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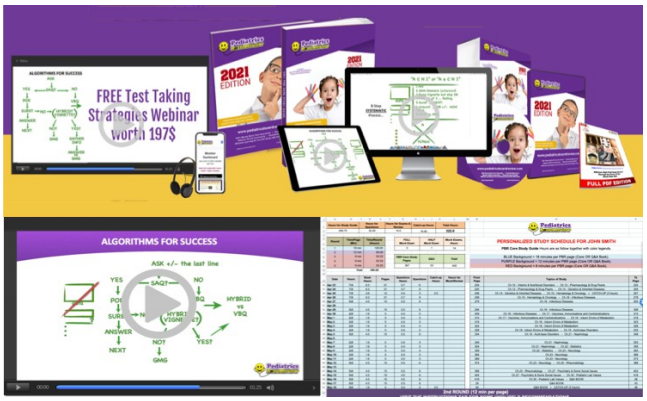

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A FEW WORDS OF THANKS TO THE PBR COMMUNITY

Every year we like to go through all of the PBR error submission and send corrections to PBR members before the **initial** certification exam. It's an EXTREMELY time consuming task (takes weeks), but 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 these corrections and clarifications have helped them correctly answer 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. JOHN COLE! John is a PBR alum, he's been an OVC Summertime Q&A Webinar panelist and he's also taken on the role of being PBR's editor! He has a HUGE heart, and he acts as our editor to help you and the PBR community.

4. A huge thanks to our 2020 Online Video Course Summertime Webinar speakers. They contributed to MANY of the chapter corrections or revisions!
   - Dr. Amar Dave
   - Dr. John Cole
   - Dr. Asalim Thabet
   - Dr. Kara Wada
   - Dr. Shamila Zawahir
   - Dr. Arpit Agarwal
   - Dr. Lina Huerta-Saenz
   - Dr. Stephanie Moses
   - Dr. Kirshma Khemani
   - Dr. Moshe Cohn
   - Dr. Raza Bajwa

NOW... WHAT IS THIS THING?

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

IN ORDER OF PRIORITY, OUR 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 questions from our Online Video Course question portals and webinars.** The summer is filled with content-based webinars, and many excellent questions, corrections and clarifications come to light during those sessions. We try to address as many of those as possible before the Initial Certification Exam.
3. **Addressing possible errors/concerns mentioned in the PBR Facebook CREW!** Yes… We 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.
4. **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 with other resources... 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.

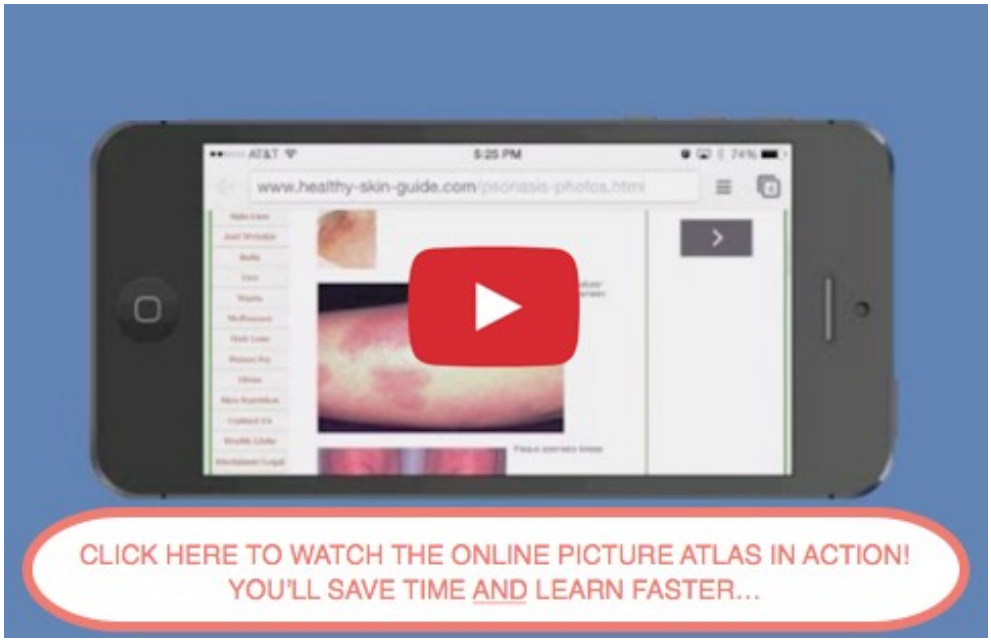
In the past we would send out replacement image links for the ones that were broken throughout the web. Because of our new systems, all of the image link corrections now happen on the BACKEND.

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:



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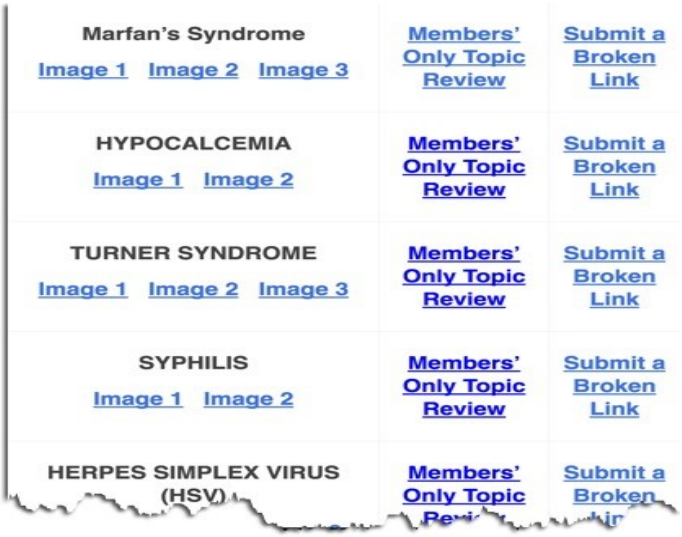

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FREQUENTLY ASKED QUESTIONS

"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. Clarifications and discussions around topics that may have been confusing to readers, or to attendees of our Live Summertime OVC Q&A Webinars.
2. A set of absolute notifications because they were true errors that we verified.

There are more submissions that we need to do additional research on, and NEW submissions for consideration that are still coming in. Those will likely result in additional changes to the next edition.

"I'm taking the exam NEXT YEAR. If I have the old book… Should I keep that one or get the new one?"

Your older edition likely has enough information in it to help you pass the initial certification (or recertification) exam. BUT, we are adding new information (new topics, new subtopics, and possibly even a new section, etc.) based on member feedback.

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

1. **IT'S FRUSTRATING TO HAVE AN OLDER BOOK. WATCH!**
    - You will see in this guide that many submissions will reference specific page numbers and specific lines within a paragraph. This happens all year long, especially in our private forum. This is NOT the time to be spending your energy cross-checking everything in this guide against your older version of the PBR. Your time is PRECIOUS and needs to be spent EFFICIENTLY and effectively.
    - 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! The purging of "the old" and the starting with "the new" is also a great MENTAL RESET.
2. **NEW CONTENT:** There is ALWAYS new content in a new release. 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 corrections that need further investigated before the next edition's release.

3. **NEW CLARIFICATIONS**: 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.
4. **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're planning on using the older version due to financial concerns, that's actually pretty silly. As your guide on this journey, I feel that it's important that I be blunt when it comes this point. 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 dollars. Plus, having a NEW and CLEAN book that you can start going through with my highlighter trick is a much better means of achieving DEEP STUDY.
5. **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?
6. **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.jpg into 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 EXCITED!**

DISCLAIMERS/WARNINGS

PLEASE READ THIS BEFORE YOU GET STARTED

- The **page numbers** in this guide refer to the **2020 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

LET'S GET STARTED WITH THE CORRECTIONS FIRST!

OKAY…. This first section is going to cover TRUE ERRORS that were in the PBR and possibly some clarifications that are going to result in CHANGES for the next edition.

Got more errors to submit? Send them over!

www.pediatricsboardreview.com/ERROR

ADOLESCENT MEDICINE

PBR States that emergency contraception can't be given three days after intercourse and be effective. There is now actually a new medication that is effective five days after intercourse to prevent pregnancy. It is called Ulipristl. I was just at a practical pediatrics conference and this was one of the post-test questions. I believe they get these questions for the post assessment from the prep self-assessment questions.

> You're right! Thank you! We've only been talking about Plan B for years. But there's a new medication called ulipristal (a progesterone agonist/antagonist). It can be used up to 5 days after intercourse. It will be added to the next edition of the text.

For secondary amenorrhea, PBR book says- 6 months without menses if the patient had previously regular periods or 12 months without menses if the patient was previously irregular. But UpToDate and AAP says absence of menses for 3 months in someone who had regular menses before and 6 months for someone who had irregular menses? Which definition is correct? Please suggest. Thanks.

Good question, we have gotten similar questions over the past few years so I think it is time to update the book in the next edition.

> Primary amenorrhea is the absence of menses at age 15 years in the context of normal growth and secondary sexual characteristics (SMR 2-5 pubic hair and breast development). However, if by 13 years of age there has base been no menses and there has been no development of secondary sexual characteristics (child is still in SMR 1), then evaluate for primary amenorrhea.

> Secondary amenorrhea is the absence of menses for more than 3 months in girls who previously had regular menstrual cycles, or the absence of menses for more than 6 months in girls with irregular menses.

Are the following 2 statements the same by any chance? Can you please clarify, especially statement two?

On page 58- no menses after the breast contour changes from a mound-on-mound to a round appearance (SMR 4-5)

On page 62- "delayed puberty can be defined as lack of MENSES within 2 years of the presence of both SMR 4 breasts and pubic hair"

> See the above question for further clarification we will correct the statement on page 62 in the next edition of the book. When breast development happens, 2-3 years later there should be menstruation. If not, it's delayed.
> Menses is usually between SMR3 and SMR4. Some not till SMR5.
> Board questions will be about the most typical scenarios. If SMR4 and no menses for 2 years, consider it to be amenorrhea and delayed puberty. So do a workup.

==========

ENDOCRINOLOGY

There were no endocrinology corrections for 2020!

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OB/GYN and some STD's

There were no ob/gyn corrections for 2020!

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ALLERGY AND IMMUNOLOGY

There were no allergy and immunology corrections for 2020!

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CARDIOLOGY

I am re-watching the video of the ask the expert webinar. The lecturer shows a picture to explain right vs left atrial enlargement. In the image it says there is peaked p waves for RAE and M shaped p wave in LAE but in lead II. Our book states that this in seen in V1.

Sorry for the confusion. This does need to be updated and we have it on our radar for the 2021 edition of the Pediatrics Board Review Core Study Guide. Here are the correct facts.

> Left atrial enlargement (LAE) has a **broad, bifid P wave in lead II**. Right atrial enlargement (RAE) has a peaked P wave in V1 but may also show peaking in the inferior leads (II, III and aVF).

> Newborns are born with right axis deviation leading to upright T waves in the V1 (right precordial leads). The T waves become negative in V1 (right precordial leads) within the first week of life as the pulmonary vascular resistance decreases. The T wave in V1 should never be positive before 6 years of age and may remain negative into adolescence. A positive T-wave in the earlier time frame can indicate right ventricular strain.

Aortic regurgitation is early diastolic; however, it is listed as an exception to the pearl on page 123, can you explain why it is an exception to this pearl?

> Thanks! Our PEARL on regurgitation murmurs is correct, and AR fits right into it and is not an exception to the rule. The “Aortic regurgitation is an exception.” has been removed from the pear. The updated PEARL from page 123 is below.

PEARLS:

“STENOSIS” murmurs generally start in the **middle** of a cycle (mid-systolic or mid-diastolic). Stenotic means something is only “half” way open!

“REGURGITATION” murmurs generally start at the **beginning** of a cycle (early systolic or early diastolic). Mitral valve prolapse is an exception (this is different from MV regurgitation).

==========

DERMATOLOGY

On page 156 of 2020 edition, there are a couple of mistakes on the scabies section. Only treat permethrin neck down because scabies does not infect face/head. As an aside it's an useful clinical giveaway when I have a patient with an itchy rash which includes the face that I am not dealing with scabies. (Thank goodness!). Also, you MUST retreat whole family in 7-10 days otherwise will have recurrence from eggs laid beneath skin that will hatch. Finally, live mites will never be found on physical exam. They are microscopic so only scraping under microscope will visualize.

Thanks! We're making some corrections. Please see the new version of this topic below.

> **SCABIES CORRECTIONS (updated content underlined):**

> Scabies presents as linear, papular, erythematous, **pruritic**, vesicular, and crusting lesions most often seen in areas with CREASES (wrist, groin, webbing of fingers). You may see burrows. Treat with permethrin overnight (8-14 hours) from neck to toe and wash off the next morning for the **entire family**. Re-treat the patient **7-10 days** later because eggs can hatch up to 10 days later. Try topical steroids or antihistamines for symptomatic cares in the interim. An alternative treatment is oral ivermectin due to ease of administration in older children.

> **PEARL**: Unlike papular urticaria, lesions are not in crops.

> **IMAGE**: www.pbrlinks.com/SCABIES1

On page 157 in the top section of Head Lice, you have to change the words "Unlike scabies" to "Similar to scabies". I think crusted scabies is important to know for exam, but maybe it's not so high yield.

Thanks! Please see the updated version below.

> **PEDICULOSIS CAPITIS (AKA HEAD LICE) CORRECTIONS (updated content underlined):**

> Pediculosis capitis (AKA head lice) results in nits/ova of the lice at the hair shafts, especially in the occipital area. Treat with permethrin. The patient will have more symptoms at night when lice tend to be more active. Itching is from the bites. Similar to scabies, **repeat permethrin again in 7–10 days** because eggs can hatch up to 10 days later.

> **PEARL**: If an African American child is pictured, it is NOT lice.

> **IMAGE**: www.pbrlinks.com/HEADLICE1

Incontinentia pigmenti to be clearer it should read Death for all males before birth on page 152.

Thank you! Please see the updated version below.

> **INCONTINENTIA PIGMENTI CORRECTIONS (updated content underlined):**
> Incontinentia pigmenti is a severe X-linked DOMINANT disease that results in DEATH for all MALES before birth. If presented with this as an answer choice, make sure the ABP vignette refers to a FEMALE patient. There are four stages of this disorder: Inflammatory vesicular phase, followed by a verrucous phase, followed by the hyperpigmentation phase noted along the **lines of Blaschko**, and finally a phase in which the hyperpigmentation disappears. This can leave atrophy or hypopigmentation behind.
> SYSTEMIC ASSOCIATIONS: **DELAYED DENTITION**, mental retardation, paralysis, **PEG teeth,** and seizures.
> **IMAGE**: www.pbrlinks.com/INCONTINENTIA1
> **IMAGE**: www.pbrlinks.com/INCONTINENTIA2
> **IMAGE**: www.pbrlinks.com/INCONTINENTIA3
> **IMAGE**: www.pbrlinks.com/INCONTINENTIA4
> **MNEMONIC**: As WOMEN age, they tend to have more "INCONTINENTs." Incontinentia = Female patient. Imagine a WOMAN on the ground having a SEIZURE. She becomes INCONTINENT of urine, which streams down her PEG legs and creates black-and-white LINEAR SKIN LESIONS. PEG refers to PEG TEETH.

The book notes erythema multiforme (pg146) is part of the spectrum of SJS and TEN. Most sources including uptodate I have read note it not considered part of the SJS/TEN. Is this still true?

Thank you! This seems to be a topic that continues to evolve. Please see the updated version below.

> Similarities in clinical and histopathologic findings have led to controversy over the distinction between EM and Stevens-Johnson syndrome (SJS). There is suggestive evidence that EM with mucous membrane involvement and SJS are **different diseases with distinct causes**.
> **ERYTHEMA MULTIFORME (EM) CORRECTIONS (updated content underlined):**
> Erythema multiforme (EM) is also a confusing topic. Evidence suggests that EM with mucous membrane involvement and SJS/TEN are different diseases with distinct causes. Distinguishing erythema multiforme minor from erythema

multiforme major is also confusing, so the terminology is not likely to be tested. Both minor and major have tiny **target** lesions (probably dusky in the middle). Sometimes you have to use your imagination to envision the target. It may just look a little darker on the inside of the lesion than the outside. Lesions usually start on the hand and/or feet and THEN progress to the trunk. There will be 0–1 mucous membranes involved (if more, it will likely be called SJS or TEN). IF you are tested on the terminology, pick minor if the patient is not toxic. Possible etiologies include HSV, Mycoplasma, and Syphilis.

