is the most common cause. Pediatrics vol 101, #2, page 296. Is there a more up to date source? Which one is correct?

MY THOUGHTS
I agree… there's confusing info out there. I just looked on UpToDate and they take a firm stance and reference multiple resources which say that Gram negatives (especially Salmonella) are responsible for the majority of osteomyelitis cases in sickle cell patients. Staphylococcus aureus is only responsible for about 25%.

So, still Staph for normal hosts, but Salmonella for sicklers.

I'm changing it. Thanks for asking!

PBR MODIFICATION p.239 – 240

SICKLE CELL ANEMIA
* **PRESENTATION**: The signs or symptoms of sickle cell anemia could include the following:

- APLASTIC CRISIS: Will be due to Parvovirus B19
- SEQUESTRATION CRISIS (aka SPLENIC SEQUESTRATION CRISIS): Look for abdominal pain, splenomegaly and signs of SHOCK. Cells get trapped in the organ (usually the spleen, sometimes the liver). The pooling of the blood results

in a lower effective volume of circulating blood which results in SHOCK. Emergently transfuse!

  - **PEARL**: If given an option of IVF or PRBCs, go with what is FASTEST to bring up the blood pressure - IVF!

- ACUTE CHEST SYNDROME: Look for the triad of PAIN + INFILTRATE + HYPOXIA. If there's any doubt about the O2 sat, confirm with an ABG. Treatment is dictated by the **hemoglobin level**. **LOW** = TRANSFUSION. **HIGH** = EXCHANGE Transfusion.
- VASOOCCLUSIVE CRISIS: Acute pain! If severe, can cause ischemia, infarction of bone, stroke or dactylitis (swelling of the hands or feet). Initial therapy is IV fluids and pain control. For suspected STROKE, get an **MRI**. For an obvious or confirmed stroke, give PRBCs.
  - **IMAGE**: www.mdnotebook.com/wp-content/uploads/2011/05/dactylitis-scd.jpg
- OSTEOMYELITIS: Salmonella osteomyelitis is MORE common than Staphylococcus aureus in sickle cell patients. Staph only accounts for about 25% of cases.
  - **PEARLS:** Remember that sickle cell patients can present with sepsis due to encapsulated organisms. Again, chose Salmonella over Staphylococcus for osteomyelitis in sicklers.
  - **(DOUBLE TAKE) MNEMONIC**: A complete list of encapsulated organisms can be recalled by remembering that "Some Nasty Killers Have Some Capsule Protection." Streptococcus pneumoniase, Neisseria meningitidis, Klebsiella pneumoniae, Haemophilus influenzae, Salmonella typhi, Cryptococcus neoformans and Pseudomonas aeruginosa. Bruton's agammaglobulinemia and sickle cell patients are especially susceptible to encapsulated organisms.
- DILUTE URINE: Due to renal damage, the urine may be dilute as evidenced by a low specific gravity. This is also known as HYPOSTHENURIA.
  - **MNEMONIC**: HYPOsthenURIA

* **DIAGNOSIS** of sickle cell anemia is done by **HEMOGLOBIN ELECTROPHORESIS** will show hemoglobin F and S (no hemoglobin A). Might be shown as SF. The hemoglobin S develops due to a "Glu-Val" amino acid swap (**Glutamic acid substitution of Valine**) in the Beta chain. Consider these patients as being functionally

asplenic. Give PCN prophylaxis through 5 years of age. Look for Howell-Jolly bodies on the smear.

* **PEARL**: These are normal sized RBCs with the Glu-Val swap that predisposes them to sickle, but they are otherwise of NORMAL size, so this is a NORMOCYTIC anemia. So, any macrocytosis or microcytosis has to be accounted for by an additional diagnosis! B12 or Folate deficiency? Iron deficiency? Concomitant thalassemia?

* **PEARL**: If the electrophoresis results come back with Hgb SA, that means the patient has sickle cell **trait**. Hemoglobin SC and S-beta thalassemia can result in a palpable spleen at the age of 6, but SF (aka SS) causes the spleen to shrivel up by then. So a palpable spleen in an 8 year old is NOT due to sickle cell anemia.

