Pediatrics Board Review

2023 Corrections and Clarifications Guide

13th Edition
Your EFFICIENCY BLUEPRINT to
Passing The Pediatric Boards

2023 EDITION

YOUR SUCCESS BLUEPRINT TO PASSING THE PEDIATRIC BOARDS

www.pediatricsboardreview.com

100% Money Back Pass Guarantee • Powerful Mnemonics
Massive Online Community • Board-Focused Content
Efficient Learning So You Can Enjoy Life And Have More Fun!

Written by Ashish Goyal, MD

PBR'S ANNUAL CORRECTIONS AND CLARIFICATIONS GUIDE

Every year, I review all the PBR submissions for potential errors and requests for corrections from my members. It's a lengthy process, taking months, but I like to do it before the initial certification exam. I do my best to address every single submission, and I include those submissions, and my thoughts in this document. Please note that other companies do NOT do this, and this NOT included in any of my packages. It's just a special "PBR touch" I like to add for you!

Please note that 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 this bonus material has helped them correctly answer questions that came up on the exam.

I hope you take a couple of hours to skim through this document. If you have to choose only one section to go through, then since "CLARIFICATIONS" section may include questions around topics that you never needed clarification on, I recommend that you go through the "CORRECTIONS" section. It's short, easy, and worth the hour that you'll put into it.

Enjoy!

Ashish & Team PBR

Ashish Goyal, MD
CEO, Pediatrics Board Review Inc.
Board Certified Pediatrician
Board Certified Internist
American Board of Pediatrics (ABP) Question Contributor
Author, Pediatrics Board Review Educational Series
Founder of AVSAR, a 501-c-3 nonprofit organization
Web: www.PediatricsBoardReview.com

BUT FIRST... AN ANNOUNCEMENT!

PREORDERS, DISCOUNTS & FREE BONUSES FOR THE NEW 2024 EDITION OF BOOKS (14TH EDITION) ARE NOW AVAILABLE!

FREE BONUS #1

To pass the boards, you're going to need a PLAN! We've helped thousands of pediatricians pass by assisting with study plans and study schedules. Having one is a key part of your prep. Team PBR will create up to THREE 90-Day Personalized Study Schedules for you with any All Access Pass or No Brainer order.

THREE 90-DAY PERSONALIZED STUDY SCHEDULES **$894 Value ($297 Each)**
FREE With Your All Access Pass or "No Brainer" PREORDER.

FREE BONUS #2

Passing the "board game" requires knowledge of the RULES to the game and how to apply them. This includes understanding the different TYPES of questions you will come across and also the different ALGORITHMS needed to process each type of question. That's what you get in our Core Concepts in Test-Taking Strategies Webinar.

CORE CONCEPTS in Test-Taking Strategies Webinar **Normally $197**
FREE With Your All Access Pass or "No Brainer" PREORDER.

WONDERING WHAT THE "**NO BRAINER**" IS?
VISIT THE NEXT 3 PAGES TO SEE EVERYTHING THAT'S INCLUDED!

THE "NO BRAINER" BUNDLE

This bundle was created to address the 3 pillars of passing the pediatric boards...

CONTENT | TECHNIQUE | COMMITMENT

So, it's not surprising that this is (by far) the
MOST COMMONLY PURCHASED PBR BUNDLE!

Money Back First-Time Pass Guarantee!

THE "NO BRAINER" BUNDLE

CONTENT SUPPORT

You'll get a 12-month All Access Pass to 100% of PBR's content-based resources to help build your pediatric fund of knowledge. You'll get hardcopy books, online books, our Online Video Course, our MP3 audio course, access to our "**ASK THE EXPERT**" question portals and MUCH MORE.

TECHNIQUE SUPPORT

You'll get access to PBR's Full Online Test-Taking Strategies Course. It's self-guided course with 7 webinars to teach you how to process questions like an expert. You will *NEVER look at board-style questions the same way again.*

COMMITMENT SUPPORT

You'll get THREE 90-Day Personalized Schedules created by Team PBR, just for you. You'll fill out a detailed questionnaire about your history with exams, your pace of reading, your work schedule, the days you are reserving to study, when you want to take practice exams and MUCH more. You'll also have access to our live "Board Success" series of webinars throughout the year with tips and guidance on how have "Deep Study."

2024 EDITION OF THE "NO BRAINER" BUNDLE

(DISCOUNTED PREORDER BUNDLE)

- Hardcopy Core Study Guide
- Hardcopy Q&A Book
- Online Core Study Guide
- Online Q&A Book
- DOWNLOADABLE MP3 Audio Course
- STREAMING MP3 Audio Course
- ONLINE VIDEO COURSE
- Private Facebook CREW Access
- FULL ONLINE TEST-TAKING STRATEGIES COURSE
- Online Picture Atlas
    - $97 Bonus & Not Available Through Our General Catalog Area
- Downloadable PDF Picture Atlas
    - $97 Bonus & Not Available Through Our General Catalog Area
- Live Summertime Q&A Webinars with Experts
    - $145 Value & Not Available Through Our General Catalog Area
- "ASK THE EXPERT" Question Portals
    - $197 Bonus & Not Available Through Our General Catalog Area
- THREE 90-Day Personalized Schedules Created by Team PBR
    - $894 Bonus & Not Available Through Our General Catalog Area

FIRST-TIME MONEY BACK PASS GUARANTEE

Click Here Now & Signup Before the Offer Ends!

~~$6473~~ VALUE FOR ONLY $1597

(75% OFF + FREE SHIPPING)

www.pbrlinks.com/PREORDER

(Limited-Time Offer)

(New Orders Only)

2024 EDITION OF THE 12-MONTH ALL ACCESS PASS

(DISCOUNTED PREORDER BUNDLE)

- Hardcopy Core Study Guide
- Hardcopy Q&A Book
- Online Core Study Guide
- Online Q&A Book
- DOWNLOADABLE MP3 Audio Course
- STREAMING MP3 Audio Course
- ONLINE VIDEO COURSE
- Private Facebook CREW Access
- Online Picture Atlas
    - $97 Bonus & Not Available Through Our General Catalog Area
- Downloadable PDF Picture Atlas
    - $97 Bonus & Not Available Through Our General Catalog Area
- Live Summertime Q&A Webinars with Experts
    - $145 Value & Not Available Through Our General Catalog Area
- "ASK THE EXPERT" Question Portals
    - $197 Bonus & Not Available Through Our General Catalog Area
- 90-Day Personalized Schedules Created by Team PBR
    - $297 Bonus & Not Available Through Our General Catalog Area

FIRST-TIME MONEY BACK PASS GUARANTEE

Click Here Now & Signup Before the Offer Ends!

~~$4882~~ VALUE FOR ONLY $1097

(78% OFF + FREE SHIPPING)

www.pbrlinks.com/PREORDER

(Limited-Time Offer)

(New Orders Only)

2024 EDITION OF THE ULTIMATE BUNDLE PACK + MP3 AUDIO COURSE

(DISCOUNTED PREORDER BUNDLE)

- Hardcopy Core Study Guide
- Hardcopy Q&A Book
- MP3 DOWNLOADABLE Audio Course
- Online Core Study Guide
- Online Q&A Book
- Online Access for 1 Full Year
- GET FREE SHIPPING

FIRST-TIME MONEY BACK PASS GUARANTEE

Click Here Now & Signup Before the Offer Ends!

~~$1388~~ VALUE FOR ONLY $347

(75% OFF + FREE SHIPPING)

www.pbrlinks.com/PREORDER

Promo Code: **PREORDER**

(Limited-Time Offer)

(New Orders Only)

HAVE YOU FAILED? IF SO, YOU NEED THE VIP BUNDLE.

If you have failed while feeling as though you had a strong handle on the content, it's likely that you have a test-taking technique or a "Deep Study" issue. Reach out to us and ask about our VIP Bundle. It includes everything from the No Brainer, but also includes a live, online 2-Day Test-Taking Strategies & Deep Study.

The Live Test-Taking Strategies & Deep Study Course covers:

- Test-taking skills to help you process every board-style question systematically
- Test-day planning to maximize your time, energy and brain power
- Anxiety management
- Diet management
- Study group coordination
- Question-answering group coordination
- One-on-one coaching at the live course
- DEEP STUDY lectures on limiting distractions, creating accountability and maximizing your focus
- AND MUCH MORE

~~$10,470~~ VALUE. PREORDER FOR ONLY $2997

(71% OFF + FREE SHIPPING)

www.pbrlinks.com/PREORDER

Promo Code: **PREORDER**

(Limited-Time Offer)

(New Orders Only)

A FEW WORDS OF THANKS TO THE PBR COMMUNITY

1. Thank you! Thanks 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. NINA AKALIS! Nina is one of my teammates, and she was instrumental in helping me create this year's Corrections and Clarifications Guide. She did a GREAT job of helping me get this to you with very high-quality answers and research!

4. A huge thanks to our Online Video Course Summertime Webinar speakers. They contributed to MANY of the chapter corrections or revisions!

   - Dr. Amar Dave
   - Dr. Asalim Thabet
   - Dr. Kara Wada
   - Dr. Shamila Zawahir
   - Dr. Arpit Agarwal
   - Dr. Lina Huerta-Saenz
   - Dr. Stephanie Felton
   - Dr. Kirshma Khemani
   - Dr. Moshe Cohn
   - Dr. Shubham Bakshi
   - Dr. Kumar Nadhan
   - Dr. Yorgo Zahlanieh

HOW DO WE MAKE THIS THING?

IN ORDER OF PRIORITY, OUR FOCUS HAS BEEN….

1. **Addressing error submissions from the PBR Error portal**. 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 "ASK THE EXPERT" 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.

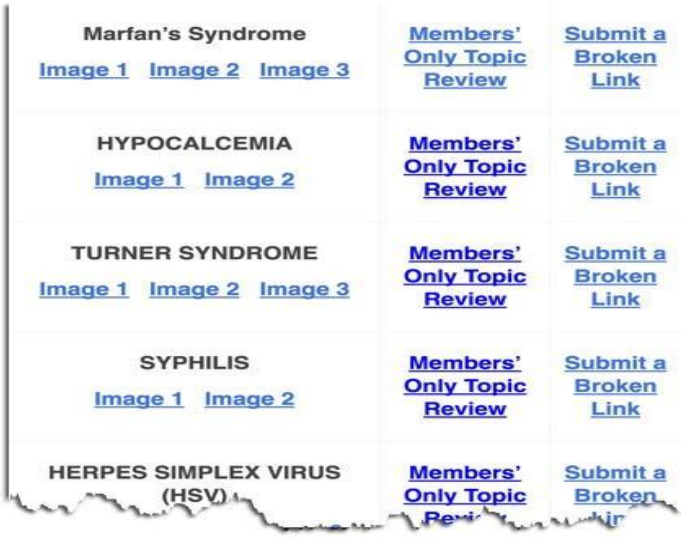
Because the PBR membership continues to grow, there has been EXCELLENT chatter in "The PBR CREW." **If you are a member of the "The PBR CREW" but you have NOT been seeing all of the posts**, please visit the private group and **make sure that your NOTIFICATION SETTINGS ARE SET TO ALL POSTS**. This is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

SHOULD I REVIEW IMAGES BEFORE THE EXAM?

Maybe! If you have a couple of hours, 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. Please note that this resource is only available to All Access Pass and No Brainer members.

Click the image below and get access to 50 free images and see how the VAPP works.

www.PediatricsBoardReview.com/VAPP



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 that will be discovered by our team. There may also be some submissions that we need to do additional research on. Those will result in additional changes to the next edition. 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.

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

It does not make sense for you to use an old, marked up book, with incorrect content to study for the biggest exam of your life. Please get the resources that are specifically for the year that you are taking the exam! We also add new topics almost every single year. If you have notes in your old book(s), transfer them over and then start fresh with my highlighting method.

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 **2023 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!

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.

Do you have more errors to submit? Send them over!
www.pediatricsboardreview.com/ERROR

CORRECTIONS FOR 2023 EDITIONS

CH. 1 – ADOLESCENT MEDICINE

There were no Adolescent Medicine corrections for 2023!

CH. 2 – ENDOCRINOLOGY

There were no Endocrinology corrections for 2023!

CH. 3 - OB/GYN & SOME STDS

PBR says absolute contraindications to OCPs include hyperlipidemia. With all due respect, can I get a confirmation please? In all the times I've learned about absolute contraindications to OCPs, I've never been told hyperlipidemia is one. Wanted to clarify for board exam, but this would also be a very helpful clarification in real life!

We checked multiple resources, and it is a contraindication. However, it's not an "absolute" contraindication. For the purposes of the boards, consider it to be a contraindication unless the hyperlipidemia is well controlled. Here's the new section!

ORAL CONTRACEPTIVE PILLS (OCPs)

* Absolute Contraindications to oral contraceptive pills (OCPS): Pregnancy, liver disease, breast cancer, and breastfeeding at < 6 weeks.

* Relative Contraindications: Hypertension, uncontrolled hyperlipidemia

* Anticonvulsants interfere with absorption, so instruct the patient to use a backup method of contraception. Consider using an OCP with more ESTROGEN.

On page 90 of the GBS section: Should every well-appearing baby with a GBS+ mom who received appropriate intrapartum antibiotics be observed for 48 hours?

If a mother is GBS positive but giving birth by C-section, no treatment is needed because the baby is not going through the birth canal. For a vaginal delivery, if a baby ≥35 weeks appears well and a mom who is GBS+ received appropriate intrapartum antibiotics, continue with routine care.

The latest AAP guidelines include **three options** for risk assessment of early-onset GBS sepsis for ≥35 weeks gestation and **an algorithm** for <35 weeks. We'll update this section in the core study guide!

CH. 4 – ALLERGY & IMMUNOLOGY

Can you please help me understand this (page 107): "If you are presented with an AIDS or immunocompromised patient who had a negative PPD, consider further skin testing (usually candida or tetanus)." Does a negative PPD mean there's an abnormal or a normal immune system in the AIDS/immunocompromised patient? And will a positive result in further skin testing mean the immune system is intact?