> **IMAGE:** www.pbrlinks.com/ERYTHEMULTI1
> **IMAGE:** www.pbrlinks.com/ERYTHEMULTI2
> **IMAGE:** www.pbrlinks.com/ERYTHEMULTI3
> **IMAGE:** www.pbrlinks.com/ERYTHEMULTI4
> **MNEMONIC**: Imagine Stevens and Johnson as two very arrogant hunters. They went TARGET shooting one day in an area that said, "Beware of BULLS." They learned their lesson the hard way when a BULL came out of nowhere and did some target practice of his own.

On page 153-155, dermatology, tinea versicolor - treatment of tinea versicolor according to critique of question 272 PREP 2019: should NOT be treated with oral ketoconazole (apprarently not FDA approved for cutaneous fungal infections...) rather 1st-line: topical selenium sulfide lotion (1-2.5%) or ketoconazole cream or shampoo, 2nd-line: oral fluconazole or itraconazole.

Thank you. You're right! Although oral ketoconazole is effective for tinea versicolor in small randomized trials, hepatotoxicity and adrenal insufficiency, along with multiple potential drug-drug interactions, have been reported with oral ketoconazole therapy, making it an unfavorable choice for the treatment of tinea versicolor. Here's our new version!

> **TINEA VERSICOLOR CORRECTIONS (updated content underlined):**
> Tinea versicolor results in hypopigmented OR hyperpigmented macules. It's caused by MALASSEZIA FURFUR. Lesions may fluoresce under Woods lamp. Treat with topical selenium sulfide lotion/shampoo (1-2.5%) or zinc pyrithione 1% shampoo. Second line treatment includes oral itraconazole or fluconazole, but NOT oral griseofulvin (use that for T. capitis).

> **IMAGE**: www.pbrlinks.com/TVERSICOLOR1
> **IMAGE**: www.pbrlinks.com/TVERSICOLOR2
> **IMAGE:** www.pbrlinks.com/TVERSICOLOR3

There is no discussion or pictures of NEC, do we need to know this?

The topic isn't specifically listed in the content specs, but we do think it's a testable topic. Thanks for bringing this up. We will add a short section to the book in the next addition.

> Necrotizing enterocolitis (NEC) is a serious complication in a newborn that happens when the mucosa of the colon gets inflamed. It is most common in premature infants < 1,500 grams. The cause is unknown but thought to be related to hypoxia to the intestines resulting in secondary bacterial infection and possible intestinal perforation. Diagnosis is with a KUB to look for air in the intestinal walls. Treatment is NPO and NG tube to suction and broad-spectrum antibiotics. Sometimes surgical removal of a portion of the intestine is required.

Can you please go over the GBS guidelines - it's given under ID in PBR text (pg 287)- but it would be great if you could please clarify it since we are doing Neo.

Thanks for the question, there are corrections and clarifications that will go into the next edition based on the latest guidelines. Here's a summary that should help.

> Intrapartum Antibiotic Prophylaxis (IAP) refers to antibiotics given to mom when she presents for delivery. IAP is indicated for:
    - Invasive GBS disease in a **previous** infant
    - Positive GBS in the urine during **THIS** pregnancy
    - Positive GBS vaginal-rectal culture (done at 36 – 37 6/7 weeks) in **THIS** pregnancy
    - Unknown GBS status PLUS any of these:
        - < 37 weeks gestation
        - ROM ≥ 18 hours
        - Intrapartum fever of ≥ 100.4'
        - Positive intrapartum NAAT testing.

> IAP is considered "adequate" when penicillin, ampicillin or cefazolin is administered ≥ 4 hours prior to delivery.

> IAP is NOT indicated for GBS positivity in previous pregnancies or for c-section done with intact membranes.

> After the delivery, pediatricians must decide if a baby will need diagnostic evaluation, antibiotics and how long the baby should be observed in the hospital. A full diagnostic evaluation includes CBC, blood culture, chest x-ray (if there are any abnormal respiratory signs) and lumbar puncture (if the procedure can be tolerated). Limited diagnostic evaluation includes a CBC and

blood culture. If antibiotics are started, baby should be monitored for 48 hours. If there were never any signs of sepsis, stop antibiotics after 48 hours. **For a neonate born to a mom for whom IAP was indicated (or the GBS status was not known), the information below should help you decide how to treat the baby:**

- FULL DIAGNOSTIC EVALUATION + ANTIBIOTIC THERAPY (FULL COURSE): If appropriate IAP was given, then do this anytime the baby HAS SIGNS SEPSIS. Baby will stay for the full course of antibiotics.
- LIMITED DIAGNOSTIC EVALUATION + ANTIBIOTIC THERAPY: If appropriate IAP was given, then do this if mom had chorioamnionitis and the baby has NO SIGNS OF SEPSIS. Discharge at 48 hours if the baby appears well.
- LIMITED DIAGNOSTIC EVALUATION + NO ANTIBIOTIC THERAPY: If the baby is doing well (NO signs of sepsis), but mom DID NOT receive adequate IAP, then do a limited workup if there was PROM (> 18 hours) OR if gestation was < 37 weeks. Discharge at 48 hours if the baby appears well.
- OBSERVE FOR 48 HOURS (**SCENARIO 1**): If the gestation was ≥ 37 weeks with ROM occurring < 18 hours prior to delivery, and if the baby is doing well (NO signs of sepsis), but mom DID NOT receive adequate IAP, then no diagnostic workup is needed and you can simply observe for 48 hours.
- OBSERVE FOR 48 HOURS (**SCENARIO 2**): Regardless of gestational age, if appropriate IAP was given and the baby is well (no signs of sepsis), you can simply observe for 48 hours.

> For the treatment of sepsis, the antibiotic choice will depend on the type of sepsis.

- Early onset (birth-DOL 6) bacteremia: ampicillin IV + gentamycin IV or penicillin G
- Meningitis: ampicillin + gentamycin + cefotaxime or penicillin G
- Late onset (DOL 6+) bacteremia: ampicillin/vancomycin + gentamycin/cefotaxime or penicillin G
- Meningitis: ampicillin/vancomycin + gentamycin + cefotaxime or penicillin G
- Cellulitis/adenitis: nafcillin/vancomycin + gentamycin + cefotaxime or penicillin G
- Septic arthritis: nafcillin/vancomycin + gentamycin + cefotaxime or penicillin G

- Osteomyelitis: nafcillin/vancomycin + cefotaxime
- UTI: ampicillin/vancomycin + gentamycin/cefotaxime or penicillin G

On page 169 in the 1st paragraph about jaundice, it says that jaundice on day one of life is bad and associated with G6PD deficiency. Then, on page 170 under G6PD - it says 'jaundice in a male baby AFTER 24 HOURS'- can you confirm please?

> You're right! There's a discrepancy here. The first statement (on page 169) is correct. Jaundice on day 1 CAN be due to G6PD Deficiency. Please have a look at the corrected topic below.

GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY (G6PD DEFICIENCY) 

Glucose-6-Phosphate Dehydrogenase Deficiency (G6PD Deficiency) is an X-linked recessive disorder (so look for a male patient!) resulting in jaundice, dark urine, and anemia due to hemolysis from oxidative injury. In newborns, this will present with jaundice in a MALE baby within the first 24 HOURS of life. In other children, it can result in hemolysis after ingestion of fava beans, malaria medications, trimethoprim-sulfamethoxazole, ciprofloxacin, or nitrofurantoin. Look for HEINZ BODIES (purple granules noted in the red cells on microscopy). G6PD Deficiency is associated with patients of African-American and Mediterranean descent.
PEARL: Do not test for the deficiency during the acute hemolytic phase. Instead, wait a few weeks because a false negative result can occur due to the build-up of G6PD in reticulocytes.
MNEMONIC: Imagine a bottle of HEINZ BODIES ketchup that has an **X** located on the exact spot that you have to hit it to make the broken RBCs come out of the bottle. Or, look at this X-shaped HEINZ BODIES bottle of ketchup.
NAME ALERT: This is not the same as glucose-6-phosphat**ase** deficiency, which can affect glucose metabolism and is associated with glycogen storage disease 1, von Gierke's disease.
>

PBR 2020 page 171. Under Neonatal Hypoglycemia, the last sentence reads "consider an IV infusion rate of D10 at 80ml/kg/hr" and I believe it should say "80 ml/kg/d".

> You're right! Thank you! We'll update the topic to read as "D10 at 80 ml/kg/day."

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DEVELOPMENTAL MILESTONES

There were no developmental milestones corrections for 2020!

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EMERGENCY MEDICINE AND TOXICOLOGY

On page 192, the anion gap formula under the "OSMOLAR AND ANION GAPS" topic is incorrect.

> Thanks for letting us know! It'll be updated in the next edition of the book

> Na - (Cl + HCO3). Normal is ≤ 12.

For near drowning on page 202, med study says the poor prognosis for drowning is flipped from what it says in PBR. Can you please verify? CPR needed for >25 min in the ER, >10 min submersion are poor prognostic indicators.

Yes, you are correct! We collaborated with other sources and this will be updated in the next edition of the text.

> **NEAR DROWNING**

> A near drowning usually requires INPATIENT monitoring because they can result in ARDS after an asymptomatic period. This can lead to DEATH from hypoxic ischemic brain injury and cerebral edema.

> **PEARLS**:

> * GOOD PROGNOSIS: Patient had good pulses on EMS arrival or required < 10 minutes of CPR. If the patient was in the water for less than 60 seconds, had NO loss of consciousness, and did not require CPR, then the patient does NOT need to be hospitalized.

> * POOR PROGNOSIS: Cold on EMS arrival (< 90° F), CPR needed for > 25 min, > 10 minutes underwater, apnea, coma, or pH < 7.1. Rewarm these patients to > 90° F and consider intubation to provide PEEP for possible ARDS.

On page 201 under the methemoglobinemia pearl, it states that "Pulse oximetry is NORMAL." However, from my understanding and outside resources, pulse ox will generally be between 82-86% despite very high arterial O2 (PaO2). The patient will also have signs of cyanosis even though the saturation is only in the 80s, and the sats remain in the 80s despite treatment with 100% FiO2.

You're right! The pulse oximeter for methemoglobinemia will be low, while the PaO2 will be high. Here's an "early version" of next year's topic.

> Methemoglobinemia is an enzyme deficiency (NADH methemoglobin reductase) that results in increased methemoglobin levels (thus reducing O_2-carrying capacity). Symptoms include BLUE or CYANOTIC SKIN without evidence of respiratory distress, hypotension/shock, and tachycardia. The blood becomes CHOCOLATE-COLORED (with less oxygen). Certain exposures increase the methemoglobinemia (this is associated with sulfa drugs and **well water** being used with formula). Treat with methylene blue, oxygen, and removal of the offending agent.

> **PEARL**: In methemoglobinemia, the pulse oximeter reading will be low. It may

show a saturation level of around 85% even if the TRUE hemoglobin oxygen saturation is higher. When the PaO2 is measured, it may be normal. The pulse oximeter will not improve with hyperoxia. Measure the methemoglobin level to get the diagnosis.

> **MNEMONIC**: To remember all of this, think of two types of hemoglobin, “regular” and “met.” The pulse oximeter gives the percentage of “regular” hemoglobin and the ABG only measures the O2 saturation for the REGULAR hemoglobin. Giving more oxygen will not change the regular:met ratio. So, the pulse oximetry doesn’t change with hyperoxia.

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VITAMINS AND NUTRITIONAL DISORDERS

There were no vitamins and nutritional disorders corrections for 2020!

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Clarification on age range for Intussusception - page 222 states 3 months to 36 months, with most less than 24 months and page 227 states Mneumonic intusSIXeption: ages 3 months to 6 years. Which is it?

Thanks for asking this. Page 222 is correct, and we'll be removing the mnemonic from page 227. There are some caveats you should be aware of and we've updated the main topic and are including it below for your review.

INTUSSUSCEPTION

Intussusception is a telescoping of the bowel into an adjacent segment of bowel, often in the ileocecal area (AKA ileocolic area). This can result in **intermittent** episodes of abdominal pain, currant jelly stools, bilious emesis, a palpable mass, and even a septic clinical picture without fever. It usually occurs in children 3 months to 36 months, and when cases present outside of that age range, you should consider a pathologic cause which has created a "lead point" (polyp, hematoma, lymphoid hyperplasia, diverticulum, etc.). Most cases occurring in the first year of life, but about 10% of cases are in patients older than 5 years of age (including teenagers and adults). Treatment options include either an air contrast enema with a small amount of barium, or a barium enema.

PEARLS: Buzz words include intermittent abdominal pain and currant jelly stools. Patient may not have ANY abdominal pain on exam. If given an option between air contrast enema and barium contrast enema, choose AIR contrast.

NAME ALERT: 

Currant jelly SPUTUM is a buzz word for KLEBSIELLA pneumonia.

It lists ranitidine as a medicine for use in infants with GERD on page 223, but as I am sure you are aware it is no longer an option.

> You're right! Ranitidine is no longer available in the US due to an FDA black box warning of possible human carcinogen effects (gastric and colorectal cancer).

> If young and generally growing well, avoid medications. Offer reassurance or try hypoallergenic breastmilk/formula prior to starting a medication in infants. If you are going to prescribe an acid suppressant, try it for 2 weeks (famotidine would be acceptable as an option) and discontinue it if there's no improvement due to concerns for side effects. Know that studies have not shown any significant improvement with acid suppression in infants.

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

Compared to pyloric stenosis, patients with gastroesophageal reflux disease (GERD) have vomiting that seems effortless. If the child is healthy, no need to treat. It will likely resolve by 18 months of age. Always consider overfeeding as a possible etiology. If there is apnea, signs of esophagitis (posturing), or poor weight gain, start a workup and also treat. A GI pH probe may help diagnose. Biopsy is unlikely to be an option, but choose GERD if eosinophils are noted on biopsy. You may treat with an H2 blocker (cimetidine, nizatidine, or nizatidine) or with a proton pump inhibitor (PPI), such as omeprazole.

PEARL: Metoclopramide and sitting upright during feeds have not been shown to decrease reflux.

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PHARMACOLOGY AND DRUG PEARLS

There were no pharmacology and drug pearls corrections for 2020!

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OPHTHALMOLOGY

There were no ophthalmology corrections for 2020!

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GENETICS

There were no genetics corrections for 2020!

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HEMATOLOGY AND ONCOLOGY

In the hematology MP3, (around minute 1) it is stated both that Shwachman-Diamond Syndrome has only "one cell line" issue and that pancytopenia is present. Is that correct or incorrect?

> Thanks for bringing this up. We'll update our MP3s to match the 2021 edition once the hardcopy books are released. For this, the book is correct on page 275. Initially, there's thrombocytopenia, pancreatic insufficiency, and **neutropenia**. These patients do have a high chance of moving forward to aplastic anemia and myelodysplastic syndrome.

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INFECTIOUS DISEASES

For chronic sinusitis, the chart on page 305 says > 90 days but in the text it says > 4 weeks, please clarify:

"CHRONIC SINUSITIS (> 4 WEEKS): Look for evidence of some other comorbidity, such as hay fever, an immunodeficiency, immotile cilia, or a history of cystic fibrosis."

Good catch! This will be updated in the next edition of the book.

> Acute bacterial sinusitis: < 30 days
> Subacute bacterial sinusitis: 30-90 days
> Recurrent acute bacterial sinusitis: < 30 days with an interval separation of at least 10 days
> Chronic sinusitis: > 90 days

On page 290 under chlamydia pneumonia, PBR states that chlamydia pneumonia can occur until 2 months of age and the preferred treatment is doxycycline/macrolides. I thought that the only exception to using doxycycline under 8 years old was for RMSF (Rocky Mountain Spotted Fever) or can you also use doxycycline in this situation? Also, as far as macrolides go - if erythromycin cannot be used under 1 month, are we then treating chlamydia pneumonia with either azithromycin/clarithromycin (not doxycycline or erythromycin) for the most common age group for chlamydia pneumonia?