* **IMAGE**: http://www.wadsworth.org/chemheme/heme/glass/slide_005_howell-jolly_bod.htm

* **MNEMONIC**: Sickle cell anemia results in **HOW**ell-JOLLY bodies. Have you ever noticed **HOW JOLLY** these patients get once they are given morphine?

* **MNEMONIC**: Regarding the glu-val swap mutation, there's a mnemonic in there somewhere about a SICKLED CELL splattering over a BED (beta) on a WALL with GLUE on it. Thoughts?

==========

QUESTION/ERROR
Clarification? Page 307 of PBR seems to imply that Fabry's Disease ALSO has a cherry red macula spot. Fabry's disease does not, it has corneal opacities. I would recommend rewriting sentence to be a little more clear for the next revision. This link is fairly complete:
http://www.fabrycommunity.com/~/media/Fabry/Files/PDF/RGDUSP212_0705_Occular_Wallchart.pdf

MY THOUGHTS
Hmm… in the version I'm looking at, I don't mention a cherry red spot. I do mention Kaney West's white sunglasses which represent the corneal opacities you mentioned.

Maybe you have an old edition?

PBR MODIFICATION – None – Topic revisited

FABRY DISEASE (aka FABRYS DISEASE)
Fabry disease (aka Fabrys disease) findings include opacities of the eye, vascular disease of the kidney, heart or brain, and ORANGE-colored skin lesions.

PEARL: There is no organomegaly. Though not included in the HuNiTaG mnemonic, it's a very common lysosomal disorder so it's likely high-yield.

MNEMONIC: Imagine Mr. Kanye West wearing his famous WHITE SUNGLASSES (opacities) and an ORANGE-colored suit (skin) made of ROUGH (rash) **FABRY**c.

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QUESTION/ERROR
Typo? See page 310. Meant to say Farber's disease, not Fabry's disease?

MY THOUGHTS
Hmm… looks like I **did** say Farber's and that **is** what I meant. Maybe you have an older edition?

Thanks GSO for the cross check.

PBR MODIFICATION – none – topic revisited

CHERRY RED SPOT DIFFERENTIAL

The differential for a cherry red spot includes Tay-Sachs Disease, Niemann-Pick Disease and Farber's Disease (Farber's Disease is low yield).

* TAY-SACHS: Ashkenazi Jews. Macrocephaly.

* NIEMEN PICKS: Hepatomegaly and neurologic problems.

* FARBER'S DISEASE (aka DISSEMINATED LIPGRANULOMATOSIS): Probably low yield. Progressive disease. Fatal within the first few years. Look for a baby with joint pain and skin nodules presenting within the first week of life.

- **MNEMONICS FOR THE ABOVE DISORDERS:**
    - Imagine a CHERRY FARMER going to NIEMEN marcus in his overalls to try and sell a SACH of cherries to them! Wow, talk about rough economy.
        - **KEY:** Cherry, Farber, Niemen Picks, Tay-SACHS.
    - CHERRY FARMER'S disease = Imagine a CHERRY FARMER working so hard that he gets ARTHRITIS and SWOLLEN BUG BITES all over his skin.

* RETINAL ARTERY OCCLUSION: Presents as painless visual loss with a **cherry red** spot at macula.

- **(NAME ALERT) RETINAL VEIN OCCLUSION:** Painless visual loss with multiple hemorrhages noted on ophthalmologic on exam, referred to as having a "thunder and lightning" appearance. Sometimes also referred to as "blood and thunder" or "pizza pie."
    - **MNEMONIC**: Venous blood is dark. Thunder and lightning occur at night.
- **(NAME ALERT) RETINAL DETACHMENT:** Visual loss with what looks like cobwebs or a curtain coming across a patient's vision.

- **MNEMONIC**: Imagine the retina being really far back in the eye in an area where COBWEBS webs are growing on old CURTAINS.

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QUESTION/ERROR
PBR Facebook Crew Post –
https://www.facebook.com/groups/pedsboardreview/permalink/330941813718433/

I haven't seen this formula before, and it seems to be wrong, unless I'm missing something.

Page 323 of PBR under hyponatremia.