When a patient has impaired T cells, such as in AIDS, you could have a false negative PPD because their T cells may not be able to mount a response. In these patients, you can use a Quantiferon gold test and CXR to check for TB. If the PPD is positive, believe it.

Everyone has been exposed to Candida, and everyone who has received the initial tetanus vaccine should have a response to tetanus. Therefore, if you don't see a response to intradermal tetanus or candida, the person's T cell's functions may not be intact, and the PPD may not be a valid test. In practice, we use a blood test instead of a skin test to stimulate lymphocytes, exposing T lymphocytes to candida or tetanus, but the ABP exam may still ask about the candida/tetanus skin test. Likewise for checking for CGD with the nitroblue tetrazolium test, which isn't used anymore but could still asked about on the boards.

CH. 5 – CARDIOLOGY

Why are diltiazem and verapamil (calcium channel blockers) absolutely contraindicated for children younger than 12 months? (page 119 of 2023 version)

Verapamil is absolutely contraindicated in children under 12 months because of the risk of severe hypotension and asystole in that age group. Diltiazem is not contraindicated in children under 12 months, so we will update the guide with that correction! Thank you!

CH. 6 – DERMATOLOGY

2023 edition, page 149: I thought you could now give doxycycline to children under 8 as they no longer believe it causes teeth staining.

You're right! There have been some recent studies supporting doxycycline use in children under 8 for short courses < 21 days. We'll update the study guide!

I was taught that primary HSV gingivostomatitis needs to be treated with PO acyclovir x 7 days and HSV reactivation can just be provided supportive treatment.

On the top of page 157, PBR says to treat with oral acyclovir, "but there is limited data supporting this in children." We should treat primary HSV in children, correct?

Primary HSV gingivostomatitis can be treated with acyclovir in children and adults. Reactivation can be treated as well. If acyclovir is started within 72 hours, it can improve symptoms and shorten duration of illness, but it will also resolve without treatment. Treatment is optional, but recommended since it's a low-risk medication and helps symptoms.

Why does fluorosis occur only in kids? And PBR says this occurs up to age 4, but I've seen other sources say up to age 8.

Fluorosis doesn't affect people older than 8 years old because the permanent teeth stop developing after that age. We've updated the Core Study Guide.

FLUOROSIS

Fluorosis is the mottled discoloration of teeth due to excess fluorine use during tooth development (up to age 8).

IMAGE: www.pbrlinks.com/FLUOROSIS1

CH. 7 – NEONATOLOGY

The PBR book says that indirect hyperbilirubinemia can be treated with phototherapy as long as the direct bilirubin does not exceed 20% of the total bilirubin. I believe the new hyperbili/phototherapy guidelines say this 20% rule no longer applies. Should we still use it for the board exam in October since I believe its content is usually a few years behind?

Yes, a direct bilirubin concentration >20% of total is no longer necessary for the diagnosis of cholestasis. Since the new AAP guidelines were released in August 2022, they could be tested on the boards this year. The new guidelines are **here**.

If the direct bili is >1 mg/dL, phototherapy is usually not the answer.

On page 167, it says premature infants need iron supplementation at 2 months old and full term infants need iron supplementation after 4-6 months. Is this talking about breastfed babies only? Or does it also refer to babies getting formula? Also, different sources say premature infants need iron supplementation starting at 1 month old, not 2 months old. What is the right answer?

Thanks for catching this! Full-term breastfed infants should start iron supplementation at 4 months old. Formula-fed full-term infants do not need iron supplementation routinely because there is usually adequate iron intake from formula and complementary iron-containing foods such as iron-fortified cereals if they have been introduced at 4-6 months of age.
Regarding premature infants, they should start iron supplementation by one month old to prevent anemia of prematurity. New version is below.

IRON SUPPLEMENTATION

Premature infants need iron supplementation by the time they are 1 month old. Full-term **breast-fed babies** need supplementation starting at 4 months, when their liver stores run out. Supplementation

DOES NOT have to be in the form of drops, and it does NOT cause constipation. Formula-fed babies do not usually need iron supplementation. If using formula, make sure it contains at least 12 mg/L.
MNEMONICS: Use this 12 lb. IRON to help you remember iron should be in formula (12 mg/L). If that doesn't work, imaging making a 12-EGG omelet on a hot IRON.

On page 165, PBR says papilledema does NOT result from macrocephaly or hydrocephalus because both conditions are usually due to slow processes, whereas papilledema is from an acute problem.
However, PBR also says that papilledema is caused by increased ICP and can develop up to 2 weeks after a head injury.
These two statements seem to contradict each other since 2 weeks does not sound acute to me.

Thanks for catching this. I shouldn't have been so definitive about it. While macrocephaly and hydrocephalus typically don't cause papilledema, they can if the ICP is high enough. This can occur in certain types of conditions. For example, if the ICP gets high enough in an obstructive hydrocephalus, it can cause papilledema.

CH. 8 – DEVELOPMENTAL MILESTONES

There were no Developmental Milestones corrections for 2023!

CH. 9 – EMERGENCY MEDICINE & TOXICOLOGY

I thought that there should be hyperactive bowel sounds in sympathomimetic toxidrome, as it says in PBR on page 207. However, the emergency medicine video (at 06:37) says otherwise.

The book table is correct. We'll work on updating the video.

CH. 10 – VITAMIN & NUTRITIONAL DISORDERS

On page 217, it says that vitamin E deficiency results in thrombocytosis. Can you explain why?

It seems we got this wrong. Even if vitamin E deficiency causes thrombocytosis, it does so rarely, and we're having a hard time locating any literature to support this. Vitamin E deficiency does seem to be associated with platelet hyperaggregability. So a patient who already has thrombocytosis and then gets vitamin E deficiency could be in a hypercoagulable state. We'll remove any mention of vitamin E deficiency being associated with thrombocytosis, and we don't think you will be tested on its association with hyperaggregability.

CH. 11 – GASTROENTEROLOGY

There were no Gastroenterology corrections for 2023!

CH. 12 – PHARMACOLOGY & DRUG PEARLS

There were no Pharmacology corrections for 2023!

CH. 13 – OPHTHALMOLOGY

There were no Ophthalmology corrections for 2023!

CH. 14 – GENETICS & INHERITED DISEASES

There were no Genetics corrections for 2023!

CH. 15 – HEMATOLOGY & ONCOLOGY

PBR says polycythemia is hematocrit >65%. Is this true for all pediatric ages?

Good question. We'll correct this because that's not true for all ages. This threshold of 65% applies to the newborn period when diagnosing neonatal polycythemia. In adults, polycythemia is defined as a hematocrit >49% in men and >48% in women. **This table** provides pediatric reference ranges for hematocrit by age and gender.

POLYCYTHEMIA

Neonatal polycythemia is defined by a hematocrit **greater than 65%**, but treatment is not required unless the HCT is greater than 70% by a VENOUS blood sample. Patients are at risk for problems associated with hyperviscosity. Potential signs and symptoms include hypoglycemia, thrombocytopenia, joint pain, clots (deep vein thrombosis or pulmonary embolus), stroke, hemoptysis, lethargy, and hypotonia. Risk factors include intrauterine growth retardation (IUGR), delayed clamping of the umbilical cord, twin-to-twin transfusion, infant of a diabetic mother (IDM), Down Syndrome, and chronic hypoxia (which refers more to an older child). Treat with partial volume exchange transfusion. Note that polycythemia in older children is rare.

PEARLS: If the patient is hypoglycemic due to polycythemia, the blood glucose may not correct as expected with the usual dextrose infusion.

MNEMONIC: Assume the RBCs are sucking up all the glucose!

CH. 16 – INFECTIOUS DISEASES

On page 297, PBR says that early-onset GBS infection refers to sepsis within the first 6 days and late-onset after 6 days and up to 90 days. On the video, she states early is within the first 3 days and late is after 3 days. Which is correct?

Early-onset GBS infection occurs within the first 6 days of life. Late-onset GBS is between 7 and 90 days. We'll update the video. Thanks!

PBR mentions tetracycline and doxycycline should not be used in kids <8 years old (except for Rocky Mountain spotted fever). I thought it only causes teeth staining with prolonged use, so is it considered okay to use now? Should we still go with this PBR guideline for the board exam?

As discussed above, doxycycline can be used in children < 8 years old as long as it's not for more than 21 days since there is limited safety data beyond 21 days for this age group. Here's the new version:

ANTIBIOTIC AGE PEARLS

AGE LIMITATIONS: When choosing an antibiotic (especially tetracycline, fluoroquinolones, and macrolides), ALWAYS look at the age of the patient.
* TETRACYCLINE may be given **after the age of 8**. DOXYCYCLINE was previously contraindicated in children under 8, but studies have supported its use in children under 8 for short courses <21 days. **PEARL**: If you diagnose a 5-year-old child with Rocky Mountain Spotted Fever, go ahead and GIVE doxycycline. That is the first-line therapy regardless of age.
* FLUOROQUINOLONES: Avoid giving to children. Wait until they are **18 years** of age if possible due to risk of possible tendonitis and tendon rupture. So, avoid choosing this as an answer for your adolescents with STDs. If no alternatives are available, you may need a quinolone for a complicated UTI or pyelonephritis. A quinolone may also be used as a second-line treatment for chlamydia pneumoniae. Lastly, ciprofloxacin is a first-line treatment for Shigella. As an FYI, evidence is now starting to suggest that avoiding quinolones in children may not be needed.
* ERYTHROMYCIN: Do not use in children younger than 6 weeks of age due to an association with pyloric stenosis. The association is strong especially during the first two weeks of life. Instead, use azithromycin. Azithromycin is also associated with pyloric stenosis, but the risk is higher with erythromycin.

On page 309, PBR says that patients with severe bronchiolitis have oxygen saturations <95%. Is that right?

It looks like multiple criteria are used to characterize the severity of bronchiolitis, but usually patients with mild bronchiolitis have an oxygen saturation >95%, moderate bronchiolitis 90-95%, and severe <90% (not <95%). Thanks for the question.

RESPIRATORY SYNCYTIAL VIRUS (RSV)

The chest X-ray of RESPIRATORY SYNCYTIAL VIRUS (RSV) BRONCHIOLITIS will show HYPERINFLATED LUNGS and DIFFUSE infiltrates. Diagnose with PCR or direct immunofluorescence testing on nasopharyngeal aspirates. Routine use of albuterol should be avoided as the wheezing is related to mucous plugging of the bronchioles and not bronchoconstriction caused by asthma in most cases. Nebulized hypertonic saline can be used in patients that are hospitalized but is not recommended for the emergency room setting. Avoid the use of racemic epinephrine, systemic steroids, chest physiotherapy, and continuous oxygen monitoring in patients hospitalized for RSV. Patients frequently need hospitalization due to the severity of symptoms, comorbidities, and possibly their social situation (family reliability).

* **SEVERE BRONCHIOLITIS**: Refers to pO2< 65 or pCO2 >40 on ABG, respiratory rate > 70 or pulse oximetry with saturations <90%. This is associated with future asthma in about half of patients.

* **PEARL:** Regarding the causes of bronchiolitis, RSV is first, rhinovirus is second and influenza is 3rd. Other causes include adenovirus, parainfluenza, human metapneumovirus.

For Entamoeba histolytica, PREP says the best diagnostic test is stool PCR, while PBR says serologic testing. Which test is more accurate?

Traditionally, E. histolytica was diagnosed by looking for cysts in stool specimens, but the test has low sensitivity and requires multiple stool samples. Stool PCR is now the gold standard, the most sensitive and specific method for diagnosis (so we'll update our materials). Serologic testing is not as useful. Negative serology can be helpful for excluding E. histolytica, but positive serology does not distinguish between acute and previous infection. New section below!

ENTAMOEBA HISTOLYTICA (AKA AMEBIASIS)

For amebiasis, caused by Entamoeba histolytica, look for a history of travel to the Southwestern U.S. or to a Native American reservation. Patients will have abdominal pain associated with diarrhea, which can be watery, bloody, or mucoid. Patients often have TENESMUS. Obtain an ultrasound to look for liver abscesses. Diagnosis with STOOL PCR is preferred over stool smears. Stool examination for cysts is supportive, but it does not allow for definitive differentiation from several other parasites. Treat with metronidazole for any signs of colitis and liver abscess. If metronidazole is not an option, choose iodoquinol.
PEARL: Tenesmus is the feeling of incomplete stool evacuation. It can be associated with pain, straining, or cramping.

IMAGE: www.pbrlinks.com/AMEBIASIS1

MNEMONIC: Imagine a METRO train filled with AMOEBAS playing TENNIS (TENESMUS).
MNEMONIC IMAGE: www.pbrlinks.com/AMEBIASIS2

CH. 17 – VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

PBR, page 339: "For immunocompromised patients, pregnant patients or those with allergies to MMR, IGIM (or IVIG) is an acceptable alternative when MMR would normally be indicated for post-exposure prophylaxis. That would not be required if MMR #1 had already been given." Does this mean that if the patient already had at least one MMR vaccine, then they do not need IGIM or IVIG post-exposure prophylaxis?

If an immunocompromised patient is exposed to measles, they should receive IVIG even if they have been fully vaccinated against measles. Pregnant women who are immunocompetent will need IVIG if they have received only one MMR vaccine since that is not sufficient for immunity. If they have been fully vaccinated, no IVIG is needed. This will also be updated in the next Core Study Guide edition!

I had a board review question about measles post-exposure prophylaxis, specifically about a 12-month-old who was out of the 72-hour window. I got the question wrong when I chose immune globulin. The Red Book says no prophylaxis if >12 months and >72 hours (even if they are non-immune). This is truly the first discrepancy I have found in the book, so I wanted to reach out and see what your thoughts were.

For immunocompetent, non-pregnant children who are at least 12 months of age, the Red Book recommends MMR vaccine if exposure was within the last 3 days. If exposure was more than 3 days ago, there is no need for post-exposure prophylaxis. We will update the table in the core study guide.

