PBR'S ANNUAL CORRECTIONS AND CLARIFICATIONS GUIDE
- 2016 EDITION -

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A FEW WORDS OF THANKS TO THE PBR COMMUNITY

Every year I like to go through all PBR error submission and send corrections to PBR members before the **initial** certification exam. It's an EXTREMELY time consuming task (takes several full days), but I believe it's worth it.

Although **the information in this guide SHOULD NOT make or break your test-experience** if you have followed *THE PBR EFFICIENCY BLUEPRINT*, several test-takers have previously said that they enjoyed reading the clarifications, and that the review of the guide even helped them correctly answer several questions that came up on the exam.

THANKS TO YOU!

1. Thank you to EVERYONE who submitted **spelling errors, typographical errors, corrections or requests clarifications** from within the PBR by visiting the ERROR page:

   www.pediatricsboardreview.com/**error**

   For everyone who provided a page number, a clear question and a reference – oh my goodness… you rock!

2. Thank you to EVERYONE who submitted **broken links** from within the PBR and the **PBR Picture Atlas** by visiting the BAD LINK page:

   www.pediatricsboardreview.com/**badlink**

3. An absolutely MASSIVE THANKS TO DR. MIKE BLYTH! Mike is a PBR alum, he's PBR's editor, and he'd done a great job of helping us streamline several systems in the world of PBR to make YOUR experience better!

NOW... WHAT IS THIS THING?

I like to address as many concerns about the PBR content BEFORE the initial certification boards in October.

IN ORDER OF PRIORITY, MY FOCUS HAS BEEN….

1. **Addressing error submissions from the PBR Error portal** (**www.pediatricsboardreview.com/error**). Basically, stuff where folks are saying, "*Ashish… I think (or I know) that this is wrong. You should fix it in the book and let folks know about it because it's more than just a spelling or grammar issue.*"
2. **Addressing possible errors/concerns mentioned in the PBR Facebook CREW!** Yes… I kind of "stalk" the group and if I see something comes up that might warrant a correction in the PBR. I set it aside for this time of year to review.
3. **Requests for content clarification through the portal or "The CREW"**. In general, the "**PBR Facebook CREW!**" is meant to help you get the help you need to understand a topic. BUT, if I see that there's a topic that could be explained *better* based on CREW conversation, I make a note of it and try to polish it up for the next edition and address the issue in this guide.

Because the PBR membership continues to grow, there has been EXCELLENT chatter in "**The PBR CREW**." **If you are a member of the "The PBR CREW" but you have NOT been seeing all of the posts**, please visit the private group and **make sure that your NOTIFICATION SETTINGS ARE SET TO ALL POSTS**. This is critical!

ARE YOU NERVOUS BECAUSE THERE ARE CORRECTIONS FOR THE PBR CONTENT?

ALL study guides have errors!

I'm simply the only author who is crazy enough, and passionate enough, to take on something like this prior the boards every year so that you can rest EASY. And instead of just giving you a one page errata sheet based on error submissions, we try to go much deeper in our explanations and we also SEEK OUT areas of improvement to share with you.

For some people, though, the idea that the PBR has errors can be anxiety provoking.

If you're one of those members, please keep in mind that there are OVER 2000 topics within the PBR, and each topic has MANY salient points associated with it. There are probably over 10,000 individual pieces of information in the PBR. Therefore, the number of corrections below is relatively TINY.

So you should rest easy knowing that there is MORE THAN ENOUGH excellent content within your PBR to get you your PASS! The PBR CERTIFICATION SYSTEM has helped pediatricians get ABOVE the national average score after MULITPLE years of failing... so you'll be fine!

WHAT ABOUT IMAGE LINK CORRECTIONS?

We have a very innovative system that allows you to view phenomenal high-yield images across the web. **We have approximately 400 image links in the PBR, but they lead to images that are not owned by PBR.** That means that any given time, an unrelated PBR website that houses a high-yield image might be down. When you notify us of this, it's a HUGE help and we can quickly replace the image link with a new, comparable image.

In the past we would send out replacement image links for the ones that were broken throughout the web. Because of our new systems, all of the image link corrections now happen on the BACKEND.

We just did a huge search and replace of the links. 97% - 99% of the links should now be working without any issues!

If you do find that there's an issue, please notify us immediately by visiting:
www.pediatricsboardreview.com/badlink.

The EASIEST way to go through all of these images is by using the online picture atlas created by Team PBR (called the **Virtual Atlas of Pediatric Pictures**). The VAPP gives you a SUPER fast and high-yield review of board-relevant images.

You can watch the video below to see how it works:

www.PediatricsBoardReview.com/**vapp**

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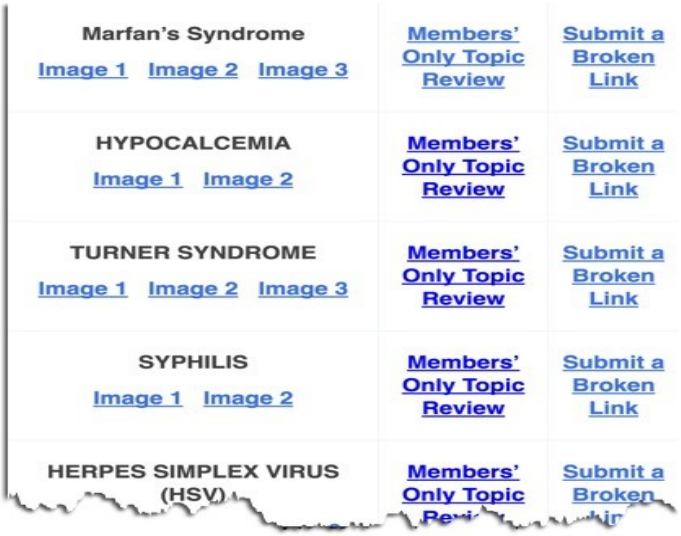

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FREQUENTLY ASKED QUESTIONS

"Is this a complete list of everything that's changing for the new edition?"

NO! The new edition will have MORE additions and modifications. This Corrections & Clarifications Guide includes:

1. A set of absolute notifications because they were true errors that we verified.
2. Several clarifications and discussions around topics that may have been confusing.

"If I have the old book… Should I keep that one or get the new one?"

If you have a 2016 Edition, it DOES HAVE enough information in it to help you pass the initial certification exam or the recertification exam, BUT we tend to add new information, and sometimes even new chapters.

Here are **the 6 main reasons to get the new edition** if you still have an old one:

1. **SAVE AND MAXIMIZE YOUR TIME**
    - If you have an older version of the book and you are taking the boards several months from now, then this is NOT the time to spending your energy cross checking everything in this guide against your older version of the PBR. Please understand that your time is PRECIOUS and needs to be spent EFFICIENTLY and effectively. Plus, there is ALWAYS new content in a new release.
    - Start with a fresh book, transfer any notes/drawings from your previous hardcopy to the new edition as you read through it the first time, and then use the new one as your bible!
2. **COST (No... I'm not just talking about money!)**
    - By cost, I mean money and opportunity cost. The cost of a new book is minimal compared to the hard **financial cost** and **opportunity cost** of FAILING the boards. The financial cost of FAILING includes over $2000 for your board fees, plus the cost of taking time off of work to study again next year (THOUSANDS of dollars of lost income). You also must include the stress and the time away from loved ones as a tremendous unmeasurable cost.
    - If you have some OTHER reason to keep studying from an older edition, so be it… but if it's due to financial concerns, that's actually pretty silly. Sorry… this is the one place in this document where I just have to be blunt. I have such a passion for efficiency and QUALITY USE OF TIME that it really **pains** me to hear about physicians that are trying to go back and forth between the corrections guide and their old study guide in order to save a few bucks.
3. **REFERENCES TO PBR IN THE CREW!**
    - The **PBR Facebook CREW!** comes alive with discussion as the boards approach. Many PBR alumni have said that the Facebook CREW! heavily contributed to their success on the boards. When your peers in "The CREW" are referring to a topic on a certain page, do you really want to (again) waste your precious time fumbling around and trying to find the topic they're referring to?