PEARL: Formula to correct = (desired Na - measured Na) x Weight in kg x 0.6. Then ADD 3 meglkg as the daily maintenance amount of sodium needed. This will give you the total amount of sodium needed for the next 24 hours to reach the desired sodium level you input into the equation, which should never be greater than 12 above the measured sodium.

Ok, I did a sample problem.

10 kg kid, measure Na+ of 130. Desired Na+ of 140.

(desired Na - measured Na) x Weight in kg x 0.6
(140-130) x 10 x 0.6 = 60 mEq Na+ needed for deficit?

Then add 3 mEq Na+ per kg, makes an additional 30 mEq for maintenance?

So now I'm up to 90 mEq Na+ needed for 24 hours, but... by the last sentence, it says it should never be more than 12 above the measure sodium? Doesn't make sense, the measure sodium is 130, which is WAY above 12. And the kid needs 90 mEq Na+ by this example. Can someone please explain this or re-edit the PERRL if a correction is needed?

MY THOUGHTS
Wow… EXCELLENT submission. Thanks for walking me through your thought process. I get many questions that are extremely vague and require a lot of digging on my part to figure out what the heck I'm being asked. You made this easy.

Basically, it sounds like you were confused about the number 12 that I mentioned. I essentially meant that the newly corrected sodium level that you are targeting should never be more than 12 above the original measured sodium level. So, in patient with a sodium level of 115, aim for a corrected sodium level of no more than 127… and if you are doing serial sodium checks and you might overshoot, SLOW DOWN.

Thanks CTM for the question and the update on how I could/should rephrase ☺

PBR MODIFICATION – No major corrections, just 1 or 2 re-phrasings – p.323

HYPONATREMIA

Hyponatremia is MUCH more common than **hyper**natremia. Look for a sodium level less than 130 mEq/L. If the level is < 120, watch out for **seizures**! Seizures can occur as free water starts moving into CNS cells at about 125 meq/L. For babies, remember **NOT** to give free water until at least 6 months of age. That's when their kidneys can handle the job of getting rid of excess water. Regarding diagnosing WHY there is **hypo**natremia, always calculate the fractional **excretion** of sodium (F**e**Na) if given the labs to do so. Also look for serum and urine osmolality values. Regarding correction of sodium, remember that rapid correction (faster than 12 meq/day) can result in central pontine myelinolysis.

* **PEARL**: Fractional Excretion of Sodium = How much Na+ is **EXCRETED** as compared to reabsorbed. So **a HIGH FeNa = HIGH excretion of Na+ into the urine!** The formula can be written in many ways. For some reason, I remember YO**U** NACR! **P**EE NACR! **FeNa+ = [(U_{Na}/U_{Cr}) / (P_{Na}/P_{Cr})]**. Division only, no multiplication.

* **PEARL**: It's possible that you will only be given a urine sodium level. For the exam, **use a urinary sodium of 20 as your cutoff** for the LOWER LIMIT of normal. So if a urine sodium level is < 20, the kidneys are either holding on to sodium tightly, or they are not holding on to water tight enough. If you get confused, it's probably in your best interest to assume that a low urine sodium reflects low/decreased sodium excretion (i.e., a low FeNA).

* **PEARL**: **Formula to correct = (desired Na – measured Na) x Weight in kg x 0.6.** Then **ADD 3 meq/kg** as the daily **maintenance** amount of sodium needed. This will give you the total amount of sodium needed for the next 24 hours to reach the **desired sodium** level you input into the equation, which should never be greater than 12 above the measured sodium. So if measured is 115, desired should be no more than 127.

- **SHORTCUT:** If the desired change in sodium is 12 meq over 24 hours, then 12 x 0.6 = 7.2, and a simpler formula for the correction of sodium = (7 x Weight in kg) + (3 meq x Weight in kg).

* DEHYDRATION:

- The dehydrated patient's blood is concentrated, so expect a **HIGH serum osmolality**.
- The Kidneys are working to retain sodium/water, so expect a **LOW FeNa**. This is a KEY POINT. Do not assume that a concentrated urine means an elevated urine sodium.

- The urine will be concentrated, so expect a **HIGH urine osmolality**.