CH. 18 – INBORN ERRORS OF METABOLISM

There were no Inborn Errors of Metabolism corrections for 2023!

CH. 19 – ACID-BASE DISORDERS

On page 360, PBR says that one should calculate the delta gap only when the overarching diagnosis is a GAP ACIDOSIS. However, question 6 in the problem set calculates the delta gap to uncover an additional metabolic alkalosis.

I'll remove the delta gap and delta bicarb from that answer because they don't belong. The triple acid-base disturbance can be uncovered through the pH, pCO2, the table for chronic metabolic alkalosis and the fact that there's a gap!

CH. 20 – FLUIDS & ELECTROLYTES

There were no Fluids & Electrolytes corrections for 2023!

CH. 21 – NEPHROLOGY

On page 377, is the section on recurrent febrile UTI talking about only the 2- to 24-month-old range? Or does any child with recurrent febrile UTIs need a VCUG?

Part of the workup requires VCUG after treatment is done, which is not specific to that age range. Repeat urine culture is no longer recommended according to the most recent guidelines, so this is something that warrants an update in the Core Study Guide. Here's the update!

URINARY TRACT INFECTION (UTI or PYELONEPHRITIS)

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 gentamicin 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**. 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).
PEARL: Cephalosporins inadequately treat Enterococcus UTIs. Add on ampicillin or amoxicillin if there is an increased chance of an Enterococcal infection due to a urinary tract anatomic abnormality, presence of a urinary catheter, or history of recent urinary tract procedure.

CH. 22 – STATISTICS

There were no Statistics corrections for 2023!

CH. 23 – NEUROLOGY

What is the current definition of status epilepticus? Does it have to be at least 5 minutes or 30 minutes long?

Historically, status epilepticus used to be defined as a seizure >30 minutes long, but now status epilepticus is considered to be continuous seizures lasting more than 5 minutes, or multiple seizures of any length without a return to baseline between them. Once a seizure has lasted more than 5 minutes, there is a high risk that it will last 30 minutes or more, so treatment for status epilepticus should not be delayed. Thanks, and we've updated this topic!

STATUS EPILEPTICUS

Status epilepticus refers to continuous seizures, or multiple repeated seizures, lasting **greater than 5 minutes** in which the child does not return to his or her baseline afterwards. This is an **emergency** and can cause long-term brain damage.

CH. 24 – ORTHOPEDICS & SPORTS MEDICINE

There were no Orthopedics & Sports Medicine corrections for 2023!

CH. 25 – RHEUMATOLOGY

There were no Rheumatology corrections for 2023!

CH. 26 – PULMONOLOGY

PBR gives a pearl to start vitamin E supplementation in CF patients prior to age 5. Why only that one? What about the other fat-soluble vitamins A, D, and K?

You're correct that CF patients will usually need supplementation of all the fat-soluble vitamins (A, D, E, and K). New topic below!

VITAMIN E DEFICIENCY (AKA TOCOPHEROL DEFICIENCY)

Vitamin E deficiency (Tocopherol deficiency) can result in hemolytic anemia, impaired reflexes, jaundice, neurologic problems, or peripheral edema.

* **PEARL**: When seen, it is almost always in association with a patient getting TPN.

* MNEMONICS:

- Tocop**HER**ol: Hemolysis, Edema and Retarted reflexes
- Vitamin EE deficiency leads to a knEE reflex deficiency.

On page 428, PBR says "When looking for an etiology [of pneumonia], obtain a blood culture (children are usually not very good about providing a sputum sample)." Is this the board question answer or also in real life? Are we really supposed to get a blood culture for every child who presents with pneumonia symptoms?

Great point, and one that needs to be more accurate in the Core Study Guide. A child with mild community-acquired pneumonia can be treated empirically without getting a blood culture. If a child is ill enough that bacteremia is a concern, or they are being admitted to the hospital, you should obtain a blood culture.

PNEUMONIA SECTION INTRO

A pneumonia can be diagnosed based on symptoms and clinical findings alone. If a patient has a fever, chills, cough, crackles in the right lower lobe, and a clear chest X-ray, s/he has a right lower lobe pneumonia. Seeing an infiltrate obviously makes the diagnosis easier. Mild cases can be treated empirically but **BLOOD CULTURE** should be obtained in **more ill children** to look for an etiology since children are usually not very good about providing a helpful sputum sample. This chapter will focus on recurrent and migrating pneumonias.

CH. 27 – PSYCHIATRY & SOME SOCIAL ISSUES

There were no Psychiatry corrections for 2023!

CH. 28 – ETHICS IN PEDIATRICS

There were no Ethics in Pediatrics corrections for 2023!

CH. 29 – PATIENT SAFETY AND QUALITY IMPROVEMENT

There were no Patient Safety and Quality Improvement corrections for 2023!

CH. 30 – PEDIATRIC LAB VALUES

PBR says thrombocytopenia in a newborn is platelets <150K in males and <200K in females. Why?

It looks like this was an error. Please use 150K for both males and females. Here's the new section:

COMPLETE BLOOD COUNT (CBC)

* **LEUKO**CYTES (AKA WBC): For newborns, the WBC can be as high as 30,000 on DOL 1.

* **LYMPHO**CYTES: The WBC differential in young children normally shows a predominance of lymphocytes over neutrophils. The opposite is true in adolescents and adults. The lower limit of normal for the *absolute* lymphocyte count in pre-adolescent children varies by age, but the minimal acceptable ALC should be at least 2000/mm^3.

* **PEARL:** Be sure to distinguish between the *percentage* and *absolute* counts of neutrophils and lymphocytes. The absolute count is the (percent/100)*(total WBC). So, for example, if the WBC = 14000/mm^3 and there are 60% lymphocytes and 35% neutrophils, the absolute neutrophil count (ANC) = (35/100)*14000 = 4900/mm^3, and the absolute lymphocyte count (ALC) = (60/100)*14000, or 8400/mm^3.

* HEMOGLOBIN

  - NEWBORN: Anemia in a newborn is indicated by any hemoglobin value **LESS THAN 13**.
  - NADIR: Occurs at approximately 6 weeks of age. The value should be about 9.

* PLATELETS

  - NEWBORN: Consider thrombocytopenia in a newborn to be any platelet count LESS THAN 150. Look for clues in the history that point towards maternal ITP as etiology (this can last weeks).
  - OLDER KIDS: For children 2 months and older, set the lower limit of normal at approximately 200–250 in your mind.

CH. 31 – PEDIATRIC VITAL SIGNS

There were no Pediatric Vital Signs corrections for 2023!

STRONG WORK EVERYONE!
THANK YOU SO MUCH FOR CALLING US OUT!

CLARIFICATIONS FOR 2023 EDITIONS
CH. 1 – ADOLESCENT MEDICINE

On pages 60-61 in the 2023 edition, peripheral precocious puberty and gonadotropin-independent precocious puberty (aka pseudoprecocious puberty) sound the same. How do we differentiate between the two?

Peripheral precocious puberty is the same thing as gonadotropin-independent precocious puberty. So, we'll change the GONADOTROPIN-INDEPENDENT PRECOCIOUS PUBERTY topic to the following to help make that more clear:

"In gonadotropin-independent precocious puberty (AKA peripheral precocious puberty or precocious pseudopuberty or pseudoprecocious puberty)..."

Is penile length in puberty from adrenal androgens or the testes? I understand that testicular growth is from testosterone from testis and pubarche is from adrenal androgens, so what about penile length?

FSH stimulates Sertoli cells, which increase testicular volume. LH stimulates Leydig cells, which release testosterone (an androgen). Penile length primarily increases because of testosterone.

On page 60, it says in gonadotropin-independent precocious puberty that the sequence of pubertal development is abnormal, but on page 61, it says everything is on a proper timeline except for "one thing." Is that the sequence abnormality it is talking about or is it something else?

Yes. On page 60, I mention that the sequence in peripheral precocious puberty (AKA gonadotropin-independent precocious puberty) is abnormal, meaning the order in which puberty normally progresses. On page 61, I'm letting you know that if even one stage is out of order, then it could be due to peripheral precocious puberty (AKA gonadotropin-independent precocious puberty). In this type of precocious puberty, the pituitary is not involved. That means breast or testicular enlargement does not happen first. When precocious puberty is caused by adrenal glands, body odor, central hair, and axillary hair can happen first.

During the first two years after menarche, how much variation can you tolerate before needing to work up for abnormal uterine bleeding? For example, if a patient's menstrual cycle > 7 days, would that trigger a workup?

If menstrual periods are lasting more than 7 days (i.e., at least 8 days), further workup is needed. This is especially true if there are other signs and symptoms that suggest certain disorders. This is mentioned on page 69.

CH. 2 – ENDOCRINOLOGY

Why would phosphorus overload be coupled with a low calcium level? Is a low PTH levl a response to high phosphorus (in the case of phosphorus overload)? Can we treat phosphorus overload with calcium?

PTH primarily responds to calcium, but it does also respond to phosphorus. So, if someone has a high phosphorus level, that can suppress PTH and therefore reduce calcium levels. High levels of phosphorus can also bind to calcium and reduce levels. Since these two go together, anytime you see a high phosphorus level, you should check the calcium level. In terms of treating a patient with phosphorus overload, the treatment is not calcium. You have to treat the underlying problem causing the increased phosphorus levels.

It seems that the thyroid gland can be involved in both primary and secondary hypogonadism (page 63). Please explain how.

This paragraph in PBR is saying that primary and secondary hypogonadism is analogous to the way we think about primary and secondary hypothyroidism, although the gonads and thyroid glands are different organs. This section is not saying that the thyroid is involved in hypogonadism. It's possible you just skipped over the word "analogous."

CH. 3 – OB/GYN & SOME STDS

I often see colleagues treat GU discharge based on clinical history without a workup. PBR says "ok to treat by clinical suspicion WHILE workup is pending." Do you always need to do a workup or can you just treat based on clinical history +/- GU exam?

We've checked ACOG, and other sites. They suggest it is standard of care to test. We do not recommend treatment with antibiotics without testing first. So, test, treat and adjust treatment if needed based on the results of the test.

PBR says first-line treatment for Neisseria gonorrhoeae is IM ceftriaxone x 1. Is there an acceptable PO first-line alternative?

There is no first-line oral medication. If an oral medication is needed due to lack of access to ceftriaxone, use PO cefixime (a second-line agent). If there's an allergy, then use gentamicin plus azithromycin.

CH. 4 – ALLERGY & IMMUNOLOGY

How would you differentiate FPIES from anaphylaxis since FPIES can also cause vomiting, diarrhea, lethargy, and shock? Is it related to timing of presentation?

FPIES and anaphylaxis are both reactions to food, but they play out differently in terms of timing and ingestions.

FPIES, or food protein-induced enterocolitis syndrome, is kind of like an upset stomach on steroids. It mostly happens in babies and toddlers. About 2-3 hours (or possibly sooner) after eating something (often it's milk, soy, or certain grains), kids with FPIES can get sick. They can start having severe vomiting and diarrhea and get so dehydrated that they go into hypovolemic shock. So it doesn't give you the typical allergy symptoms like a rash or trouble breathing. It's all about the belly.

Anaphylaxis, on the other hand, is like your classic allergic reaction. The onset is typically much sooner (often within 30 minutes). There can be hives, itching, swelling, coughing, trouble breathing, abdominal pain or vomiting. Diarrhea and dehydration are not part of the picture, but you can go into shock and have syncope if your blood pressure drops (but this would happen more quickly than the dehydration-related hypovolemia/shock in FPIES). Common triggers are peanuts, tree nuts, shellfish, fish, milk, and eggs.

Do bone marrow transplants only work as a cure if the thymus is present?

This is definitely out of the scope of the peds exam, but BMTs can work in an athymic patient for some complicated reasons. But if someone doesn't have a thymus, then the T-cell recovery can be delayed or incomplete.

Why is CVID associated specifically with HSV infections, VZV infections and EBV-associated lymphoma?

Patients with CVID have a higher risk of infections. HSV and VZV are usually controlled by both cellular and humoral (antibody-mediated) immunity, both of which are affected in CVID. Regarding EBV-associated lymphoma, it's more complex. EBV can cause many diseases (from mono to cancer). For patients with normal immunity, the EBV infections are typically well controlled by the immune system. But, in patients with CVID, EBV can go unchecked, leading to chronic infection and a higher risk for all its potential complications, including EBV-associated malignancies.

PBR says the first-line treatment for allergic rhinitis is intranasal steroids. Is this still true? I am seeing many allergists prescribe intranasal antihistamines as first-line treatment.

PBR is correct for now, but there may be upcoming changes in the future.

I have seen many well-seasoned doctors give IM epinephrine as soon as they see any lip/tongue/throat swelling with hives for "anaphylaxis." Is that wrong?

It's wrong to do that on the boards, and in real life.

Why do you need to suggest a peanut allergy test in a baby with an EGG allergy prior to introducing peanuts? (page 99)

The LEAP trial showed that kids with an egg allergy had an increased risk of peanut allergy.

PBR says if a reaction occurs >24 hours after taking penicillin, it is NOT a penicillin allergy and there is NO indication for skin testing. Is this only true for board exam questions?
I have seen several kids referred to allergy with a reaction >24 hours after amoxicillin, and the allergist diagnosed them with amoxicillin allergy.
PBR is correct. There have been many changes in the field. So if pediatricians are testing for PCN allergy for reactions that occur after 24 hours, it's possible they are not up to date yet. The chance of an IgE-mediated allergic reaction to PCN occurring >24 hours later is very low.

In a suspected B-cell deficiency, when obtaining antibody titers for something a child was already immunized against, do you only get IgG and IgM titers? Or also IgA and IgE?

You will likely get a panel, but if given a choice, get IgG. IgG is the main memory immunoglobulin and there should be a high level for anything the child has already been exposed to through immunizations if the B cells are working properly.