4. **UPGRADED FORMATS**: Every edition is MUCH better than the previous.
    - **Corrections**
    - **Clarifications**
    - **New image links**
    - **NEW, TIMESAVING INNOVATIONS**. For example, our links used to be EXTREMELY long. Now we have a system that turns http://upload.wikimedia.org/wikipedia/commons/4/45/Aphthous_ulcer.jpginto something easy like www.pbrlinks.com/aphthous1. **HOW COOL IS THAT! Try typing out the 2 different links and see HOW FAST you get to review images using the new PBR link ≡ - these things get me SO GEEKED!**
5. **NEW CORRECTIONS**: There WILL be other corrections that will make their way into the new edition. MANY of the corrections below were included in this guide because of help from the PBR community, and many were done on my own. But there are more that need further investigation before the next edition is released.
6. **NEW CLARIFICATIONS**: Again, there was ACTIVE discussion within the **members' only PBR Facebook CREW**! about board review topics that I THOUGHT were explained well within the PBR. That discussion leads me to believe that I can be EVEN MORE clear in future editions. There will be many additional clarifications and updates in the next edition.

DISCLAIMERS/WARNINGS - PLEASE READ THIS BEFORE YOU GET STARTED

- The **page numbers** in this guide refer to the **2016 Editions of the Pediatrics Board Review** books (covers shown below).

- **DEAR NON-PBR MEMBERS… the PBR Facebook CREW! is a private, members-only area** for anyone who has signed up for a qualifying product. **YOUR REQUESTS TO JOIN WILL BE REJECTED** if you have only signed up to get free info from PBR (free GI & DERM study guides, free emails about new PBR web article, free Q&A discounts, free MP3, etc). **We cross-check all requests** to join "The CREW" before clicking the APPROVE button. This is done in order to keep it a spam-free, private and intimate area.
- **Reminder…** I LOVE being told I'm wrong (sort of), so keep the comments coming! Just keep in mind that the best place to submit error submissions, corrections, requests for clarifications, etc. is here:

www.pediatricsboardreview.com/**ERROR**

LETS GET STARTED WITH THE CORRECTIONS FIRST!

OKAY…. This first section is going to cover TRUE ERRORS that were in the PBR.

There were a few spelling errors this year. Thanks for bringing those to our attention. There are still several celebrity names and common names purposely spelled wrong for mnemonic reasons. For example, Kalvin instead of Calvin was used for K in Klinefelter. These should be pretty obvious. If you see others that don't make sense, let us know.

When it comes to the Corrections & Clarifications Guide, our primary goal is to make sure that we evaluate any content error submissions. Thank you guys SO much for these submissions! I REALLY appreciate it. Got more? Send 'em over!

www.pediatricsboardreview.com/error

ADOLESCENT MEDICINE

The Core Study Guide says, "Estrogen is often given to little old ladies (post-menopausal) to protect them from osteoporosis!" Little old ladies are NOT given estrogen for osteoporosis prevention anymore!!!!

> Partly correct, at least. The offending sentence has been dropped!

No one should be teaching their patients, adult or pediatric to do breast exams! The USPSTF recommends AGAINST self breast exams for all women.

> True. That paragraph has been changed to the following:

BREAST EXAMS

Teaching breast self-examination to women is no longer recommended.

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ENDOCRINOLOGY

I don't understand the "Hawaii 5-0" rule.

> The "Hawaii 5-0" rule has been removed from the 2017 edition since it can be confusing and also because it gives more glucose than is now recommended. The recommended bolus now is 2.5 ml/kg of D10. Slow administration (2–3 ml/min) is preferred in order to avoid hyperglycemia. Thanks for the submission. NEW hypoglycemia section below:

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 minutes 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 **2.5 ml/kg of D10** at a rate of 2-3 ml/min. For adolescents, simply give ½ –1 ampule of D50 (12.5 - 25 grams of dextrose), though D10 is preferred if available.

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.

PEARL: Fifteen grams of sugar can be found in one tablespoon of sugar, one tablespoon of honey, four ounces of fruit juice, or six ounces of soda.

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EMERGENCY MEDICINE & TOXICOLOGY

NO 2016 CORRECTIONS!

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OB/GYN

NO 2016 CORRECTIONS!

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CARDIOLOGY

Diagnosis of Rheumatic Fever

The PBR has been updated to reflect the latest guidelines for the diagnosis of acute rheumatic fever. Two significant differences are:

- Evidence of a previous Group A Strep infection is necessary whether using one or two major criteria.
- When a patient has had a previous episode of ARF, three minor criteria are enough to make the diagnosis.

JONES CRITERIA FOR RHEUMATIC FEVER

The two common ways to diagnose Acute Rheumatic Fever (ARF) using the Jones criteria are listed below. Note that evidence of a previous Group A Strep infection is required in all cases.

* Patient meets 2 of the MAJOR Jones criteria.

* Patient meets 1 major AND 2 minor criteria.

PEARL: Antibody testing such as ASO titers, the Streptozyme test or the anti-DNASE B should be ordered to better correlate with recent infection since a negative screen or culture does NOT rule out a recently resolved pharyngitis. If those options are NOT available, order a Streptococcal screen or culture. Since these can be positive in carriers.

MAJOR JONES CRITERIA FOR ACUTE RHEUMATIC FEVER

Major Jones Criteria for acute rheumatic fever include:

* An asymmetric, migratory, polyarthritis of the large joints (ankles, knees, wrists)

* Signs of carditis: Valves, myocardium, and pericardium can be affected so look for new murmurs, CHF, cardiomegaly, and pericarditis.

* Painless, firm subcutaneous nodules (wrists, elbows, knees)

*** (DOUBLE TAKE) ERYTHEMA MARGINATUM**

- Erythema marginatum is a transient, erythematous, macular and light colored. It is described as being “SERPENTiginous” (snakelike) and the MARGINs are noted progress as the center clears. It is part of the Jones criteria for Rheumatic Fever.
- IMAGE: http://pbrlinks.com/ERYTHEMA1
- MNEMONIC: The E in Erythema is part of the E in jonEs, and the name MARGINatum should remind you to look for an interesting description of the rash’s MARGINs. Erythema MARGINatum.

* Sydenham’s Chorea: Movements of the face and/or extremities without purpose. Some describe it as “purposeless dancing.”

MINOR JONES CRITERIA FOR ACUTE RHEUMATIC FEVER

Minor Jones criteria for acute rheumatic fever include:

* ARTHRALGIAS: Refers to PAIN without inflammation. Note that this is a MINOR criterion.

* Elevated ESR or CRP

* Fever

* Prolonged PR

Note that joint and cardiac criteria can only be used once so, for example, you can’t use arthritis as a major and arthralgia as a minor criterion.

MNEMONIC for the MAJOR JONES CRITERIA: J O N E S

* Joints: asymmetric, migratory, polyarthritis of the large joints (ankles, knees, wrists)

* O looks like a HEART (♥): Carditis = new murmurs, CHF, cardiomegaly and pericarditis.

* Nodules: Painless and firm subcutaneous nodules (wrists, elbows, knees)

* Erythema MARGINatum: The name MARGINatum should remind you of the interesting facts regarding the MARGINs of the rash.