* DIURETICS: May vary depending on the diuretic, but in general, if a diuretic is at fault for hyponatremia, that means it's forcing sodium out. Expect a **HIGH FeNa** and probably a **LOW urine osmolality** since free water is being forced out as well.

* ACUTE TUBULAR NECROSIS & RENAL FAILURE: Expect a **HIGH FeNa** because the kidneys are not able to do their job of holding on to sodium.

* GASTROENTERITIS & DIARRHEA: This usually does NOT cause a hyponatremia **until** the patient is only given (or is only tolerating) **hypotonic liquids** (like free water). The patient is volume depleted due to GI losses, so the kidneys hold on to as much sodium as they can. Therefore, the **FeNa and the urine Na+ are LOW** (very low, often < 10).

* PSYCHOGENIC POLYDIPSIA: Refers to patients who just tend to drink a lot of water on a fairly chronic basis. Serum becomes dilute, and the kidneys are excreting as much water (using sodium to do it) as possible. So look for the serum osmolality **and** urine osmolality to be low. Since the kidneys are pushing water out with sodium, the **FeNa should be HIGH**.

* ACUTE WATER INTOXICATION: This refers to drink lots of water over a **very short time**. Look for a healthy, afebrile child having change in mentation or a seizure after being around a body of water (tub/pool). Think of it as a VERY acute issue, so total body sodium is normal. This is a **hyper**volemic hyponatremia, though the child will not be edematous and will **look** euvolemic. Urine sodium will again be high since there's no drive to retain it.

* NEPHROTIC SYNDROME: Patients are edematous due to 3rd spacing from hypoalbuminemia. The total body water (TBW) is normal, BUT the intravascular department is depleted. Patients can get hyponatremia. Assume it's the same as being dehydrated. **FeNa and urine sodium are LOW** (very low, often < 10).

* SYNDROME OF INAPPROPRIATE ADH SECRETION (SIADH): Excess ADH results in excess water retention and an INCREASED total body water (and sometimes BP). Urine output may be decreased to < 1 cc/kg/day. There is serum **hypo**smolality (<300 osms) and **hypo**natremia because of retained water. Urine is concentrated/**hyper**osmolar (>300 osms). **Urine sodium is > 25 (normal to high)**, so the **FeNa is high.** Possible etiologies of SIADH include brain tumor/bleed, pneumonia or other lung disease, cancer, infection, recent surgery and Guillain Barre. It's a diagnosis of exclusion, so other etiologies of hyponatremia should be ruled out first. Treat with fluid

RESTRICTION. Use Demeclocycline (diuretic) or Fludrocortisone if fluid restriction is not working and you note that the sodium level is persistently < 120.

- **MNEMONIC**: Assume the FeNa is high in SIADH because there is no drive to hold on to sodium given the hypervolemic state. Part of the kidney must hold on to water due to the ADH, but there is no problem with sodium excretion, so that part of the kidney is trying to EXCRETING sodium in an effort to excrete water.

* PSEUDOHYPONATREMIA: Total body sodium is normal, but elevated triglycerides or proteins cause an increased oncotic pressure which pulls excess water into the intravascular space. Also, triglycerides and proteins are big, and sodium is measured as the amount of sodium per overall volume (not per volume of water). This could be seen in a nephritic with hyperlipidemia.

* DIABETIC KETOACIDOSIS (DkA): Hyperglycemia results in increased osmolality, and thus increased oncotic pressure. This leads to water being drawn in and causing a hyponatremia. No treatment is needed since the sodium level will correct as the glucose corrects with insulin. To check if the drop in sodium is appropriate, add 1.6 meq/L to the sodium level for every 100 mg/dL of glucose above 100 to see if the final sodium value is normal. If it's still low, then there's also something else going on. So for a glucose of 300, the sodium should've dropped by only 3.2.

* CEREBRAL SALT WASTING: This is **low yield** and complicated. Look for increased urine output, increased salt excretion (and thus an elevated FeNa and urine sodium) and hyponatremia. The key finding (which is confusing) is that the serum osmolality is **higher** than the urine osmolality.