I'm trying to decipher the difference between a true milk protein allergy vs. food protein-induced enteropathy or food protein-induced proctocolitis. On page 101, the book explains that to have a true allergy, you must have GI symptoms and extraintestinal symptoms (e.g., wheezing or hives). When we see infants clinically with bloody stools and diagnose them with milk protein allergy, is this a misdiagnosis because of the absence of extraintestinal symptoms?

Yes. It's likely a misdiagnosis because in a milk protein allergy, there is a true allergy to the protein and you will have similar reactions to those of a child with a peanut allergy. Bleeding will not be part of the case presentation.

In what clinical scenarios would you utilize skin testing for candida, mumps, and tetanus (page 116) to diagnose delayed-type hypersensitivity anergy?

Delayed-type hypersensitivity (DTH) anergy refers to the body's lack of response to antigens that would typically provoke a cell-mediated immune response. This type of immune response is largely mediated by T cells, so DTH testing can help assess T cell function and overall cell-mediated immunity when you suspect immunocompromise due to HIV, malnutrition, medications that could possibly be suppressing the immune system, etc.

Why does PCP occur in SCID and hyper IgM but not CVID or Bruton's?

SCID causes T-cell dysfunction and hyper IgM does too. CVID and Bruton's are predominantly going to result in B-cell dysfunction.

Is page 98 of the PBR book stating that skin testing is the most specific test for inhalants, with low specificity for food allergies?

Yes, but due to ethical concerns and parental concerns, it's not always done. Regarding food allergies, remember that testing should be interpreted in the context of clinical history.

The book says that skin testing is the most sensitive screening test for both foods and inhalants. Is that correct?

Classically, yes, but serum testing is starting to become more equal to skin testing.

Where does RAST testing fall in being a more sensitive or specific tool?

It has high specificity for inhalants but low specificity for food allergies (more false positives with food allergies).

When do you utilize RAST testing vs. skin testing vs. oral food challenge?

This is discussed in the Core Study Guide, but something else to consider is that if someone is on medications that may affect skin testing or oral food challenge, such as a tricyclic antidepressant with antihistamine properties, serum testing is likely a better choice.

If a person has a known food allergy that was diagnosed as a young child, is it appropriate to retest them if you suspect that they may no longer be allergic to that particular food anymore? If so, what test would you do?

Yes. For food allergies, RAST is likely a better choice. Not all food allergens have skin tests available. Oral food challenges are complicated and wouldn't be recommended for someone with a previously diagnosed food allergy.

CH. 5 – CARDIOLOGY

Why does AV canal defect cause left axis deviation on EKG?

This is more of a cardiology fellow level question, but the posterior position of the AV node and bundle of His with an AV defect causes left axis deviation.

Why does tricuspid atresia cause left axis deviation?

Patients with tricuspid atresia develop left ventricular hypertrophy, which leads to left axis deviation on EKG.

Regarding page 117, does hypo or hypermagnesemia cause prolonged QT interval and prolonged PR interval?

Hypomagnesemia causes prolonged QT interval, but hypermagnesemia causes prolonged PR interval.

I know that hand grip maneuver is what can help differentiate between mitral valve prolapse and hypertrophic (obstructive) cardiomyopathy. However, I don't understand the mechanism behind why/how hand grip maneuver increases MVP vs. decreases HOCM murmur.

The hand grip maneuver increases afterload, meaning the blood pressure in the arterial system that is located "after" the heart, including in the aorta. By increasing afterload, the hand grip maneuver delays closure of the mitral valve because the left ventricle now needs more time to eject blood against the increased resistance. This increased time of ejection causes a more pronounced prolapse of the mitral valve, thereby accentuating the murmur associated with MVP.

The murmur of hypertrophic obstructive cardiomyopathy (HOCM) isn't directly caused by a faulty valve, but rather by the turbulent flow of blood through the left ventricular outflow tract (LVOT). This turbulence is a result of the obstruction created by the hypertrophied septum, especially when the ventricle contracts during systole. With the increased afterload associated with the handgrip maneuver, the left ventricle pumps harder, which leads to a longer period of ejection. This means the ventricle has more time to eject blood before it contracts fully. The more gradual ejection of blood results in less turbulence in the outflow tract and, consequently, a reduction in the intensity of the HOCM murmur.

On page 140, regarding stage 2 HTN:

It specifies that a low-dose single medication should be started for persistent stage 2 HTN without a risk factor, such as obesity.

Wouldn't you start meds for persistent stage 2 HTN even if there was a risk factor like obesity?

It's true that you would start meds for stage 2 hypertension, regardless of whether obesity is present. But if a patient also has obesity, it's important to address that as well and try to help the patient lose weight to improve BP.

A board review question stated that stage 1 hypertension is a systolic blood pressure between 130 and 139 and stage 2 is a systolic pressure of 140 or greater. Is this a good reference to use?

No, pediatric classification of hypertension depends on age, sex, and height. Please have a look at this link (https://www.pbrlinks.com/BPGUIDE) from the Core Study Guide to see what kind of information should be given to you on the boards.

I had a practice question that asked which Kawasaki treatment is more effective to prevent arterial damage. The answer was aspirin, but in another question, the answer was IVIG. Can you please clarify? Which would be the BEST treatment to prevent arterial damage and coronary artery aneurysms?

Both aspirin and IVIG are beneficial for Kawasaki disease, but IVIG is the treatment of choice to prevent coronary artery aneurysms and has to be given within the first 10 days of symptoms.

Does right or left heart obstruction cause hepatomegaly, pulmonary edema, and pedal edema? How do we decipher between left heart obstruction signs vs. right heart obstruction CHF signs? Can you give specific signs/symptoms of both right and left heart obstruction?

Left heart obstruction, such as from aortic stenosis, creates increased pressure in the LV, LA, and the lungs, causing pulmonary edema. This can eventually lead to increased pressure in the RV and RA as well, and symptoms that are also seen in right heart failure, but this is over the long term.

If there is a right heart obstruction, such as pulmonary artery stenosis, the pressure in the RV, RA, SVC, and IVC will increase, causing swelling in the extremities, hepatomegaly, and swelling in the head and neck area.

In VSD, is the single S2 because the shunt is increasing pulmonary blood flow, causing a widened split and therefore a single heart sound heard during S2? If not, why is S2 single?

Most of the time, a VSD does not cause a single S2. It's only when a VSD is very large and associated with pulmonary hypertension (as in Eisenmenger syndrome) that there would be a single S2.

What is the significance of RBBBs or LBBBs? What do we need to understand about them for boards?

Bundle branch blocks can occur because of surgery or diseases like myocarditis. If they are found incidentally in a healthy person, there is typically no clinical significance. However, since they can occur due to certain diseases/disorders (e.g., myocarditis, ischemia, etc.), it's important to do a full history and physical if one is discovered. For the exam, it's probably enough for you to just be able to identify them.

I'm having a hard time understanding the cause of a difference between preductal and postductal saturations. Can you explain further? And also provide a resource that offers a visual explanation?

Normally the blood flow through a PDA is from the aorta to the pulmonary artery, with no difference in preductal and postductal saturations in the aorta. In patients with pulmonary HTN, such as in premature babies with respiratory distress syndrome or babies with Down syndrome, some blood can go from the pulmonary artery to the descending aorta, lowering the postductal oxygen saturation.

Preductal oxygen saturation is almost always equal to, or higher than, postductal saturation. For the exam, remember that the only condition where postductal saturation is higher than preductal saturation is transposition of the great arteries with pulmonary hypertension.

Why is the PFO not kept open due to high RA pressures from heart defects like tricuspid atresia or pulmonary atresia but patent in hypoplastic left heart?

For pulmonary atresia and tricuspid atresia, the high pressures DO frequently result in a PFO remaining patent. This is mentioned in the cardiology chapter for the pulmonary atresia and the tricuspid atresia topics. But, a child with these conditions may also be born without a PFO. In those patients born without a PFO, a septectomy is done urgently to increase blood flow between the right and left sides of the heart.

CH. 6 – DERMATOLOGY

2023 edition, page 156: I believe Pap smears are not recommended until 21 years of age in any risk person (not just average risk).

Yes, the likelihood of getting cervical cancer before 21 is very low, so Pap smears are not recommended except in rare cases where cervical cancer is suspected.

Is it possible to differentiate between HSV gingivostomatitis vs. eczema herpeticum vs. impetigo from the picture alone? The lesions all look honey-colored to me.

While the morphology is similar, you can differentiate based on the location and distribution.
HSV gingivostomatitis occurs on the gingiva and labia, and a herpetiform cluster of punched-out circles is classic.

Eczema herpeticum also has a punched-out look, but it's usually widespread with up to hundreds of lesions, mainly on the cheeks.

Impetigo looks very golden and affects a small area, mostly right around the mouth and about the size of a quarter.

PBR says that you do NOT need any baseline labs when treating tinea capitis with oral griseofulvin. Is this standard of care now? I thought you needed a CBC and LFTs at baseline and six weeks into treatment to monitor for side effects.

Baseline labs are not recommended when using oral antifungals because we can't predict hepatic damage from bloodwork.

How would the descriptions for nummular eczema and tinea corporis be different on the ABP exam? They look similar to me in pictures.

Tinea corporis is annular with a central clearing. The red, scaly border has a leading edge as the fungus burrows and moves outward, leaving the center clear. It also causes intense itching.

Nummular eczema is coin-like, a lichenified or weepy patch with dry, cracked skin throughout, including the center. Other distinguishing features are pinpoint satellite lesions that surround the coin and the potential for an overlying golden crust if a staph infection causes impetiginization.

What kind of light is used to treat pityriasis rosea? Is that first-line treatment? I thought in general it would resolve on its own, or topical steroids could be offered for pruritus.

Pityriasis rosea is a self-limited condition. A single treatment with UV light or a 30-minute sunbathing episode may stop itching and shorten duration of the rash. If you don't do those things, the condition will still resolve.

When should we obtain Lyme antibody titers? Erythema migrans is an easy clinical diagnosis, but what about the other scenarios/symptoms?

Lyme is a clinical disease, but we order Lyme serology to verify. Start antibiotic treatment empirically and adjust treatment depending on lab results. If serology is negative, stop doxycycline. Other scenarios may include when lyme disease is in the differential and also possibly to monitor treatment efficacy.

Can you explain why large or multiple hemangiomas can be dangerous? How do they cause high-output cardiac failure?

Patients with 5+ hemangiomas should be screened for multifocal infantile hemangiomas, aka diffuse neonatal hemangiomatosis, with thyroid studies, an echocardiogram and a liver ultrasound. Diffuse neonatal hemangiomatosis can cause high-output cardiac failure, bleeding, liver damage, and hypothyroidism. If the heart is unable to meet the increased demand for blood flow through large hemangiomas, high-output cardiac failure may result.

How do you differentiate among tinea corporis, pityriasis rosea, and granuloma annulare?

Tinea corporis is very itchy and has a scaly border with a clear center.

Pityriasis rosea starts with a herald patch on the chest or back followed by multiple small, pink, scaly patches in a Christmas-tree distribution on the trunk. The herald patch will usually be shown on the exam. It causes mild to moderate itch.

Granuloma annulare is not scaly at all because it affects the dermis. The border looks like a series of pebbles in a ring shape with central clearing.

CH. 7 – NEONATOLOGY

Please explain why a mom who is a CMV carrier can breastfeed, but not if she is a recent CMV converter (page 166).

A mom who has been a CMV carrier for a while has antibodies to CMV that may lower the risk of CMV transmission during breastfeeding. A recent CMV converter may not have developed protective CMV antibodies yet, so it's a relative contraindication to breastfeed.

PBR says a full-term baby should gain 20-30 g/day and a preterm baby should gain 15-20 g/day. In residency, I learned 30-35 g/day for full-term and 20-30 g/day in preterm. Which is correct?

I would go with 30g/day for term infants and 20g/day for preterm infants. In reality, the ABP is unlikely to test these very small differences.

I submitted a question for the Ask The Expert Q&A regarding direct hyperbilirubinemia. The expert answered my question but added that he would define direct hyperbilirubinemia as >1 mg/dL in a newborn, along with >20% of total bili. I just realized that PBR defines direct hyperbilirubinemia as >2 mg/dL in the lab values chapter, and I got a question bank question wrong because I went with >1 (like the expert said) instead of PBR's >2. So should I stick with >2 mg/dL?

In a young infant, the threshold for initiating a clinical evaluation for cholestatic liver disease is direct bilirubin >1 mg/dL. Milder elevations of conjugated bilirubin in the neonatal period are abnormal and warrant workup and close follow-up. However, clinically significant direct hyperbilirubinemia is usually defined as direct bilirubin >2 mg/dL, such as in infants with intestinal failure-associated liver disease from TPN.

CH. 8 – DEVELOPMENTAL MILESTONES

It seems like you are saying in the study guide that it doesn't include EVERY milestone (updated AAP and CDC 2022 version), but if we just learn what's in the tables (both "surveillance milestones" and "milestones to keep on your radar," pages 181-194), then that should be enough for the boards. Is that correct?

The core study guide includes most of the CDC surveillance milestones that you need to know. The mnemonics might not include all. The "milestones to keep on your radar" includes information that members have found "very useful" in the past, but are not included in the recent surveillance milestones released by the AAP and CDC. What you have in the Core Study Guide should be enough for the boards. But, if you have EXTRA time to study because you have cemented 100% of the Core Study Guide in your brain, then here are links to the AAP and CDC milestones:
http://www.pbrlinks.com/2022-milestones-video
https://www.pbrlinks.com/cdc-milestones

Milestones were recently updated, and it seems like PBR has updated some milestones from last year in the review book, but some question banks haven't updated any milestones. Will the exam have updated milestones as testable material?

What are the best resources to review milestones to supplement PBR? Are there any other resources, or should I just focus on the PBR review book?

Yes, the 2023 exam should include updated milestones. See the above answer please. It's best to focus only on the PBR chapter and to avoid learning from multiple outside sources, which may not even have the updates.