* Sydenham's Chorea: Movements of the face and/or extremities without purpose and sometimes described as "purposeless dancing."

PEARLS:

* Rheumatic fever is only caused by Streptococcal PHARYNGITIS! For the exam, assume that it is NOT associated with skin infections.

* EXCEPTIONS to JONES Criteria:

- CHOREA alone, in the context of a recent Strep infection, may be considered diagnostic.
- So-called "indolent" carditis, i.e. not acute, may be the only manifestation months after a Strep infection.
- Patients with a previous history of ARF or rheumatic heart disease with evidence of recent Strep infection can be diagnosed with ARF recurrence with 3 minor criteria.

The section on negative T waves (p. 107 in 2016 edition) states that "The T wave is negative in newborns in lead V1. The T wave becomes positive/upright after the first week of life." From what I've read, the opposite is true.

> Right, good catch! The book now says:

NEGATIVE T WAVE

The T wave is positive/upright in newborns in lead V1. The T wave becomes negative after the first week of life.

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DERMATOLOGY

The book says that lamellar ichthyosis is associated with *inverted* eyelids, but this should actually be *everted* eyelids.

> Right, the book has been changed to read, "Eyelids seem everted (ectropion)."

==========

NEONATOLOGY

The book says to give 2% fat milk after 2 years but I read elsewhere that it should be 1%. Which one is right?

> The latest recommendation is to give **1% or skim milk** after 2 years. Also, children from 12-24 months who have a family history of obesity or cardiovascular disease, or those who are at risk of being overweight, should be given 2%, not whole milk. The corrected section now reads:

WHOLE MILK

Whole milk should NOT be given to babies until they are 1 year of age. It can cause anemia, renal damage and electrolyte abnormalities, such as hypocalcemia.

DEFINITELY give whole milk from 1−2 years of age. The fat content helps with nervous system development. However, 2% milk is recommended for children who are at risk of becoming overweight, or those who have a family history of obesity or cardiovascular disease. After 2 years of age, SWITCH to 1% or skim milk in order to LIMIT the fat content.

==========

GASTROENTEROLOGY

I thought with pancreatitis bowel sounds were usually decreased (and not increased as noted in the book/website material).

> Correct, the book has been changed.

PEARL: Many GI diseases result in hyperactive bowel sounds. With pancreatitis, bowel sounds can be DECREASED.

==========

PHARMACOLOGY

I came across a part on the Magnesium sulfate that stated on page 214 that it could possibly cause HYPOmagnesemia in neonate when mother has received mag sulfate as tocolytic. However, when I looked it up it appears to cause HYPERmagnesmia which gives the neonate the respiratory depression, etc. Can you please clarify this for me?

> True! Hypermagnesemia is commonly found since the magnesium easily passes the placental barrier. The baby's tone and respirations may be decreased, but this is rarely an issue. The accompanying *hypocalcemia* can be a problem, however. The Core Study Guide has been changed to the following:

PEARLS: **(DOUBLE TAKE) Magnesium Sulfate Infusions** (for t**ocolysis or** preeclampsia) can result in severe hypocalcemia. Treat with calcium gluconate, but keep in mind that they may not respond as quickly as would normally be expected. So if you are given a vignette about a hypocalcemic baby not responding to calcium gluconate, consider magnesium tocolysis as the etiology.

==========

OPHTHALMOLOGY

The PBR says, "If you are given a patient with a cherry red spot on the macula and hepatomegaly**, this is your answer! Fabry and Tay-Sachs do not have organomegaly!" Does this mean Fabry has a cherry red spot?**

> No, *Fabry* does not have a cherry red spot and we've removed it from that sentence. *Farb**er*** disease may have a cherry red spot but it can also have organomegaly. It is so rare that you really don't need to know about it in the differential of a cherry red spot.

==========

HEMATOLOGY

Hemangioma treatment- it says systemic steroid, however propranolol is first line therapy.

> Correct! Thank you! The book has been changed and now reads as follows:

HEMANGIOMAS

Hemangiomas are an abnormal build-up of blood vessels. They eventually self-involute but are dangerous during PROLIFERATION PHASE. They are otherwise benign. They usually look red, but can appear blue if deep (CAVERNOUS HEMANGIOMAS). Proliferation is greatest during the first 6 months, and lesions are largest around 1 year of age. Lesions start to involute around 2 years of age and disappear by 5–10 years of age. If in a benign area, they can be left alone. If in a more sensitive area (near the eyes, ears, nose, throat, or spine), they may require medical treatment with propranolol being the first line drug. Second line therapies include systemic steroids, pulsed dye laser therapy and surgery.

The PBR lists alkalinization with sodium bicarbonate as one of the main treatments for tumor lysis syndrome. I understand this is no longer accepted.

> Correct! The book has been changed to reflect this, and also to add *rasburicase* as an alternative to allopurinol. Thanks. New section is below!

TUMOR LYSIS SYNDROME

Tumor lysis syndrome is an **ONCOLOGIC EMERGENCY**. It occurs due to lysis of large tumors or "blood tumors" (lymphoma). Potassium, phosphorus, and LDH levels are elevated due to release from lysed cells. Treat with allopurinol or rasburicase, aggressive hydration, and careful monitoring and management of electrolytes. **Urine alkalinization by giving sodium bicarbonate is no longer generally recommended**.

==========

INFECTIOUS DISEASES

In the section on measles post-exposure prophylaxis, PBR says that measles immune gobulin (MIG) can be given after the 72 hour window for ~~immunization~~. The PBR does not specify that the MIG can only be given up to 6 days after the exposure.

> Correct! good catch. The book now reads as follows:

- If > 3 days but < 6 days since exposure in a high-risk, unimmunized child, including those who are immunosuppressed or under 5 years old, give measles immunoglobulin (MIG). Also give this if the patient's vaccination status is unknown. Anyone given MIG will need a first-time vaccination, or possibly a re-vaccination, 5 months after the MIG dose.

MEASLES (AKA RUBEOLA)

"COUGH, CONJUNCTIVITIS, and CORYZA" are the classic symptoms of measles (AKA rubeola). Coryza refers to rhinorrhea. Also look in the mouth for KOPLIK SPOTS and on the skin for a rash. The three C's come first, then the Koplik spots, and the LAST symptom to appear is the RASH. The rash starts at the head (around the hairline) and progresses down. The rash resolves after about 5 days. The major cause of death is PNEUMONIA. The virus is transmitted via droplets and is so contagious that patients require negative pressure isolation. Patients are contagious from FOUR days prior to the onset of the rash until FOUR days after the rash appears.

* POSTEXPOSURE PROPHYLAXIS:

- Give the **vaccine** (MMR) if it has been < 3 days since exposure. This includes unimmunized children (or children with an unknown immunization status) who are > 6 months and immunocompetent. If the MMR was given for prophylaxis and the child was only 6 to 12 months of age, s/he will need it again at one year of age.
- The measles **immunoglobulin** (MIG) is reserved for **unimmunized** individuals and those with an unknown immunization status with exposure > 3 days ago, but < 6 days ago. MIG is not given after 6 days. It is also not given to immunocompetent children that have had at least 1 MMR at 1 year of age or later. Any nonimmune person (no evidence of immunity) who received the MIG will need an MMR 6 months after the MIG dose.

- **IVIG** is given to any immunocompromised individual (immunodeficiency, on chemo, low CD4, etc.), regardless of immunization or vaccination status.

* TREATMENT: The treatment for measles is supportive.