Thanks! I think this section can be further clarified and updated. For young children, you have to pick the "best of the worst." The CDC says to treat with a macrolide in the young kids, so the exception you mentioned (using doxycycline in kids under 8 only if you're dealing with RMSF) is correct. Here are some modifications we'll be making.

> First-line therapy for children of all ages is a macrolide (erythromycin or azithromycin). For a child > 8 years of age, doxycycline or a fluoroquinolone would be acceptable, but as a second-line treatment.

> Both erythromycin and azithromycin are associated with increased risk of infantile hypertrophic pyloric stenosis (IHPS), particularly in infants younger than two weeks old, but the benefits outweigh the risks in this case.

On page 294, in the aspergillosis section, it says to treat with amphotericin B for invasive disease in an immunocompromised child. Uptodate notes to treat with Voriconazole as drug of choice. What is going on here?

> Yup. There's been a change!

> Aspergillus is usually only invasive in immunocompromised patients. Regardless of whether a patient is immunocompetent or immunocompromised, if a patient has invasive disease, then treat with either voriconazole or isavuconazole.

For Brucellosis on page 317, I found that treatment for someone greater than 8 years of age is doxy +amnioglycide/rifampin, and treatment for those less than 8 years old is bactrim and rifampin. Is this correct?

> Thanks for the question. It looks like we need an update here.

> For brucellosis WITHOUT focal disease from spondylitis, neurobrucellosis, or endocarditis:

- If < 8 years, use TMP-SMX + Rifampin.
- If ≥ 8 years use Doxycycline + either Rifampin OR Streptomycin OR Gentamicin.

> For brucellosis WITH focal disease, there are different regimen based on whether the disease is due to spondylitis, neurobrucellosis, or endocarditis. So, here are some key points to remember:
    - If ≥ 8 years, use Doxycycline + Rifampin + "a 3rd medication" (an aminoglycoside for spondylitis or endocarditis, and ceftriaxone for neurobrucellosis).
    - If < 8 years, the regimen is the same as above, but replace Doxycycline with TMP-SMX.
    - The 3rd antibiotic OR Streptomycin OR Gentamicin.

On page 295, it reads that Coxsackie virus and enterovirus are related. More accurately, Coxsackie IS an enterovirus. There are (traditionally) four enteroviruses, Coxsackie A, Coxsackie B, poliovirus, and echovirus.

> Good catch! Thank you. You're right, and we'll update the book to reflect this since coxsackie is a subtype of the Enterovirus family.

On page 296, "paramyxovirus is second line" for causes of bronchiolitis. The term second line refers to medications, not causes. And multiple sources state that rhinovirus is the second most common cause, which is a picornavirus, not a paramyxovirus. Influenza is probably third. Here's one source: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3880140/

> Thanks for the info! The book said that paramyxovirus is second "IN line" (not "IS line"), but everything you mentioned is correct. Regarding the causes of bronchiolitis, RSV is first, rhinovirus is second and influenza is 3rd. Other causes include adenovirus, parainfluenza, human metapneumovirus.

On page 306 of the 2020 edition of PBR it says "you should also GIVE TOPICAL STEROIDS..." for Otitis Externa. However, a Cochrane review found that there was insufficient evidence to add corticosteroids to topical antibiotic preparations.

> Thank you for this! Yes, it looks like for otitis externa, topical antibiotics are sufficient and there's no significant increase in cure rate to add on topical steroids. Topical Steroids do decrease time to symptom resolution by a day, but are not required.

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VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

In PREP 2020 in a question about tetanus booster they mention that if required, use DTap in children <7, Td in children 7-10 and Tdap if > 11. This is different than what is recommended in PBR page 325. Please clarify if possible.

> Thank you for the question. Great catch! If Tdap is inadvertently given between ages 7-10 years, then a repeat Tdap should be given at 11 years old. So you are correct in saying that a TD is recommended for ages 7-10 years of age. We'll update the next edition of PBR to state the following on page 325:

> **TETANUS BOOSTER**
> **DTaP** and **DT** are for kids under 7. Babies are **immunized** with **DTaP**. If a tetanus **booster** is needed for possible exposure to a child under the age of 7, only the **DT** portion needs to be given. If a **booster** is given for possible exposure (or as a routine booster) **from age 7-10 years,** give **TD** and repeat the routine **Tdap** at age 11-12 years old or if **> 11 years old**, simply give **Tdap**.

On page 324 under measles post-exposure prophylaxis section, it states if the patient is at least 6 months old and the exposure was less than 72 hrs ago, the vaccine or MIG may be given. From my understanding, the second MMR vaccine given at well child visits is given not as a booster, but for any non-responders to the first dose. So, if the patient has not received their first MMR dose yet and there is a chance that he/she is a non-responder, why wouldn't we always give MIG for post-exposure prophylaxis?

> I believe a simplified version of your question is along the lines of, "If it's possible that some people may not respond to the MMR vaccine, why not just always give MIG." There are a few reasons. MMR administration as a form of post-exposure prophylaxis (PEP) for those over 6 months of age who were exposed < 72 hours may "prevent or modify disease" AND it provides permanent protection in the vast majority of children. Being a non-responder is the expection rather than the norm. So if the MMR was still as helpful at preventing disease when given at 4 – 6 days after exposure, I'm guessing that the guidelines would recommend giving the MMR instead of IMIG (or IVIG) in even more cells within the table below.

Here's the table we'll be including in the next edition to provide clarification and information that's more correct.

Age	3 days or less	4-6 days	> 6 days
< 6 months old	IGIM (or IVIG)	IGIM (or IVIG)	No PEP indicated because it's too late to prevent the onset of the disease
6 – 11 months old	**MMR**	IGIM (or IVIG)	
> 12 months old	**MMR** **MMR #1** if never received. **MMR #2** as per routine guidelines if it's been at least 28 days since MMR #1.	IGIM (or IVIG) if MMR #1 never received, or if vaccination status unknown. **MMR #2** may be given as per routine guidelines if it's been at least 28 days since MMR #1.	

> For immunocompromised patients, pregnant patients or those with allergies to an MMR component, IGIM (or IVIG) is an acceptable alternative when MMR would normally be indicated for PEP. That would not be required if MMR #1 had already previously been given.

> **PEARLS**: DO NOT GIVE MMR for at least 5 months after giving IGIM, and DO NOT GIVEN MMR and IGIM at the same time.

> **MNEMONIC**: Mom's antibodies can linger and "fight" the MMR vaccine until about 6 months, which is why we give IGIM (or IVIG) as post-exposure prophylaxis to those under 6 months of age.

New topic on COVID-19 for the PBR Core Study Guide?

I would be very, very surprised if the ABP asked you anything about COVID. It's a new and evolving topic with many unanswered questions and is therefore unlikely to be tested. But, I still feel it's worth having a quick review of some known and undisputable facts.

> **Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)** is the novel virus that causes coronavirus disease 2019 (COVID-19). The incubation period is thought to extend up to 14 days, but most people will exhibit symptoms in 4-5 days from exposure. Its effect is variable, but it is generally well tolerated by children who lack comorbidities. Findings may include fever, chills, cough, fatigue, body aches, shortness of breath, headache, new loss of

taste or smell, sore throat, congestion, runny nose, diarrhea, nausea or vomiting. Lab abnormalities may include a transaminitis, lymphopenia, abnormal coags, elevated inflammatory markers or an elevated LDH. In rare cases, the disease can progress rapidly and cause COVID-19-associated multisystem inflammatory syndrome in children (MIS-C). This has been reported to occur at 2-4 weeks after the onset of COVID-19 and can result in severe complications (cardiac dysfunction, shock, myocarditis coronary artery dilatation or aneurysm, and acute kidney injury) or death. Diagnosis is made by RT-PCR samples of the nasopharynx for SARS-CoV-2 RNA. Most SARS-CoV-2 cases are treated at home with mask use to prevent household spread, isolation to a single room when possible, a 14-day quarantine, and supportive measures. For severe cases, steroids may help in the ICU setting. Various existing and new medications are currently being investigated. As of 10/2020, no vaccine has been approved by the FDA.

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INBORN ERRORS OF METABOLISM

With Fabry's disease, are the skin lesions in Fabry's disease reddish-purple or orange? The core study guide mentions both.

> Reddish purple for sure. But, in our mnemonic we mentioned orange. We'll change this to purple. Thank you!

With Fatty acid oxidation disorders, are you looking for disease with the name Acyl-Co-A-Dehydrogenase (core study guide) or carnitine (audio series)?

> Thank you for the question. You're right. There's a discrepancy. The Core Study Guide is correct, and the audio course has it wrong. We'll be addressing this in the next audio course!

On page 336 under "disorders of carbohydrate metabolism", the Core Study Guide mentions Aldolase Deficiency but not mentioned further... should we know this?

> Hmm... maybe. But, in reading more about this, it feels like something out of left field. It's extremely rare. We're still undecided on this and we may actually REMOVE "aldolase deficiency" from the book. For now, we've created a topic for POSSIBLE insertion into the 2021 study guide.

> **GLYCOGEN STORAGE DISEASE DUE TO ALDOLASE A DEFICIENCY**: This long-named disease is also called Glycogen Storage Disease XII (GSD12) and is characterized by hemolytic anemia, possible myopathy and possible intellectual deficit. The myopathy can be so severe that it can trigger a fatal

rhabdomyolysis.

> **PEARL**: A family history of episodic rhabdomyolysis should make you think of this condition.

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ACID-BASE DISORDERS

There were no genetics corrections for 2020!

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FLUIDS AND ELECTROLYTES

On page 354 PBR states: "The dehydrated patient's blood is concentrated, so expect a HIGH serum osmolality, above 300 mOsm/l."

This sentence is under the HYPONATREMIA section, I do not think this would be possible with HYPONATREMIA and I've looked at several references and hyponatremic hypovolemia (dehydration) will have a low serum osm <300. This sentence could be true with HYPERNATREMIA and dehydration, maybe it is mean to be in the HYPERNATREMIA section?

Based on the equation alone it would be I think impossible for serum osm to be >300 with hyponatremia, so maybe this sentence is meant to be in the hypernatremia section? Or is the sentence supposed to say high URINE osm under HYPONATREMIA with dehydration?

You are correct, the text will be updated to note low serum osmolarity (less than 270 mOsm/kg) in hyponatremic dehydration.

> Children with hyponatremic dehydration have hypotonic body fluids with serum osmolarity less than 270 mOsm/kg (270 mmol/kg) that can lead to fluid shifts from the extracellular to the intracellular space. The degree of dehydration may be overestimated because these patients have diminished intravascular volume that is manifested by more severe clinical symptoms. They are very likely to require immediate circulatory support.

> On the other hand, children with hypernatremic dehydration have hypertonic body fluids with serum osmolarity, often in excess of 300 mOsm/kg (300 mmol/kg). Fluid shifts from the intracellular to the extracellular space to maintain intravascular volume. The degree of dehydration in these children is often underestimated, contributing to late presentation for medical care. Assume 10% dehydration when the child comes into the ED.

> Dehydration causing diminished intravascular volume (hyponatremic dehydration) increases vasopressin (ADH) to the kidney release leading to water conservation and urine concentration. Vassopressin (ADH) works on the

kidneys by getting rid of urine sodium thus, **hyponatremic dehydration has low serum osmolality and high urine osmolality and low urine sodium.**

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NEPHROLOGY

In the 2020 PBR book (p. 361, paragraph 3), it discusses oral outpatient treatment of UTIs. There has been a movement to use first-generation cephalosporins, such as cephalexin for oral outpatient treatment. I agree that we've used more 3rd generation in the past, but more recent UTI hospital pathways are recommending 1st generation (cephalexin). Here is the link to the CHOP UTI pathway: https://www.chop.edu/clinical-pathway/urinary-tract-infection-uti-febrile-clinical-pathway.

> Thanks for having us review this! I do think a correction is needed and we've made it below.

URINARY TRACT INFECTION (UTI or PYELONEPHRITIS) (Corrections are underlined)

If a urinary tract infection (UTI or pyelonephritis) is diagnosed and the urinalysis shows POSITIVE NITRITES, this is virtually diagnostic of a GRAM-NEGATIVE organism (E. coli, *Klebsiella pneumoniae*, and Proteus). If nitrites are negative, the UTI could be due to a gram-positive organism (especially Enterococcus or *Staph. saprophyticus*). For most UTIs, give outpatient therapy. If the patient is not septic, has a low likelihood of renal disease (no fever or back pain) and can tolerate orals, **give a 1st generation cephalosporin (cephalexin)**. If there is concern for possible renal disease (fever with or without back pain), give a 2nd generation (cefuroxime) or 3rd generation cephalosporin (cefixime or cefdinir). If there is a severe allergy to penicillin/cephalosporins then treat with trimethoprim-sulfamethoxazole. If allergic to sulfa medications, then treat with ciprofloxacin. Treat for 10 days if < 6 months old and 7 days if > 6 months old. Early treatment is key to prevent renal damage. If the patient has nausea and vomiting from pyelonephritis, they cannot tolerate outpatient therapy and should be hospitalized. Inpatient treatment should be with an IV cephalosporin (ceftriaxone, cefepime or cefotaxime). Ampicillin and gentamycin as a combination may also be acceptable.

* FIRST FEBRILE UTI (2-24 months of age): Treat with antibiotics and obtain renal and bladder ultrasound. A renal ultrasound will look for structural problems (kidneys, ureters, bladder). Since there is still ongoing debate between pediatric urologists, as of this time, it is not recommended to routinely obtain a VCUG after the first febrile UTI.

* RECURRENT FEBRILE URINARY TRACT INFECTIONS: Further evaluation is needed, including VCUG if not previously done. A VCUG will look for ureteropelvic junction obstruction (UPJ obstruction), vesicoureteral reflux (VUR), and posterior urethral valves (PUV). Wait to order the VCUG until **at least a week after UTI resolution** as evidenced by a **repeat urine culture showing negative results**. Routine antibiotic prophylaxis is no longer recommended. Rather, parents should be instructed to seek medical attention for future febrile episodes within 48 hours so that a prompt workup can be done for possible UTI.

PEARL: Febrile UTI in a young child is associated with a HIGH rate of vesicoureteral reflux (VUR).

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STATISTICS

There were no statistics corrections for 2020!

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NEUROLOGY

In the neurology MP3 lecture, around minute 11, Guillain Barre is associated with "clostridium jejuni" rather than campylobacter jejuni. I think this is an error. Do you agree?

> Thank you! This is probably more of a clarification. It was a bad abbreviation on our end as “C. jejuni” in the book and I think the speaker misspoke. We’ll be sure to spell out “Campylobacter” in the next edition and also redo that audio chapter for 2021.

Can you add topics like Flaccid myelitis as well to the neurology section where it explains about Guillian Barre?

We’ll likely add that this topic to the Core Study Guide ☺

> Acute flaccid myelitis (AFM) is uncommon and causes weakness and decreased reflexes. Most cases are occurring in young children and seem to be occurring more frequently since 2014. Not caused by polio. Symptoms include: hypotonia, hyporeflexia, difficulty moving eyes or droopy eyelids, facial droop or weakness, difficulty with speech or swallowing, pain in arms and legs, and pain in the neck or back. Severe symptoms can cause respiratory failure or body temperature and blood pressure instability. Etiology is unknown but thought to be related to a viral infection with enteroviruses being the most common. Diagnosis includes MRI, CSF, blood, stool, respiratory fluid for viral etiologies along with clinical findings. Treatment is supportive along with rehab including PT and OT.

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ORTHOPEDICS AND SPORT MEDICINE

There were no orthopedics and sport medicine corrections for 2020!

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RHEUMATOLOGY

In Juvenile Idiopathic Arthritis do we need to differentiate between mild to moderate vs. severe disease? What is first line treatment? Should we be using DMARDs such as methotrexate or steroids? Our text on page 399 notes second line therapy is steroids. Please clarify.

Good question! This topic will need to be corrected and updated. See updated content below that will be in the next edition of the book.

