Pediatrics® Board Review

2024 Corrections & Clarifications Guide

14th Edition
Your EFFICIENCY BLUEPRINT to
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2024 EDITION

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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

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A FEW WORDS OF THANKS TO THE PBR COMMUNITY

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

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

THANKS TO YOU!

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

   www.pediatricsboardreview.com/error

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

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

   www.pediatricsboardreview.com/badlink

3. An absolutely MASSIVE THANKS TO DR. SHAZIA LATIF! Shazia is a PBR alum and is now a PBR Content Contributor and teammate! 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

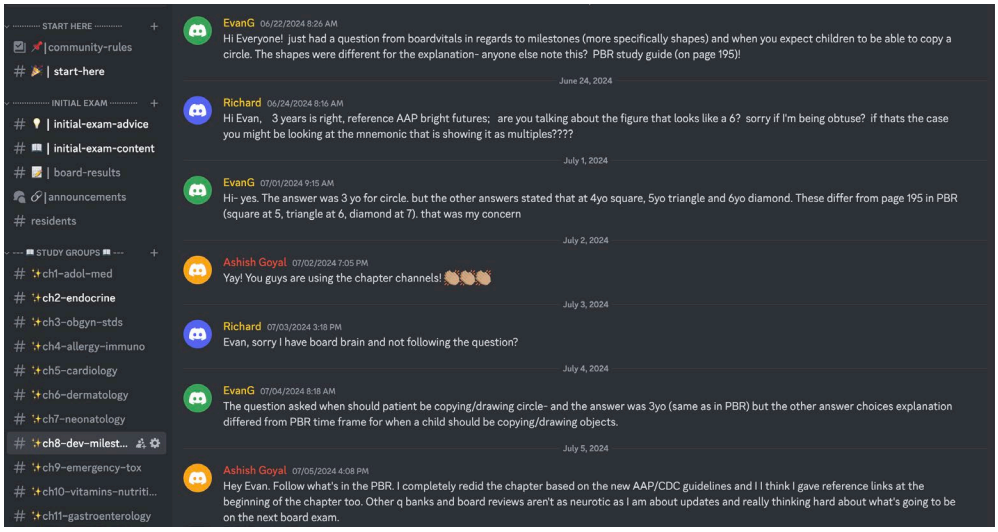
NOW… WHAT IS THIS THING?

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

IN ORDER OF PRIORITY, OUR FOCUS HAS BEEN….

1. **Addressing error submissions from the PBR Error portal**. Basically, stuff where folks are saying, "*Ashish… I think (or I know) that this is wrong. You should fix it in the book and let folks know about it because it's more than just a spelling or grammar issue.*"
2. **Addressing questions from our Online Video Course question portals and webinars.** The summer is filled with content-based webinars, and many excellent questions, corrections and clarifications come to light during those sessions. We try to address as many of those as possible before the Initial Certification Exam.
3. **Addressing possible errors/concerns mentioned in the PBR Discord Community!** 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 "Discord community"**. In general, the "PBR's Discord Community!" 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 the community's 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 "Discord Community" **If you are a member of "The PBR Discord Community".**



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

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

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

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

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

WHAT ABOUT IMAGE LINK CORRECTIONS?

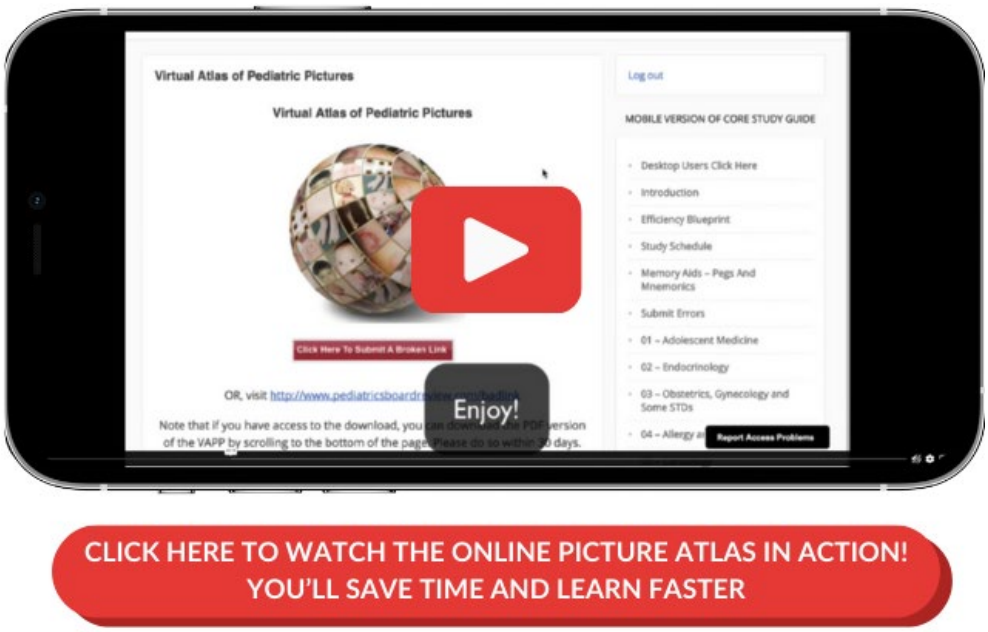
We have a very innovative system that allows you to view phenomenal high-yield images across the web. **We have approximately 400 image links in the PBR, but they lead to images that are not owned by PBR.** That means that any given time, an unrelated PBR website that houses a high-yield image might be down. When you notify us of this, it's a HUGE help and we can quickly replace the image link with a new, comparable image. At this time, 98% - 99% of the image links should be working without any issues!

If you do find that there's an issue, please notify us immediately by visiting:

www.pediatricsboardreview.com/badlink.

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

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



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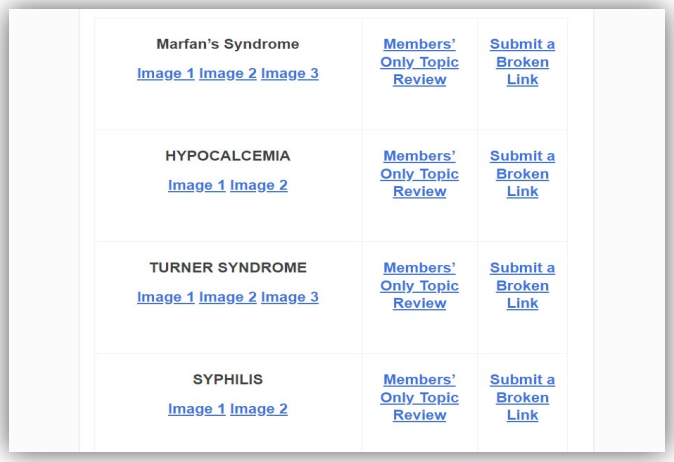

FREQUENTLY ASKED QUESTIONS

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

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

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

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

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

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

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

1. **IT'S FRUSTRATING TO HAVE AN OLDER BOOK. WATCH!**
    - You will see in this guide that many submissions will reference specific page numbers and specific lines within a paragraph. This happens all year long, especially in our private forum. This is NOT the time to be spending your energy cross-checking everything in this guide against your older version of the PBR. Your time is PRECIOUS and needs to be spent EFFICIENTLY and effectively.
    - Start with a fresh book, transfer any notes/drawings from your previous hardcopy to the new edition as you read through it the first time, and then use the new one as your bible! The purging of "the old" and the starting with "the new" is also a great MENTAL RESET.
2. **NEW CONTENT:** There is ALWAYS new content in a new release. MANY of the corrections below were included in this guide because of help from the PBR community, and many were done on my own. But there are more corrections that need further investigated before the next edition's release.

3. **NEW CLARIFICATIONS**: There was an ACTIVE discussion within the Discord Community! 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 COMMUNITY!**
    - The PBR Discord Community comes alive with discussion as the boards approach. Many PBR alumni have said that the Discord COMMUNITY! heavily contributed to their success on the boards. When your peers in "The COMMUNITY" 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 https://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 **2024 Editions of the Pediatrics Board Review** books (covers shown below).