* PEARL: Most measles exposures result in MIG being given when prophylaxis is indicated because the most obvious and scary symptom (the RASH) is the LAST thing to present. Late presentation to the doctor means MIG is the only prophylaxis option for anyone exposed.

* NAME ALERT: 

Note that this is not the GERMAN MEASLES caused by the RUBELLA VIRUS.

* (DOUBLE TAKE) MNEMONIC: Imagine a patient stuck in a NEGATIVE PRESSURE ISOLATION room. He's watching MTV and gets pissed because he's bored and all they ever show is reality shows. He grabs the phone and throws it at the "M.T.V." – Negative pressure isolation is required for Measles, Mycobacterium Tuberculosis and Varicella. For VZV, droplet precautions are sufficient if only one dermatome is involved. As mentioned in the Aspergillus section, that, too, requires negative pressure isolation.

In the immunizations section, pg 303-304, PBR calls varicella a pox virus. Though it is the causative agent of chickenpox, varicella is NOT a pox virus. It is a herpes virus.

> Correct, the book has been changed to eliminate the reference to pox virus. New version:

LIVE VACCINES

The live vaccines have traditionally been Measles, Rubella, "Sabin" (AKA oral polio, OPV), Varicella/VZV, Adenovirus (for military only), Mumps, and Yellow Fever. Oral polio is not used in the U.S., so you're unlikely to be tested on it. The newer live vaccines are the inhaled influenza (virus) vaccine (FluMist—currently off the market) and the rotavirus vaccine. So the list includes **MMR, OPV, VZV, Adenovirus, Yellow Fever, inhaled Influenza, and Rotavirus**.

PEARL: For the purposes of the exam, do not give any of these vaccines to a **pregnant teen (see below)** or someone who is **severely immunocompromised**. This includes patients with HIV and a low CD4 count, as well as many of those patients with disorders reviewed in the Allergy & Immunology chapter.

PEARL: If an HIV patient is generally healthy, do NOT hold back any vaccines.

MNEMONIC: MR. SPAMY is ALIVE! This mnemonic includes the older live vaccines, including Measles, Rubella, Sabin (Polio if **oral** OPV), Pox viruses (Varicella/VZV), Adenovirus (for military only), Mumps, and Yellow Fever. This does not include FluMist or Rotavirus! Also, VZV is actually a herpes virus and not a pox virus.

MNEMONIC: MR. FARM-SPY is ALIVE includes all of the live vaccines. Creating a story around one or both of these mnemonics will help even more.

MNEMONIC: Here's a mnemonic that covers some (not all) of the DEAD vaccinations. "**RIP,** Hepatitis **A** & **B**!" = Rest In Peace Hepatitis A & B = Rabies, Influenza, Polio (IPV), Hepatitis A, and Hepatitis B.

Why is answer B correct In the Q & A book, question 39? The explanation says "If mom was given ERYTHROMYCIN, TREAT the baby because it doesn't cross the placenta." The mom in the scenario was given erythromycin, but the correct answer is listed as B.

1. A healthy-appearing child is born to a mother that had poor prenatal care. Late in her pregnancy, she was found to have syphilis that was confirmed with a positive FTA. She was treated with erythromycin 6 weeks prior to the delivery. Non-treponemal titers are obtained from both the mother and the neonate. Both are positive. What's the next best step in management?
    a. Treat the mother and the baby with penicillin.
    b. Treat the mother with penicillin and check the FTA in the baby.
    c. Do not treat the mother. Treat the baby with penicillin.
    d. Recheck the baby's non-treponemal titers in one month.

> Correct. Answer B is wrong, and C is the most correct. The mother has been treated already, but the baby has not, so the baby needs treatment. Checking the FTA is useless since it may remain positive long after the infection is cleared. The baby still needs some clinical and lab evaluation to determine the exact regimen of penicillin required, but will require the drug in any case. Therefore, C is correct.

The book says that the most common cause of pneumonia in children with cystic fibrosis is Strep. pneumoniae. Elsewhere I've seen that it's Staph. aureus. Which is correct?

> Staphylococcus aureus would be the best answer. Pneumococcus is not a common cause of pneumonia in CF patients. The book has been changed:

PNEUMONIA

Overall, Streptococcus pneumoniae (pneumococcus) is the most common etiology of pneumonia in children.

PEARL: If asked what the most common etiology of pneumonia is in a patient with cystic fibrosis, pick Staphylococcus aureus or Pseudomonas aeruginosa, not pneumococcus.

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FLUIDS AND ELECTROLYTES

NO CORRECTIONS!

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STATISTICS

NO CORRECTIONS!

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NEUROLOGY

The first-line medication for pseudotumor cerebri (idiopathic intracranial hypertension, IIH) is acetazolamide (Diamox), NOT steroids.

> Correct. The book has been updated to read,

- (DOUBLE TAKE) PSEUDOTUMOR CEREBRI (AKA IDIOPATHIC INTRACRANIAL HYPERTENSION or BENIGN INTRACRANIAL HYPERTENSION)

  The symptoms associated with **increased intracranial pressure** (ICP) predominate for Pseudotumor Cerebri (AKA Idiopathic Intracranial Hypertension or Benign Intracranial Hypertension). These include headache, nausea, vision changes, and bulging fontanelle. Late findings are papilledema and loss of vision. The disease is **not** benign, and although CT or MRI of the head will not show herniation, a lumbar puncture will show increased intracranial pressure. It can be associated with **excessive vitamin A**, isotretinoin, tetracycline, and thyroxine. Treatment may require acetazolamide, diuretics, and sometimes even a shunt or a surgical procedure to relieve pressure on the optic nerve.

Myasthenia gravis: Tensilon testing is no longer performed due to potential cardiac complications and its subjective nature. The diagnosis is by testing for acetylcholine receptor antibodies; if negative, look for muscle specific receptor tyrosine kinase (MuSK) antibodies; if negative, use EMG with repetitive stimulation.

> THANKS! We interpret this as you bing “partly right.” Tensilon testing has mostly been replaced by serologic testing for autoantibodies. Tensilon may still be useful in some bedside situations. The book has been changed! See blow.

MYASTHENIA GRAVIS (MG)

Myasthenia gravis (MG) findings will include either a baby with **variable** ptosis (not constant, occurring at different times), or an older child with evidence of muscle **weakness that waxes and wanes**. On exam, reflexes are diminished but **not absent**. Myasthenia gravis is an autoimmune disease in which antibodies to postsynaptic acetylcholine receptors **block them from functioning**. Tests include serology for antibodies to the acetylcholine receptor (AChR-Ab) or to muscle specific receptor tyrosine kinase (MuSK-Ab). In some cases the edrophonium (Tensilon)

test is also used. A positive test occurs when administering edrophonium causes improvement in ptosis or eye paralysis. If required, treatment may include **pyrido**stigmine, steroids, plasmapheresis, or worst case scenario, a thymectomy to permanently get rid of the antibodies.

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ORTHOPEDICS

NO CORRECATIONS!

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RHEUMATOLOGY

The book says that the lesions of Henoch Schonlein purpura blanch, but I think this is wrong as they are vasculitic and do not blanch.

> Partly right. Early lesions blanch, but later they become purpuric and non-blanching. The book has been changed.

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 early pink, blanching, flat or urticarial lesions that later become **PALPABLE** and **TENDER** purpura. 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 in the 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://pbrlinks.com/HSP1

STRONG WORK EVERYONE!

THANK YOU SO MUCH FOR CALLING ME OUT!

NOW LET'S GO OVER THE CLARIFICATION REQUESTS!

Again… I've tried to be as concise as I can this year because I know your time is short.

This section is going to cover CLARIFICATION REQUESTS from members, as well as anything that we felt might warrant a clearer explanation.