> Treatment should be directed towards underlying synovitis and inflammation. Initial treatment should start with disease-modifying antirheumatic drugs (DMARDs) such as methotrexate or a tumor necrosis factor (TNF) inhibitor in addition to methotrexate for more severe disease. NSAIDS are not recommended for monotherapy but can be used for symptom management.

> **PEARL**: Children receiving methotrexate should be supplementing with folic acid or leucovorin (folinic acid).

> **PERAL**: Long term, high dose use of steroids should be avoided for patients with JIA. Short term, low dose steroids may be helpful in some patients, but DMARDs such as anti-TNF agents are the preferred treatment for children with JIA.

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PULMONOLOGY

In the book on page 403, it states that " There is a strong association between Trisomy 21 and Cystic Fibrosis." Is this statement accurate? In the article below, CF is not mentioned as a Pulmonary complication of Down Syndrome. Also, the North American Cystic Fibrosis Foundation's guidelines do not mention screening for CF specifically for Down Syndrome patients.

J Pediatr. 2011 Feb;158(2):319-25. doi: 10.1016/j.jpeds.2010.07.023. Epub 2010 Sep 16. Pulmonary complications of Down syndrome during childhood.

> You're correct! There was historically an increased number of false positives in Down Syndrome kids. It's possible that we somehow mixed something up. We'll remove this from the Core Study Guide.

I was doing a question and the correct answer for a question about most common trigger for asthma exacerbation was URI. On page 406 of the 2020 PBR book it says dust mites (which was an answer choice in the med study question and was wrong). Can you please help clarify? Thanks.

> This was a tough one, even for our pulm/critical care doc.

> URIs are considered the leading cause of an asthma exacerbation (**based on some poor studies**). These may specifically be in the context of exacerbations **leading to hospitalizations**. Small exacerbations managed at home are likely more often due to dust allergies. Our recommendation is to look at the context of the question when answering, but to choose URIs if you're simply being asked about the leading cause of asthma exacerbations.

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PSYCHIATRY AND SOME SOCIAL ISSUES

There were no psychiatry and some social issues corrections for 2020!

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ETHICS

There were no ethics corrections for 2020!

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PATIENT SAFETY AND QUALITY IMPROVEMENT

There were no patient safety and quality improvement corrections for 2020!

STRONG WORK EVERYONE!

THANK YOU SO MUCH FOR CALLING US OUT!

NOW LET'S GO OVER THE CLARIFICATION REQUESTS!

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

This section is going to cover CLARIFICATION REQUESTS from members and anything that we happened to find on our own that we felt might warrant a clearer explanation.

ADOLESCENT MEDICINE

What does the bone age look like in GH deficiency? Is it delayed like in congenital GH deficiency?

> Yes. Most of the times it's delayed. This goes for both acquired and congenital.

Can you discuss when to start OCP use for dysmenorrhea/irregular/PMS?

> After discussing this with our endocrinologist, we felt that this is probably beyond the scope of the ABP Initial Certification exam. That's good news!

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ENDOCRINOLOGY

What do we do next after finding a cold thyroid nodule? Complete excision or partial?

> You would start with a thyroid ultrasound to get the size and other characteristics (echogenicity, calcifications, etc.). Based on that data, you then possibly do a fine-needle aspiration (FNA) if a malignancy is suspected.

What is the most common thyroid malignancy in children?

> The most common thyroid malignancy is papillary carcinoma.

For low calcium and high phosphorus etiologies: shouldn't renal failure be listed here? Book content/info slightly confusing because you list renal failure as a cause of hypocalcemia on page 77. Yet you list renal disease (not mentioned in content) as etiology for normal calcium and high phosphorus. Once phosphorus high from renal disease, I would think you would also have low calcium?

> Thanks for the question. This likely requires a clarification on our part, and we'll likely change "renal disease" to "renal failure" in the table on page 79.
>
> In general, this topic is VERY complicated (even the experts say so). So, this may not be completely accurate, but it should help for the exam.
>
> The phrase "renal failure" is now typically reserved for patients who need to go onto dialysis. On page 77, we list "renal failure" as a potential cause of "late hypocalcemia." If we substitute "renal failure" with LATE STAGE CHRONIC KIDNEY DISEASE, that may help. In later stages of CKD, lower production of Vitamin D can lead to hypocalcemia.
>
> Later in the course of renal disease, the kidneys fail and are unable to excrete phosphorus. PTH restores calcium balance but is unable to help the kidneys remove excess phosphorus.

What do you do with an abnormal newborn screen for hypothyroidism?

> Since this is a screen, you need to confirm the diagnosis before starting treatment. So, check TSH and FT4 immediately (that day, or the very next morning). If there is a high TSH and a low Free T4, begin treatment.
>
> If asked which lab test to obtain, check the TSH level because sometimes the FT4 can still be normal or low normal.

Do children with Familial Hypophosphatemic Rickets require treatment with phosphorus and calcitriol, or just phosphorus?

> They need both phosphorus and calcitriol treatment.

On page 78 of the core study guide, can you explain why the ALP is elevated in Familial Hypophosphatemic Rickets?

> **Bone-specific** alkaline phosphatase increases when there is increased bone turnover.

What is the most common heart condition in Turner Syndrome: Coarctation or bicuspid aortic valve?

> The most common heart condition in Turner Syndrome is a bicuspid aortic valve.

For hypocalcemia in babies, we give magnesium sulfate as treatment when hypomagnesemia is the cause of the hypocalcemia. But magnesium tocolytic use in mother causes hypocalcemia in babies. Please explain?

> Great question! Magnesium is required for the production and release of PTH. So, if there's low magnesium, you get low calcium, and treatment is magnesium. However, if magnesium sulfate is being administered to the mom as a tocolytic for longer than 5-7 days, it can SUPPRESS the secretion of PTH in the fetus, leading to decreased levels of calcium in the baby. This can potentially lead to osteopenia and fractures.

How high is alkaline phosphatase if having liver disease?

> It's on a spectrum. What's important is to keep in mind that it is a nonspecific lab test. Look at the rest of the picture to determine if it's bone disease (any calcium or phosphorus problems?) or liver disease (elevated LFTs or GGT?).

For a child with Type 1 DM with no IV access who is unconscious, can you please include information for treatment?

> Check blood glucose and if BG is < 70 mg/dL, proceed with the **administration of glucagon IM injection** based on age/weight:
>
> - a) If weight < 45lbs and age > 5 yrs old → administer 0.5 mg IM or SC
> - b) If weight > 45lbs and age > 5 yrs old → administer 1 mg IM or SC

> Re-check blood glucose levels every 15 minutes.

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OB/GYN and some STD's

Midway down page 91 "In general, when you think the diagnosis is due to Chlamydia species, choose doxycycline if the child is >8 or macrolide (usually erythromycin) ..." Is this speaking specifically to lymphogranulooma venerum? I think I was thrown off by Chlamydia species. As I would treat with Azithromycin for Chlamydia trachomatis.

> The book is correct. It depends on the age of the child. Typically, since this is an STI it will occur in an older child > 8 years old. So, the first line treatment would be doxycycline. However, if a younger child is infected with Chlamydia then use azithromycin or erythromycin. A review from the PBR Core Study Guide ☺

CHLAMYDIA TRACHOMATIS

Chlamydia trachomatis can cause urethritis, conjunctivitis and pelvic inflammatory disease (PID). In neonates, it can cause pneumonia associated with a staccato cough. PID can lead to ectopic pregnancies and infertility. Eye infections can lead to blindness. Conjunctivitis in a neonate (less than a month old) should raise concern for this as the etiology (vertical transmission). It's an obligate intracellular anaerobe. Getting cultures is difficult, so order PCR of CELLS, secretions, or urine. Chlamydia can also cause lymphogranuloma venereum (LGV), which is an STD that initially starts with small, nontender papules or shallow ulcers that resolve. Then a TENDER UNILATERAL INGUINAL lymph node appears that can rupture, relieve the pain, and then possibly drain for months.

* SEXUALLY TRANSMITTED DISEASE (STD): Treat with Azithromycin x 1. If cost is a concern, then treat with DOXYCYCLINE for 7 days. If the patient is given doxycycline and there is recurrence, treatment failure, or a concern for noncompliance, treat with AZITHROMYCIN x1. Other possible medications include erythromycin, levofloxacin, or ofloxacin for multiple doses/day x 7 days. Test for other STDs including gonorrhea, syphilis, and HIV and treat if positive.

* CONJUNCTIVITIS: Treat with oral erythromycin to eradicate nasopharyngeal colonization, which can lead to pneumonia.

* (DOUBLE TAKE) LYMPHOGRANULOMA VENEREUM SEROVAR is an STD caused by Chlamydia trachomatis. It is rare in the U.S. but more common in tropical areas. It starts as small nontender papules or shallow ulcers that resolve. Eventually, a TENDER UNILATERAL INGUINAL lymph node appears. Pain is relieved when it ruptures. The node can continue to drain for months. Treat with DOXYCYCLINE or erythromycin.

* PEARL: In general, when you think the diagnosis is due to a Chlamydia species, choose doxycycline if the child is > 8 years of age, or choose a macrolide (usually erythromycin). Also, this is an intracellular organism. Look for the phrase "intracytoplasmic inclusions."

* PEARL: While chlamydia is often said to be the most common STD, that's not the case. It's the most common BACTERIAL STD, and it's the most commonly REPORTED STD. HPV is the most common STD.

In what scenarios would you do a PAP in a < 21 year old? What are the risk factors? Does sex alone in a 14yo for example count for reason to do earlier PAP smear? I'm afraid of being tricked on a question to the "Exceptions" of PAP smear guidelines.

> The new guidelines state that no PAP smear should be done for any child under the age of 21 years old.
> Women aged 21–29 years should have a Pap test alone every 3 years. HPV testing is not recommended.
> Women aged 30–65 years should have a Pap test and an HPV test (***co-testing***) every 5 years (preferred). It also is acceptable to have a Pap test alone every 3 years.

What is the treatment duration of labial adhesions?

> The topic from the Core Study Guide is included below, but you should NOT be asked to remember the duration of treatment. In general, you will be required to know the names of medications, but not the doses, frequency or duration.

LABIAL ADHESIONS (PENILE ADHESIONS for boys)

LABIAL ADHESIONS (or PENILE ADHESIONS for boys) usually resolve without intervention. If they are present in a girl and she has dysuria, treat using ESTROGEN cream.

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ALLERGY AND IMMUNOLOGY

Most infants and toddlers are going to outgrow their allergies by age 5. When is it an appropriate time to start to reintroduce certain foods back into the diet (cow's milk/egg/wheat)?

> This is usually based on an individual basis if it's a true food allergy. Various questions come to mind. How is the patient doing? Has repeat skin or blood testing been done? Should we now do a food challenge in the office? So, as you can see, there is no particular age that is recommended, rather working with the allergist and reviewing the clinical scenario is required on an individual basis.

How do they classify as mild, moderate, severe eczema? How do they treat based on severity? What do they recommend for the face?

> Many studies will use a scoring system that may take body surface area into account. The scoring system is unlikely to be tested. First line is always topical steroids. Pick the lowest potency to effectively calm it down. Low to mid potency for the face. Desonide is very commonly used on the face.
> It's possibly they could ask about steroid sparing drugs, but those are usually prescribed by a specialist. Such medications include topical calcineurin inhibitors (TCIs), such as pimecrolimus or tacrolimus, or topical PDE4 inhibitor, such as crisaborole.

Could you kindly elaborate on the differences between True milk protein allergy, Food Protein Induced Enteropathy, Food Protein Induced Proctitis/colitis and FPIES? Are they on a continuum or are they different entities?

Great question on some confusing topics. Thanks for asking.

> True milk protein allergy → This is just like a peanut allergy, but with milk. You can get anaphylaxis.
> Food Protein Induced **Enter**opathy → Think of "enter" as it's entering the bowel cells. The wall is location of the problem. The dysfunctional bowel wall interferes with normal absorption, so this can present as failure to thrive.
> Food Protein Induced Proctitis/col**itis** → "-ITIS" of lower GI tract. These kids appear generally well, but usually present with blood and/or mucus in the stool. It's due to an intolerance of a protein in their diet. Can be seen in breastfed infants who have a cow milk intolerance. Moms must remove milk from their diet. Kids generally do well later in life when cow's milk is brought back into their diet.
> Food Protein-Induced Enterocolitis Syndrome (FPIES) → The exact mechanism is still not known. Seems to be an allergic type of sensitization. Kids have a delayed reaction at 1-3

hours after exposure which results in tons of vomiting, diarrhea and more. Can even look listless and septic. Cow's milk and soy are the biggest culprits. For older kids, rice and some other solids can be the culprits. They usually to outgrow it.

Can an initial presentation of IgA deficiency present as a blood transfusion reaction?

> Yes, but it would be rare. Rates of transfusion reactions related to this are so low that it would likely fall under a case report type of scenario. It's not likely to be the correct answer on the boards.

I keep getting SCID, Brutons, and CVID confused due to all of them having abnormal immunoglobulin levels. Is there a way to differentiate them besides age of onset? I know SCID and Brutons both have no thymus, but that makes them even more difficult to differentiate for me. How do you differentiate these two on a question?

Let's see if we can help!

> BRUTON'S → This is also called "agammaglobulinemia." Meaning there are NO antibodies. There are NO tissues to make those antibodies (nodes, tonsils, etc.). These kids have more bacterial infections because the humoral immunity is affected. This is X-linked, so look for a boy.
> SCID → This is Severe COMBINED Immune Deficiency. Missing humoral immunity AND it's missing the T cell functionality. You can think of it like Bruton's AND HIV combined. There will many more types of infection, PCP, fungal, viral, etc. because both sides have been hit.
> CVID → The name has VARIABLE in it. It's most similar to Bruton's, but usually not as severe. It's "variable" so the severity is often not as bad as Bruton's. The block where the deficiency happens can happen at different places. Bruton's will present around 6 months, while those with CVID can present much later (even in adulthood). Because of this, there can be a huge lag before it's diagnosed because infections are chalked up to "life." Also, many older kids and adults may be going to an urgent care center for an infection and not treated by a primary care doctor or medical home, so it is often missed.

At what point do we refer to an allergist for a skin test - egg, peanut, bee sting?

> SKIN TESTING FOR PEANUTS: If a child has a history of any eczema or a known egg allergy, and they do not have peanuts in their diet, then they are at increased risk for having a peanut allergy and they require skin testing.
> ECZEMA: If no peanuts in their diet, we now check for peanut allergy via skin testing before introducing peanuts.
> EGG ALLERGY: If no peanuts in their diet, check for peanut allergy via skin testing before introducing peanuts.
> AEROALLERGENS: If you're maxing out with your antihistamines, montelukast, and nasal steroids, then refer (even on the ABP exam).
> PEARL: Do NOT restrict foods ONLY based on IgE results. Food allergies are clinical diagnoses. You must have the history (reaction) to support the diagnosis of a food allergy. Also, by avoiding the foods a child can actually DEVELOP a true food allergy.

If an immunological work up is being done for frequent sinopulmonary infections, what labs should be ordered initially?

> CBC with differential, quantitative immunoglobulins (IgA, IgM and IgG), and vaccine titers. The titers are obtained to see if the patient has developed the appropriate IgG antibodies to the vaccines.

What is first line therapy for allergic rhinitis?

> Oral antihistamines followed by intranasal steroids if the child can participate.

If a patient with questionable penicillin allergy, should I order a penicillin panel IgE? Or refer for skin testing?

> The lab test is extremely unreliable. Send them to an allergist. They have a defined protocol to put the patient through that includes penicillin skin testing.

For Chronic Granulomatous Disease (CGD) is there a preferred test for diagnosis? DHR vs. NBT?

> DHR is the preferred test.

If a patient develops a rash consistent with drug eruption while using a penicillin anabiotic, like amoxicillin, is this considered a true penicillin allergy? Should the physician stop using amoxicillin or other penicillin's in the future? Or should we refer to an allergist for penicillin skin testing at that point? Is it safe to use the drug in the future?