- **DEAR NON-PBR MEMBERS… the PBR Discord Community! 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.).
- **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 2024 EDITIONS

CH. 1 – ADOLESCENT MEDICINE

No corrections!

CH. 2 – ENDOCRINOLOGY

I noticed in the congenital hypothyroid question, it states to start levothyroxine right away if given an abnormal NBS. I had learned previously to repeat labs (TSH, free T4), prior to starting levo. Could you clarify if we should start meds right away?
The latest AAP recommendations say that if the TSH on a newborn screen is > 40 mIU/L, T4 treatment should be started right away after drawing new labs, without waiting for the results. If the newborn screen TSH is ≤ 40 mIU/L, wait for the new test results (preferably with a 24-hour turnaround time) before starting T4 treatment. Great question. Thanks!

In familial hypophosphatemic rickets, if there is a defect in converting 25 vit D to 1,25 vitamin D, why would you not see an excess of 25 vitamin D and a low 1,25 vitamin D? If you have a defect in conversion, wouldn't 25 vit D build up?
Familial hypophosphatemic rickets can cause mildly decreased conversion of 25 vitamin D into 1,25 vitamin D, but that doesn't mean an excess of 25 vitamin D levels. 25 vitamin D reflects body stores of vitamin D, which would be normal with adequate oral intake and sun exposure, and a high level would be more commonly expected in cases of excessive intake of high-dose supplements. The key point for X-linked familial hypophosphatemic rickets is that the mutation in the PHEX gene causes increased production of a growth factor that inhibits phosphate reabsorption in the kidneys. Oral phosphate supplementation and vitamin D used to be the treatment, but now the treatment of choice is burosumab, a monoclonal antibody against the growth factor that inhibits phosphate reabsorption. This information has been updated in the study guide. Thanks!

CH. 3 - OB/GYN & SOME STDS

No corrections!

CH. 4 – ALLERGY & IMMUNOLOGY

Should all children that have an anaphylactic reaction be sent to the ED? If so, is it sufficient to monitor the patient for 4-6 hours?
Previously these children were all sent to the ED, but the latest anaphylaxis practice parameters say that not all children need to be sent to the emergency department after an anaphylactic reaction because management doesn't often change. This is unlikely to be asked about on the boards because it's often a judgement call and shared decision-making with the family is key. It's acceptable for children to be monitored at home if the family has a second dose of epinephrine available and can effectively manage the situation at home. The typical monitoring period, if needed, is 4-6 hours. Thanks for asking about this!

CH. 5 – CARDIOLOGY

No corrections!

CH. 6 – DERMATOLOGY

No corrections!

CH. 7 – NEONATOLOGY

No corrections!

CH. 8 – DEVELOPMENTAL MILESTONE

No corrections!

CH. 9 – EMERGENCY MEDICINE & TOXICOLOGY

On page 211 of the study guide, it states that a button battery lodged in the esophagus is a medical emergency requiring immediate removal. It suggests leaving the battery to pass naturally if it has moved beyond the esophagus. However, NASPGHAN guidelines advise assessing and potentially removing the battery within 24-48 hours in children under 5 years old, even if asymptomatic, and observing older children with repeat X-rays. Should age-related differences be considered in managing button battery ingestion, and how do these guidelines impact the decision to leave or remove a battery that has passed into the stomach?

If a button battery is lodged in the esophagus, it is a medical emergency requiring immediate removal. For children over 1 year old, administer honey or sucralfate to reduce esophageal injury. According to the NASPGHAN guidelines, if the battery is < 20 mm and has passed beyond the esophagus, and the child is asymptomatic, observation is appropriate, even for children under 5 years of age. However, if the battery is > 20 mm or there is evidence of injury (e.g., symptoms or endoscopic findings), remove the battery, even if it is in the stomach. We've updated the study guide with these details. Thanks!

CH. 10 – VITAMIN & NUTRITIONAL DISORDERS

Can you please explain the difference between vitamin D2 and D3 supplements and when each type of supplementation should be used? Why do we first supplement with vitamin D2 and then switch to vitamin D3 once levels are normalized?

Either vitamin D2 or D3 supplements can be used to treat vitamin deficiency, but studies suggest that vitamin D levels normalize a little faster with vitamin D3 supplements. Therefore, if both forms are available, vitamin D3 supplements are preferred. The information in PBR is based on previous recommendations when the only high-dose formulation of vitamin D available was D2. Now that high-dose formulations of D3 are available, vitamin D3 is preferred for both initial supplementation and maintenance therapy. We've updated the Core Study Guide to reflect this. Thanks!

CH. 11 – GASTROENTEROLOGY

Regarding the H. pylori test of cure on page 229, I understand that patients need to be off PPIs for 2 weeks prior to testing for H. pylori to avoid a false-negative result on the biopsy. However, the book mentions that testing for cure too early (before 1 month after completing treatment or 2 weeks after stopping PPIs) can result in a false-positive result. Could you please clarify this?
Yes, you're right! The pearl should read that testing too early after treatment can cause a false **negative** instead of false positive. We've changed this line in the Core Study Guide. Antibiotics, bismuth products, and PPIs can suppress H. pylori growth and initially lead to negative results, but H. pylori may not be completely eradicated and resume growth once treatment is stopped. If you test at least 4 weeks after treatment and the results are negative, it is more likely to be a true negative and a reliable indicator of eradication than if you tested immediately after completion of therapy. Thanks!

CH. 12 – PHARMACOLOGY & DRUG PEARLS

No corrections!

CH. 13 – OPHTHALMOLOGY

I recently encountered a question that asked which test was the best indicator of strabismus, and the answer was the cover test, not the light reflex, since the cover test is more reliable. However, there's no mention of the cover test for strabismus in PBR (pages 246-247).
Both tests are used to test for strabismus, but you're right. The cover test is more reliable. It's recommended that it be used in conjunction with other tests, such as the light reflex. We've updated the study guide to include the cover test. Thank you!

CH. 14 – GENETICS & INHERITED DISEASES

No corrections!

CH. 15 – HEMATOLOGY & ONCOLOGY

When do we treat ITP with IVIG versus steroids?
Most children with ITP can be managed with just watchful waiting. For moderate bleeding symptoms, most patients can be treated with oral steroids, but IVIG is recommended when the platelet count needs to be brought up quickly, such as for urgent surgery or head trauma. For severe or life-threatening bleeding, both IVIG and steroids may be used together. Previous guidelines suggested bone marrow testing before starting steroids, but that is no longer necessary for children with typical features of ITP. We've updated the study guide with this information. Thanks!

CH. 16 – INFECTIOUS DISEASES

I noticed a discrepancy between the infectious diseases video and the book regarding early-onset sepsis. The book states that early-onset sepsis occurs within the first 6 days of life, while the

video says the first 3 days of life. Other resources such as the Cleveland Clinic also say the first 3 days of life. Could you clarify why the book says 6 days? Is this an error?
Early-onset sepsis is from birth through day 6, so the book is correct. Our editing process for the videos and audios are always based on changes made in the Core Study Guide. So, when in doubt, definitely ask and notify us, but also keep in mind that if a discrepancy exists, it's likely that the book is correct and the particular info that you're seeing was modified but didn't make it into the A/V resources. We will work on updating the video. Thanks for the heads up!

Page 293 states that varicella requires airborne precautions "with contact precautions." But page 309 states that for VZV, droplet precautions are sufficient if only one dermatome is involved. So if only one dermatome is involved, should we use airborne, contact, and droplet precautions? And if more than one dermatome is involved, should we use airborne and contact precautions and a negative pressure room?
There are two types of varicella manifestations: chickenpox and shingles/zoster. Chickenpox and disseminated shingles require airborne and contact precautions. Localized shingles only needs standard precautions. We've corrected this information in the Core Study Guide. Thanks!