"Do you think we should study tympanograms?"

> I don't usually like to answer these types of questions in this guide, BUT… I think my answer could help a lot of you from straying too far off of the PROVEN PBR PATH right now.

It's **possible** you'll get ONE question on this. That's too low yield for you to pursue studying now. **Focus on the rest of PBR, which covers ALL of the very LOW HANGING AND HIGH-YIELD FRUIT!... AND SO MUCH MORE!**

"I've seen a couple of practice questions about XXXXXXX and about XXXXXXX. I think you should consider maybe including these topics in the PBR."

Thanks so much for ALL of your submissions. My answer to this question is a resounding "NO." Trust me when I say that it's VERY realistic/possible to pass the exam with a first edition of the PBR from 2011. PBR will NEVER be a mini-Nelsons. Meaning, it'll NEVER be a book where you can turn to for every pediatric diagnosis known to man. It's not meant to help you be the most well-rounded and knowledgeable pediatrician in the world. It is DEFINITELY meant to give you MORE than what you need to pass the boards with a score that is ABOVE the national mean!

Know it inside and out… and ignore everything else. Otherwise, you'll find that there's ALWAYS going to be more information that you could potentially chase. It'll lead you down a rabbit hole… or the BLACK HOLE of Google Search.

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ADOLESCENT MEDICINE

Delayed puberty. What's workup? LH, FSH, estrogen, progesterone?

> The workup can be complex, but focus on the basics. Don't forget the importance of history, family history, growth chart, and physical exam. This quick list has been added to the Study Guide:

BASIC WORKUP OF DELAYED PUBERTY

- **History, family history and physical exam** (Family history of delay? Growth curve suggests slowing or cessation of development? Eating & nutritional issue? Physical signs of a syndrome? Absent sense of smell?)
- **Bone age** helps determine whether the delay is constitutional.
- **Other imaging** if there is a suspicion gonads are abnormal or absent**.**
- **FSH, LH, and either estrogen or testosterone** (according to sex) to distinguish primary and secondary problems.
- TSH, T4, and prolactin
- **Karyotype** in patients with primary hypogonadism.

==========

ENDOCRINOLOGY

In testicular feminization, why are breasts present given that there are no ovaries to produce estrogen?

> The defect in the androgen receptor gene prevents the development of male external genitalia. Even though no ovaries are present, there are pathways through which testosterone is converted to estrogen.

I'm a bit confused with the relations between LH & FSH & prolactin & thyroid hormones in the amenorrhea workup.

1.Progesterone challenge positive. If high LH level = PCOS: is that because the ovaries aren't functional and aren't producing estrogen

2.Low LH: is that because a prolactin tumor SIZE is suppressing LH?? Or is it a feedback of prolactin to suppress LH?? Or both?

3.Same question as 2, for the TSH

> 1. A positive progesterone challenge suggests that estrogen is present but ovulation is not occurring. Two situations causing anovulation are PCOS and low LH. It's not that normal-high LH indicates dysfunctional ovaries, but that it rules out low LH as the cause of ovulatory failure.

2. Prolactin suppresses GnRH. The size of the tumor is not the issue.
3. Hypothyroidism is a known cause of hyperprolactinemia, but the mechanisms are unclear.

How would you know if a patient with a history of diabetes and very high blood sugars is in DKA or hyperosmolar coma?

> Ketoacidosis is the key. Think of the ways to diagnosis that: an increased anion gap metabolic acidosis (electrolytes, ABG, hyperpnea); ketone odor on breath (fruity); ketones on the urine dipstick. All those can be quickly determined from the exam and stat labs.

==========

ALLERGY & IMMUNOLOGY

I'm not sure what the paragraph on penicillin desensitization is saying. "Desensitize patients with a penicillin allergy if they have SYPHILIS IN PREGNANCY or if you are presented with a CYSTIC FIBROSIS (CF) patient with resistant Pseudomonas."

> Other antibiotics can usually be found for someone who is allergic to penicillin. However, there are certain cases, such as syphilis in pregnancy, where penicillin is the ideal drug. In such cases, a patient can be rapidly desensitized to penicillin by receiving graduated doses.

Clarified paragraph:

PENICILLIN (PCN) ALLERGY

In most cases, alternative antibiotics can be used when patients are allergic to penicillin. In some cases, however, such as syphilis in pregnancy, penicillin is the best treatment. ***Desensitize patients with a penicillin allergy if they have*** *SYPHILIS IN PREGNANCY or if you are presented with a CYSTIC FIBROSIS (CF) patient with resistant Pseudomonas (needing an anti-pseudomonal penicillin such as ticarcillin).*

The section in B-cell deficiencies says, "Because of problems with antibody production, you will NOT FIND THE EXPECTED TITERS for bacteria we typically immunize against, including Tetanus, Diphtheria, and Streptococcus (AKA pneumococcus)." Can you clarify how this helps diagnose B-cell deficiencies?

> Children immunized with these antigens should produce the corresponding antibodies if they have normal B-cell function. If the titers are *low* or undetectable, then a B-cell deficiency is likely!

On video of immunology section it is shown capillaries do not blanch, whereas in the 2016 book page 99 it says they blanch. What is the correct answer?

> The correct answer is that they do blanch. We'll work on fixing the video.

==========

CARDIOLOGY - ???

Page 122 first says Strep viridans is the most common cause of endocarditis in children. The next page says Staph aureus is the most common etiology of bacterial endocarditis in children. If the distinction is important, which is right?

> These are two different issues. Strep viridans is the most common cause of all cases of endocarditis as well as *subacute* endocarditis in children. However, when it comes to *ACUTE bacterial endocarditis,* Staph is the most common cause. Have a look at these 3 sections below.

ENDOCARDITIS DEFINITION

Endocarditis is defined as an infected heart valve. In general, STREP VIRIDANS is the most common etiology in children, followed by Staph aureus and then Staph epidermidis (especially if the valve is prosthetic). Findings may include fever (possibly up to 2 weeks), new murmurs (especially mitral or aortic regurgitation), petechiae, splenomegaly (from CHF), tender nodules on fingers/toes (Osler's nodes), and **nontender** macules or nodules on the palms/soles (Janeway Lesions).

ACUTE BACTERIAL ENDOCARDITIS

Patients with acute bacterial endocarditis present acutely toxic with high fever and possibly a septic picture. The most common etiology is **STAPH AUREUS**.

SUBACUTE BACTERIAL ENDOCARDITIS

Patients with subacute bacterial endocarditis present with vague symptoms lasting for weeks, rather than acutely toxic. The most common organism is **STREP VIRIDANS**. Aortic valves are especially vulnerable.

MNEMONIC: "VERDE sounds like VIRIDANS and means GREEN in Spanish. Imagine VERDE DECAY in teeth (a common hideout for viridans) entering the blood stream during flossing and settling on heart valves."

On page 112 you say that a LBBB has an RsR' pattern in the lateral precordial leads. A RBBB has that pattern, a LBBB does not. It has negative (rightward forces) initially followed by delayed leftward forces causing a widened and late QRS in the lateral precordial leads (V5-V6).

> The book says that a right *or* left bundle branch block may have this pattern. You're right that a widened QRS is part of the criteria for LBBB. But it can also have a "rabbit ears" appearance, rsR, in the lateral leads.

In WPW, there is a re-entrant tachycardia that bypasses the AV node. Why do adenosine and vagal maneuvers work to abort an AVRT associated with WPW? I thought we are bypassing the AV-node? At some point, when the tachycardia becomes re-entrant, does it go through the AV node?