New milestone checklist vs. old milestone checklist: The Q Banks still have questions on the old checklist, and I'm getting some of them wrong, which makes me nervous. Any thoughts or advice?

Unlike my Core Study Guide, most board review materials are not updated every year, including question banks. Also, as I like to preach, do not study from question banks. Only use them to practice your test-taking strategies.

I realized that under cord catheters, it says complications include "hepatic infection (omphalitis)." Since omphalitis is an infection of the umbilical cord, it's a little misleading the way it's currently phrased.

CH. 9 – EMERGENCY MEDICINE & TOXICOLOGY

What is charcoal's mechanism of action?

Charcoal works by absorbing toxins in dissolved liquid and in direct contact with the charcoal.

Please point out the key differentiating factors between the effects of different illicit substances.

Differentiating symptoms of illicit substances can be challenging. Here's a table outlining key differences that may be emphasized in board exams (created by our speaker for the ER "Ask the Expert webinar"):

Stimulant:	
1. Amphetamines	HTN, Euphoria
2. Cocaine	HTN, Euphoria, Aggression
Hallucinogens	
1. PCP	Vertical nystagmus, Rhabdo
2. LSD	Hallucinations, Hyperthermia
Sedative	
1. Benzo	CNS Depression, Respiration Depression
2. Barbiturates	Respiration Depression, Psychosis
3. Opioid	Miosis, Indifference to Pain
4. Marijuana	Gynecomastia, Mydriasis, Emesis

How does alcohol ingestion cause hypokalemia?

Alcohol ingestion can lead to hypokalemia in several ways, including dehydration and magnesium deficiency.

Which brain imaging is preferred for concussions, CT or MRI?

A concussion can't be confirmed by imaging. Concussion grading scales are used to assess the severity of a concussion. A CT scan would look for bleeding or skull fractures.

Is isopropyl alcohol the only alcohol ingestion that will cause ketones?

Yes, isopropyl alcohol creates ketones when metabolized. Other alcohol ingestions may also result in ketones through other mechanisms, such as starvation ketosis in chronic ethanol ingestion, but isopropyl alcohol creates them as a breakdown product.

Why is immediate removal needed for button batteries in the esophagus, but not for button batteries in the stomach? Also, does the specific location within the esophagus dictate if the battery will be removed or not?

A button battery in the esophagus is considered a medical emergency because of the potential complications of leaving it in place. The esophageal tissue is very fragile and can be damaged by electrical discharge or pressure necrosis. The concern is that it may be lodged in place due to the high-pressure environment, and the complications of leaving it in place can be severe (fistula formation, strictures, bleeding, etc.). Once it's in the stomach, it's unlikely to become lodged because it can move around and is in contact with one part of the stomach for a shorter amount of time. The stomach's mucus also protects it from damage.

Would magnet ingestion need to be emergently removed regardless of where it is, or is it location dependent?

If there are two or more magnets, they need to be removed because the magnets may get attracted to each other, creating a fistula. If there's only one magnet, even if it's in the esophagus, it is not considered a medical emergency and can be followed by serial x-rays to ensure that it's progressing through the esophagus.

CH. 10 – VITAMIN & NUTRITIONAL DISORDERS

Why is vitamin K deficiency more common in breastfed babies than in formula-fed babies?

All infants are born with very small amounts of vitamin K and have immature livers that don't efficiently utilize vitamin K, which is why we give intramuscular vitamin K at birth. Breast-fed infants are at higher risk for vitamin K deficiency because breast milk has less vitamin K than formula.

Why does TPN increase the risk for vitamin E deficiency?

Vitamin E can get degraded on the plastic of the bag it's hung in, and its stability in emulsion and absorption can vary depending on which oils are used. That's why Vitamin E levels should be monitored for kids on chronic TPN.

How does an excess of Vitamin C lead to hemolysis in patients with G6PD deficiency (page 221)?

Vitamin C is an oxidative agent that crosses RBC membranes, so excess vitamin C can oxidize the hemoglobin in RBCs and make hemolysis more likely.

Please explain how essential fatty acid deficiency causes thrombocytopenia (page 223).

Essential fatty acids must be ingested because they can't be synthesized. Omega-3 and omega-6 fatty acids are important for platelet aggregation and healthy cell membranes, so a deficiency in essential fatty acids may lead to thrombocytopenia.

CH. 11 – GASTROENTEROLOGY

What is the difference between gallbladder hydrops and acalculous cholecystitis?

Gallbladder hydrops is a dilatation of the gallbladder caused by obstruction of the cystic duct from sterile mucin. A patient with hydrops will present with pain and possibly a palpable mass, but no signs of infection (no fever, no leukocytosis, no inflammation of the gallbladder wall).

Acalculous cholecystitis is due to the stasis of bile within the gallbladder, causing ischemia and necrosis in the gallbladder wall, leading to infection and sometimes perforation. Patients usually have predisposing risk factors. These kids are much sicker, presenting with fever, tenderness, a positive Murphy's sign, leukocytosis and an elevated direct bilirubin, alk phos, and AST/ALT.

What type of inflammatory bowel disease is more prevalent in Ashkenazi Jews?

Ashkenazi Jews are at increased risk for both ulcerative colitis and Crohn's disease.

Why is congenital hepatic fibrosis associated with polycystic kidney disease?

The association between congenital hepatic fibrosis and polycystic kidney disease is rooted in genetic mutations that affect both kidney and liver cells.

How do you know when to order a HIDA scan, cholangiography, or ultrasound? Or a barium swallow vs. an upper GI series?

- HIDA (technetium-99m hepatobiliary iminodiacetic acid) scan: This dynamic imaging traces bile flow from uptake of a tracer to excretion into the biliary tree and eventually the intestine. It's primarily used for diagnosing biliary atresia, but it can also evaluate abdominal pain arising from biliary dysfunction or look for a biliary leak.
- Cholangiography: Provides a detailed view of the biliary tree's structure. It's frequently done after a HIDA scan, especially in patients being evaluated for biliary atresia.
- ERCP: Injects dye into the biliary tree and combines endoscopy and fluoroscopy, which can be both diagnostic and therapeutic.
- MRCP: Provides similar information as an ERCP, but only diagnostic since it uses magnetic resonance imaging.
- Ultrasound: Shows the size of the gallbladder and thickness of the gallbladder wall. Edema can suggest infection and cholecystitis.
- Barium swallow: Assesses the swallowing mechanism into the esophagus.
- Esophagram: Looks only at the esophagus, evaluates esophageal peristalsis and checks for potential foreign bodies.
- Upper GI series: This imaging sequence extends until the ligament of Treitz, which can identify conditions such as malrotation or hiatal hernia.

Why is urine copper high in Wilson's disease? If there is low ceruloplasmin, how does copper get carried to the kidneys? Or are the kidneys one of the tissues that copper accumulates in?

Wilson's disease disrupts copper metabolism so that it is not excreted properly, leading to its accumulation in various tissues, including the kidneys.

CH. 12 – PHARMACOLOGY & DRUG PEARLS

Why is diazepam contraindicated during pregnancy, as mentioned in the pharmacology chapter video?

Benzodiazepines like diazepam are contraindicated during pregnancy because they can cross the placenta. This may lead to respiratory depression and a loss of muscle tone in the fetus after birth.

What is the difference between hepatic inducers and hepatic inhibitors? Why are they important?

Hepatic inducers enhance the activity of drug-metabolizing enzymes in the liver, leading to faster drug metabolism and potentially reduced effectiveness. Conversely, hepatic inhibitors suppress these enzymes, slowing drug metabolism, which can increase drug concentrations and potential adverse effects. Recognizing these interactions is essential in clinical practice for optimizing drug therapy and preventing adverse drug reactions.

How do vincristine and vinblastine potentially cause SIADH?

The exact mechanism is not known, but vincristine may cause SIADH by affecting nerve cells in the hypothalamus that regulate ADH release. The disrupted feedback mechanism from the drug's impact on these cells might lead to an excessive release of ADH, causing water retention and dilutional hyponatremia.

How does valproic acid, an inhibitor, increase the concentration of carbamazepine, also an inhibitor?

Valproic acid can increase carbamazepine levels by inhibiting the enzymes responsible for breaking down carbamazepine. Carbamazepine is an inducer, which typically accelerates drug metabolism. When both are present, the inhibitory effects of valproic acid can dominate, leading to increased carbamazepine levels and potential side effects.

CH. 13 – OPHTHALMOLOGY

I got a practice question wrong about a 4-month-old infant with tearing, clear discharge, photophobia, no fixation or tracking, opaque red reflexes, and mild corneal clouding. What are the eye exam differences between cataract and glaucoma that could have helped me get the question right?

The patient in the question likely had congenital cataracts. The opaque red reflex is the key finding because the lens is normal in glaucoma but opacified with cataracts.

Can you please explain how to visually differentiate between cataracts and retinoblastoma? They look similar in photo examples.

It's unlikely that you'll have only an image to distinguish between cataracts and retinoblastoma. For cataracts, the vignette may include steroids at a young age. A retinoblastoma case might describe a toddler with an absent red reflex noted on a family picture (though cataracts can also have an abnormal, or absent, red reflex). But one way to think about it is that retinoblastoma originates from the retina, which is located at the back of the eye. That's why the abnormal white reflection (leukocoria) is noticed when light is shone directly into the eye or when captured in flash photography. Cataracts, on the other hand, involve clouding of the lens, which is closer to the front of the eye, making the cloudiness more easily seen without the need for direct examination with a light.

PBR says that cataracts on the ABP exam will be due to one of three things: galactosemia, rubella, or NF2. What about the other TORCH infections?

Yes, other TORCH infections can cause cataracts as well, but it's most commonly asked about in relation to rubella.

On page 248, PBR says "nystagmus is an abnormal finding regardless of age." Do you mean just on the board exam? I learned in residency that not all nystagmus is pathologic.

Nystagmus is not normal, so it's an abnormal finding regardless of age. However, if it presents early in life, it may be "congenital" or "infantile" nystagmus. For these children, the nystagmus is usually a lifelong condition, and the focus is often on maximizing visual potential and ensuring they can function as normally as possible. When it develops later in life, there's usually an underlying cause that should be investigated.

CH. 14 – GENETICS & INHERITED DISEASES

Is it important to know the workup and management of the genetic and inherited diseases listed in this chapter?

Identifying whether a genetic disease is autosomal dominant vs. X-linked is important. An AD disease often includes an immediate family member with a similar disease, and X-linked diseases usually affect males. As far as whether you need to know all the details for diagnosis and management of all these diseases, I would say that's too much. I would not go chasing those tidbits until you know the core study guide and workups very well that are higher yield.

I got a pedigree question last year and was wondering if there was a faster way to approach them? Or when y'all see a pedigree, how do you approach the question to answer it in 1:30 minutes?

Steps to Identify Inheritance Patterns from Pedigrees

1. Autosomal vs. Sex-linked:
    - Establish whether the trait is located on autosomes (non-sex chromosomes) or sex chromosomes (X and Y). Autosomal traits can be autosomal dominant or autosomal recessive, while sex-linked traits can be X-linked dominant or X-linked recessive.
2. Autosomal Dominant Inheritance:
    - Typically affects both males and females equally.
    - Affected individuals have at least one affected parent.
    - Affected individuals have a 50% chance of passing the trait to each offspring.
    - Unaffected individuals do not pass the trait to their offspring.
3. Autosomal Recessive Inheritance:
    - Typically affects both males and females equally.
    - Unaffected individuals can have affected offspring if they are carriers.
    - Affected individuals often have unaffected parents (carriers).
    - Two carrier parents have a 25% chance of having an affected child.
4. X-linked Dominant Inheritance:
    - Affects both males and females, but females are often more severely affected due to having two X chromosomes.
    - Affected males pass the trait to all of their daughters but none of their sons.
    - Affected females can pass the trait to both sons and daughters.
5. X-linked Recessive Inheritance:
    - Affects more males than females since males only have one X chromosome.
    - Affected males pass the trait to all of their daughters but none of their sons.

- Carrier females have a 50% chance of passing the trait to their sons, and a 50% chance of having carrier daughters.

6. Y-linked Inheritance:
    - Only affects males and is passed directly from father to son along the Y chromosome.
7. Mitochondrial Inheritance:
    - Involves genes carried in the mitochondrial DNA (mtDNA).
    - Passed from the mother to all of her offspring.
    - Both males and females can be affected, but only females can pass the trait to their children.

I'm confused by translocation Down's syndrome. Is it essential to understand this concept, or is the summary table on page 260 enough to work through the questions and answer them correctly? If not, please explain the key points that must be understood.
The summary table is adequate, but I'd also like to share a useful YouTube link that goes over Robertsonian translocation: https://www.youtube.com/watch?v=wJfe0-5uHZo

CH. 15 – HEMATOLOGY & ONCOLOGY

In Von Willebrand disease, PBR differentiates between treatment for minor bleeds vs. major bleeds. Can you give examples of what counts as a minor vs. major bleed? And when would you monitor vs. give DDAVP?

Minor bleeds include nosebleeds, gum bleeding, and longer bleeding after scrapes. DDAVP is used for minor bleeding that is not stopping on its own and for patients undergoing minor surgeries. Major bleeding is hematemesis, bloody stools, head injuries, etc., which can be treated with factor 8 concentrate or cryoprecipitate.

How does steroid treatment help with Diamond Blackfan anemia?

Steroids have the potential to drive erythroid precursors in the bone marrow.

On page 283, in the "pearls" section, PBR states that "if tested on a thalassemia it will likely be beta thal major." However, it also mentions that if a board exam offers a low MCV (60-70) that's disproportionate to "fairly mild" anemia, we should choose thalassemia, but explicitly NOT beta thal major. This seems contradictory; is there a discrepancy?

Additionally, earlier details specify that Beta Thal Intermedia typically presents with an Hb range of 6-9. Is this Hb range indicative of "fairly mild" anemia? Therefore, if a board question provides an MCV of 60-70 with an Hb between 6-9, should we select beta thal intermedia?