In a recent board review question, a 3-week-old with fever on ampicillin and gentamicin had a culture with Gram-positive cocci in clusters, negative for Staph aureus by molecular testing. The answer for the next best step was no further testing (no need to repeat culture) and no medication changes since the organism was most likely coagulase-negative Staph, which in a term baby without an indwelling device is likely a contaminant. However, PBR (page 298) suggests treatment or at least multiple cultures in such cases. Could you clarify the best practices in this scenario?
The practice question is a specific situation because there was molecular testing which ruled out Staph aureus so you know that this is most likely coagulase-negative staph. And if the child doesn't have any prosthetic materials such as a central line, then you know that this is likely a contaminant and you don't need to treat it. However, if you don't have molecular testing, you don't know whether this is Staph aureus or coagulase-negative Staph, and you have to assume the worst, which is having a Staph aureus infection. In that case, you need to repeat blood cultures and treat with vancomycin for possible MRSA. We'll update the Core Study Guide to reflect that coagulase-negative Staph does not need to be treated when it is likely a contaminant. Thanks!

Can you please clarify the differences in impetigo types between strep and staph as described on page 299? Which bacteria cause bullous versus non-bullous impetigo? I encountered a review question elsewhere that states staph causes non-bullous impetigo, which seems contrary to what is in PBR. Page 299 says that staph bullous impetigo is "less common than GAS." Is this referring to the overall incidence of staph impetigo or specifically the bullous pattern?
There are two types of impetigo, non-bullous and bullous. The non-bullous type is the more common type, and non-bullous impetigo can be caused by either Staph aureus or group A strep. However, bullous impetigo, which is the less common one, is only caused by Staph aureus. We've updated the table so that this distinction is clearer. Thanks!

CH. 17 – VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

Page 341 says that both the inactive and live flu vaccines can be administered to individuals with chicken or egg allergies. However, the mnemonic just below this suggests that it's a contraindication. Could you clarify what the correct approach would be?

It's not a contraindication to give the flu vaccine to people with chicken or egg allergies, even in cases of anaphylaxis. We've updated the Core Study Guide to reflect this. Thank you for the question!

Page 311 says, "DO NOT GIVE MMR for at least 6 months after giving IGIM," but page 339 says to wait 5 months. Which is correct? From what I researched, MMR should be delayed by 6 months after administration of IGIM or by 8 months after IVIG.
Yes, you're right, live attenuated vaccines like MMR should be given at least 6 months after IGIM and 8 months after IVIG. We've updated the Core Study Guide so that it's correct on both pages. Thanks for catching this!

There seems to be a discrepancy in the guidelines for measles post-exposure prophylaxis. On page 311, it states that for those older than 12 months who were exposed 4-6 days ago, PEP is too late, but they should receive the MMR vaccine if they never received it. However, on page 339, it indicates that those older than 12 months who were exposed 4-6 days ago should receive IGIM (or IVIG) if they never received MMR or if their vaccination status is unknown. Could you clarify which guideline is correct for individuals exposed to measles 4-6 days ago?
The table on page 311 is correct, and we've updated the table on page 339 to be the same. For immunocompetent children older than 12 months who were exposed to measles 4-6 days ago, it is too late for measles PEP. They may receive MMR#1 after home quarantine for 21 days if they have never had MMR before. Great eye for catching this. Thanks!

For Hep A postexposure prophylaxis, PBR says to give Hepatitis A immunoglobulin to unimmunized family members (page 338), but the ACIP recommendations state that healthy people over 12 months old should get Hep A vaccine for PEP instead of immunoglobulin.
For Hep A postexposure prophylaxis, the Hep A vaccine should be given to all exposed individuals who are at least 12 months of age as long as they are not immunocompromised. However, if a patient is less than 12 months old, they should get the immunoglobulin instead because the Hep A vaccine cannot be given for children less than 12 months of age. If a patient is immunocompromised, they should be getting both immunoglobulin and Hep A vaccine if they are > 12 months. We've updated the Core Study Guide with this information. Thank you!

CH. 18 – INBORN ERRORS OF METABOLISM

No corrections!

CH. 19 – ACID-BASE DISORDERS

No corrections!

CH. 20 – FLUIDS & ELECTROLYTES

What is the normal range for serum osmolality? What values are considered high and low serum osmolality? What is the normal range for urine osmolality? The book states that a high urine osmolality is >100, but what is considered normal and low urine osmolality?
The normal range for serum osmolality is 275-295 mOsm/kg, so a high osmolality would be > 295 and a low osmolality would be < 275. Urine osmolality can vary much more, ranging from 50-1200 mOsm/kg, in

response to fluid and electrolyte changes in the body. The Core Study Guide stated that a high urine osmolality is > 100 mOsm/kg, but that is actually a relatively low urine osmolality. In the setting of dehydration, urine osmolality would usually be > 400, and we've corrected the guide to reflect this. Thanks!

CH. 21 – NEPHROLOGY

No corrections!

CH. 22 – STATISTICS

No corrections!

CH. 23 – NEUROLOGY

No corrections!

CH. 24 – ORTHOPEDICS & SPORTS MEDICINE

I saw another resource that said that skeletally immature children with a Cobb angle between 10° and 20° can be observed and rechecked in 6 months. PBR says that observation is ok for a curvature < 25°. Could you clarify the best approach?
There are multiple guidelines available. We've had a difficult time finding one by the AAP, but what you found seems to be a common recommendation. For the exam, we recommend observation with x-rays every 6 months for a Cobb angle between 10° and 19°, and a referral to orthopedics for a possible brace for a Cobb angle greater than 20°. Thanks for the question!

CH. 25 – RHEUMATOLOGY

No corrections!

CH. 26 – PULMONOLOGY

No corrections!

CH. 27 – PSYCHIATRY & SOME SOCIAL ISSUES

The study guide mentions that ADHD must be diagnosed after the age of 6, with symptoms present before the age of 12. However, I found that the diagnostic criteria for ADHD can apply to children as young as 4 years old. Could you clarify the correct age requirements for diagnosing ADHD?
Yes, you're right. The most recent AAP clinical practice guideline about ADHD states that ADHD can be diagnosed in children as young as 4 years old. When diagnosing older patients, the DSM-5 states that

symptoms should have started before the age of 12. We've corrected this in the study guide. Thanks for the question!

CH. 28 – ETHICS IN PEDIATRICS

No corrections!

CH. 29 – PATIENT SAFETY AND QUALITY IMPROVEMENT

No corrections!

CH. 30 – PEDIATRIC LAB VALUES

No corrections!

CH. 31 – PEDIATRIC VITAL SIGNS

No corrections!

QUESTIONS & ANSWERS BOOK

No corrections!

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

CLARIFICATIONS FOR 2024 EDITIONS

CH. 1 – ADOLESCENT MEDICINE

In the adolescent medicine video at the 1 hour 15 minute mark, the lecturer said that penile inclusion epidermal inclusion cysts are small, white, benign papules on the glans of the penis. Is the more common description "pearly penile papules"?
Yes. You're right, so we'll update the Core Study Guide to include the term "pearly penile papules."

Can you please clarify the difference between pseudoprecocious puberty and peripheral precocious puberty? They're both under the gonadotropin-independent topic.
The names have changed over the years. Both are the same entity and refer to gonadotropin-independent precocious puberty due to adrenal tumors or other non-pituitary sources. The study guide says that gonadotropin-independent precocious puberty is also known as peripheral precocious pseudopuberty or pseudoprecocious puberty.

CH. 2 – ENDOCRINOLOGY

Can you explain thyroid storm?
Thyroid storm occurs in the context of untreated hyperthyroidism, typically in autoimmune thyroid disease. Symptoms include tachycardia, fever, hypertension, neurological symptoms (e.g., agitation, psychotic symptoms), and systemic symptoms (e.g., excessive sweating, diarrhea). The management of thyroid storm requires ICU admission with telemetry, beta blockers to manage cardiac symptoms, and iodine solution to rapidly treat symptoms. Methimazole may also be used. This condition is rare in children but should be treated with urgency due to its life-threatening nature.