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QUESTION/ERROR
PBR Facebook Crew Post –
https://www.facebook.com/groups/pedsboardreview/permalink/331158493696765/

could someone please confirm the following
1 . most common area for intususception is_____
2. most common area for NEC IS _____. pbr is confusing

MY THOUGHTS
Ileocecal (aka ileocolic, ileocolonic area) as mentioned in the current version of the PBR. So, basically near the junction of the small and large intestine.

PBR MODIFICATION - none

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QUESTION/ERROR
Correction on page 369 under asthma. It has "mild intermittent asthma". This was rename "intermittent asthma" in 2007. This reference has the details. Don't read it till after the exam, it's very long, 73 pages.
http://www.nhlbi.nih.gov/guidelines/asthma/asthsumm.pdf

MY THOUGHTS
Thanks SO MUCH for the:

- clarification
- page number
- and the reference link (big help)

I was glad to see the change in nomenclature b/c it never made sense to me to include the word mild. Now that they've removed it… we can simply focus on INTERMITTENT vs MILD vs MODERATE vs SEVERE

Also, thanks to GSO for confirming.

PBR MODIFICATION - p.369

PEDIATRIC ASTHMA CLASSIFICATION
The shortcuts and mnemonics for pediatric asthma classification below may not cover every possible combination of symptoms, but it comes **extremely close** and should be plenty for the pediatric board exam.

* **INTERMITTENT** ASTHMA: Daytime symptoms twice or less per week.

* **MILD** PERSISTENT ASTHMA: Three times or more per week (during the day).

* **MODERATE** PERSISTENT ASTHMA: Daily symptoms

* **SEVERE** PERSISTENT ASTHMA: Continuous symptoms (throughout the day or multiple times/day)

* **MNEMONICS:**

  - **DAYS PER WEEK CUTOFFS: 2-3-D-C or 23DC** provides the cutoffs for classifying asthma based on the frequency of **daytime** symptoms per **week**. 2 or less = intermittent. 3 or more = mild. Daily = moderate. Continuous = severe.
  - **NIGHTS PER MONTH CUTOFFS: 2-3-W-N** provides the cutoffs for classifying asthma based on the frequency of **nighttime** symptoms per **month**. 2x or less per month = intermittent. 3x or more = mild. **W**eekly or more = moderate. Nightly = severe.

* **PEARL**: For **nighttime** symptoms in young children (0–4 years of age), the classifications are more aggressive and shifted by 1 category. So it becomes 0-2-3-W, where once weekly at night is **severe**.

* **SPIROMETRY** - %FEV1 OF PREDICTED: Use the cutoffs below to classify:

- INTERMITTENT - MILD ASTHMA: > 80% of predicted.
- **MODERATE ASTHMA: 60-80% of predicted.**
- SEVERE ASTHMA: < 60% of predicted.

* **SPIROMETRY** & REVERSIBILITY

REVERSIBILITY refers to an **FEV1 that improves by 12% with bronchodilator** use. If it does, that diagnoses a reversible obstructive process (asthma). If it does not, then it **does not rule out** the possibility of asthma. The test has a **high positive predictive value** only.

* **TREATMENT SHORTCUTS/PEARLS**

- **INTERMITTENT ASTHMA:** For a patient with **intermittent** asthma, **intermittent** use of inhalers as needed is appropriate.
- **PERSISTENT ASTHMA: Persistent** use of inhaled corticosteroids (fluticasone, budesonide, etc.) is the **mainstay of asthma treatment** for anyone with PERSISTENT asthma. This includes mild, moderate and severe **persistent** asthma. Inhaled steroids help with both inflammation **AND** hyperresponsiveness. Side effects include oral candidiasis, dysphonia (dysphonia), and cough.
- **LONG-ACTING BETA AGONISTS:** These should be used in anyone with **MODERATE to severe asthma** patients who have not achieved control with inhaled steroids alone. For the purposes of the pediatric board exam, it's probably safe to assume that anyone with at least moderate asthma should be on both an inhaled steroid and a long-acting beta agonist.
- **PEARLS:** Males, patients with low socioeconomic status, and patients who have had prior intubation have higher mortality rates. Indicators of poor control include frequent ER visits and use of an inhaler 2 or more times per month. Montelukast should be used in patients of any age if the parents don't want to give **inhaled** steroids. Do not choose to give long-term oral steroids. If a patient has sputum production, that **does not** rule out asthma.