> The re-entrant tachycardia is *circular*, passing through the AV node in one direction and the accessory path in the other. The AV node is part of the cycle. That is why blocking it is enough to interrupt the tachycardia. See http://pbrlinks.com/WPW_DIAGRAM, which shows how the circle can go in either direction (prodromic or antidromic).

What are the EKG findings for LVH?

> Lateral chest leads. V4-V6, esp V5 and V6. If you want ONE lead, then V6. Various criteria based on age. Use the EKG as a screening test but NOT as a diagnostic test for LVH. Too many criteria for too many leads.

==========

DEVELOPMENTAL MILESTONES

Last minute question as I am having a brain freeze. For developmental milestones, do we choose the age based on the most common milestones or the oldest milestone?

> For the exam, when we're given a patient with various milestones, we look at the milestone that is most ADVANCED and then we choose the child's age based on that developmental milestone. For the rest of the milestones, we assume that the child is either on track or delayed in certain areas.

In preparing for my MOC exam, I went through some self assessment questions. One dealt with a "stiff legged run." From my studying , I marked 18 months - WRONG. The answer is not 24 months either. I deduced that the answer was 15 months. Looking in UpToDate, a 15 month "runs stiff legged" and a 16 month old scribbles, though the PBR assigns this to an 18 month old. Any comments?

> As the PBR says, "Lastly, there are many sources available for your review on this topic. The timelines do vary since milestones are achieved on a spectrum in reality. Memorize one source (this one) and do your best."

==========

NEONATOLOGY

I always remembered the risk factors for severe hyperbilirubinemia in newborn as High risk: preterm (<37 weeks) + risk factors, but PBR says <38 weeks + risk factors Which one is right?

The correct answer is that the high risk group is defined as gestational age < 38 weeks with risk factors.

RISK FACTORS FOR DEVELOPING HYPERBILIRUBINEMIA

Risk factors for developing hyperbilirubinemia include G6PD deficiency, asphyxia, temperature instability, sepsis, acidosis, albumin < 3, ABO incompatibility, Rh disease, sibling with history of phototherapy, bruising, cephalohematoma, and exclusive breastfeeding that is not going well. SO ALMOST ANYTHING IS A RISK FACTOR. Focus instead on identifying neonates that are at high and medium risk of developing hyperbilirubinemia, as well as memorizing the bilirubin levels at which phototherapy should be initiated.

* ***HIGHEST RISK BABIES:*** *35–37 + 6/7 weeks WITH risk factors*

* *MEDIUM RISK BABIES: 35–37 + 6/7 weeks WITHOUT risk factors OR ≥ 38 weeks WITH risk factors.*

* *LOW RISK BABIES: ≥ 38 weeks and well*

* *START PHOTOTHERAPY ON HIGH RISK NEONATES IF:*
    - *TOTAL bilirubin > 8 at 24 hours*
    - *TOTAL bilirubin >11 at 48 hours*
    - *TOTAL bilirubin >13 at 72 hours*

SHORTCUTS FOR WHEN TO START PHOTOTHERAPY IN MEDIUM AND LOW RISK NEONATES:

* *MEDIUM risk: The bilirubin threshold increases by 2 (so approximately 10, 13, and 15).*

* *LOW risk: The bilirubin threshold increases by 4 (so approximately 12, 15, and 18).*

PEARL*: NONE OF THE ABOVE-MENTIONED RISK FACTORS AND BILIRUBIN THRESHOLDS APPLY TO PREMATURE NEONATES < 35 WEEKS.*

PEARL*: If there is ANY concern that the child will develop hyperbilirubinemia, have the family follow up at 48 hours.*

==========

GASTROENTEROLOGY

In Wilson's disease, is the serum copper level high or low? I thought it was high.

Understandably a confusing topic...

Ceruloplasmin is low and copper is not properly incorporated into ceruloplasmin, so the serum copper serum level is low, but the tissue copper level is high.

H. PYLORI TREATMENT – are you sure it's right in the PBR?

>Everything looks okay in the PBR. PPI + Amox + Metro is acceptable since some kids don't tolerate Clarithro. Another option is a PPI + Clarithro + Metro.

HELICOBACTER PYLORI

* *PEPTIC ULCER DISEASE (AKA H. pylori induced PUD)*

If a patient is diagnosed with an ulcer of any type that is found to be positive for H. pylori, treatment will require a proton pump inhibitor (PPI) and antibiotics. Possible regimens include:

- PPI + Amoxicillin + Clarithromycin
- PPI + Amoxicillin + Metronidazole (good if the patient can't tolerate clarithromycin)
- PPI + Clarithromycin + Metronidazole (good if the patient is allergic to penicillin)

* *NODULAR GASTRITIS: The most common etiology is H. pylori. An EGD with biopsy (samples sent for pathology) is the gold standard for diagnosis. This can also be found in Crohn's disease.*

  - ***IMAGE:**http://pbrlinks.com/HELICOBACTER1*

* *CAMPYLOBACTER-LIKE ORGANISM TEST (AKA CLO test or Rapid Urease Test): Just know that this can be used at the time of an EGD to help diagnose. It's faster and cheaper than sending a biopsy specimen to pathology, but it is not as specific as an EGD with biopsy.*

* *UREASE BREATH TEST: This is a noninvasive means to attempt diagnosis of Helicobacter pylori.*

==========

GENETICS & INHERITED DISEASES

What are the current recommendations for Down syndrome and sports participation screening for atlantoaxial instability (AAI)? I found conflicting things on Google. That is, Special Olympics we do screen with x-ray, but not otherwise?

> Of course you will find conflicting things on Google! The answer to your question is that screening asymptomatic children with Down syndrome is **not** recommended by AAP guidelines, but it is required for participation in the Special Olympics. More important is the need to be vigilant for possible *symptoms* of cord compression in *all* children with Down syndrome, and to do imaging on those patients in a timely way. The risk of a child with asymptomatic AAI having a devastating injury because of it is very low, not even well-defined, hence the controversy. In other words... It's controversial, and not in the PBR... so save the brain power for something else ^

I think Angelman Syndrome should be maternal imprinting, because the maternal gene on chromosome 15 is missing, but PBR says paternal imprinting.

>The book is right on this one but this is a fairly tricky topic. *PATERNAL imprinting* refers to a situation where the MATERNAL gene is defective or missing. In Angelman Syndrome, the maternal gene is missing or dysfunctional, resulting in PATERNAL imprinting. The reverse is true in Prader-Willi.

ANGELMAN SYNDROME (AKA ANGELMAN'S SYNDROME)

Angelman syndrome (AKA Angelman's syndrome) patients can be MALE or FEMALE so don't let the name fool you! These patients tend to be happy, "angelic" and laugh frequently. They are developmentally delayed, and have delayed speech. They are easily excited, can have seizures, and are ataxic with what is referred to as a "puppet gait." The disorder results from the absence or the dysfunction a gene on the MATERNAL copy on chromosome 15. It's a special type of

gene. In normal children, both maternal and paternal copies function but they have DIFFERENT effects. If the maternal copy is MISSING, or dysfunctional, or if it has a mutation that makes it behave like the PATERNAL gene (called paternal imprinting), then Angelman Syndrome develops. Also, if the child gets TWO paternal copies of chromosome 15 (paternal disomy) and NO maternal copies of chromosome 15, then Angelman Syndrome develops. The gene location is probably not important for the test, but the fact that it is KNOWN means that this disorder can be diagnosed by FISH.