Can you please explain transient thyrotoxicosis (page 75)?
Transient thyrotoxicosis, also known as Hashitoxicosis, occurs in the context of autoimmune thyroid disease, where there is a transient period of hyperthyroidism during the initial inflammation and destruction of the thyroid gland. This leads to the release of thyroid hormones into the bloodstream, causing hyperthyroid symptoms such as tachycardia, weight loss, and sweating. This condition is usually self-limited and eventually resolves or develops into hypothyroidism. Antibody testing (TSI, TPO, thyroglobulin) helps confirm the diagnosis, and temporary treatment can include beta blockers to manage symptoms while thyroid levels are high.

What happens to vitamin D levels with hypoparathyroidism and pseudohypoparathyroidism?
In hypoparathyroidism and pseudohypoparathyroidism, 25 vit D can be normal while 1,25 vit D is low. PTH stimulates the renal production of 1,25 vit D, so 1,25 vit D levels can be low from the low levels of PTH in hypoparathyroidism or from the end-organ resistance to PTH seen in pseudohypoparathyroidism.

The book mentions that familial hypophosphatemic rickets has a high alkaline phosphatase in bold letters. Don't most forms of rickets have elevated alkaline phosphatase, or is this an important diagnostic finding for FHR that helps us distinguish this on the boards?

Alkaline phosphatase is elevated in all types of rickets, so it's not something that would help distinguish familial hypophosphatemic rickets from other types of rickets.

Why isn't there hypertension in 21-hydroxylase deficiency?
21-hydroxylase deficiency usually causes a buildup of androgens but not mineralocorticoids. The lack of mineralocorticoids means that there is no hypertension in this type of congenital adrenal hyperplasia.

When discussing congenital adrenal hyperplasia, page 82 says that there is no hypertension in 21-hydroxylase deficiency. Since hypotension is a hallmark of 21-hydroxylase deficiency, can we use hypotension to distinguish this type of CAH from other types of CAH associated with hypertension (17-hydroxylase and 11-hydroxylase deficiency)?

Hypotension is not a hallmark feature of any form of CAH. Hypotension from an adrenal crisis or inadequate treatment may occur with multiple types of CAH, including 11-hydroxylase deficiency. The key distinguishing factor for 21-hydroxylase deficiency is ambiguous genitalia in girls and potential salt-wasting crises in boys. While hypotension can be seen in some cases, it's not a definitive feature for distinguishing types of CAH.

On the boards will they use the term "male pseudohermaphroditism," or should we expect to see "ovotesticular disorder of sex development"? And will we see "Müllerian inhibitor hormone deficiency" or "persistent Müllerian duct syndrome"?
"Male pseudohermaphroditism" is not used anymore, and "ovotesticular disorder of sex development" or "disorder of sexual development" is preferred. "Persistent Müllerian duct syndrome" is a specific disorder of sexual development that is caused by a deficiency of anti-Müllerian hormone or defect in its receptor.

How does maternal hyperparathyroidism cause early hypocalcemia?
TH can cross the placenta. The high PTH levels in the mother cause transient underdevelopment of the baby's parathyroid gland, resulting in lower PTH production and low calcium levels in the newborn.

How does hyperphosphatemia cause hypocalcemia? I thought high phosphorus increases PTH, which would then increase calcium and decrease phosphorus.
Hyperphosphatemia does not cause hypocalcemia. Phosphorus and calcium levels must be understood in the context of the primary defect. For example, the table on page 80 shows a variety of conditions associated with low or high phosphorus levels, along with either low or normal calcium levels. It's essential to identify the underlying cause to understand what happens with phosphorus and calcium levels.

Page 81 says Addison's disease can cause hyponatremia. But then it says that ADH can be elevated and that it's a normal response. Can you please explain that part about the ADH and how that does or doesn't cause hyponatremia?

In Addison's disease, adrenal insufficiency leads to decreased production of cortisol and aldosterone. The low levels of aldosterone lead to hyponatremia. Additionally, the low cortisol levels trigger an increase in ADH, which causes the kidneys to retain more water, further diluting the sodium levels in the blood and exacerbating the hyponatremia. This elevation of ADH is a compensatory mechanism in response to the low cortisol levels.

Can you please explain how sodium and potassium are normal in secondary adrenal insufficiency?
In secondary adrenal insufficiency, the main defect is insufficient ACTH release, which primarily affects cortisol levels. The adrenal gland does not have a primary problem. Therefore, sodium and potassium levels are typically normal because aldosterone production remains unaffected. Aldosterone secretion is largely controlled by the renin-angiotensin-aldosterone system and not directly dependent on ACTH.

When monitoring primary hypothyroidism, do you monitor treatment response with TSH or FT4? And what about in other forms of hypothyroidism?
Both TSH and free T4 should be used for monitoring of primary hypothyroidism. However, TSH is the preferred test when used for screening of primary hypothyroidism.

For central hypothyroidism, only free T4 should be monitored since the TSH level is not reliable.

If I have a patient in the ED who presents with adrenal crisis, what is the first hormone lab test that we should order to confirm the diagnosis other than electrolyte abnormalities?
A low serum cortisol level during stress or suspected adrenal crisis suggests adrenal insufficiency. An ACTH level should also be drawn to differentiate between primary and secondary adrenal insufficiency. Giving ACTH as part of an ACTH stimulation test and measuring cortisol levels afterwards can confirm the diagnosis of adrenal insufficiency in uncertain cases. However, glucocorticoid treatment should not be delayed for adrenal crisis since it can be life-threatening.

In what situations is it appropriate to obtain T3 levels in addition to TSH and FT4?
T3 levels can be useful when assessing for hyperthyroidism. Graves' disease can cause T3 thyrotoxicosis (elevated T3 with normal T4), and measuring T3 levels can help diagnose hyperthyroidism when TSH is low and free T4 levels are not significantly elevated.

In euthyroid sick syndrome, critically ill patients may have mildly abnormal thyroid tests, such as low T3 and free T4, although their thyroid is functioning adequately. These patients often have normal thyroid function tests after recovering from their illness. Measuring a T3 can be helpful in distinguishing between suspected hyperthyroidism and nonthyroidal illness in a critically ill patient. The T3 is usually high in hyperthyroidism but low or normal in nonthyroidal illness.

CH. 3 - OB/GYN & SOME STDS

For fibroadenomas, why does PBR say to refer to gynecology if they last greater than 3 months? Is it because of concern for an oncological process?
Most fibroadenomas in adolescents decrease over time, and some disappear completely. If the mass persists, an ultrasound can help confirm whether a mass has the characteristics of a fibroadenoma. Since the definitive diagnosis of fibroadenoma is through biopsy or excision, a referral to gynecology can be useful for characterizing a persistent or growing breast mass, especially as an adolescent approaches adulthood and the risk of breast cancer increases.

CH. 4 – ALLERGY & IMMUNOLOGY

In cases of anaphylaxis, what is the timeline we should observe a child to identify rebound anaphylaxis?
Rebound anaphylaxis typically occurs within a 4-6 hour window after the initial reaction. Approximately 20% of anaphylactic reactions may have a second wave, but early use of epinephrine can reduce this likelihood. The most recent anaphylaxis practice parameters offer more flexibility regarding whether a child should be observed in the emergency department or at home, especially if epinephrine was used early and a second dose of epinephrine is available at home.

How do you differentiate symptoms related to a rebound from anaphylaxis versus ongoing symptoms from the original reaction?
Rebound anaphylaxis will first show a temporary **resolution** of symptoms after initial treatment with epinephrine and supportive medications (antihistamines, steroids, albuterol).