> Approximately 10% of the population is labeled with a penicillin allergy, however over 90% of these people are negative to skin allergy testing and can tolerate penicillin.
> Allergic reactions to penicillin occur quickly within 1 hour of receiving a dose, even if they have tolerated the medication in the past. Typical symptoms include an urticarial rash and/or angioedema.
> Anaphylaxis to penicillin is rare and can include swelling of the tongue, throat, and lips, respiratory distress, or light-headedness and loss of consciousness due to low BP.
> If a patient shows any of these signs, it is recommended that the child stop using penicillin antibiotics and seek consultation with an allergist for penicillin skin testing. There is only about a 1% cross-reactivity issue with first generation cephalosporins (due to a similar R1 side chain) so switching the antibiotic to cefdinir (3rd-gen) or cephalexin (1st-gen) is completely reasonable.

C1-C4 complement deficiency is presented similarly to Bruton's hypogammaglobinemia. How could I distinguish between the two in a test question scenario? Is it just by the CH50?

> Bruton's is found in males (X-linked recessive) and it presents as severe bacterial infection around 6 months of age since B-cells are absent (no immunoglobulins). C1-C4 complement deficiency also results in bacterial infections, but not as severe. CH50 is a good lab test to differentiate these two, but also review the text from PBR for more details. CH50 is low in C1-C4 complement deficiency and normal is Bruton's agammaglobulinemia.

GENERAL PEARLS

* Complement can help clear pathogens in multiple ways, including by binding to Ag-Ab complexes and also by binding directly onto some types of bacteria, and thus promoting phagocytosis.

* NEISSERIA MENINGITIDIS (AKA meningococcus or meningococcal disease): Look for recurrent and overwhelming infections due to this bug in children under the age of two (after that age, they have antibodies towards various meningococcal serogroups). NEISSERIA GONORRHEA infections can also be severe.

* CH50: If this is normal, then ALL complement pathways are okay (C1–9).

C1–4 COMPLEMENT DEFICIENCY

C1-C4 complement deficiency may look similar to Agammaglobulinemia (AKA **B**ruton's = **B**oys = anti**B**odies = **B**acterial infections) because this part of the complement system works closely with anti**B**odies.

AGAMMAGLOBULINEMIA (AKA X-LINKED AGAMMAGLOBULINEMIA, AKA BRUTON'S AGAMMAGLOBULINEMIA)

Agammaglobulinemia (AKA X-linked agammaglobulinemia or Bruton's agammaglobulinemia) is X-linked, so it is seen in **B**oys. There is a total absence of **B** cells, which means there are **NO IMMUNOGLOBULINS. NO Igs**! Labs may show **high T-cell counts**. Patients have **tiny or absent tonsils and no palpable lymph nodes**. It results in recurrent **B**acterial infections and presents around 6 months of age. You might be presented with a child who has a history of "many antibiotic courses." This could refer to recurrent infections with enCAPSulated organisms, especially Pseudomonas, Streptococcus pneumonia, and Haemophilus influenza. Look for sepsis, meningitis, and recurrent pneumonia. Pneumocystis pneumonia (AKA PCP) does NOT occur in this disorder. If you see PCP, think Hyper-IgM or SCID! TREAT with IVIG for life and give prophylactic antibiotics. BMT is curative.
MNEMONIC: The age of presentation (6 months) happens to be around the same age when the mother's immunoglobulins/antibodies begin to wane!

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CARDIOLOGY

In the 2019 webinar replay min 34, the question asked was the most common cause of endocarditis (Strep Viridans vs. Staph aureus). The cardiologist said, 'Staph aureus.' In the 2020 PBR book, most common cause of infectious was listed as Strep Viridans, followed by Staph Aureus. Further, for acute bacterial most common cause is Staph Aureus and subacute bacterial most common causes is strep Viridans. Page 134-135. Can you clarify the most common cause of endocarditis?

> The greatest number of cases are due to Strep viridans, which typically causes a SUBACUTE infection. The most common cause of an ACUTE endocarditis is Staph aureus. Along with the acuity of the infection, also consider the entire history. If it's post-surgical, associated with a central line or related to IV drug use, choose Staph aureus.

In the discussion of HOCM on page 137 of the 2020 core study guide it states that HOCM results in septal hypertrophy leading to LVOT obstruction and mitral regurgitation. However, the video states LVOT obstruction and pulmonary stenosis. Are both correct or is one a typo? I'm not sure if the videos correspond to the 2020 materials or if they are from an older version.

> Thanks for asking. The videos are a little older than the 2020 books, so when there's a discrepancy between the two, please go with the more recently updated BOOK content ☺

Can you discuss the difference between critical vs severe pulmonary stenosis?

> Severe becomes "critical" once the patient becomes cyanotic. It's a clinical assessment. If it's critical, they need urgent care.

Is the shunt in truncus arteriosus bidirectional, or right to left?

> It's technically a right to left shunt because the aorta is supposed to carry "red" blood. But, when you are asked about a shunt, read the question very carefully to understand the SITE that is being discussed because a condition can have multiple types of shunts associated with it. For example, in a patient with truncus arteriosus, the shunt at the level of the VSD is a bidirectional shunt with mixing of the blood at the VSD. But, at the level of the aorta (and overall), this is a right-to-left shunt and results in a blue baby.

I'm reading the prophylaxis guidelines for SBE on page 136 of 2020 and I'm confused. It says prosthetics valve or prosthetic material use to repair valve: lifelong. Then it says repaired cyanotic CHD with prosthetic material: ppx for 6mo after surgery unless prosthetic valve material use, then lifelong ppx not needed. I'm confused what the difference between prosthetic valve and prosthetic material is because that seems to be a determining factor. Could you please clarify?

Great request for clarification. We learned something new as we addressed this question and discussed it with our cardiologist too! We'll likely be including a new PBR "PEARL" to help future members too. Have a look below.

> **PEARL**: When a procedure uses prosthetic "material," the general expectation is that the material will end up being covered with endothelium (become endothelialized) within 2-4 months. After that, no prophylaxis is needed to prevent endocarditis (hence the 6-month rule). However, any foreign material used to repair a valve (including prosthetic "material") will not undergo endothelialization and will therefore require lifelong prophylaxis. Also, if a repair is done with "material" but it leaves a residual defect, such as a VSD repair with a residual leak, then it's assumed that endothelialization will not happen, and therefore prophylaxis should continue.

On page 136 can you clarify which conditions require only 6 months of SBE prophylaxis? Is it lifelong prophylaxis anytime the VALVE is repaired, but only for 6 months if prosthetic MATERIAL is used for any other NON-VALVE heart defect that is CYANOTIC? What about repaired and un-repaired non-cyanotic heart conditions?

> Any unrepaired, non-cyanotic heart disease does NOT require prophylaxis.
> There's also no indication for prophylaxis in repaired cyanotic heart disease, UNLESS prosthetic material is used and it is NOT expected to endothelialize (or does not endothelialize completely due to a persistent stress).

I'm having trouble keeping the following different heart sounds straight; is there a way to

logically think through them or is just a matter of memorization? In particular paradoxical S2 split, wide split S2, wide pulse pressure, pulsus paradoxus

This is a pretty broad request, and not one that we can help with through written text. BUT, I found some YouTube videos. One is "great" and the other two are pretty "good." ☺

> Great video about murmurs, maneuvers and cardiac buzzwords.

> "Good" video #1 with mnemonics.

> "Good" video #2 with mnemonics.

What is a normal pulse pressure? What is considered to be a wide pulse pressure?

> Subtracting the diastolic BP from the systolic blood pressure gives you your pulse pressure. So, if the BP is 120/80, then the pulse pressure is 40.

> A normal PP is usually 33% - 50% of the systolic BP. A wide PP is generally considered to be a PP that is > 50% of the systolic blood pressure.

On page 118 in relation to WPW and adenosine, is adenosine normally used to treat WPW, but contraindicated in WPW with a fib and a flutter?

> Adenosine is used to **stop** SVT (which can happen acutely due to WPW) but not to "treat" WPW. We usually use beta blockers or ablation for **treatment** of WPW.

Tricuspid atresia and AV canal defect. Just wanted to confirm if this is also seen in Pompe's disease (as said in pg 334- key finding- cardiomegaly from deposition in the heart- look for LAD on an EKG)

> Yes, left ventricular hypertrophy causes left axis deviation. The book is correct.

> A good mnemonic for causes of Left axis deviation – **Left Her VILLA Project**

> Left = Left axis deviation

> Her = Hyperkalemia

> V = Ventricular hypertrophy (Left)

> I = Inferior wall MI

> L = Left bundle branch block

> LA = Left Anterior hemiblock

> Project = Primum type Atrial septal defect

Can you provide a general overview on how we should approach questions about pediatric HTN? How to diagnose and how to treat?

> For someone with a normal BP, do an **annual BP screening** at a well child check and routine lifestyle counseling about weight and nutrition.

> "ELEVATED BP": Offer counseling on lifestyle modifications and schedule a second BP measurement **in 6 months**. If the 2nd measurement is also high, check upper and lower extremity BPs. If it still falls in the "elevated BP" rage, get a third BP measurement in **another 6 months**. If it's still elevated, start ambulatory blood pressure monitoring (ABPM), start a diagnostic evaluation and consideration for subspecialty referral.

> STAGE 1 HTN WITH INITIAL BP MEASUREMENT: Lifestyle counseling followed by a second BP measurement in **1-2 weeks**. If it's still at Stage 1, check upper and lower extremity BPs and repeat BP measurements in **3 months**. If it's still at Stage 1, start ambulatory blood pressure monitoring (ABPM) to confirm HTN, start a diagnostic workup, initiate pharmacologic

treatment and consider a subspecialty referral.

> STAGE 2 HTN WITH INITIAL BP MEASUREMENT: Check upper and lower extremity BPs and offer lifestyle counseling. The second BP measurement should occur **within 1 week** OR you can make a subspeciality referral immediately for consultation within 1 week. If you check the second BP measurement yourself in about a week and it still fits the Stage 2 criteria, start ambulatory blood pressure monitoring (ABPM) and start pharmacologic treatment, or refer to a subspecialist to be seen within 1 week. If the patient is ever symptomatic, or the BP is very high (> 30 mm Hg above the 95th percentile), refer to the ED.

> Click HERE if you want to see the AAP guidelines, BUT don't get lost in the document! Look at TABLE 3 and TABLE 11. That's it ☺

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DERMATOLOGY

In the section of Tinea, there should be a treatment section (how to manage regular versus infected)

> Treat with antifungal creams such as clotrimazole, ketoconazole, terbinafine, or luliconazole.

Vitiligo vs Tinea versicolor vs Pityriasis alba. I am having a difficult time differentiating between vitiligo, tinea versicolor, and pityriasis alba. Can you please help point out how to keep these straight?

> These rashes can all look similar because they can all be hypopigmented, but the description, location, and pattern of the rash on the body should be a dead giveaway to the correct answer.

> **Vitiligo** is thought to be caused by an autoimmune process that destroys melanocytes and likely has a genetic component as well. Treatment includes corticosteroid creams, immune modulators such as calcineurin inhibitor ointments and light therapy (UVB). It can be associated with other autoimmune conditions such as Hashimoto's disease or celiac disease.

> **Tinea Versicolor** is a fungal infection of the skin mostly surrounding the trunk and shoulders. This rash can be hypo-/hyper-pigmented and resolves with anti-fungal treatments.

> **Pityriasis alba** is usually associated with eczema or seborrheic dermatitis found mostly on the face and upper extremities. The rash is hypo-pigmented with a fine scale on it and is thought to possibly change into vitiligo after a period of time.

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NEONATOLOGY

The video and audio say ROP is <32 weeks and the book says <30 weeks, for differing context between the book and video, which one should I be trusting?

> The book is more updated ☺

Why does ABO incompatibility occur only in a FIRST pregnancy and not the others following? Wouldn't the same risk of anti-A or anti-B IgG antibodies still be there in future pregnancies?

> Sorry for the confusion. It can definitely happen in ANY pregnancy if the mom is O and the fetus is A or B. But, as opposed to rhesus disease (where the exposure during pregnancy #1 leads to problems in pregnancy #2), ABO incompatibility can cause problems even in the very first pregnancy due to pre-made anti-A or anti-B antibodies.

> **PEARL**: RH disease is never present in a "first" pregnancy and ABO incompatibility can occur in any pregnancy.

ABO INCOMPATIBILITY

ABO incompatibility usually occurs in mothers with an "O" blood type. Naturally occurring "anti-A" or "anti-B" IgG antibodies may be present. This can result in hemolytic disease of the newborn in a FIRST pregnancy. So for hemolysis in a G1P1 baby, consider ABO incompatibility as the etiology.

RHESUS DISEASE (AKA RH DISEASE)

When checking for Rhesus Disease (AKA RH Disease), look for an Rh- mom in her SECOND pregnancy: Maternal IgM antibodies are made during the FIRST pregnancy and are too large to cross over into the fetal circulation. During the SECOND pregnancy, IgG antibodies are present that are small enough to cross. (This can cause ERYTHROBLASTOSIS FETALIS if they cross early in pregnancy.) Rh- moms are supposed to get RHOGAM at 28 weeks gestation, and then again after delivery if the baby is found to be Rh+.
* KLEIHAUER BETKE TEST: Check MATERNAL blood to see if there are FETAL red cells present.

How high yield is it to be able to estimate gestational age by physical exam? I've had several MedStudy practice questions that are more detailed (ie - determining a 31-32 week old vs a 34-35 week old) than what is presented in PBR. Is it worth taking time to memorize these small details and do you have any recs to make this easier?

> We don't think it's worth memorizing the smaller details you're probably seeing in that resource (our neonatology expert agrees). Please continue to focus on the core content in the PBR resources.

Single umbilical artery - pg 174. Is renal ultrasound still recommended when isolated single umbilical artery is found on exam and no other abnormalities?

> Single umbilical artery is found in 0.4%-0.6% of live births and most have no coexisting anomalies so routine imaging is NOT recommended. Monitor clinically for dysmorphic features or other anomalies on exam.

> Renal anomalies are found in 4%-16% of newborns with a single umbilical artery of which most are minor and insignificant.

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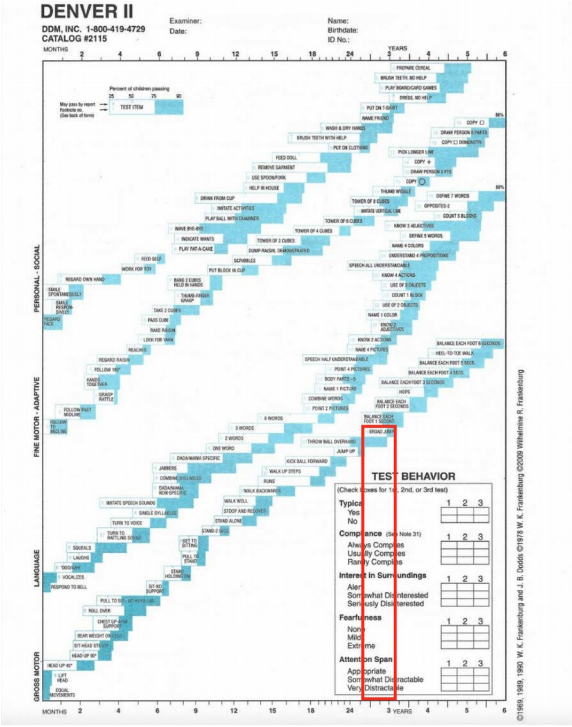
DEVELOPMENTAL MILESTONES

Broad jump happens at 4 years, not at 2 years.

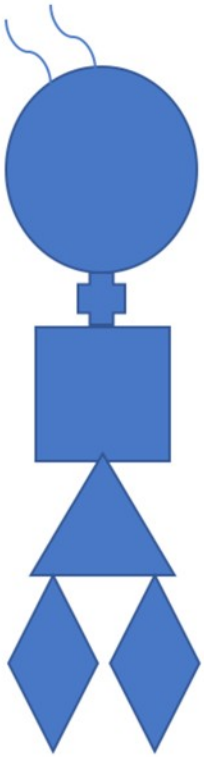
> According to PBR, broad jump occurs at age 2-3 years. This is correct. There's a lot of variability out there between different sources. If you look at the DENVER II, you'll see that the age ranges actually line up with PBR fairly nicely. And if you do an online search for milestones, you'll see that they are extremely variable depending on where you go. **Our chapter in PBR, as is, has served the author (me), our editor (John), and thousands of other PBR alumni VERY well. Their scores in that section have SKYROCKETED.** My recommendation is that you use ONE resource for this section (PBR's) and know it inside and out!