Lastly, could you clarify the criteria for mild, moderate, and severe microcytosis and anemia levels?

The text is actually correct, but we'll plan on updating it to make it more clear. Here's what I meant:

1. First Sentence - "if tested on a thalassemia it will likely be beta thal major": This means that if a board exam focuses on a specific type of thalassemia for a question, it'll often be referring to beta thalassemia major due to its significant clinical implications and unique presentation.
2. Second Sentence - "If board exam gives low MCV (60-70) out of proportion to 'fairly mild' anemia, pick thalassemia (but NOT beta thal major)": This is highlighting a scenario where there's a mild anemia but a significantly reduced MCV. In these cases, the boards want you to think of thalassemias other than beta thal major, like beta thalassemia minor or even alpha thalassemia trait, which can present with mild anemia and a disproportionately low MCV.

3. Regarding a Hb of 6-9 in Beta Thal Intermedia: A hemoglobin level of 6-9 is not "fairly mild." It's not as severe as what you might see in beta thal major, it's still a significant anemia.

Regarding your question “If a board question provides an MCV of 60-70 with an Hb between 6-9, should we select beta thal intermedia?,” yes. That's a reasonable interpretation. Beta thalassemia major typically presents with a more profound anemia (often Hb <6) and is associated with earlier clinical manifestations (often in infancy). Beta thalassemia intermedia is, as its name suggests, intermediate in severity between major and minor forms.

In terms of criteria for microcytosis and anemia categorization, this may help:

Microcytosis Classification (based on MCV):

1. **Mild**: MCV slightly below age-specific reference range.
2. **Moderate**: MCV noticeably reduced but not extremely so.
3. **Severe**: Markedly reduced MCV, often with MCV values <70 fL in older children.

Anemia Classification (based on Hemoglobin, Hb):

- For infants aged 6-12 months:
    1. **Mild**: Hb 9.5 - 11 g/dL
    2. **Moderate**: Hb 7 - 9.5 g/dL
    3. **Severe**: Hb <7 g/dL
- For children aged 1-5 years:
    1. **Mild**: Hb 10 - 11.5 g/dL
    2. **Moderate**: Hb 7 - 10 g/dL
    3. **Severe**: Hb <7 g/dL
- For children aged 5-12 years:
    1. **Mild**: Hb 11 - 11.5 g/dL
    2. **Moderate**: Hb 8 - 11 g/dL
    3. **Severe**: Hb <8 g/dL
- For teenagers aged 12-15 years:
    1. **Mild**: Hb 11 (for females) or 11.5 (for males) - 12 g/dL
    2. **Moderate**: Hb 8 - 11/11.5 g/dL
    3. **Severe**: Hb <8 g/dL

What is the typical age range for physiologic anemia in infants? PBR says 6-16 weeks old, but I learned something different.

Physiologic anemia typically occurs around 6-10 weeks in term infants, but it can be earlier and longer in preterm infants.

I understand why there are more leukocytes in leukemia, but can you explain why there are normal/high leukocytes and high or low lymphocytes in Hodgkin's lymphoma, while leukocyte count may be normal in non-Hodgkin's lymphoma (page 271-272)?

In reality, all cell lines can be affected in leukemia and lymphoma. Leukemia will often show abnormal cell lines with blasts on the peripheral smear. Hodgkin’s and non-Hodgkin’s lymphoma can’t be diagnosed from the CBC, so biopsy of the lymph node is essential. Reed-Sternberg cells are indicative of Hodgkin’s lymphoma, which distinguishes it from non-Hodgkin’s lymphoma.

Can you please explain what MCHC is (hereditary spherocytosis section on page 279)?

MCHC is the concentration of hemoglobin relative to RBC size. In hereditary spherocytosis, cells can be dehydrated, leading to an elevated MCHC.

For acute chest syndrome, why is treatment for high hemoglobin level exchange transfusion?

Both simple and exchange transfusions can improve oxygenation in sickle cell anemia patients with acute chest syndrome, but high hemoglobin levels increase the risk of hyperviscosity. Exchange transfusion is preferred when hemoglobin levels are high because a large volume of blood can be transfused — lowering the percentage of Hb S and decreasing vaso-occlusion — without risking hyperviscosity.

CH. 16 – INFECTIOUS DISEASES

Under febrile infant and septic workup for 29- to 60-day-old babies, PBR says, "If CSF results are negative (regardless of the urinalysis results), consider giving IV or oral antibiotics and consider the setting of observations (i.e. home or in the hospital)." What are we treating for and with?

If initial CSF results are negative, you can treat with either oral or IV antibiotics pending CSF and urine cultures. You can also observe at home or in the hospital without treatment if CSF and U/A results are negative. For more details (if you have lots of free time), see the AAP guidelines for well-appearing febrile infants between 8 and 60 days old.

For the febrile workup, can the same recommendations about urine cultures and observation at home be applied to the 8-21 day old group (and 22-28 day old group)? I was wondering if the younger 8-21 day old group (and 22-28 day old group) would need IV antibiotics and stay admitted, while the older 29-60 day old group can be observed at home with oral antibiotics.

Febrile infants who are 8-21 days old should always get a full sepsis workup (U/A, blood culture, and LP) and be admitted for IV antibiotics and observation in the hospital. Infants who are 22-28 days old may potentially be observed at home if initial CSF results are normal, but they would first receive parenteral antibiotics and be reassessed in 24 hours. A detailed algorithm for both age groups is available in the AAP guidelines mentioned above.

Why do certain respiratory viruses, such as parainfluenza and RSV, require contact precautions but not droplet precautions?

There are some viruses that are spread by direct contact with respiratory secretions, which is why parainfluenza, RSV, metapneumovirus and enterovirus require contact precautions. Viruses with heavy droplets (such as influenza and adenovirus) require droplet precautions. Lighter particles (as in measles and varicella) travel farther and require airborne precautions.

PBR says post-strep glomerulonephritis treatment is mainly IV fluids. Don't we also need to treat the strep infection with antibiotics?

Post-strep glomerulonephritis is an immunologically mediated post-infectious process that occurs after the initial strep infection. Since it's not caused by streptococcus itself, no antibiotics are needed.

PBR mentions avoiding fluoroquinolones until >18 years old if possible. I thought a new study showed 1 tendon rupture would be caused for every 50,000 kids treated so it's now considered safer to use. Should we still avoid picking it as an answer choice on the board exam?

You should still avoid using fluoroquinolones in children <18 years old unless they are the only option for treatment. While the risk of tendon rupture is small, it still exists, and tendinitis is also a risk (1 case of tendinitis for every 4400 adolescents treated with fluoroquinolones).

Why do you look for low IgG when a patient has spontaneous bacterial peritonitis?

Patients with nephrotic syndrome lose proteins including IgG in the urine, which make them more prone to infections. They can also develop ascites and are at increased risk of SBP, which is why SBP and low IgG are associated.

PBR says treatment for cryptosporidium is supportive care. I had learned in the past that it was nitazoxanide. Which is correct?

Immunocompetent people with cryptosporidium might only need supportive care. If they have severe diarrhea, nitazoxanide can be used if the patient is at least 1 year old. All immunocompromised patients who have cryptosporidiosis should receive a course of nitazoxanide.

On page 300, PBR says that if MRSA is susceptible to clindamycin but resistant to erythromycin, give something else. Why is that?

MRSA isolates that show resistance to erythromycin but susceptibility to clindamycin in lab tests may have inducible clindamycin resistance. Those isolates can develop clindamycin resistance in vivo once clindamycin is given to the patient. Therefore, an alternative antibiotic should be given instead, such as vancomycin, trimethoprim/sulfamethoxazole, or doxycycline.

Please review the RBC to WBC ratio for traumatic spinal taps (pp. 321-322) and any tips for remembering the ratio, along with the pathophysiology to help us differentiate between bacterial and viral meningitis.

The expected ratio of RBC/WBC in a traumatic tap is 500. So for every 500 RBCs, we allow 1 WBC. Every WBC above that number can suggest bacterial meningitis. In the PBR example, 10,000 RBCs/500 = 20 WBCs expected. But the patient has 130 WBCs, which is 110 WBCs more than 20 and concerning for bacterial meningitis.

I seem to get TORCH infections mixed up when answering questions. How do you differentiate these infections?

The three most important TORCH infections to know about are toxoplasmosis (triad of chorioretinitis, diffuse intracranial calcifications, and hydrocephalus), CMV (periventricular calcifications, chorioretinitis, sensorineural hearing loss, thrombocytopenia, hepatosplenomegaly, jaundice, and anemia), and rubella (cataracts, patent ductus arteriosus). One important difference to know is that the intracranial calcifications in toxoplasmosis are diffuse, whereas the calcifications in CMV are periventricular.

Congenital varicella is associated with skin scarring and extremity deformities.

In other resources, I've seen that oral metronidazole should be used for first-time C. diff colitis and first recurrence or that oral vancomycin or fidaxomicin should be used for first-line therapy. Please let me know what I should mark on the boards if this question comes up.

Oral vancomycin, metronidazole, and fidaxomicin are all acceptable choices for first-line treatment of mild or moderate C. diff colitis. Fidaxomicin was approved by the FDA in 2020 to treat C. diff colitis in children.

On page 297, PBR says "Overall, Streptococcus pneumoniae (pneumococcus) is the most common etiology of pneumonia in children." Should this say the most common cause of bacterial pneumonia? I thought viruses were the most common cause of pneumonia in children.

Viruses are the most common cause of pneumonia in children under 5 years old, but bacterial causes are more common in children older than 5, in whom Strep pneumoniae is the most common etiologic agent.

CH. 17 – VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

When do you give IVIG vs. IGIM for measles post-exposure prophylaxis?

IVIG is given to anyone at least one year old. IMIG is given only to infants <12 months old. All infants <6 months old receive IMIG. Immunocompetent infants between 6 and 12 months can receive the MMR vaccine, and immunocompromised infants between 6 and 12 months get IMIG.

I thought Neisseria meningitidis post-exposure prophylaxis followed the same guidelines as H. influenzae in terms of how many cases are needed to start prophylaxis: at least 1 in household or at least 2 in daycare or at least 2 in school. On page 338, PBR says N. meningitidis needs only 1 case of exposure. Can you clarify?

To start N. meningitidis post-exposure prophylaxis, the criteria are:

- At least one case in daycare or preschool, regardless of how close the contact was
- One case in a regular school setting if there's close contact, such as shared secretions
- One case within a household

For H. influenzae, the requirement is two cases in daycare or preschool within a 60-day period or a single household case, but other criteria such as age and vaccination status also have to be met to start chemoprophylaxis.

CH. 18 – INBORN ERRORS OF METABOLISM

Please explain what is meant by the note in the Core Study Guide near the PKU section about a newborn screen being valid only after protein intake.

Any problem with processing amino acids will be seen only after consumption of proteins (amino acids). Therefore, newborn screens should not be done until the child has consumed proteins. The same concept applies for the galactosemia screen (prior lactose intake is needed for the screen to be valid).

CH. 19 – ACID-BASE DISORDERS

Diarrhea causes metabolic acidosis, but PBR also says laxatives cause metabolic alkalosis. Can you please explain?

Diarrhea usually causes metabolic acidosis, but chronic laxative abuse can result in metabolic alkalosis through excessive loss of K+ in the stool, intravascular volume depletion, and increased bicarbonate reabsorption. I'll discuss this a little more in an upcoming question.

On page 361, when PBR discusses the delta gap for bicarbonate, is the question "Is the bicarb lower than expected?" another way of saying delta bicarb > delta gap?

Yes. If the delta gap is 4 (4 higher than the expected gap), then the delta bicarb should be 4 (4 lower than the expected bicarb). If delta bicarb > delta gap, that means the bicarb is lower than expected and there is an additional non-gap metabolic acidosis.

Conversely, is the question "Is the bicarb higher than expected?" another way of saying delta bicarb < delta gap?

Yup! See the above. It's just the opposite. You have an additional metabolic alkalosis. The bicarb will be higher than expected.

CH. 20 – FLUIDS & ELECTROLYTES

PBR says that laxatives cause metabolic alkalosis. I am doing some board review questions, and they mention that laxatives cause metabolic acidosis. Can you clarify please?

Laxatives can cause metabolic acidosis or alkalosis depending on the clinical scenario and how long the laxatives have been used. Chronic laxative use is most commonly associated with metabolic alkalosis. On the test, they should give you additional information to determine the acid-base abnormality, but it's more likely that the boards will present a patient with chronic laxative abuse.

CH. 21 – NEPHROLOGY

In IgA nephropathy, patients typically present with the proteinuria/hematuria DURING a URI, correct?

The proteinuria/hematuria in IgA nephropathy can begin during a URI or shortly after.

Please explain the role chloride plays in non-anion gap metabolic acidosis. Also, please explain why chloride responsiveness is a factor in metabolic alkalosis.

In a non-anion gap metabolic acidosis, chloride levels go up to replace lost bicarbonate and maintain electrical neutrality. This is often called "hyperchloremic metabolic acidosis."

Metabolic alkalosis has increased chloride and decreased bicarbonate; metabolic acidosis has increased bicarbonate and decreased chloride.

In a metabolic alkalosis, you're losing anions. In certain situations, like vomiting and diuretic use where chloride is low, adding it back can fix the alkalosis ("chloride-responsive"). If adding chloride doesn't help, it's "chloride-resistant" alkalosis, and you'll likely need to treat the root cause, like hormone imbalances.

So the responsiveness, or resistance, can be important to help guide treatment and even to get to the root cause if it's unknown.

Page 380 says that any time you are giving long-term steroids, you should consider getting a PPD first. Could you explain why?

You should screen for latent TB before starting steroids because of the increased risk of reactivating TB while on immunosuppressants.

What are the main differences between multicystic dysplastic kidney and polycystic kidney disease?

Instead of writing this out, maybe a table would be more helpful!