Should we treat an anaphylactic rebound reaction by continuing to administer epinephrine?
Yes, continue to administer epinephrine if a rebound anaphylactic reaction occurs. Having multiple doses of epinephrine is important for managing any residual or recurring symptoms effectively. Additional therapies can include antihistamines and steroids.

Should you restrict foods if a screening test for food allergies is positive but the child has never had that food?
No, do not restrict foods solely based on a positive screening test for food allergies if the child has never had a clinical reaction to that food. Food allergy is a clinical diagnosis supported by testing, with the gold

standard being an oral food challenge. Screening tests can yield false positives, and unnecessary food restrictions might lead to loss of tolerance.

Page 98 says, "Initially, the potential food allergen is eliminated from the diet…" I thought we discussed not eliminating foods based on only skin and RAST testing. Am I misunderstanding this?
That sentence is referring to an elimination diet done prior to an oral food challenge. If symptoms improve on an elimination diet, it supports the *clinical* diagnosis of a food allergy. The gold standard is an oral food challenge, but it does come with the risk of triggering anaphylaxis.

If a parent tells you that a child had a limited reaction to a food, such as only a rash or only vomiting, what would be the next step?
If the reaction is limited to a rash, instruct parents to try feeding the food again. If the rash is around the mouth, certain foods can be irritating, such as tomato sauce or strawberries, and an emollient can be applied around the mouth as a barrier before giving the food. For vomiting, assess the severity and timing to see if it matches the clinical picture for food protein-induced enterocolitis syndrome (severe vomiting and dehydration 1-3 hours after ingestion), which is not IgE-mediated and requires clinical diagnosis.

How can we differentiate a severe milk protein allergy from a severe presentation of FPIES in a clinical vignette?
FPIES and IgE-mediated food allergies 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.

A severe allergy to milk can present with classic symptoms of anaphylaxis, which typically start 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).

CH. 5 – CARDIOLOGY

On the bottom of page 117, it states that hypomagnesemia causes both prolonged QT and prolonged PR intervals. However, doesn't hypermagnesemia also cause a prolonged PR interval?
Yes, both hypomagnesemia and hypermagnesemia can cause repolarization abnormalities and QT prolongation. Hypomagnesemia can cause a prolonged PR interval due to impaired AV node conduction, although it's more commonly associated with QT prolongation. Low magnesium disrupts ion balance, slowing electrical signals in the heart. We've added a clarification in the Core Study Guide that hypermagnesemia can also cause prolonged QT and PR intervals. Great question. Thanks!

Is transposition of the great arteries a ductal-dependent lesion? Also, what's the correct order of management steps for TGA? Should the first step be administering prostaglandin E or performing a balloon septostomy?
TGA can be ductal-dependent or not depending on other defects. For example, ASDs and VSDs are commonly associated with TGA, and the patient is not ductal-dependent if they have these defects. Without these defects, the TGA is ductal-dependent. The next best step depends on the specific anatomy. However, PGE is usually the answer if the child has cyanosis.

Will the boards expect us to know surgical details for tetralogy of Fallot? For example, should we know the timing of correction and types of procedures, such as the initial vs. staged modified Blalock-Taussig shunt followed by full correction at a later date? I'm guessing this is more detail than I need to know, but I would appreciate your input.
No, the ABP won't ask about these specific details. The management and intervention depend on the patient's presentation and the severity of the condition, which is beyond the scope of what you'll need to know for the boards.

Is LDL cholesterol still the primary metric used for deciding between lipid-lowering medications and lifestyle changes for 6 months, or has the guidance shifted to using non-HDL cholesterol?
The current guidelines recommend using non-fasting, non-HDL cholesterol for initial screening in children between 9 and 11 years old. If the screening is abnormal, a fasting lipid profile is done. The management of hypercholesterolemia is then based on LDL cholesterol and triglyceride levels. While non-HDL cholesterol is important for screening, LDL remains the primary metric for deciding on lipid-lowering medications and lifestyle changes.

For EKGs, can you explain the normal RS pattern in different age groups and the idea of R' please?
In the first one or two years of life, the right-sided leads have a very significant R wave because the right-sided forces are prominent due to high right-sided pressure. The right ventricle of a newborn or a baby in the first year of life is thick because the pulmonary pressures are high, but the right-sided forces gradually decrease over time. The R' pattern (RSR or M-shaped) occurs due to delayed conduction and can indicate right-sided or left-sided conduction delays depending on which leads show the pattern.

Can you talk about T-wave patterns at different ages?
T-wave patterns change with age. In newborns, T waves are generally inverted due to high right-sided pressures. As the child grows, the T wave transitions to being upright, typically becoming fully upright by around 10 to 11 years of age.

Can you explain depolarization and repolarization on EKG. Where it starts and where it ends?
The P wave shows atrial depolarization. The Q wave represents depolarization of the septum. Ventricle polarization is the R and S pattern depending on the lead. If it's a lead two, generally it's the left ventricle depolarization and then the right ventricle depolarization. The T wave represents the repolarization of the ventricles.

How can we navigate board questions for pediatric hypertension and its stages? It's very confusing, and the chart is very detailed, which we won't have access to on the exam. Any suggestions for this topic and what we should focus on?
If the exam asks you to make a diagnosis of hypertension, it will give you the normal parameters because the limits change with the gender, age, and height. It won't ask you that without giving you the normal parameters for that age and height, so you don't have to memorize those. You do have to remember the definitions for elevated blood pressure and stage one and two hypertension, which are given in PBR.

Is hypertrophic obstructive cardiomyopathy a risk for commotio cordis?
HOCM is not a direct risk factor for commotio cordis, which can result in sudden death due to arrhythmias after a sudden, blunt impact to the chest. While HOCM increases the risk of sudden cardiac death due to arrhythmias, this is related to the structural abnormalities of the heart, not external trauma like commotio cordis. Therefore, the two conditions are distinct, though both involve risks of fatal arrhythmias.

CH. 6 – DERMATOLOGY

Could you concept map biotin deficiency with inborn errors of metabolism?
The main similarity is that both conditions can cause neurological symptoms like seizures and developmental delays. But beyond that, their presentations diverge.

Biotin deficiency typically comes with a specific set of symptoms, including skin issues like dermatitis and hair loss (alopecia), along with conjunctivitis. Though neurological symptoms can occur, they often come with these distinct skin and eye signs, which you don't usually see in most IEMs. Also, IEMs present with a wide range of systemic symptoms that reflect the underlying metabolic problem, which can include metabolic acidosis, hypoglycemia, failure to thrive, and organ dysfunction like an enlarged liver (hepatomegaly) or heart problems (cardiomyopathy). The neurological symptoms in IEMs also tend to be more severe, depending on the specific metabolic pathway involved.

In general, when do hemangiomas need treatment?
Hemangiomas typically don't require treatment and often resolve on their own, but intervention is necessary in specific situations. Treatment is needed if a hemangioma ulcerates, interferes with vital functions (such as vision, breathing, or feeding), risks causing permanent disfigurement, or is located in an area that may lead to structural distortion, like the nose or lips. Additionally, treatment may be needed for a complex syndrome (e.g., PHACE) or if its presence causes significant psychosocial distress.

Another question bank asks a lot of questions about dyshidrotic eczema. How important is this topic and is there anything we should focus on for eczema?
This is unlikely to be a major focus on the exam. The unusual thing to know about dyshidrotic eczema is that it's more responsive to stronger topical steroids, so you'd want to use something like triamcinolone or fluocinonide rather than hydrocortisone. For eczema more generally, focus on understanding how to treat it and what kind of patients are more predisposed to having eczema.

For keratosis pilaris, PBR says no treatment is required. Is there any role for lactic acid/glycolic acid?
No treatment is required for keratosis pilaris because it does not have any morbidity or mortality associated with the condition, and oftentimes it's asymptomatic. But for patients who find it bothersome,

you could use a lactic acid or glycolic acid. Ammonium lactate is very commonly used. Urea and retinoids are other options as well.