> 

Page 176 - I'm seeing some discrepancies from other sources (including the Peds in Review article) with the ages that toddlers are expected to draw certain shapes. 3 - circle, 4 - square/cross, 5 - triangle, 6 - diamond. Can you confirm if PBR is correct, or if the other sources are?

> Yes, PBR is correct and please only use 1 source (we would recommend PBR).

- 2 Hairs: Squiggly lines at 2 years of age
- Head: Circle at 3 years of age
- Neck: Cross at 4 years of age
- Torso: Square at 5 years of age
- Hips: Triangle at 6 years of age
- Legs: Diamonds at 7 years of age



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EMERGENCY MEDICINE AND TOXICOLOGY

In lead poisoning I was wondering when does the child need to be retested according to the lead level?

> This is unlikely to be tested since the guidance is based on the level. Higher levels warrant a more prompt recheck.

Are stimulants considered sympathomimetics? If so, why are there bowel sounds from stimulants? My understanding is that sympathetic responses result in a flight or fight response (dec GI motility/activity).

> We don't know the science behind it, but we did confirm that this is correct ☺.

Can you add a high osmolar gap mnemonic?

> ME DIES: Methanol, Ethylene glycol, Diuretic, Isopropyl alcohol, Ethanol, Sorbitol?

What is the difference between carbon monoxide poisoning vs methemoglobinemia poisoning, especially in terms of giving oxygen and what shows up on the pulse oximeter?

> Pulse oximetry is artificially high in CO poisoning because a pulse oximeter can't differentiate between **oxyhemoglobin** and carb**oxyhemoglobin**. So, giving O2 won't change the reading much in CO poisoning. PaO2 measures O2 dissolved in blood, which is not affected by CO, and is therefore normal. Dx is based on the exam and the CARBOXYHEMOGLOBIN measurement.

> In methemoglobinemia, the pulse oximeter will be low. Possibly showing around 85% even if

the TRUE hemoglobin oxygen saturation is higher. The pulse oximeter will not improve with hyperoxia. The PaO2 may be normal. Measure the methemoglobin level.

For CO poisoning, if not improving on 100% O2, do you 1) treat with 100% hyperbaric chamber or 2) treat cyanide poisoning for Na Thiosulfate?

> Treatment for possible cyanide poisoning in this scenario is reasonable. Cyanide poisoning results in hypoxia due to the body's cells not being able to take up or use oxygen from the bloodstream resulting in death. Giving 100% oxygen will not improve the condition and lactate levels will be elevated. Death will be rapid, unless treated with a small inhaled dose of amyl nitrite, followed by IV sodium nitrite, and finally IV sodium thiosulfate.

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VITAMINS AND NUTRITIONAL DISORDERS

On page 208, it states that Familial Hypophosphatemic Rickets is AKA Vitamin D Resistant Rickets. However, I had a practice question on Hereditary Vitamin D Resistant Rickets, and now I am confused. Can you please clarify the differences, and why in the Hereditary, there is high PTH and low calcium, but in the Familial, there can be normal PTH, calcium, and vitamin D values?

> Focus on Familial Hypophosphatemic Rickets as we don't cover Hereditary Vitamin D Resistant Rickets as it is low yield for the initial certification exam.
> Familial Hypophosphatemic Rickets has the following labs = Normal (or low) calcium, LOW serum phosphorus, HIGH ALKALINE PHOSPHATASE, normal Vitamin D 25, and NORMAL PTH since calcium is usually normal
> The key here for Familial Hypophosphatemic Rickets is that it has normal vitamin D 25 levels, but NOT normal vitamin D 1,25 levels.
> PEARL: For the test, they probably want you to focus on FAMILIAL Hypophosphatemic Rickets. The differentiating lab would be a low Vitamin D level (25) in early Vitamin D depletion, versus a normal level in Familial Hypophosphatemic Rickets.

In Vit D deficiency - can you please clarify- why should we start Rx with Vit D2 first and then Vit D3- why don't we just do Vit D3? i.e., cholecalciferol?

> Studies show that D3 is more effective than D2 in raising blood levels of calcifediol, so choose Vitamin D3 (cholecalciferol) if given the choice.
> Vitamin D2 (ergocalciferol) is found mainly from plant sources (mushrooms, fortified foods, dietary supplements) whereas Vitamin D3 (cholecalciferol) is found in animal-sourced foods (fish oil, liver, egg yolk, and butter).
> Calcifediol (25-hydroxyvitamin D2 + 25-hydroxyvitamin D3) is the main form of vitamin D circulating in the body.

The liver metabolizes vitamin D2 and D3 differently and vitamin D2 tends to yield less calcifediol than an equal amount of vitamin D3. GASTROENTEROLOGY

Does Gardner's Syndrome have the same lifetime cancer risk as plain Familial Adenomatous polyposis? Also, do they require the same treatment?

> You're unlikely to be asked on the exam to choose which disease has a higher risk of cancer.

PHARMACOLOGY AND DRUG PEARLS

On page 231 in reference to malignant hyperthermia, is the tachypnea a reaction to the acidosis and increased pCO2?

> Yes ☺ That causes a compensatory tachypnea.

Reading the last two bullet points on page 346 under ABG and Chemistry shortcuts, could you possibly explain this more or provide an example? Also, do you know any good resources to practice acid/base disorder questions?

> Great question. The 2nd to last one is simply referring to mixed acid base disorders. If it's a mixed disorder but the "strongest" or "overarching" disorder is a metabolic acidosis, you should have a low bicarb. If you do not, then you likely have extra base in the system.
> For the final bullet point, the opposite is true.

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OPHTHALMOLOGY

There were no ophthalmology clarifications for 2020!

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GENETICS

What percentage of Downs syndrome babies have CHD and other malformations?

	Prevalence (%)	References
Congenital heart defects	44–58	[34]
Vision disorders	38–80	[21], [27]
Hearing disorders	38–78	[21]
Obstructive sleep apnoea syndrome	57	[25]
Wheezing airway disorders	30–36	[4]
Congenital defects of gastrointestinal tract	4–10	[9]
Coeliac disease	5–7	[37]
Obesity	30–35	[30]
Transient myeloproliferative disorder	10	[39]
Thyroid disorders	28–40	[15], [28]
Atlanto-axial instability	10–30	[12]
Urinary tract anomalies	3.2	[16]
Skin problems	1.9–39.2	[18], [23]
Behaviour problems	18–38	[15], [21]

Prevalence of medical problems in children with Down syndrome

On page 249, the risk of future Down's children calculation (maternal age-related risk + 1%) - Is it maternal AGE or age-related RISK? If it's the risk, is this a constant number the question will give me? or should I not focus much on this calculation aspect?

> It is an age-related risk of having a child with Downs Syndrome. But, if there's been a previous Downs pregnancy, then add 1% to the age-related risk to get your new percentage.

The section on Duchenne Muscular Dystrophy (DMD) doesn't mention much on cognitive deficits. Are there any that we should focus on for the boards?

> There can be varying degrees of nonprogressive cognitive deficits in children with DMD with intelligence quotients (IQ) shifted downward one standard deviation below the normal range. So, if a case kind of sounds like DMD and includes mention of some "memory problems or trouble in school" pick DMD.

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HEMATOLOGY AND ONCOLOGY

What are some common peripheral smears?

> Our heme expert put together a few links for you to click on. **CLICK HERE** to see the links!

What are the most distinguishing features of these 3 disorders: Diamond Blackfan Anemia (DBA), Fanconi's Anemia (FA), Schwachman Diamond Syndrome (SDS)?

> Diamond Blackfan Anemia (DBA): Just RED CELL line effected resulting in significant anemia. They also show bone marrow defects and physical abnormalities (triphalangeal thumbs, cleft palate, microcephaly, ptosis). More risk of going into myelodysplastic syndrome (MDS).
> Fanconi Anemia (FA): All cell lines can be affected so pancytopenia can be present. Other features include bruising, recurrent epistaxis, fatigue, shorter stature, and hypopigmented patches.
> Schwachman Diamond Syndrome (SDS): All cell lines can be affected so pancytopenia can be present. Initially thrombocytopenia, pancreatic insufficiency, and neutropenia. These patients do have a high chance of moving forward to aplastic anemia and myelodysplastic syndrome.
> Neutropenia and FTT are prominent in SDS.
> Anemia is prominent in DBA and FA.

Can you review the important distinguishable labs for the different anemia? Also, when we see low retic count, we should think of iron def anemia and aplastic anemia, correct?

> Much of it depends on the clinical presentation and when you get your labs. For example, anemia of chronic disease (ACD) can have either microcytosis or normocytosis depending on when you get the labs. So, you can't use MCV alone to make these diagnoses.
> Alpha/Beta thalassemia: Low MCV. Lower in Alpha thalassemia than Beta thalassemia. Alpha thalassemia has a more homogenous cell size.
> Iron deficiency anemia: Low MCV. Hypochromic. RDW might be slightly elevated.

Differentiate the presentation of Maternal Immune Thrombocytopenia purpura vs Neonatal alloimmune Thrombocytopenia?

> Maternal Immune Thrombocytopenia Purpura (MITP): the mother usually has low platelets.
> Neonatal Alloimmune Thrombocytopenia (NAT): the mother creates antibodies to the FETAL

platelets. Mom's platelet count is normal.

I have come across a couple of questions in regard to Factor 5 Leiden mutation, but I do not see it in the book, can you please discuss if you think it is pertinent for the boards?

Great idea for a topic addition. I think Factor 5 Leiden is pertinent, so we'll add it to the new edition of the book.

> Factor 5 Leiden is an inherited condition that causes thrombophilia (increased tendency to form clots). Patients can be homozygous or heterozygous for the gene.
> HETEROZYGOUS: The majority of these patients go through life without any issues. If discovered, it's usually an incidental finding.
> HOMOZYGOUS: These are the patients who can require anticoagulation because of thromboses, DVTs, PEs, etc. These patients often have a high number of spontaneous abortions/still births.

New mnemonic of pediatric bone tumors.

You guys are great. Thanks for sending in such greate mnemonics. Here's one we might be adding to the next edition.

> osteogenic **S**arcoma - **S** - **S**unburst
> osteo**C**hondroma - **C** - **C**artilaginous **C**ap
> Osteoid Osteoma - O - Owl's eyes - sclerotic bone
> Ewing's Sarcoma - onion skin lesions which make your eyes hurt (painful lesions)

I don't think this is an error as much as it perhaps clarification.

From PBR on page 271, "If tested on a thalassemia, it will likely be beta thalassemia major. If you are provided with an MCV that is low (60–70) and "out of proportion" with the fairly mild anemia, pick a thalassemia (but not beta thal major). Look for microcytosis and target cells on a blood smear."

I thought that if target cells seen on a blood smear, that WAS beta thal major?? But, is this saying to not pick beta thal major?

> Unfortunately, target cells are not pathognomonic for beta thalassemia major. There are many other reasons you can see target cells. Usually if the question wants you to go towards thalassemia major , there will probably be something about receiving chronic transfusion/candidate for transplant/iron overload, elevated ferritin levels, or high HbA2 levels on electrophoresis.

Bleeding: Von Willebrand Disease may be autosomal recessive, right?

> Yes, usually Type 2N/2A and type 3 can be AR.

Do we need to know pediatric lead screening? It shows up in subject exams and ITEs.

> I think the key pieces of information to know for this topic include:
    - Screen everyone at around 9-12 months and then again at 24 months. If no screening was done prior to 36-72 months, screen at that time.
    - How you screen varies, and can range from a simple questionnaire to a blood lead test. If the lead exposure is widespread in a community, universal blood lead testing is recommended at ages 1 and 2. If exposure is not widespread, use questionnaires to guide "targeted" blood lead screening. If a parent or guardian can't answer the questionnaire, do blood lead screening.

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INFECTIOUS DISEASES

On page 289, *Citrobacter freundii* can cause a brain abscess. See below for a cool mnemonic to remember this fact!

Thanks for the mnemonic!

> *freundii* looks like Freud, so Sigmund Freud who was a neurologist and he is famous for his contributions to modern psychology and all things in the brain like an abscess

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A question about strep pharyngitis in absence of coughing/sneezing/rhinorrhea (page 284) - Is this for all kids or just older? I was told that some younger kids will not follow this rule absence of cough. For boards, should we assume if they are having viral symptoms, avoid strep diagnosis?

> You got it ☺
> Clinically, a cough can still be Strep pharyngitis. But, for boards, Strep patients are not going to present with a cough.
> Strep Rx: Penicillin/Amoxicillin, Azithromycin/Clarithromycin (resistant to penicillin), Cephalexin or Clindamycin.

On page 285 for the treatment of penicillin allergic patients using clindamycin/erythromycin -- is this only with true confirmed allergy? Can you use cephalosporin first if allergy is suspected/not confirmed?

> According to Norton AE, et al. Antibiotic Allergy in Pediatrics. *Pediatrics, 2018, 141*, 75% of patients were labeled penicillin allergic before the age of 3, however, when tested and undergoing drug challenges, > 90% of those children were able to tolerate penicillin.
> Most children labeled penicillin allergic do not have a true penicillin allergy.
> Penicillin, cephalosporins, and carbapenems all share a 4-membered beta lactam ring, therefore resulting in a theoretical risk of cross-allergy between the antibiotic groups. Given the low incidence of true penicillin allergy it would be safe to try a cephalosporin first unless the patient has had confirmatory skin testing for penicillin allergy.

On the top of page 290, it talks about Chlamydia trachomatis causing infection in a less than one-month old infant (concern for vertical transmission) and the treatment is oral erythromycin to eradicate nasopharyngeal colonization. Is it still okay to use this macrolide, given a child less than one month due to the risk of pyloric stenosis (page 281)? Are there alternatives to use to avoid this side effect? What would be the correct board answer?

> AAP Redbook & CDC recommend oral erythromycin QID for 14 days for either chlamydial conjunctivitis or chlamydia pneumonia or azithromycin once daily x 3 days.
> Both erythromycin and azithromycin are associated with increased risk of infantile hypertrophic pyloric stenosis (IHPS), particularly in infants younger than two weeks old, but benefits outweigh risks. So it's reasonable to simply advise parents of this increased risk.
> AAP Redbook 2018 says to treat chlamydial ophthalmia with oral erythromycin or oral azithromycin.
> Topical antibiotics for conjunctivitis are not effective and there is a high failure rate compared with oral antibiotics.

What medication can be given in less than 1 month? I picked azithromycin and that was the wrong answer (explanation was due to increased risk of pyloric stenosis), the correct answer was Linezolid. Can you explain further as PBR says azithromycin is safe less than 1 month (erythromycin is not).

> PBR is correct and I don't agree with that board vitals answer. I would have picked oral erythromycin
> AAP Redbook & CDC recommend oral erythromycin QID for 14 days for either chlamydial conjunctivitis or chlamydia pneumonia or azithromycin once daily x 3 days.
> Both erythromycin and azithromycin are associated with increased risk of infantile hypertrophic pyloric stenosis (IHPS), particularly in infants younger than two weeks old, but benefits outweigh risks, just advise parents of this increased risk.
> According to the CDC the established treatment for neonatal chlamydial ophthalmia is oral erythromycin syrup: 50 mg/kg body weight/day in 4 divided doses for 14 days. There is no indication that topical therapy provides additional benefit. If inclusion conjunctivitis recurs after therapy, erythromycin treatment should be reinstituted for an additional 1-2 weeks.
> Neonatal chlamydia pneumonia should also be treated with oral erythromycin syrup: 50 mg/kg/day in 4 divided doses for 14 days.

Could you please confirm that the treatment for sinusitis > 90 days is cefazolin (IV) - page 306? Thanks!

Sure!