	MCDK	PKD
Time of Diagnosis	Usually diagnosed early, sometimes prenatally	Usually diagnosed later, sometimes in adulthood
Number of Cysts	Multiple cysts but usually fewer in number	Multiple cysts that increase over time
Affected Kidneys	Usually affects only one kidney	Usually affects both kidneys
Renal Function	Usually poor from the beginning	Gradually worsens over time, leading to kidney failure as more cysts form

---

Why is vesicoureteral reflux the only one that's highlighted to cause UTIs and HTN in PBR (page 376)? Couldn't ureteropelvic junction obstruction, vesicoureteral reflux, and posterior urethral valves all lead to these complications?

You're right. Other urologic abnormalities can predispose a child to urinary tract infections (UTIs) and hypertension (HTN). VUR is just the one that seems to be most likely to appear on the boards. Here's a summary of the disorders.

1. **Vesicoureteral reflux (VUR)**: VUR is a common finding in children who experience UTIs, especially recurrent ones. Pediatric patients with recurrent UTIs are often screened for VUR due to the risk of renal scarring, which can lead to hypertension. Early detection and management of VUR can help prevent these complications.
2. **Ureteropelvic junction obstruction (UPJO)**: While UPJO is typically recognized in the neonatal period due to prenatal ultrasounds showing hydronephrosis (swollen kidney), not all cases cause symptoms early on. Those that do manifest later can present with UTIs, flank pain, or even

palpable abdominal masses in more severe cases. Chronic obstruction can certainly damage renal parenchyma and lead to hypertension.

3. **Posterior urethral valves (PUV)**: PUV is the most common cause of significant lower urinary tract obstruction in male neonates. PUV can lead to bladder dysfunction, UTIs, and renal insufficiency. As renal damage accrues from repeated infections and back pressure, there's a risk of hypertension.

On page 376, PBR says "renal ultrasound shows decreased renal parenchyma." What is "renal parenchyma"?

Renal parenchyma refers to the functional part of the kidney.

CH. 22 – STATISTICS

There were no Statistics clarifications for 2023!

CH. 23 – NEUROLOGY

What are the initial signs of tuberous sclerosis?

Seizures or hypomelanotic macules (white patches) on the skin will usually be the first signs of tuberous sclerosis.

Is the word "benign" in benign childhood epilepsy with centrotemporal spikes a misnomer? It doesn't seem so benign.

Yes, the term "benign" can be misleading because the condition may not be entirely benign, but it is eventually outgrown.

Which annual screening tests are recommended for NF2 patients?

An annual MRI of the brain and spine is recommended starting at 10 years of age, with spinal MRIs every other year. Regular checkups and monitoring for hearing loss to screen for bilateral acoustic neuromas are also essential.

Page 391 mentions somatosensory evoked potential tests are for demyelinating processes. Why is a SEP not used to diagnose GBS instead of nerve conduction studies if GBS is a demyelinating process?

Nerve conduction studies are preferred for diagnosing Guillain-Barré Syndrome (GBS) because GBS primarily affects the peripheral nerves. Nerve conduction studies directly assess the function and integrity of these nerves. While somatosensory evoked potentials can detect demyelinating processes, they are more suited for conditions affecting the central nervous system.

Why is a stat MRI instead of a CT ordered for a spinal epidural abscess if it's an emergent condition (page 394)?

MRI offers superior soft tissue visualization, allowing for a clearer identification of the abscess and associated inflammation. It also provides detailed insights into any spinal cord compression or involvement by the abscess, and when enhanced with gadolinium, it differentiates the abscess from

surrounding tissues. Even though it's an urgent situation, the diagnostic accuracy of MRI makes it the preferred choice for ensuring appropriate management.

Why do carbamazepine, phenytoin, and oxcarbazepine worsen juvenile myoclonic epilepsy, a generalized seizure disorder, even though they are useful for treating tonic-clinic seizures (page 402)?

The mechanism of action involves stabilizing neuronal membranes and reducing the ability of neurons to fire at high frequencies. This can help prevent the spread of seizure activity seen in tonic-clonic seizures, but it may not be as effective, or could even be counterproductive, for the more specialized neuronal firing patterns seen in JME.

How do you diagnose acute cerebellar ataxia (page 403)?

Acute cerebellar ataxia is characterized by gait abnormalities, speech abnormalities, nystagmus, and impaired coordination of voluntary movement. If a patient presents with typical symptoms, diagnosis can be made clinically from history and exam after excluding other serious illnesses and obtaining a toxicology screen. A diagnosis of acute cerebellar ataxia would be supported by a rapid onset of symptoms, prodromal illness during the previous two to three weeks, and absence of other signs or symptoms that would suggest an alternative diagnosis.

Do ataxia telangiectasia and Friedreich's ataxia have the same disease process? Are they both nerve fiber degeneration diseases? They appear similar (page 403).

They are both associated with nerve fiber degeneration but are distinct entities. One leads to problems with DNA repair (AT), and one leads to problems with mitochondrial function (FA).

CH. 24 – ORTHOPEDICS & SPORTS MEDICINE

PBR says reassurance is the answer in most cases of metatarsus adductus, but sometimes cast or surgery is required. When are interventions required?

This is definitely out of the scope of what the exam would be asking for from a pediatrician. If interventions are needed, orthopedics will be involved. Even so, here is some information that you might be interested in for curiosity's sake.

The treatment options vary, largely depending on the severity of the condition and the child's age. The options range from reassurance and observation to non-operative and operative management.

Reassurance and Observation: This option is often chosen for mild cases of MTA, especially when they are flexible and detected early in infants. Most mild deformities may resolve spontaneously by the time the child is a toddler due to natural correction. It is important to monitor the condition regularly to ensure that it is indeed self-resolving.

Non-Operative Management: Non-operative interventions become necessary when the condition does not improve with time, especially if the deformity is moderate to severe or if it is semi-rigid or rigid. These interventions primarily include stretching exercises, serial casting, or use of special shoes or braces to gradually correct the alignment of the foot. Physical therapy might also be included to help improve muscle balance and strength. These methods aim to manipulate and maintain the foot in the correct alignment over time, allowing the bones to grow out of the deformity.

Operative Management: Surgery is generally reserved for severe cases of MTA or those that don't respond to non-operative treatments. The aim of surgery is to correct the deformity and restore normal alignment and function to the foot. This usually involves procedures to release tight structures and realign the bones of the foot. The precise surgical procedure will depend on the specific nature of the deformity and the age of the patient.

The choice of management depends on several factors, including the child's age, the severity and flexibility of the deformity, and how the deformity affects the child's function. The child's overall health and any other associated conditions would also be considered. As such, the management of metatarsus adductus should be individualized, considering the particular circumstances of each child.

PBRC says all newborns have femoral anteversion but also says that all newborns are born with genu varum. I'm having a hard time understanding how both can be present at the same time. One seems to bring your knees closer together (femoral anteversion) and the other your knees further apart (genu varum). Please help me understand! Thanks

Both conditions can be normal findings in infants and young children, depending on their age and the severity of the condition.

Femoral Anteversion: Femoral anteversion refers to a medial (or inward) rotation of the femur, leading to the knees and feet turning inwards ("pigeon-toed"). It's normal for babies to be born with some degree of femoral anteversion, and it often becomes more apparent when the child begins walking. The condition often peaks at around age 4-6 and then gradually improves. By the time most children reach adolescence, the femoral rotation has typically corrected itself. Most of the time, femoral anteversion doesn't cause any problems and doesn't require treatment.

Genu Varum (Bowed Legs): Most infants start with a degree of genu varum due to their positioning in the womb. This is quite normal and the curvature typically starts to correct as the child starts to walk and bear weight on their legs. By the ages of 2-3, most children's legs will have naturally realigned to the typical slightly knock-kneed (or genu valgum) alignment. This then tends to straighten out by late childhood or early adolescence. However, if the genu varum persists past the age of 2 or is severe or asymmetrical, it may warrant further investigation and potential intervention as it could be a sign of an underlying condition, such as Blount's disease or rickets.

Genu Valgum: It's normal for children to have some degree of genu valgum between the ages of about 2 and 5, and this typically corrects itself as the child grows. By late childhood or early adolescence, most children will have a nearly straight alignment of the legs.

As with femoral anteversion and genu varum, if the genu valgum is severe, seems to be getting worse, or is causing symptoms such as pain or difficulty walking, it would be a good idea to consult with a healthcare provider for further evaluation. Conditions like rickets or skeletal dysplasias, although less common, could cause persistent or severe genu valgum.

What is the best salmonella osteomyelitis treatment? PBR described a case in a sickle cell anemia patient. I am finding various answers online.

You're extremely unlikely to be tested on this in a normal patient because seeing salmonella osteomyelitis outside of sickle cell is RARE. So if you see it, think sickle cell disease. For treatment, choose ceftriaxone. If that's not an option, choose a fluoroquinolone.

What is a good resource to practice recognizing radiological images of pediatric ortho conditions? There are good images in PBR, but radiological imaging is a weak area. I would like to be able to see more radiological examples of typical peds ortho conditions to quickly reference.

In recent years, the American Board of Pediatrics has had fewer images on the exam. I would strongly recommend against trying to do too much when it comes to studying images. Until you can tell yourself that you know the entire study guide and question book completely, it will serve you best if you keep your studying to those two resources. If you have questions or ideas that you would like to pursue further, I suggest you write them in a separate notebook and only pursue them once you can tell yourself that you are 100% crystal clear on everything in these two books. With that being said, if you want to look at Ortho Bullets, you could. But know that it can be overwhelming since it's not meant for general pediatricians.

CH. 25 – RHEUMATOLOGY

You state that there can be periarticular joint involvement in IgA vasculitis and HSP, but soft tissue only. What does that mean?

Those conditions can affect the soft tissues surrounding a joint but don't involve the joint space itself.

On page 418, PBR states that yellow fluid for arthrocentesis is normal. I thought you could also have yellow fluid in inflammatory and even infectious etiologies.

You are correct that yellow fluid obtained during arthrocentesis does not necessarily indicate normal joint fluid. While synovial fluid is typically clear and straw-colored (similar to the color of egg whites), it can vary in color depending on various factors, including the underlying cause of joint inflammation or disease. Therefore, while yellow synovial fluid can be a normal variation, it can also be indicative of various pathological conditions, including inflammation and infection. The interpretation of synovial fluid color should always be considered in conjunction with other clinical findings, such as the patient's symptoms, physical examination, laboratory tests, and imaging studies, to determine the underlying cause of joint symptoms accurately.

Should we pick arthrocentesis (as the answer and in real life) even if we think a child has transient synovitis and not true septic arthritis?

It depends on how the question is asked and other clinical findings. If inflammatory markers are high, with a history of fever, and there is definite joint involvement, arthrocentesis could be the correct answer.

Septic arthritis and transient synovitis are two different conditions that can cause similar symptoms, such as joint pain, swelling, and limited joint movement. However, they have distinct causes, characteristics, and treatments. Here's a comparison of the two:

Septic Arthritis:

- **Cause:** Septic arthritis, also known as infectious arthritis, is caused by a bacterial, viral, or fungal infection within the joint space. Bacterial infections are the most common cause.
- **Symptoms:** It typically presents with a sudden onset of severe joint pain, swelling, redness, warmth, and fever. The affected joint may also be tender, and the person may experience decreased range of motion.
- **Diagnosis:** Diagnosis often involves joint aspiration (arthrocentesis) to analyze synovial fluid for evidence of infection. Blood tests and imaging may also be used to support the diagnosis.
- **Treatment:** Septic arthritis is a medical emergency and requires prompt treatment with antibiotics. Joint drainage through aspiration or surgery may be necessary to remove infected material.

Transient Synovitis (Transient Hip Synovitis in the Hip Joint):

- **Cause:** Transient synovitis is a self-limiting condition that primarily affects the hip joint, especially in children. Its exact cause is often unknown, but it may be associated with viral infections or minor trauma.
- **Symptoms:** It typically presents with sudden hip pain, limping, and occasionally low-grade fever. Symptoms often improve on their own within a few days to weeks.
- **Diagnosis:** The diagnosis of transient synovitis is usually clinical, based on the characteristic symptoms and ruling out other conditions. Imaging studies may be used to rule out more serious causes of hip pain.
- **Treatment:** Transient synovitis often resolves on its own with rest, over-the-counter pain relievers, and physical therapy. In some cases, a short period of bed rest or crutches may be recommended to relieve pressure on the joint.

PBR says to use NSAIDs as first-line treatment for oligoarticular JIA/JRA and systemic JIA. Why not start with NSAIDs for polyarticular JIA too?

In general, polyarticular JIA is considered more severe and may involve more joints than oligoarticular JIA. Because of this, the treatment approach often begins with DMARDs, such as methotrexate or biologic agents, rather than NSAIDs. DMARDs are more effective at controlling inflammation and preventing joint damage in polyarticular JIA.

CH. 26 – PULMONOLOGY

For bronchopulmonary dysplasia, PBR says to watch out for electrolyte abnormalities, especially hypocalcemia. Why does BPD cause hypocalcemia?

This is an association and not a cause. Preemies are predisposed to BPD and often have electrolyte imbalances. For those with BPD, acid-base disturbances can contribute to this as well as the use of diuretics (which are used in the treatment of BPD).

On page 428, PBR says "if a patient has a fever, chills, cough, crackles in the RLL, and a clear CXR, s/he has a RLL pneumonia." Wouldn't the CXR have to show something that would confirm what you were hearing in the RLL on physical exam for it to be pneumonia?

Chest x-rays can look different due to penetration, technique and even hydration. Pneumonia is a clinical diagnosis. Even if the CXR was clear, this would not prevent you from diagnosing pneumonia based on history and exam.

What is the diagnostic test for vascular rings? I learned MRI but PBR says CTA. Please help clarify.

You can use either for diagnosing vascular rings. The case may give you additional information to direct you to one imaging modality over another. CTA is quicker and does not require sedation, so may be useful if a child is experiencing respiratory distress.

Why do children with alpha-1 antitrypsin deficiency have jaundice at birth when cirrhosis doesn't develop until later?