Is there a good resource to look at derm pictures?
DermNet New Zealand's website is highly recommended. VisualDx is the best catalog for images if you have access. Google images can also be helpful but sometimes misleading. Additionally, *Weinberg's Color Atlas of Pediatric Dermatology* and a pediatric atlas by Dr. Koh are great resources, particularly for concept mapping. However, since there has been less emphasis on pictures on the exam in recent years, the image links provided in the PBR Core Study Guide and Virtual Atlas of Pediatric Pictures should be sufficient.

CH. 7 – NEONATOLOGY

On page 177, it mentions that Smith-Lemli-Opitz syndrome and Opitz syndrome are synonymous. Can you confirm if this is correct?
No, page 177 provides a "name alert" to highlight that Smith-Lemli-Opitz syndrome is different from Opitz syndrome. Smith-Lemli-Opitz syndrome causes defective cholesterol synthesis and is associated with cognitive deficits, microcephaly, syndactyly, and various organ malformations. Opitz syndrome is characterized by midline birth defects, such as agenesis of the corpus callosum, hypertelorism, cleft lip, heart defects, and hypospadias.

Do you think the ABP will update the recommendations on HIV breastfeeding with the recent changes suggesting that breastfeeding should be supported for mothers who are compliant with treatment and have undetectable viral loads?
The official AAP recommendations came through recently and probably wouldn't make it onto the 2024 boards.

Do you have any helpful tips such as mnemonics for how to estimate gestational age by physical exam?
No mnemonics came to mind for our content expert, but he tends to focus on the ear's flexibility and springiness. The closer to term they are, the easier it is to fold and unfold their ears. The creases on the foot and the testicular rugae in males are two other key things I look at.

For car seat safety, the PBR says to have the child stay in a rear-facing seat until 24 months or until the manufacturer's height and weight limits are reached. The AAP has its own guidelines for weight. Should we focus on the AAP guidelines?
The most recent AAP policy statement states that all infants and toddlers should stay in a rear-facing car seat for as long as possible until the highest weight or height allowed by the manufacturer. It does mention that most convertible car seats can be used rear-facing to at least 40 pounds, allowing most children to be rear-facing for at least 2 years. However, the AAP is not using 40 pounds as a limit; it still recommends using the manufacturer's height and weight limits. Specific height and weight limits for car seats are unlikely to be tested on the boards.

CH. 8 – DEVELOPMENTAL MILESTONES

The milestones in another question bank differ from those in the PBR. What should I do?
When encountering discrepancies in developmental milestones between different question banks and the PBR, prioritize the PBR materials. The PBR updates its developmental milestones chapter annually to align with the latest CDC and AAP recommendations. This ensures that the information is current and reliable. Other question banks may not update their content as frequently, which can lead to inconsistencies. Focus on the PBR materials for the most accurate and up-to-date information.

Since last year's exam, have you received any feedback or information that could confirm that the updated milestones were actually on the board exam? Do you know which question banks are the most up to date?
Developmental milestones are a heavily tested area on the board exams. Feedback from members who took last year's exam confirms that these topics were included. It remains uncertain which question banks are the most up-to-date, but it's suspected that many take years to update. Therefore, relying on the PBR materials, which are updated annually, is recommended for the most accurate preparation.

At 12 months, an infant should be able to say "mama," "dada" or another specific name. If an infant says "baba" for the word bottle, is that a specific name? Or would they actually have to be using the word "bottle"? I'm having trouble with a question about whether or not an infant who says "mama," "dada" and "baba" (for bottle) is more likely to be 12 months versus 15 month old. Thank you!
An infant saying "baba" for the word "bottle" is naming a specific object, similar to saying "mama" or "dada." Therefore, count "baba" as a specific name for developmental milestones. This is more likely to be characteristic of a 12-month-old than a 15-month-old.

Can a 15-month-old follow simple commands with or without gestures? PBR and CDC say with gestures, while another resource says without gestures. Could you clarify this discrepancy?
The updated CDC guidelines say that a 15-month-old should be able to follow directions WITH gestures. Use these guidelines for the most recent developmental milestones.

I'm finding discrepancies between PBR and other resources about the ages that children can draw shapes. Which is correct?
You will find variability about developmental milestones among resources. The lack of consensus among subject matter experts is why these milestones may not have been included in the surveillance milestones checklists updated by the AAP and CDC. I suggest sticking with one resource. The PBR!

I'm still confused about the age cutoff for developmental milestones. As I've encountered different questions in various question banks and the PBR book, my plan is to memorize what's in the book and move on. Could you please provide your thoughts?
Yes, memorize and learn the information inside the PBR materials. When dealing with developmental milestones in exam vignettes, if a child's abilities span different age ranges, assume the child is older if they exhibit more advanced skills. It's more likely for a child to be behind in some areas rather than advanced for their age. You should generally assign an age to a child based on the most advanced milestones they exhibit.

Should we know any anticipatory guidance for topics like social media and gun safety?
Please see page 71 of the Core Study Guide for recommendations about guns. Detailed guidance about social media is provided in this AAP Bright Futures PDF. It includes recommendations to stop device use at least 1 hour before bedtime to minimize disruptions to sleep and guidance on conversations about responsible social media use. Parents should discuss internet safety, such as checking with a parent before interacting with any strangers online. In addition, adolescents should be counseled that nothing online is truly private, potentially leaving a permanent digital footprint, so they should be careful about anything they post or send online, especially sexual content.

Should a four-year-old who speaks 97% intelligibly instead of 100% intelligibly be diagnosed with mild speech delay and referred to therapy?
No, a four-year-old speaking 97% intelligibly does not necessarily indicate a speech delay requiring therapy. A slight deviation from 100% intelligibility is generally acceptable and likely does not warrant a referral. The specific threshold for speech delay diagnosis can vary, but a small percentage difference is typically not significant. One study suggests referring 4-year-olds with less than 66% intelligibility (2 standard deviations below the mean) for speech therapy.

CH. 9 – EMERGENCY MEDICINE & TOXICOLOGY

No clarifications!

CH. 10 – VITAMIN & NUTRITIONAL DISORDERS

Can you please explain why alkaline phosphatase becomes elevated? I find the concept of alk phos confusing, particularly its roles in both bone and liver function.
Alkaline phosphatase is an enzyme that is present in various tissues, including bone and liver. It can become elevated when there is increased bone turnover (e.g., bone growth, remodeling) or when there is damage or obstruction in the liver or bile ducts. If ALP levels are elevated and the source is unclear, checking a GGT can help determine if the elevation is liver-related. An elevated GGT along with ALP suggests a hepatic origin, whereas a normal GGT with elevated ALP suggests a bone-related cause.

Can you please explain the difference between vitamin D2 and D3 deficiencies?
Vitamin D2 comes from plants, while vitamin D3 comes from sun exposure and animal sources, such as egg yolks, cheese, and fish. Intake of either vitamin D2 or D3 can help raise vitamin D levels, so a lack of both leads to vitamin D deficiency. A vitamin D deficiency can be treated with either vitamin D2 or D3 supplementation, although the D3 form is preferred.

If oral vitamin K does not effectively prevent hemorrhagic disease of the newborn, what is the rationale behind administering it at birth?
Vitamin K is given **intramuscularly** at birth and not orally so that it is effective in preventing hemorrhagic disease of the newborn.

CH. 11 – GASTROENTEROLOGY

What are the indications for loperamide in diarrhea?
Loperamide can be used for post-surgical causes of diarrhea, short bowel syndrome, diarrhea-type irritable bowel syndrome, and mild antibiotic-associated diarrhea. But it should not be used when there are red flags for inflammation, such as for a child who is not growing well, losing weight, or having bloody stools.

CH. 12 – PHARMACOLOGY & DRUG PEARLS

No clarifications!

CH. 13 – OPHTHALMOLOGY

No clarifications!