> Acute < 30 days
> Subacute 30-90 days
> Chronic > 90 days
> IV antibiotics are recommended if orbital and intracranial complications, ICP, or nonverbal patients.
> PBR is correct on page 306 below.
> **CHRONIC SINUSITIS** (> 4 WEEKS): Look for evidence of some other comorbidity, such as hay fever, an immunodeficiency, immotile cilia, or a history of cystic fibrosis. < 90 DAYS: The usual HMS bugs. Treat with Amoxicillin. > 90 DAYS: Think **STAPHYLOCOCCUS AUREUS**. Treat with Cefazolin.

How long is the triple therapy for TB?

> Triple antibiotic therapy includes rifampin, isoniazid, pyrazinamide (quadruple therapy add ethambutol)
> Duration depends on type of TB: pulmonary (6mo) vs. meningitis (9-12 months) vs. osteoarticular (9-12 months).

On page 301, treatment for babesiosis is azithro + atovaquone. Clindamycin and quinine are alternatives, not first line.

> Babesiosis treatment: 1st line is Azithromycin + Atovaquone, but standard treatment for severely ill is Clindamycin + quinine

Under vertically transmitted diseases, there is nothing on HIV testing in kids. What tests at what age do you use in a child of a mom with HIV?

> HIV testing in an exposed baby at 6 weeks is DNA PCR, then a confirmatory antibody test. If > 6 months old, do HIV antibody testing followed by DNA PCR if HIV+.

VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

IVIG/Steroids delay in live vaccines - Can you clarify the time interval for delaying live vaccines (specifically MMR) in the setting of receiving Steroids or IVIG. I'm seeing different intervals. I understand that steroids is dependent on dose receive and with systemic (ie inhaled would not exclude) but do we wait 2wks or 4wks? I'm also seeing different intervals for period of waiting when receiving IVIG. Could you please clarify? Thanks!

> Delay vaccines (MMR or Varicella) for **11 months** in kids who get IVIG for Kawasaki.
> For high dose steroid (> 20 mg/day) or chronic steroid use (> 14 days use) wait **1 month** after stopping glucocorticoid therapy to give live virus vaccines (MMR or Varicella)

I may have missed it, but I didn't see the rec to give 1 dose of mmr and hep a between 6-12 months for international travel. Thanks.

> Depends on where you are traveling in the world and these recommendations are constantly changing based on the state department recommendations due to local outbreaks. This is beyond the scope of the pediatric certification exam and I would recommend discussing each case with the travel clinic at your local county health department.

Can you expand on when to give VZIG? Is there a certain time frame? Which infants receive it?

> Newborns of mothers who develop varicella five days before to two days after delivery.
> **PEARL**: VZIG (VZV immunoglobulin) is given for prophylaxis to newborns if the mom developed symptoms within FIVE days prior to delivery and TWO days after delivery. If symptoms started six days prior to delivery, NO PROPHYLAXIS IS NEEDED. Congenital varicella syndrome can result in low birth weight as well as CNS, eye and skin abnormalities.

When you give the meningococcal vaccine to a sickle cell patient? Would it be at 11 or do you start at 2 months?

The answer is that "it depends" on the age of the child when you see them. Because of the MANY age cut offs, this is probably something that's beyond the scope of the boards. And while the regimen is unlikely to be tested, it "might" be reasonable to expect to know that if NO vaccine has been given before, then vaccination SHOULD occur.

> Ages 2 - 18 months: Give Menveo at ages 2, 4, 6, and 12 months.
> Ages 19 - 23 months and have not received any Menveo vaccine prior: Give 2 doses of Menveo, 3 months apart.
> Ages 2 - 9 years and not received any Menveo vaccine prior: Give 2 doses of Menveo or Menactra, 2 months apart.
> Ages 10 - 55 years and not received any doses of Menveo or Menactra: Give two doses of Menactra or Menveo, 2 months apart **and** either Trumenba (three doses administered at 0, 1 to 2, and 6 months) **or** Bexsero (two doses administered at least one month apart)

I've had several practice questions on the combined MMRV vaccine and the increased risk

of febrile seizures. Is the risk only increased if the MMRV is given as the first dose, or is it age-dependent? (ie - if an older, unvaccinated child needs MMRV, should it be given as a combined dose or separate; and if a child younger than age 4 needs their second MMRV earlier, can they get the combined dose?)

> The increased risk for febrile seizures is based on the age of the child. The combined MMRV vaccine is not recommended for the 1st vaccine in the series. So, for the 1 year well child check use – MMR, Varicella, and Hep A. OK to use combination MMRV vaccine after 2 years old for the booster series as the risk for febrile seizures decreases dramatically after that age.

Can you clarify which exposures need post-exposure prophylaxis even in immunized individuals?

> Depends on the exposure criteria as summarized below. Also, if the patient is unimmunized, you will often catch them up on the vaccine as well.

POSTEXPOSURE PROPHYLAXIS

The following discusses postexposure prophylaxis 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 one case of exposure is required to start prophylaxis in contacts.

  H. INFLUENZAE B: <u>One close contact case (home/daycare/school) is required</u>. If the immunization status is unknown, also give the Hib vaccination. If you are presented with a question about the first child to be diagnosed with H. flu (the index case), then be sure to also immunize his/her household members.

- PERTUSSIS: Give a macrolide to all household contacts and all exposed people who are at high risk (including infants, pregnant women in their third trimester, those with a pre-existing illness like asthma, and those with contact with infants and pregnant women.

  <u>PEARL</u>: 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: Give 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.

  <u>PEARL/SHORTCUT:</u> 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 <u>newborns of mothers who develop varicella five days before to two days after delivery.</u>

- MEASLES:

Age	3 days or less	4-6 days	> 6 days
< 6 months old	MIG (IM)	MIG (IM)	None
6 – 11 months old	MMR	MIG (IM)	None
> 1 year old	MMR	None	None

PEARL: DO NOT GIVE MMR for at least 5 months after giving MIG

PEARL: DO NOT GIVEN MMR and MIG at the same time If the child is < 1 year and receives the MMR, then they will need another MMR according to the routine vaccine schedule

PEARL: If injection volume is a concern then use MIG (IV) instead of MIG (IM)

On page 324, there is discussion about chemo-prophylaxis for H. Flu and it states that there must be 2 cases prior to starting prophylaxis. I had a question during studying that asked about this and I missed it because I chose no prophylaxis was needed since the vignette made mention of only one case/exposure. I then proceeded to research to see if the question was possibly out of date, but according to the CDC close contact is all that is needed, so household, daycare or school to start ppx.

> Thanks for bringing this up. I think the book is generally correct, but it could be worded better. When you're talking about a daycare setting, the CDC and AAP say that 2 cases are needed within a 60-day window. However, for any case of H. Influenzae B, all HOUSEHOLD contacts should receive chemoprophylaxis. So in the setting of household contacts, only 1 case is needed because there's a much higher chance of spreading the disease. We'll change the topic to read as follows:

* > 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 one case of exposure is required to start prophylaxis in contacts.
- H. INFLUENZAE B: For school and daycare settings, TWO documented cases are required. However, only ONE case of exposure is required when considering prophylaxis for household contacts. So, if you are presented with a question about the first child to be diagnosed with H. flu (the index case) at a school, the household members of the index patient should receive prophylaxis, but the classroom contacts will not get prophylaxis until there is a second confirmed case. If the immunization status is unknown for someone receiving chemoprophylaxis, also give the Hib vaccination.

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INBORN ERRORS OF METABOLISM

On page 328 on the IEM table, it says that galactosemia in the newborn period has LOW glucose but NO ketones. I'm wondering why there are no ketones in galactosemia- won't there be fatty acid breakdown in this baby for energy, given the low glucose?

> Good question, but in galactosemia, it's the processing of LACTOSE that's the problem. Not glucose. Therefore, there's no need to breakdown fatty acids.

Sphingolipidoses - Can you please review the distinguishing features of the sphingolipidoses?

> The sphingolipidoses are inherited lipid storage diseases caused by defects in genes encoding proteins of the lysosomal catabolism. All sphingolipidoses are inherited in an autosomal recessive mode, with the exception of Fabry disease, which follows an X-linked recessive mode of inheritance.

TAY-SACHS DISEASE

Tay-Sach disease (AKA Tay-Sach's disease) **is a deficiency of Hexosaminidase A enzyme** (a lysosomal enzyme) resulting in **progressive neurologic deficits**. Patients can develop normally until about **9 months** but then begin to show signs of lethargy and hypotonia. They are also found to have macrocephaly, an **exaggerated startle reflex,** and a **cherry red spot** on the retina. Children usually die by the age of four. You are **REQUIRED TO SCREEN all kids born to Ashkenazi Jews for this, and may do so by amniocentesis or chorionic villus sampling.** It's autosomal recessive, which means both parents are carriers.
PEARL: **There is no organomegaly.**
MNEMONIC: Do you know anyone named Tay? Tate? Taz? Imagine TATE becomes famous and develops a HUGE HEAD (macrocephaly) from his success. He's now a smooth and LOOSE talking player. Everything is fine until a jealous PREGNANT ASHKENAZI JEWISH RABBI shoves a NEEDLE in his belly (amniocentesis) and a CHERRY in his EYE. He is HUGELY STARTLED (startle reflex) by how his fame has done him in.

GAUCHER DISEASE (AKA GAUCHER'S DISEASE)

Patients with Gaucher disease (AKA Gaucher's disease) have **hepatomegaly**, thrombocytopenia, easy bruisability, **osteosclerosis and lytic lesions with bone pain,** and often 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!

FABRY DISEASE (AKA FABRY'S DISEASE)

Fabry disease (AKA Fabry's disease) findings include opacities of the eye, vascular disease of the kidney, heart or brain, and reddish-purple skin lesions (angiokeratomas).
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 Kanye West wearing his famous WHITE SUNGLASSES (opacities) and an ORANGE-colored suit (skin) made of ROUGH (rash) **FABRY**c.

NIEMANN-PICK DISEASE

Niemann-Pick disease is a **sphingomyelinase deficiency** that results in accumulation of **sphingomyelin** in macrophages within the liver and lungs. Patients have **neurologic problems**, **hepatosplenomegaly,** and a **cherry red spot** on the macula.
PEARL: If you are given a patient with a cherry red spot on the macula and **hepatomegaly**, this is your answer! Tay-Sachs does not have organomegaly!
MNEMONIC: Imagine a mall socialite who is stumbling around Niemann Marcus taking shots of tequila with cherries on top. Niemann Marcus = niemann pick, stumbling around = neurological deficits, shots of tequila = hepatosplenomegaly, cherries on top = cherry red spots

Can you please review the distinguishing features of organic acidemias, urea cycle defects, mitochondrial d/o, fatty acid oxidation d/o, glycogen storage dz, aminoacidopathies?

> This chart on page 327 was created for this specific purpose. I would recommend reviewing it often and making flashcards to learn all the distinguishing features between different metabolic diseases.

Chapter 18: INBORN ERRORS OF METABOLISM & MISCELLANEOUS METABOLIC DISORDERS

NOTE: HIGH YIELD! This section is difficult. Focus on the absolutes for each category. If the chart is overwhelming, start on the next page and refer back.

	Glucose	NH3	Ketones	Acidosis?	Lactate	Notes & Names	Diagnosis/Treatment
Organic ACIDemias	LOW	HIGH (usually)	YES	+ GAP **acid**osis from organic **acid** build up	HIGH Except in Isovaleric acidemia.	- Presents by **DOL 2.** - CAN have THROMBOCYTOPENIA and Granulocytopenia (→ low WBC)! **ALL LABS ARE MESSED UP** - Isovaleric acidemia (foot odor/seizure), Glutaric acidemia (odor), MMA, Propionic academia	**Dx:** Urine Organic Acid Levels **Tx:** Hydration! Also change diet to a HIGH CARNITINE diet (helps to get rid of the built up organic acids).
Urea Cycle	OK	HIGH NH3 & LOW BUN	NO	NO Serum pH is normal or shows a mild **resp. Alkalosis**	- Usually normal, but can be slightly elevated	- **Hypotonia**! - Can have RESP. ALKALOSIS - Hyperammonemia without ketosis or liver dysfunction - Ornithine Transcarbamylase (OTC), Citrullinemia, Argininosuccinic Aciduria (ASA)	**Dx**: **Low Arginine** in all. No citrulline and high urine orotic acid in OTC. No arginine in citrullinemia. **Tx**: Restrict protein intake. Benzoate & phenylbutyrate (NH3 scavengers).
Fatty Acid Metabolism **Mitochondrial disorder (energy problem)!**	LOW Because the hypoglycemia starts the chain of events!	OK or HIGH	**NO** Can't make any!	MILD From anaerobic metabolism and protein breakdown	HIGH Lactic Acid & Uric Acid	- Induced by physiologic stress, illness, & **fasting** (low glucose). - Since it is a disorder of fatty acid metabolism, the fatty acids cannot be broken down. That means no ketone production. During fasting states, or when carbohydrate reserves have been used up, there is a **NONKETOTIC Hypoglycemia!** - MCAD	**Dx**: Low fasting glucose **without** appropriate rise in ketones. Obtain Carnitine or Acylcarnitine levels. **Tx**: IV GLUCOSE
GSD I (von Gierke) **Mitochondrial disorder (energy problem)!**	LOW	- Usually OK	YES	+ GAP acidosis +Lactic acidosis & +Ketoacidosis	HIGH Lactic Acid & Uric Acid Depends on anaerobic metabolism & protein breakdown	- GSD 1 is a true glycogen storage disease. Patients are unable to break down glycogen or perform gluconeogenesis. They depend on fats, protein, and anaerobic metabolism for energy. Glycogen builds up in organs, causing organomegaly. - GSD 2 = "pompLay" = a Lysosomal issue of glycogen accumulation (heart, liver). It's **not** a glycogen breakdown issue so there's no hypoglycemia, ketonuria, or acidosis.	**Dx**: Low fasting glucose with + ketones. **Tx**: Cornstarch to allow for slow carbohydrate breakdown
Amino Acidopathy	**OK** Except in MSUd (Low)	**OK** Except in MSUd (High)	**NO** Except in MSUd (High)	**NO** Except in MSUd (High)	**OK** Except in MSUd (due to Ketoacidosis from protein breakdown)	- For each specific disorder, there is a specific amino acid that cannot be broken down. - PKU (smell), Alkaptonuria (black urine), Tyrosinemia, Homocystinuria (clots, Marfanoid) - MSUd is a disorder in which ketoacids formed during the catabolism of Valine, Isoleucine and Leucine cannot be broken down. Patients are hypoglycemic at presentation because that is when there is a need for protein catabolism.	**Dx**: Look for high levels of specific amino acids in the serum and urine. **Tx**: Limit intake of specific amino acids based on the disorder.
GaLactosemia	LOW	OK	NO	**NO**	-	- Galactose-1-phosphate **uridyltransferase (AKA GALT)** deficiency, so galactose (a product of lactose) can't be broken down, and galactose-1-phosphate builds up and deposits in the liver, kidney, and brain. Patients often **present with GNR sepsis (especially E. coli).**	**Dx**: ↑galactose-1-phosphate, non-glucose reducing substances in urine, ↓GALT activity in RBCs **Tx**: Lactose & gaLACTOSE-free diet
HypER-GLYCINEmia	OK	OK	NO	NO	-	Unique because most labs are normal! Look for seizure activity (burst suppression on EEG), lethargy, or "hiccups."	**Dx**: High GLYCINE **Tx**: Limited tx options. Restrict diet. Poor prog.

What are distinguishing features to look for in glutaric acidemia?

> Low yield topic. Remember sweety feet! Those with early onset glutaric acidemia, type 1 (GA-1) will develop an encephalopathic crisis (due to bilateral striatal injury resulting in progressive neurologic movement disorders) by 2 years of age. Late onset GA-1 occurs after the age of 6 years old and results in chronic headaches, macrocephaly, epilepsy, tremor, and dementia. Glutaric acidemia, type 2 (GA-2) results is hypotonia, liver dysfunction, muscle weakness and cardiomyopathy. Glutaric acidemia, type 3 has no clinic phenotype.

> https://www.ncbi.nlm.nih.gov/books/NBK546575/

GLUTARIC ACIDEMIA

Glutaric acidemia can also present with the smell of SWEATY FEET.
MNEMONIC: If you can connect ERIC with SMELLY FEET, you're set: Glut-ERIC.