***PEARL**: The "puppet gait" terminology may have fallen out of favor but refers to an unstable and jerky gait like that of a puppet. The concept of unilateral parental disomy can be confusing. This is a great exam question for the pediatric boards. Hopefully the mnemonic below helps.*

***MNEMONIC:** Rename the disease from AngelMAN to AngelWOMAN to remind you that it can ALSO occur in females.*

***MNEMONIC:** Imagine a FEMALE ANGEL with wings. She has a SMILE on her face and is wearing a t-shirt that says "DADDY'S LITTLE ANGEL." Even though she has wings, the man upstairs decides he is going to pull her to the clouds with some PUPPET STRINGS.*

** **KEY:** FEMALE ANGEL represents that this is a male OR female disorder, SMILE represents frequent laughter, DADDY'S LITTLE ANGEL represents paternal disomy in which she is carrying two sets of Dad's genes, and the PUPPET STRINGS is to remind you of the "puppet gait."*

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 be found in BOYS or GIRLS. This mechanism of this disorder is the mirror image of that of Angelman's. It occurs due to the absence or dysfunction of the PATERNAL copy of a gene in the same region of chromosome 15 as in Angelman syndrome. It also occurs when two maternal copies of chromosome 15 were received and no paternal copies (maternal disomy), and also when a mutation of the paternal gene makes it behave like the MATERNAL gene (called maternal imprinting). 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://pbrlinks.com/PRADERWILLI1*

** **IMAGE**: http://pbrlinks.com/PRADERWILLI2*

* ***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. Imagine a HUGE, OBESE, and DUMB FISH/WHALE named FREE 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 this big DUMB FISH is stuck on the dock. He's HUNGRY. He's thrashing back and forth, and he's FLOPPING his tiny WILLY all around.*

* ***MNEMONIC IMAGE****: http://pbrlinks.com/PRADERWILLI3*
    - ***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 fins.*

What's the major valve problem in Marfan's: Aortic or Mitral problem?

> Can actually have AR, MR, or MVP. Focus on the MVP for the boards.

(DOUBLE TAKE) MARFAN'S SYNDROME (AKA MARFANS SYNDROME)

Classic features of Marfan's Syndrome (AKA Marfans Syndrome) include tall stature with long and thin upper extremities, long fingers, a pectus deformity, joint flexibility/hypermobility, and possible cardiac problems. Cardiac problems may include ***mitralvalve prolapse (MVP), aortic dissection,*** *and mitral or aortic regurgitation. Patients may have a high arched palate and a speech disorder, but do NOT have cognitive deficits. Patients are also at risk for esophageal perforation.*

**** PEARLS:*** *Patients can have subluxation of the lens, which may also be seen in Ehlers Danlos and homocysteinuria. If they mention SUPERIOR subluxation of the lens, pick Marfan's. Any patient with Marfan's should not be cleared for sports participation until they have had an echocardiogram and an evaluation by a cardiologist. If they mention "arm span greater than height," you're done.*

**** IMAGE****: http://pbrlinks.com/MARFANS1 - Please do not get distracted by the reading. Look at the images and move on.*

**** IMAGE****: http://pbrlinks.com/MARFANS2*
* ***MNEMONIC****: http://pbrlinks.com/MARFANS3 - Michael Phelps won several gold medals. Isn't that just like winning the Most Valuable Player (MVP = Mitral Valve Prolapse)?*

"Is retinitis pigmentosa AD, AR, X-linked?"

> Honestly… b/c it's so many things, it doesn't matter b/c you won't be tested on it. In the book it's listed under "other autosomal dominant disorders" b/c regardless of the proportion of identified mutations, most of the **clinically relevant** cases tend to be AD… but again, even if I'm wrong… it doesn't matter. Just look for a family history.

** RETINITIS PIGMENTOSA: Retinitis pigmentosa is a retinal dystrophy that eventually leads to blindness.*

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

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INFECTIOUS DISEASES

The PBR says that a TB skin test reaction *>10 mm* is considered positive for ANY risk factor. Any thoughts on what those risk factors are? The things I thought were risk factors, immunocompromised and close contact exposure, are said to make a *5–10 mm* reaction qualify as positive.

> The CDC lists two categories of risk factors. If a person has one of the higher risks, then a 5–10 mm reaction counts as positive. If they have only the lower risks, then they need a >10 mm reaction. The higher risk factors are:

- People who are potentially immune suppressed
- Those who are recent contacts of someone with TB
- Those with CXR findings consistent with previous TB

The lower risk factors, which cause a reading of >10 mm to be positive, are basically situations where the risk of exposure is higher than normal:

- Recent immigrants from high-incidence countries;
- Children less than 4 years old
- Residents and employees of prisons etc.
- Children and adolescents exposed to high-risk adults.

Note that these categories just make sense, so you don't have to spend a lot of time memorizing them. Clearly, people who are immune suppressed, have signs of TB on their X-ray, or are recent contacts with *known* TB patients are at particularly high risk, while those with other factors only have a general, population-based increased risk.

"What is the consensus on pertussis prophylaxis after 6 wks of age? The PBR says EES, but PREP says azithromycin. What is your take on this?"

> Even the Red Book doesn't really seem to have a preference, so I don't think they could give you both options. For less than 1 month of age, definitely Azithro due to concerns for pyloric stenosis.

I stand by the info below.

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 breathe, 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. The CDC and AAP say "any" macrolide is okay between 1 month and 6 months of age. Recommendations for > 6 months of age are similar. 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). If dose NOT decrease the "whooping," or paroxysmal stage. Erythromycin, clarithromycin and azithromycin are all acceptable agents. Use Azithromycin for children < 6 months of age due to concerns for erythromycin-induced pyloric stenosis. Any macrolide should be okay for after 1 month of age, and because of FDA regulations and* ***much*** *uncertainty as to exactly which macrolide "should" be used in infants, you will NOT be expected to choose one macrolide over another for infants > 1 month of age.*
MNEMONIC*: If it ends in –ELLA, it's probably a Gram-negative organism! Brucella, Shigella and Salmonella fit better elsewhere in this chapter.*

"Medstudy says also not to give IPV if pregnant"

> It's a precaution only, so you won't be tested on that nuance - http://www.cdc.gov/vaccines/recs/vac-admin/contraindications-vacc.htm

Btw… why are your reading that?!? And what super-complicated clinical scenario would even require IPV in an unvaccinated teenage pregnant patient? Meaning, is that even a board relevant piece of information that belongs in a board review book for pediatricians?!?

Umm… no. Stick close to your PBR and close all of your other books.

In the PBR in the C5-C9 deficiency it says something along the lines of, "give meningococcal vaccine early." How early should it be given?

> Give in the first year of life at the usual visits. Exact schedule depends on the type of vaccine. Newly edited section for 2017:

C5–9 COMPLEMENT DEFICIENCY

C5–9 complement deficiency has an unusual predilection towards NEISSERIA infections (both MENINGITIDIS and GONORRHEA). Prevent by early meningococcal vaccination, during the first year of life.

Is there a treatment for sporotrichosis (rose picker's disease)?

Yes. There are multiple. The primary treatment would be **itraconazole**, or amphotericin B for those who are severely ill. Other treatments include terbinafine, fluconazole, and potassium iodide ("saturated solution of potassium iodide," "SSKI").

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FLUIDS AND ELECTROLYTES

I am LOST when people start talking about Anion Gap and Osmolar Gap. Yes, yes, I understand the formulas BUT WHAT THE HECK DOES IT MEAN TO THE PATIENT????

> The anion gap and osmolar gap are quite different and are not related to one another. An elevated (aka "positive") *anion gap* refers the presence of a gap metabolic acidosis. MUDPILES is the mnemonic that you typically go through to find the cause of the gap acidosis when you see a + gap.