Infants with alpha-1 antitrypsin deficiency often have cholestatic jaundice in the first two months of life because of impaired bile flow, not cirrhosis. Cirrhosis is usually a later finding that occurs after chronic disease.

CH. 27 – PSYCHIATRY & SOME SOCIAL ISSUES

What key points should we know about treating transgendered patients for the boards?

When we talk about gender-affirming care for someone who is transgender, we mean all the different ways we can help a person live as the gender they identify with, not necessarily the one they were assigned at birth. This may include getting support from a mental health professional, taking hormones like testosterone or estrogen, having surgery, working with a speech therapist to change how they talk, making social changes like switching names or pronouns, and getting legal help or advocacy support. The idea is to help ease any discomfort someone might feel because their gender identity doesn't match what they were assigned at birth, and to improve their mental health and quality of life. It's important to remember that not everyone will want, or need, all these types of support. It's more about what makes sense for each person's own situation and feelings.

What mood issues do we screen for when a patient is on ADHD medication?

It's unlikely that you would be tested on this since there are multiple medications. However, look for side effects that could result in an elevated mood (anxiety, psychosis, etc.). Also, some medications may cause depression and other side effects.

Do we need to know about psychiatric conditions like bipolar disorder, mania, manic episode, depression, things like that for the pediatric boards? Diagnostic criteria, risk factors, treatment, etc.?

Bipolar disorder, including the manic episodes that go along with it, is rare in the pediatric population and unlikely to be tested. Depression is more common. We'll expand the section for next year:

Depression is a very nebulous diagnosis in kids, and on the exam, because symptoms can present differently from the classic "SIGECAPS" findings of Sleep issues, loss of Interest, feelings of Guilt, loss of Energy, diminished Concentration, change in Appetite, Psychomotor agitation and Suicidality. Children may be more irritable than usual, grades may suffer, or they may not have adequate weight gain. Diagnosis requires these symptoms to be present every day. First-line treatment for children is cognitive behavioral therapy (CBT) or interpersonal psychotherapy (IPT). For the exam, consider choosing depression over drug abuse as an answer unless there is something specifically suggestive of drug abuse.

CH. 28 – ETHICS IN PEDIATRICS

Page 438-439 in 2022 book: What is quality of life? What is futility? Could an action be futile for the patient, but not futile for the parents?

Quality of life refers to a patient's ability to enjoy and participate in normal life activities.

There are two kinds of futility:

1. Physiological - intervention that won't improve a disease process
2. Qualitative - intervention that would not improve quality of life

Futility is determined by the medical team and pertains to the futility of interventions for the patient only.

I had a board review question where parents refused treatment for a child who had a tick bite (with a risk for Rocky Mountain spotted fever) based on religious grounds. The recommended next step was to report to CPS due to risk of imminent harm without treatment. I had picked an ethics consult but that was marked as incorrect.

Our ethics expert mentioned that she would obtain an ethics consult when there is more ambiguity about whether refusing to do something will result in bodily harm. In this case, refusing care would result in harm, so care should not be withheld. A physician may need to take custody in life-threatening situations when parents refuse care.

CH. 29 – PATIENT SAFETY AND QUALITY IMPROVEMENT

There were no Patient Safety and Quality Improvement clarifications for 2023!

CH. 30 – PEDIATRIC LAB VALUES

Why do newborns have a prolonged PT? Is a prolonged PT for a newborn a variant of normal?

PT is usually prolonged (>15) in the first nine months of life for multiple reasons. One easy way to remember this is by thinking of the low physiological vitamin K levels that newborns have at birth. Since many clotting factors are dependent on vitamin K, the PT is expected to be longer than in older children.

CH. 31 – PEDIATRIC VITAL SIGNS

There were no Pediatric Vital Signs clarifications for 2023!

QUESTIONS & ANSWERS BOOK

There were no corrections or clarifications requested for the PBR Q&A Book for 2023!

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)!

IF YOU ARE NOT A PBR MEMBER, YOU SHOULD BE!

PBR has an estimated pass rate of over 98% for first-time test takers of the ABP **initial certification** exam. This is based on the insanely low number of requests we get for the 100% Money Back First-Time Pass Guarantee (<1%).

PBR also has a 10+ year history of pass rates in ~100% range for general pediatricians taking their recertification board exams.

Also, the "PBR Certification System" has **helped pediatricians pass the boards after they previously FAILED 7-9X**. One VIP Bundle member finally passed with PBR's help on his TENTH attempt. Many others have passed on their last and final chance to take the boards. DO NOT wait until fail to join PBR. Join now!

What Are YOU Waiting For?

Pediatrics Board Review...

YOUR RESOURCE FOR EFFICIENCY IN STUDYING…
ESPECIALLY DURING CRUNCH TIME!

As the final days of preparing for the initial certification exam come up, there's often a sense of **PANIC**. Please BREATHE! If you've been studying the PBR thoroughly and you have a fair handle on your test-taking skills, you should be in GREAT shape!

What can you do to MAXIMIZE your remaining study time? Go over your PBR over and over again. Focus on the areas that you still need to master and SKIP the areas that you know well.

Get the DOWNLOADABLE MP3 audio course from PBR so that you can eat, sleep and breathe PBR no matter where you are. Yes… even between seeing patients!

www.PediatricsBoardReview.com/MP3

YOUR RESOURCE FOR EFFICIENCY IN STUDYING…
ESPECIALLY DURING CRUNCH TIME!

You can also WATCH 31 videos that cover ALL of the PBR through PBR's Online Video Course, Conference & Live Webinars in a single day!

www.PediatricsBoardReview.com/VIDEOS

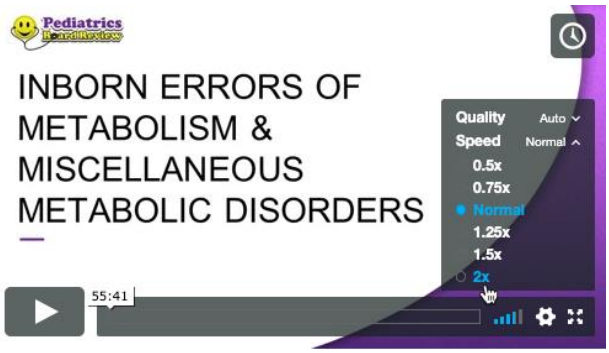
You'll get chapter videos PLUS our webinar replays from this year's Summertime Q&A Webinars.

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… through these talks and practice sessions, we get comments like, "This is so much easier!... The light bulb just went off!"

www.PediatricsBoardReview.com/VIDEOS

And if you want to CRANK THROUGH the videos and webinars in just 12-14 hours, you can do so at **double speed** using our SPEED CONTROL function.



Okay! That's it! Now go study in whatever way is going to give you the MOST REPETITION AND LEARNING.

PREORDERS, DISCOUNTS & FREE BONUSES FOR THE NEW 2024 EDITION OF BOOKS (14TH EDITION) ARE NOW AVAILABLE!

FREE BONUS #1

To pass the boards, you're going to need a PLAN! We've helped thousands of pediatricians pass by assisting with study plans and study schedules. Having one is a key part of your prep. Team PBR will create up to THREE 90-Day Personalized Study Schedules for you with any All Access Pass or No Brainer order.

THREE 90-DAY PERSONALIZED STUDY SCHEDULES **$894 Value ($297 Each)**
FREE With Your All Access Pass or "No Brainer" PREORDER.

FREE BONUS #2

Passing the "board game" requires knowledge of the RULES to the game and how to apply them. This includes understanding the different TYPES of questions you will come across and also the different ALGORITHMS needed to process each type of question. That's what you get in our Core Concepts in Test-Taking Strategies Webinar.

CORE CONCEPTS in Test-Taking Strategies Webinar **Normally $197**
FREE With Your All Access Pass or "No Brainer" PREORDER.

WONDERING WHAT THE "**NO BRAINER**" IS?

VISIT THE NEXT 3 PAGES TO SEE EVERYTHING THAT'S INCLUDED!

THE "NO BRAINER" BUNDLE

This bundle was created to address the 3 pillars of passing the pediatric boards...

CONTENT | TECHNIQUE | COMMITMENT

So, it's not surprising that this is (by far) the
MOST COMMONLY PURCHASED PBR BUNDLE!

Money Back First-Time Pass Guarantee!

THE "NO BRAINER" BUNDLE

CONTENT SUPPORT

You'll get a 12-month All Access Pass to 100% of PBR's content-based resources to help build your pediatric fund of knowledge. You'll get hardcopy books, online books, our Online Video Course, our MP3 audio course, access to our "**ASK THE EXPERT**" question portals and MUCH MORE.

TECHNIQUE SUPPORT

You'll get access to PBR's Full Online Test-Taking Strategies Course. It's self-guided course with 7 webinars to teach you how to process questions like an expert. You will *NEVER look at board-style questions the same way again.*

COMMITMENT SUPPORT

You'll get THREE 90-Day Personalized Schedules created by Team PBR, just for you. You'll fill out a detailed questionnaire about your history with exams, your pace of reading, your work schedule, the days you are reserving to study, when you want to take practice exams and MUCH more. You'll also have access to our live "Board Success" series of webinars throughout the year with tips and guidance on how have "Deep Study."

2024 EDITION OF THE "NO BRAINER" BUNDLE

(DISCOUNTED PREORDER BUNDLE)

- Hardcopy Core Study Guide
- Hardcopy Q&A Book
- Online Core Study Guide
- Online Q&A Book
- DOWNLOADABLE MP3 Audio Course
- STREAMING MP3 Audio Course
- ONLINE VIDEO COURSE
- Private Facebook CREW Access
- FULL ONLINE TEST-TAKING STRATEGIES COURSE
- Online Picture Atlas
    - $97 Bonus & Not Available Through Our General Catalog Area
- Downloadable PDF Picture Atlas
    - $97 Bonus & Not Available Through Our General Catalog Area
- Live Summertime Q&A Webinars with Experts
    - $145 Value & Not Available Through Our General Catalog Area
- "ASK THE EXPERT" Question Portals
    - $197 Bonus & Not Available Through Our General Catalog Area
- THREE 90-Day Personalized Schedules Created by Team PBR
    - $894 Bonus & Not Available Through Our General Catalog Area

FIRST-TIME MONEY BACK PASS GUARANTEE

Click Here Now & Signup Before the Offer Ends!

~~$6473~~ VALUE. PREORDER FOR ONLY $1597

(75% OFF + FREE SHIPPING)

www.pbrlinks.com/PREORDER

(Limited-Time Offer)

(New Orders Only)

2024 EDITION OF THE 12-MONTH ALL ACCESS PASS

(DISCOUNTED PREORDER BUNDLE)

- Hardcopy Core Study Guide
- Hardcopy Q&A Book
- Online Core Study Guide
- Online Q&A Book
- DOWNLOADABLE MP3 Audio Course
- STREAMING MP3 Audio Course
- ONLINE VIDEO COURSE
- Private Facebook CREW Access
- Online Picture Atlas
    - $97 Bonus & Not Available Through Our General Catalog Area
- Downloadable PDF Picture Atlas
    - $97 Bonus & Not Available Through Our General Catalog Area
- Live Summertime Q&A Webinars with Experts
    - $145 Value & Not Available Through Our General Catalog Area
- "ASK THE EXPERT" Question Portals
    - $197 Bonus & Not Available Through Our General Catalog Area
- 90-Day Personalized Schedules Created by Team PBR
    - $297 Bonus & Not Available Through Our General Catalog Area

FIRST-TIME MONEY BACK PASS GUARANTEE

Click Here Now & Signup Before the Offer Ends!

~~$4882~~ VALUE. PREORDER FOR ONLY $1097

(78% OFF + FREE SHIPPING)

www.pbrlinks.com/PREORDER

(Limited-Time Offer)
(New Orders Only)

2024 EDITION OF THE ULTIMATE BUNDLE PACK

+

MP3 AUDIO COURSE

(DISCOUNTED PREORDER BUNDLE)

- Hardcopy Core Study Guide
- Hardcopy Q&A Book
- MP3 DOWNLOADABLE Audio Course
- Online Core Study Guide
- Online Q&A Book
- Online Access for 1 Full Year
- GET FREE SHIPPING

FIRST-TIME MONEY BACK PASS GUARANTEE

Click Here Now & Signup Before the Offer Ends!

~~$1388~~ VALUE. PREORDER FOR ONLY $347

(75% OFF + FREE SHIPPING)

www.pbrlinks.com/PREORDER

Promo Code: **PREORDER**

(Limited-Time Offer)
(New Orders Only)

HAVE YOU FAILED? IF SO, YOU NEED THE VIP BUNDLE.

If you have failed while feeling as though you had a strong handle on the content, it's likely that you have a test-taking technique or a "Deep Study" issue. Reach out to us and ask about our VIP Bundle. It includes everything from the No Brainer, but also includes a live, online 2-Day Test-Taking Strategies & Deep Study.

The Live Test-Taking Strategies & Deep Study Course covers:

- Test-taking skills to help you process every board-style question systematically
- Test-day planning to maximize your time, energy and brain power
- Anxiety management
- Diet management
- Study group coordination
- Question-answering group coordination
- One-on-one coaching at the live course
- DEEP STUDY lectures on limiting distractions, creating accountability and maximizing your focus
- AND MUCH MORE

~~$10,470~~ VALUE. PREORDER FOR ONLY $2997

(75% OFF + FREE SHIPPING)

www.pbrlinks.com/PREORDER

Promo Code: **PREORDER**

(Limited-Time Offer)
(New Orders Only)

EXAM DAY PREPARATION

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

www.PediatricsBoardReview.com/EXAMDAY

GOOD LUCK!

From everyone here at Team PBR, we hope that our efforts have been well-received. Your success is truly our primary focus, and we hope that we've supported you successfully throughout this journey.

Please finish off with a ton of energy as you go through another round of content review and several strong test-taking strategy sessions.

You're going to do great!

Trust me, if I can do it… so can you!

Best of luck on your board exam!

Sincerely,
Ashish & Team PBR