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ACID-BASE DISORDERS

Can you please give scenario examples of each type of acid base derangement - metabolic acidosis, metabolic alkalosis, resp alkalosis, and resp acidosis? Thank you.

> That's a bit too broad of a request. However, on the Acid-Base video page, I created a very good review of the chapter and I also go through about 7 practice questions. Look for the "*ACID BASE TUTORIAL & QUESTIONS FROM ASHISH GOYAL*." You can download a PDF of the questions and practice them on your own during the lesson ☺

On page 192, which mnemonic is for anion gap acidosis and which is for osmolar gap? I learned in medical school that MUDPILES was for high anion gap acidosis. Please clarify because both mnemonics state "high anion gap acidosis".

> Sorry for any confusion! Both mnemonics are for high anion gap metabolic acidosis. We'll update from MUDPILES to CAT MUDPILES to be more inclusive and remove CUTE DIMPLES due to incorrect inclusion of ethanol (see below question).
> **MNEMONIC**: CAT MUDPILES: Carbon monoxide/Cyanide/Congenital heart failure, Aminoglycosides, Teophylline/Toluene (glue sniffing), Methanol, Uremia, Diabetic ketoacidosis, Propylene glycol, Isoniazid, Lactic acidosis, Ethylene glycol, Salicylates for high anion gap metabolic acidosis.

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FLUIDS AND ELECTROLYTES

There were no fluids and electrolytes clarifications for 2020!

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NEPHROLOGY

What's the differential diagnosis for persistent hematuria?

> Hematuria is defined as the presence of 5 or more red blood cells (RBCs) per high-power field in 3 of 3 consecutive centrifuged specimens obtained at least 1 week apart.
> Glomerular etiologies = Alport Syndrome, HUS, Post-infection glomerulonephritis, Lupus nephritis, HSP, IgA nephropathy, and thin basement membrane disease (benign familial hematuria).
> Non-glomerular etiologies = fever, exercise, menstruation, foreign bodies, UTI, hypercalciuria/urolithiasis, sickle cell disease/trait, tumors, coagulopathy, drugs (NSIDs, anticoagulants, cyclophosphamide, antivirals), anatomic abnormalities (hydronephrosis, PCKD, vascular malformations)

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STATISTICS

There were no statistics clarifications for 2020!

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NEUROLOGY

Can you please confirm or clarify the following statements with regard to increased intracranial pressure (ICP)? (1) ICP can cause dilated and nonreactive pupils in one or both pupils and (2) papilledema seen in ICP is a bilateral phenomenon.

> Your question said that “ICP can cause...” ICP means intracranial pressure. Increased ICP can cause a dilated and nonreactive pupil on one or both sides. It can also cause papilledema. If papilledema is present, it will be noted on both sides.

When a patient presents with bilateral lower extremity weakness – should they be sent for imaging (MRI) right away, or lab work up first?

> Do a quick imaging study first. A CT would be most likely to be done first. An MRI will often require sedation and would be done once the initial workup has been done. Regarding labs vs. imaging, you should read the question that the question writer is asking to figure out which is the likely answer. If you are forced to choose between CT, MRI and labs, go with a CT unless there is history to suggest something like an electrolyte imbalance. But even then, that would likely cause a more generalized problem.

> Also consider whether the weakness is proximal versus distal. Proximal weakness is characteristic of muscle disease (muscle or neuromuscular junction), whereas distal weakness is suggestive of a neuropathy. Bilateral weakness of lower extremities, even if asymmetric, would suggest a cord lesion.

For migraines, after starting amitriptyline or topiramate for prophylaxis, when and how should it be weaned off? Should these pts be referred to neurology?

> These patients should definitely be referred to neurology and you should let them wean the medication. For the exam, it’s likely not going to be tested. What “could” be board-relevant for a general pediatrician is to know what the effects of sudden discontinuation of these meds could be. For amitriptyline, side effects can last 1-3 weeks after being abruptly taken off the medication and includes dizziness, headaches, vomiting, decreased appetite, diarrhea, muscle/joints aches, fever, chills, and sweating. For topiramate, this could include difficulty concentrating, anxiety, anger, depression, dizziness, fatigue, insomnia, mood swings/irritability, seizures, changes in vision, or weight gain.

On page 384 for the tx of infantile spasms it lists ACTH. I believe the correct treatment is now actually orapred (prednisolone). I wasn't sure for the exam (knowing that content many times is delayed by 1-2 years) however, this has been treatment now for over 3 years, and feel that it is likely free game for the test.

> According to uptodate.com that was last updated July 17, 2020 – Corticotropin (ACTH) is still preferred as first-line treatment in most patients. Glucocorticoids are insufficient as first-line treatment in most consensus statements and guidelines reviewed. The antiseizure medication, Vigabatrin, is also effective as initial treatment for infantile spasms, but best when used in combination with ACTH.

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ORTHOPEDICS AND SPORT MEDICINE

For scoliosis, what do you do if the degree is > 25 degrees and the child is done growing?

Because I know you brace if they are still growing but what if growth is completed?

> The brace helps to keep the curve from getting worse as the child grows. After they are no longer growing, you watch. So, no brace would be needed in your proposed scenario. Surgery would be the treatment for complications or if > 40 degrees.

On page 394 in relation to developmental hip dysplasia, the book states to ultrasound between 6 weeks to 4 months; however, webinar states ultrasound up to 6 months. When I skimmed Up to date it also said u/s up to 6 months. Please clarify.

> Our book addresses this. Please see the PEARL after the bullets. Both ultrasound and x-ray can be used for 4-6 months of age, but x-ray is recommended during this window as stated in the CSG.

In toddlers fracture you don't talk of normal spiral fractures that happens to kids. It makes it seem like all spiral fractures are related to abuse.

> Ahh... In the book, we were making sure that you knew of the red flags to look for when the full history and clinical picture seems "suspect." Spiral fractures can definitely happen to toddlers with no abuse.

Should we spend time on learning common presentations of knee ligament tears such as ACL, MCL, PCL, or meniscus? ACL tears are the only tears mentioned. Also, should we spend time learning about other overuse injuries i.e. tennis elbow etc?

You know, that seems reasonable. So, here we go...

> Knee injury
    - MCL is the most common ligamentous injury of the knee
        - How: Valgus stress (contact), Skiing (non-contact)
        - History: POP reported at time of injury
        - Symptoms: medial joint line pain, difficulty ambulating
        - Diagnosis: MRI
        - TREATMENT: NSDAIDS, bracing, rest, surgical repair
    - ACL
        - How: ACL injuries most commonly occur during sports that involve sudden stops or changes in direction soccer, Basketball, skiing, football
        - History: POP reported at time of injury, immediate swelling
        - Symptoms: effusion, lack of full extension
        - diagnosis Lachman's test, MRI
        - TREATMENT: NSDAIDS, bracing, rest, surgical repair
    - Meniscal injury
        - How: sports related, squat and twisting motion
        - Medial tears more common than lateral
        - Symptoms: locking and clicking when squatting
        - Test: Apley compression, McMurray test
> Lateral epicondylitis (Tennis Elbow)
    - Overuse injury involving eccentric overload at origin of common extensor tendon
    - Symptoms pain with resisted wrist extension, pain with gripping activities
    - Physical exam: point tenderness at ECRB insertion into lateral epicondyle
    - Diagnosis based on physical exam
> Little League Shoulder
    - Overuse injury resulting in epiphysiolysis of proximal humerus → Salter Harris Type I
    - Who: adolescent pitchers (baseball or softball)

- Dx: XR

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RHEUMATOLOGY

After reviewing the Pediatric Board content on the ABP website, under rheumatology they list "psoriatic arthritis, fibromyalgia and inherited d/o of connective tissue" which are not in PBR. Can you briefly touch on these topics as it pertains to the boards?

> PSORIATIC JUVENILE IDIOPATHIC ARTHRITIS (psJIA): Younger children (< 6 years old) tend to be female, ANA+, may have dactylitis and arthritis of the wrists, hands and feet. Older children have an even ratio of male-to-female and may have enthesitis (pain at insertion points of ligaments and tendons), spinal disease or sacroiliac disease. Any child with psJIA can have uveitis. About 50% of children can have psoriasis (usually mild). For the boards, you will not be tested on the consensus criteria for diagnosis, but you might be given a child with some of the above findings and mildly elevated inflammatory markers (ESR, CRP, platelets) and a slightly elevated ANA titer. The goal of treatment is to ultimately have normal labs, normal imaging and to avoid having any bony or cartilaginous damage. For mild cases, NSAIDS may be enough, but for those with widespread disease NSAIDS plus DMARDS can help to induce remission. If the patient has psoriasis, treat like any other patient with psoriasis.

> FIBROMYALGIA: Look for a child who complains of "hurting everywhere" due to chronic, widespread pain. There may be points of tenderness, fatigue, depressed mood or subjective joint swelling. A limited workup can be pursued (CBC, chemistry, ESR, CRP, TSH, UA). If there are joint complains, an ANA and a rheumatoid factor can be obtained. There are multiple, controversial diagnostic criteria, but they all seem to include the presence of many tender points and the presence of widespread pain. Treatment may involve recommendations on having structure for living with this disease, routine exercise that is slowly increased in intensity over time, and cognitive-behavioral therapy (CBT). Medications are usually not needed, but SSRIs may be prescribed.

> For a review of connective tissue disorders, please focus on Marfan Syndrome and Ehlers-Danlos Syndrome in the PBR Core Study Guide.

MARFAN SYNDROME (AKA MARFANS SYNDROME)

Classic features of Marfan 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 **mitral valve 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 homocystinuria. If they mention SUPERIOR subluxation of the lens, pick Marfan. Any patient with Marfan 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**: www.pbrlinks.com/MARFANS1 - Please do not get distracted by the reading. Look at the images and move on.

* **IMAGE**: www.pbrlinks.com/MARFANS2

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

EHLERS-DANLOS SYNDROME

Ehlers-Danlos syndrome has many similarities to Marfans Syndrome, including joint hypermobility, possible mitral valve prolapse (MVP), and aortic dissection. Skin laxity (loose skin) is a key differentiating feature. Other unique features include contractures, skin nodules, **easy bruisability** and poor wound healing. Also, after getting wounded, patients form "cigarette paper" scars. (Skin, skin, skin.)

IMAGE: www.pbrlinks.com/EHLERSDANLOS1

IMAGE: www.pbrlinks.com/EHLERSDANLOS2

IMAGE: www.pbrlinks.com/EHLERSDANLOS3

MNEMONICS: Rename it as "That feller's *daaamn* loose!" = "That fella is damn loose," said with a southern drawl). Also, if you get confused between Marfans and this one, remind yourself that Michael Phelps doesn't have any skin findings.

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PULMONOLOGY

I get a lot of practice questions on vocal cord dysfunction. Can I get a brief summary of this disorder?

> Vocal Cord Dysfunction (VCD) also known as Paradoxical Vocal Fold Movement (PVFM) is after confused with asthma and occurs when the vocal cords don't open correctly. It is possible to have both VCD and asthma, but the treatments are very different. VCD presents as **difficulty with inhalation** while asthma is an obstructive lung disease that cause **difficulty with exhalation.**

> Symptoms of VCD include difficulty breathing, throat tightness, hoarse voice and voice changes, wheezing, and chronic cough.

> Symptoms worsen when triggers such as exercise, lung irritants, viral URI, or GERD

> Diagnosis is challenging and often requires spirometry or laryngoscopy. Referral to an allergist/immunologist or pulmonologist can be helpful.

> Treatment consists of activities that relax the throat muscles such as controlled breathing exercises and speech therapy.

Pg 404 Under the "Congenital Diaphragmatic Hernia" section, "Chest X-ray will show a small atelectatic "right lung"....Would this be left lung, since most hernias occur on the left side?

> The left side with have hypoplastic lung tissue due to bowel herniation into the left chest wall cavity whereas the right side with have atelectasis due to shifting of the mediastinal contents to the right.

> https://www.nature.com/articles/jp2009129.pdf?origin=ppub (good CXR pictures on page 2 of this article)

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PSYCHIATRY AND SOME SOCIAL ISSUES

What is the time length that separates grief reaction, from adjustment disorder, from major depressive episode?

- The timeline is not exact. Hopefully this will help.
- **Grief reaction** is a healthy and normal response after the loss of something to a person. It can last 6-12 months, or even longer, but should not greatly affect activities of daily life.
- Symptoms of an **adjustment disorder** start within three months of a stressful event and last no longer than 6 months after the end of the stressful event. Adjustment disorders also can sometimes turn into major depressive episodes in people who are at risk for developing mood disorders.
- **Major depressive disorder** is characterized by a persistent feeling of sadness and loss of interest and can interfere with your daily functioning (screen with PHQ-9 form) and requires therapy and medications (SSRIs) and can last for years.

On child abuse, page 417 says "never make an accusation on the initial meeting" and later says "notify authorities immediately", so which one is it?

- The idea is to build trust with the family and child. However, if there suspicions of child abuse, it is your duty to report your suspicions to the authorities.

COMMON ABUSE-RELATED FRACTURES

Fractures of the scapula, sternum, spinal processes, multiple ribs, skull, and any spiral fracture should raise suspicion for abuse-related fractures.
IMAGE: www.pbrlinks.com/CHILDABUSE1 – Healing rib fractures

BUCKET HANDLE FRACTURES AND CORNER FRACTURES

Bucket handle fractures and corner fractures are **highly specific for abuse**. They are fractures of the metaphysis caused by sudden pulling, which causes avulsions.

IMAGE: www.pbrlinks.com/CHILDABUSE2 – Bucket handle fracture
IMAGE: www.pbrlinks.com/CHILDABUSE3 – Corner fracture

NAME ALERT/PEARL: 

Buckle fractures are NOT associated with child abuse. Other fractures/conditions that are more likely due to an **accident** include linear skull fractures, supra-condylar fractures of the elbow, and clavicle fractures.

RETINAL HEMORRHAGE (AKA SHAKEN BABY SYNDROME)

Caused by shaking a baby with force resulting in abusive head trauma from repetitive acceleration-deceleration injury. Injury can cause a subdural hematomas (SDH) and vision loss. Close follow-up with an ophthalmologist is recommended.

Can you clarify whether a person with a single kidney should avoid contacts sports or can play contact sports with appropriate protection? The book (page 417, bottom) clearly states avoid BUT when I listened to the previous webinar it stated they can play contact sports with protection.

- The book is more updated so please go with those recommendations. In general, kids with a single kidney are allowed to play contact sports, but nephrologists definitely don't like that idea. Therefore, the recommendation is to "avoid" contact sports.

ETHICS

Can you give HPV vaccine (which is technically sexually related) to a child without parental consent or overt parental objection?

> This is not something that is standardized in different states. Some states say “yes,” and some say “no.” Therefore, you will not be tested on this.

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PATIENT SAFETY AND QUALITY IMPROVEMENT

There were no patient safety and quality improvement clarifications for 2020!

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!
4. If you're feeling pretty good about your pediatric KNOWLEDGE/CONTENT, then work on your TEST-TAKING STRATEGY by going through the **CRASH COURSE on Test-Taking Strategies** (it has been a HUGE HIT)!

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For the Acid Base chapter, I've even included a short impromptu practice question session as well as additional practice questions for our Online Video Course and All Access Pass members to enjoy.

The Acid Base discussions and resources show how confused pediatricians are about the delta delta, when to check for compensation, Winter's formula, etc… but by these talks and practice sessions, we get comments like, "This is so much easier!... The light bulb just went off!"

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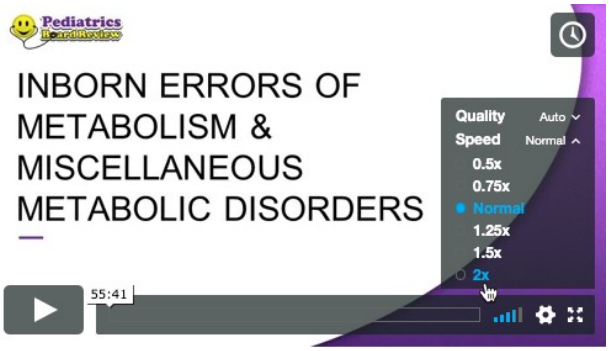

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