The *osmolar gap* is used to determine whether there are significant amounts of "stuff" in the serum that should NOT to be there. Ordinarily, most of the osmoles in the serum should be sodium (plus its balancing anions), some glucose, and some urea (BUN). There are, of course, some other things in relatively small amounts. If the *measured* osmolarity is close to the *calculated* osmolarity (using sodium, glucose and BUN), then we know that there isn't too much "extra stuff" around. If, however, the measured osmolarity is much higher than the calculated one, then there must be something else: alcohol, ketones, organic acids, lipids, or non-glucose sugars are some of the main causes.

An *anion gap metabolic acidosis* means a metabolic acidosis when the anion gap is *abnormally large*. This means that some anion other than chloride is present in excess. It's a "gap" only because those other anions are not routinely measured as serum electrolytes. Bicarbonate and chloride are measured, but lactate and organic acids are not. So if you add lactic acid to the blood, then you'll get a "gap" acidosis. If you add hydrochloric acid, or replace bicarbonate with chloride, then there will be no "missing" anions and no increased gap.

So, a non-gap acidosis is basically due to loss of bicarbonate or addition of chloride, while a gap acidosis is due to other acids such as those produced by poisoning. Again, see the MUDPILES mnemonic for the list.

In the explanation of the delta gap, on page 327 in the 2016 Core Study Guide, what is meant by, "Is the bicarb is lower than expected?" Do you mean that the bicarb is lower than 24 mmol/l, or that it is lower than predicted by the delta gap?

> The issue is whether the bicarbonate is lower than **predicted** by the delta gap. If there is *only* a gap acidosis, then the delta gap should equal the delta bicarb. For example, if the anion gap is 16, then it's 4 more than normal, and delta gap = 4. In a pure anion gap acidosis, the bicarbonate should fall by that much. So it should fall by 4. Predicted bicarb would be 20. If the measured bicarb is even *lower than that*, then there's LESS base around than expected, which means there's an ADDITIONAL acidosis present. It's a

NON-GAP acidosis.

Why does hyperventilation cause low calcium?

> Hyperventilation causes low *ionized* or free calcium in the blood because hydrogen ions and calcium compete for the same negatively charged binding sites on albumin. Take away hydrogen ions (alkalosis), and more calcium can bind to the albumin, reducing the amount of ionized calcium in the blood.

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NEPHROLOGY

Could you please explain a little more why ACE inhibitors should NOT be used in renal artery stenosis???

> In renal artery stenosis, the kidney perfusion is LOW due to the stenotic renal artery. The autoregulatory response is for angiotensin II to constrict the POST-glomerular arterioles, thus raising the glomerular pressure. **Angiotensin**-converting-enzyme **inhibitors** blunt this response.

> Typically, the greatest danger is when there is BILATERAL renal artery stenosis since neither kidney can "pick up the slack."

Proteinuria: If the urine protein to creatinine ratio is less than 0.2 with first AM void, are we assuming it's normal?

> Well, we're not *assuming* it's normal. The protein:creatinine ratio of < 0.2 is normal, so we know that the child does not have persistent, non-orthostatic proteinuria. If the history, exam and the rest of the urinalysis is normal, then the recommendation is to retest the urine in a year.

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ORTHOPEDICS AND SPORTS MEDICINE

Regarding developmental dysplasia of the hip (DDH), is imaging required at 2 weeks of age or older for both boys and girls with history of breech presentation?

> Girls with breech presentation have the highest risk of DDH and definitely require imaging. For boys with breech presentation, imaging is optional, since the risk of DDH is lower, but still substantial at 26/1000.

DEVELOPMENTAL DYSPLASIA OF THE HIP (DDH)

Infants with developmental dysplasia of the hip (DDH) may be noted to have a leg-length discrepancy, extra creases at the thigh, "clunks" or "clicks" on exam. A majority of newborn

"clunks" resolve by 2 weeks of age. So do NOT obtain any imaging at the time of birth. ALL patients with an unequivocal Barlow or Ortolani maneuver (meaning there's no question in your mind on exam) should be referred to an orthopedist for evaluation. There is ***only*** *one category of infants in which the American Academy of Pediatrics guidelines REQUIRES further imaging. It's the category of* ***girls with a breech presentation****. There are two categories in which imaging, or referral to an orthopedist, seems strongly recommended. These include* ***boys with a breech presentation*** *and* ***girls with a history of an affected first-degree relative****. For the purposes of the exam, you should order imaging for any child that fits into one of these three categories or has a positive exam. Everyone else can be followed up with serial exams if the exam is negative. One equivocal exam is okay but if it is equivocal on repeat examination at 2 weeks, then refer to orthopedics or obtain imaging. For children* ***under 4 months of age, ultrasound*** *should be used. After* ***4 months of age, hip x-rays should be used****. Treatment of DDH requires a Pavlik harness.*

* ***PEARL:*** *The guidelines seem to suggest that referral to an orthopedist is the preferred option when there is concern. On the exam this may not be an option since the ABP likes for YOU to make such decisions instead of leaning on consultants. In that case, you will need to decide on the modality of imaging. The age cut-off for ultrasounds is 4 months,* ***NOT*** *6 months. NEVER image before 2 weeks of age. For a child with no clinical signs of DDH on exam but with a NEED for evaluation based on high risk factors, you can ultrasound at 6 weeks or obtain radiographs at 4 months of age. When it comes to the Barlow and Ortolani signs,* ***if EITHER of them are positive, refer or send for imaging*** *(after 2 weeks of age)! ALL children should be "screened" periodically at the well-child visits by EXAM! Meaning, if you're asked if you should "screen" a child for DDH at the 2-month visit, the answer is always going to be YES. Lastly, if you encounter an asymptomatic child that was supposed to get imaging (e.g., breech girl) but never did, and the patient is now 5 or 6 months old, GET IMAGING even if the exam is normal!*

* ***IMAGE:*** *http://pbrlinks.com/DDH1*

* ***IMAGE:*** *http://pbrlinks.com/DDH2*

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AWESOME! YOU'RE DONE! – WHAT NOW?

1. Read the PBR "Exam Day" article. It's a MUST read. It will give you a great deal of insight into your exam day. I'll list the link at the end of the document.
2. **Go back to your core PBR study material!** At the end of the day, THAT is what will help you pass the boards.
3. If you're not a PBR member yet, this is a GREAT time to join!

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You'll also get access to some GREAT webinars covering content from each of the chapters.

Some of my favorites include the Acid Base chapter webinar of 2014 and the Fluids & Electrolytes webinar of 2015. I decided to do an impromptu 30-minute session of going over six awesome acid base questions to demonstrate how to easily tear apart acid base questions. People were confused about the delta delta, when to check for compensation, Winter's formula, etc… but by the end, I was getting comments like, "the light bulb just went off!"

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And if you want to CRANK THROUGH the videos and webinars, you can do so at **double, or even TRIPLE, speed** using an awesome tool that's FREE to try. It's called MySpeed and you can check it out here: **http://pbrlinks.com/myspeed**. Or, you can watch me demo the OVC and the speed tool here:

http://youtu.be/YLqLJkArceI

Okay! That's it!

Now go study in whatever way is going to give you the MOST REPETITION AND LEARNING. If that's simply reading over and over again, GREAT!

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Lastly, don't forget to read some excellent information about your **exam and how you should structure your big day**. The article below is a MUST read, so please click the link below and take 5 minutes to read it:

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Alright guys… Team PBR has worked **VERY HARD** this year to help you with the tools and guidance you needed to prepare for the boards. If you followed our lead, then I'm sure you're going to rock your exam! Trust me, if I can do it… so can you!

Best of luck on your board exam.

Sincerely,

Ashish & Team PBR