==========

QUESTION/ERROR

Please clarify on pg 370. . In 23wf. What is f?

MY THOUGHTS

Great question! I wish I knew!!! I think I meant FREQUENT. I've now changed it to 2-3-W-N under the MNEMONICS area for nighttime cutoffs. Thanks for the catch!

PBR MODIFICATION – p.370 (see asthma section above)

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QUESTION/ERROR
PBR Facebook Crew Post –
https://www.facebook.com/groups/pedsboardreview/permalink/332620740217207/

So DTap vaccine is not indicated in patients 7 years and older, correct? ….. As far as I know, we only give Tdap boosters to adults if they haven't had it previously.

Different PBR Facebook Crew comment:
https://www.facebook.com/groups/pedsboardreview/permalink/333431203469494/

For TDap booster, per Old PREP, it requires 2 years interval from last Td. BUT, new CDC update (6/2012) says "REGARDLESS the interval..."

MY THOUGHTS
I think the PBR was okay… but needed more details including 1 from above. I've modified the topic and also included a new MNEMONIC.

Thanks SS for the help.

PBR MODIFICATION – p.291

TETANUS BOOSTER

DTaP and **DT** are for kids < 7 years of age (DTaP at 2, 4, 6, 15 months and 5 years, and DT for possible tetanus exposure). **Tdap** and **Td** are for older children. If a child is > 7 years old and did not complete the DTaP series, give Tdap. Generally, **Tdap** is given once around 11-12 years of age to continue protecting against tetanus **and** pertussis. **Td** is then given every 10 years after that as a tetanus booster. When a booster is being given **after the age of 7**, give Td. As an FYI… Tdap can be given no matter when Td was last received.

* CLEAN WOUND: If the last shot was < 10 years ago, no need. If it was > 10 years ago, they are due for a Td booster anyways, so go ahead and give it!

* DIRTY WOUND: If the last shot was > **5** years ago, **give a booster**. If the wound is dirty and the immunization status **unknown or incomplete**, give **IMMUNOGLOBULIN + BOOSTER.**

*** MNEMONIC**: Do the D's and T's confuse you? Just remember that the D's get smaller with age. DTaP and DT are for kids < 7 years old. Tdap and Td are for older kids. Also, remember that boosters are only 2 letters.

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QUESTION/ERROR

On page 45; Obesity Pearl states that Obesity is a risk factor for depression, avascular necrosis of the hip

MY THOUGHTS

I'm not sure what I'm addressing… but I did note an error with the help of MK. I've switched out Legg Calve Perthes with SCFE. See below…

PBR MODIFICATION – p.45

OBESITY

The high caloric intake in obesity CAN also result in tall stature with advanced bone age. If obesity is due to hormonal/endocrine issue, patient is usually fat but short + delayed bone age. If no bone age is provided in the question, consider Cushing's as the diagnosis.

PEARL: Obesity is a risk factor for depression and Slipped Capital Femoral Epiphysis (SCFE)

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QUESTION FROM ASHISH
Has this been of help to YOU?

MY THOUGHTS
I really hope so!

PBR REQUEST
I work very hard year round to do everything I can within the constraints of being a practicing physician, a father, a husband, an entrepreneur, and the Executive Director of a non-profit organization.

I truly hope that the PBR books, this Corrections & Clarifications guide, the discounted review questions (from **BoardVitals** and **Exam Master**), the PBR website, the coaching and the PBR Facebook Crew help you pass your board exam!

I hope…

- I've gone above and beyond your expectations
- I've provided an experience unique from any other board review experience you have ever had
- I've given you much more value than the cost of the materials you purchased

So, keeping that in mind... how much has the PBR experience been worth to you?

ONLY if you feel I've accomplished the above… I'd like to ask you to help my non-profit efforts by donating to **AVSAR Volunteers - http://www.avsarvolunteers.org/about**

I have personally donated over $70,000 to date and thousands of hours of my time. Our goal is to raise $50,000 in the coming months, but if you would be willing to donate something… **anything**, it would mean the world to me.

To donate, all you have to do is visit

http://www.avsarvolunteers.org/donation

Thank You!