CH. 14 – GENETICS & INHERITED DISEASES

Retinitis pigmentosa is listed under autosomal dominant disorders, but the mnemonic refers to X-linked, and a quick search states that autosomal recessive is the most common cause. What should we focus on?
There are multiple patterns of inheritance, so you won't be asked which is most common. You could be asked to identify an inheritance pattern, but then they'll have to give you a pedigree and more information.

I've gotten lots of practice questions on abetalipoproteinemia, which is associated with retinitis pigmentosa. Should this be on our radar or in our side notebook of fun facts?
It's not commonly tested. Put it in your fun facts notebook, which you should only look at again for "fun" or because you've become a PBR Core Study Guide ninja!

Could you discuss how the boards have tested or would likely test for retinitis pigmentosa? Do we need to recognize retinal images?
The boards may describe symptoms such as difficulty seeing at night in a preadolescent or adolescent child, or they might talk about developing tunnel vision. The ABP uses fewer images than it used to, so you're unlikely to be tested on recognizing retinal images specifically. But a quick Google search will show you unique pictures that should be easy to recognize on the boards.

How do you know when to choose FISH vs. CMA vs. karyotype? I understand that the CMA is the most detailed cytogenetic test, but a practice question asked which of the following is the best confirmatory diagnostic test and the options are CMA vs. karyotype?
Chromosome microarray (CMA) provides the most comprehensive, detailed results and is the first-line test for children with multiple anomalies that don't fit within a specific genetic syndrome, global developmental delays, or intellectual disability. Karyotyping is most useful for detecting larger chromosomal changes, such as aneuploidy (missing or extra chromosomes) and large translocations and deletions. FISH testing involves specific targeted probes that can be used to confirm a suspected diagnosis such as Williams syndrome. The most appropriate testing depends on the clinical context and whether a specific genetic disorder is suspected.

CH. 15 – HEMATOLOGY & ONCOLOGY

Could you provide an example of a sample question where neonatal alloimmune thrombocytopenia would be the correct answer? Or would that likely be a distractor answer choice?
Since the thrombocytopenia is caused by maternal antibodies attacking fetal platelets, the baby would be expected to show signs of thrombocytopenia at or soon after birth. A fetal intracranial hemorrhage or a platelet count below 100,000 without another explanation should have you suspecting neonatal alloimmune thrombocytopenia.

The book contains only a few sentences on brain tumors. Is this limited information sufficient for the board exam, or should I consider a more in-depth study of this topic?
There may be a few questions on the board exam, but the big thing to focus on in a vignette would be symptoms like sudden ataxia, imbalance, or headaches that wake the child up at night and cause vomiting. The key is recognizing when to consider brain tumors as part of the differential. I don't think you need to study all the different types of brain tumors or their details. The study guide should be sufficient.

When would we do a platelet transfusion for ITP?
A platelet transfusion is almost never used for ITP unless that patient is acutely bleeding out or in the OR and you're unable to stop the bleeding. Transfusing platelets is usually not effective in ITP since antibodies quickly attack them.

For hemophilia A and B, are there clinical differences we should be aware of?
The main difference to remember is that hemophilia A is caused by factor VIII deficiency, and hemophilia B is caused by factor IX deficiency. If the vignette says a patient with a history of hemophilia A is presenting with a brain bleed, you'd infuse factor VIII. As for severity, I don't think they're going to give you specific factor activity levels; that would be too detailed.

How can lupus anticoagulant cause a prothrombotic state and also cause prolonged PTT? When I think of prolonged PTT, I think bleeding. Does the prothrombotic state just predominate?
The term "lupus anticoagulant" is a bit of a misnomer because it is a prothrombotic antibody and not associated with bleeding. The term came about because of its *in vitro* properties since it can prolong coagulation times in laboratory tests like PTT, but this does not reflect what the antibody actually does in the body. Think of it as a lab artifact rather than a true tendency to bleed more.

CH. 16 – INFECTIOUS DISEASES

If a pregnant woman tests negative for GBS at 36 weeks and the baby is term, but there is a history of invasive GBS disease in a previous pregnancy, should intrapartum antibiotic prophylaxis still be administered?
Yes. If a mom has a child with GBS infection, then she should get IAP in subsequent pregnancies even if she tests negative for GBS.

When should I use IV ceftriaxone for neurological presentations of Lyme disease? For example, if the patient only presents with Bell's palsy, should I choose doxycycline, and for any other neurological symptoms, should I select ceftriaxone?
Oral doxycycline can be used for all stages of Lyme, including Bell's palsy, meningitis, and other neurological symptoms. However, some patients are clinically unstable or hospitalized at the beginning of the illness and may need IV ceftriaxone and can then be switched to oral doxycycline once stable and ready for discharge. IV ceftriaxone can also be used in persistent arthritis if not responding to doxycycline. We've updated the Core Study Guide with this information to make it more clear. Thank you!

The book mentions using doxycycline to treat Lyme disease but doesn't specify the age. Should we give doxycycline instead of amoxicillin to children under 8 years old?
Doxycycline is preferred over amoxicillin because it treats the other bacteria carried by the same tick, such as Anaplasma and Ehrlichia. Amoxicillin doesn't cover for Anaplasma and Ehrlichia. For children who are less than eight years old, we can use doxycycline for up to 21 days. We've added this clarification to the Core Study Guide too!

Regarding the treatment of peritonsillar abscess, page 296 of the book suggests giving both clindamycin and ampicillin-sulbactam, while the summary table on page 299 says that either can be given. Could you please clarify the correct treatment approach?
Either clindamycin or ampicillin-sulbactam can be used for a peritonsillar abscess. We've rewritten the sentence in the Core Study Guide to make this clearer.

CH. 17 – VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

Regarding vaccines that contain gelatin, what is the recommendation for patients who follow Halal or Kosher dietary guidelines?
Most religious leaders who follow a Halal or Kosher diet believe that the use of gelatin in vaccines does not break religious dietary laws since these products are purified and inhaled or injected rather than ingested orally.

CH. 18 – INBORN ERRORS OF METABOLISM

Can you please explain how carnitine is a treatment for organic acidemias and why?
Carnitine binds to toxic organic acids, forming acylcarnitine compounds that can be excreted in the urine. This reduces the accumulation of harmful substances in the body, alleviating symptoms and preventing metabolic crises.

CH. 19 – ACID-BASE DISORDERS

Where can I find the recording video and instructional video on acid-base questions?
The recording video and instructional video on acid-base questions can be found on the video page where the webinar replays are located. This page includes the acid-base chapter video and the instructional video on tackling acid-base questions.

What kind of acid-base disturbance does a patient have when they are on diuretics and also have diarrhea?
Diarrhea typically causes a non-gap metabolic acidosis, but different diuretics can lead to different types of acid-base disturbances, so there isn't one answer. You would get numbers from an ABG and chemistry panel to analyze any complicated, mixed acid-base disturbances.

How should one approach complex acid-base disturbance questions on the board exam?
To approach complex acid-base disturbance questions on the board exam, focus on the steps taught in the acid-base chapter. Start with the ABG and chemistry panel, identify the primary disorder, and look for compensation. If there's a gap acidosis, calculate the delta gap. Use the provided numbers to work through the problem methodically.

Page 365 says that in chronic respiratory acidosis, bicarbonate increases by 5 mmol per 10 mmHg, but it should be 3.5 mmol per 10 mmHg. This discrepancy is also noted at the beginning of the chapter. Could you clarify this?
Page 365 says that a *decrease* of 5 mmol of bicarbonate is expected for every 10 mm Hg decrease of pCO_2 in chronic respiratory *alkalosis*. In chronic respiratory *acidosis*, bicarbonate is expected to *increase* by 3.5 mmol per 10 mm Hg increase in pCO_2. These are written correctly in the study guide, but you may have been looking at the chronic respiratory alkalosis section.

CH. 20 – FLUIDS & ELECTROLYTES

I am going over diabetes insipidus and wanted to understand specific gravity a bit more. Why is the diagnosis unlikely if SG is >1.008? How is specific gravity even calculated and what is considered normal?
Specific gravity is calculated in a laboratory and reflects how dilute or concentrated a urine sample is, typically ranging from 1.005 to 1.030, with the higher number signifying more concentrated urine, such as when a person is dehydrated. Diabetes insipidus is caused by a lack of or resistance to antidiuretic hormone (ADH), which means that the body only produces dilute urine and is unable to concentrate urine. Therefore, if a urine sample has a specific gravity higher than 1.008, it means that the body is able to concentrate urine and diabetes insipidus is unlikely.

What is the normal range for urine sodium? The book mentions 20 as the lower limit, but what is the upper limit for urine sodium?
The normal range for urine sodium is 20-40 mEq/L, so a high urine sodium is > 40.

CH. 21 – NEPHROLOGY

The core study guide states that hemolysis in HUS is caused by a verotoxin and is Coombs negative. Could you please explain what Coombs negative means and how it relates to the hemolysis in HUS?
The Coombs test detects antibodies against red blood cells that are attached to the cells (direct Coombs) or floating in the serum (indirect Coombs). In HUS, the Coombs test is typically negative because HUS is not caused by an immune-mediated process where the antibodies are attacking the red blood cells. Instead, the hemolysis in HUS results from the mechanical destruction of the red blood cells from clots and damage to the blood vessels rather than from an immune response. So a negative Coombs test would help differentiate HUS from conditions like autoimmune hemolytic anemia.

Can you explain briefly about Fanconi syndrome, cystinosis, and RTA II?
Cystinosis is an autosomal recessive lysosomal disease that may cause renal tubular dysfunction, in addition to other problems from the buildup of cystine in organs and tissues. It is one of the most common causes of Fanconi syndrome, which is the term for generalized proximal tubular dysfunction, including phosphaturia, glucosuria, and type II renal tubular acidosis. RTA type II, also known as proximal RTA, causes a fall in serum bicarbonate because the kidney is not able to reabsorb bicarbonate well in the proximal tubules. RTA II can present on its own or be a part of Fanconi syndrome.

CH. 22 – STATISTICS

I understand how to set up the table for sensitivity/specificity and PPV/NPV, but I am struggling to grasp the concepts of Spin and Snout. Could you explain why sensitivity rules out a disease and specificity rules in a disease?
A test with high specificity means that a person who tests positive is likely a true positive, so you could use a specific test as confirmation to rule IN a disease if the test result is positive. A test with high sensitivity means that a person who tests negative is likely a true negative, so it can be used as a screening test to rule OUT a disease if the test result is negative.

CH. 23 – NEUROLOGY

No clarifications!

CH. 24 – ORTHOPEDICS & SPORTS MEDICINE

No clarifications!

CH. 25 – RHEUMATOLOGY

No clarifications!

CH. 26 – PULMONOLOGY

PBR says that obstructive sleep apnea can lead to pulmonary hypertension. Can you explain how pulmonary hypertension occurs, as well as the sequelae and management of it?
Forced inspiration against a blocked airway during apneic episodes in OSA leads to a large negative intrathoracic pressure, which can result in elevated venous return and RV overload. The increased blood in those blood vessels can lead to thicker and stiffer vessel walls and increased pulmonary blood pressure. As the RV works harder, it can develop RV hypertrophy over time and also bulge the septum into the LV. Management of pulmonary hypertension secondary to OSA usually involves treating the underlying OSA with CPAP. Additional treatments may include oxygen therapy, nitric oxide, and medications like sildenafil.

For congenital diaphragmatic hernia (page 423), is the most common location on the left?
Yes, most congenital diaphragmatic hernias occur on the left side.

Can you please compare vocal cord paralysis vs. vocal cord nodules vs. exercise-induced bronchoconstriction?
Vocal cord paralysis is where the vocal cords aren't moving, which can be caused by damage to the recurrent laryngeal nerve or physical damage. Vocal cord nodules can result from overuse and present as a hoarse voice or a cry that's better in the morning or worse with use. Hoarseness in vocal cord paralysis is usually constant over the course of the day as the vocal cords remain paralyzed.

Exercise-induced bronchoconstriction causes asthma signs and symptoms such as wheezing during exercise, which is usually heard just with a stethoscope.

For the different congenital pulmonary diseases on page 423, will we be expected to know all the names for the exam?
Yes. We recommend you aim to know everything in the PBR materials. If you're cramming at the end of the study season, consider focusing more on the more common diseases rather than rare ones.

I've gotten practice questions on ventilator management through a question bank that I'm going through, but I haven't seen any information on ventilator management in the PBR materials. Can you provide a review of ventilator management content that's required for the boards?
Thanks for asking. I know it can be stressful to see information like this in practice questions, but this is not relevant for the ABP initial certification exam. It would be extremely rare for something about vent management to show up on the initial certification exam for **general** pediatricians. So, I don't think it's worth your time to learn this information.

CH. 27 – PSYCHIATRY & SOME SOCIAL ISSUES

Page 437 of the study guide mentions that frenotomy is required if tongue-tie results in feeding problems. However, there has been a significant push by the AAP to reduce the use of frenotomy. Is frenotomy still considered the correct answer on board exams for infants with feeding issues due to tongue-tie?
Yes, the AAP just published an article in August 2024 that calls for avoidance of unnecessary frenotomy. However, if there are symptoms, such as not gaining weight, nipple pain, and feeding difficulties after working with lactations consultants, surgery is still recommended even if it's to help avoid a mom giving up on breastfeeding because of pain or difficulties while feeding.

CH. 28 – ETHICS IN PEDIATRICS

No clarifications!

CH. 29 – PATIENT SAFETY AND QUALITY IMPROVEMENT

No clarifications!

CH. 30 – PEDIATRIC LAB VALUES

What lab values will the ABP provide on the exam? For example, will they provide the normal ranges for different urine studies and coagulation tests?
We reached out to the ABP to ask about this. The ABP will not provide a reference table for common labs. You are expected to know normal values for common tests (e.g., electrolytes, CBC, etc.). For uncommon tests, values will typically be given with reference ranges or described qualitatively (e.g., elevated, reduced).

How would a question be presented if it asks about when to order specific labs? For example, would they say "'all of the above need to be ordered except"?
They usually won't ask in an all-of-the-above-except format. Instead, they'll typically ask something like "Which lab value would you expect to be low?" For example, in a question about Wilson disease, the answer might be ceruloplasmin. They'll give you a set of options, and you choose the correct one. So don't worry about all-of-the-above-except-style questions.

CH. 31 – PEDIATRIC VITAL SIGNS

On page 141, PBR says the cuff bladder should "encircle 3/4 or more of the patient's arm circumference and 40% of the mid upper arm circumference." This doesn't make sense. I think it should say that the cuff should "cover 3/4 of the pt's ARM LENGTH from axilla to antecubital fossa, and encircle 40% of the mid upper arm circumference." Is that correct?
No, the cuff bladder should encircle 80% to 100% of the patient's arm circumference and the cuff bladder width should equal 40% to 50% of the upper arm circumference. We've rewritten the sentence in the core study guide to make this clearer!

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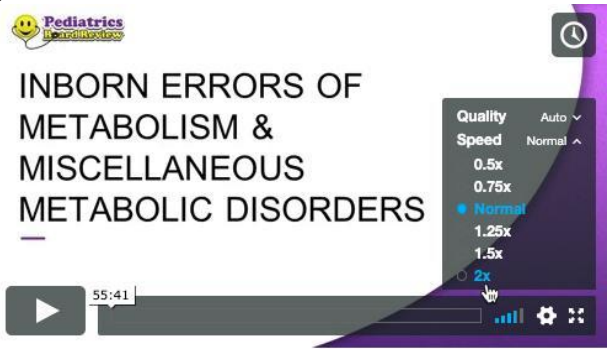

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