Pediatrics® Board Review

2025 Corrections and Clarifications Guide

2025 EDITION

15th 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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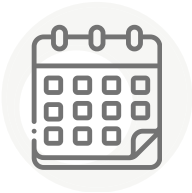

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A FEW WORDS OF THANKS TO THE PBR COMMUNITY

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. 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. Moshe Cohn
- Dr. Shubham Bakshi
- Dr. Kumar Nadhan
- Dr. Yorgo Zahlanieh

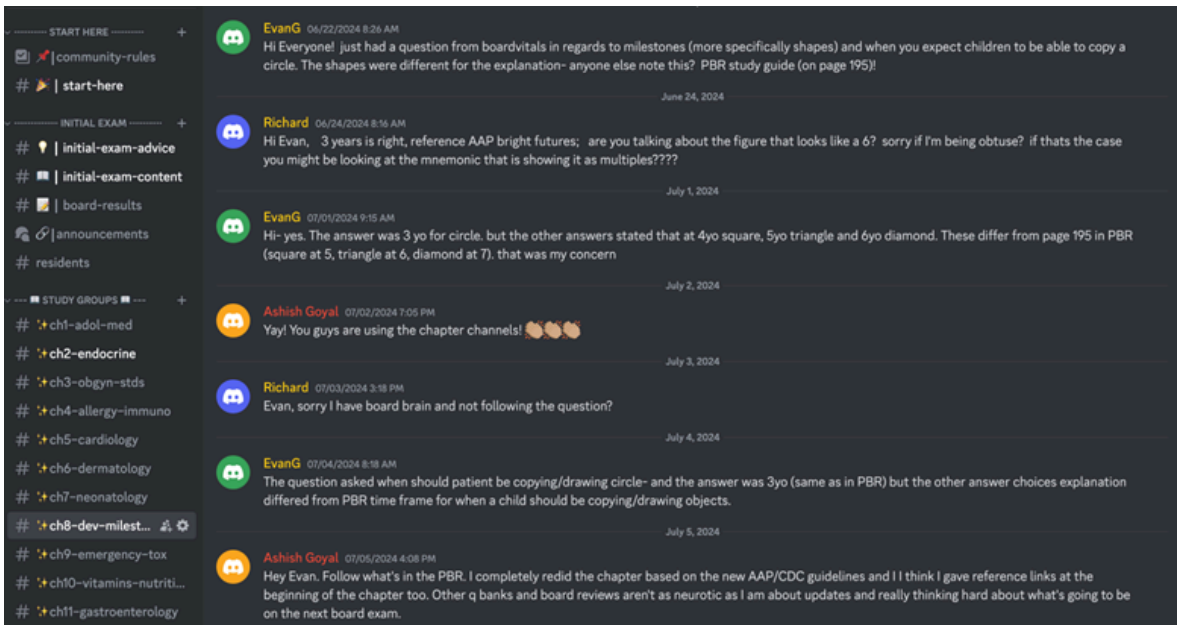
NOW… WHAT IS THIS THING?

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" i**f 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!

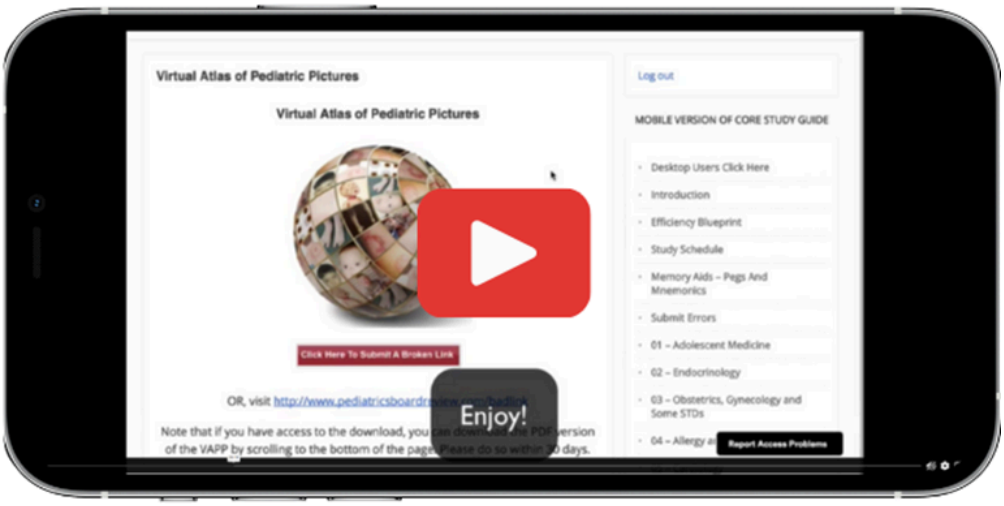
WHAT ABOUT IMAGE LINK CORRECTIONS?

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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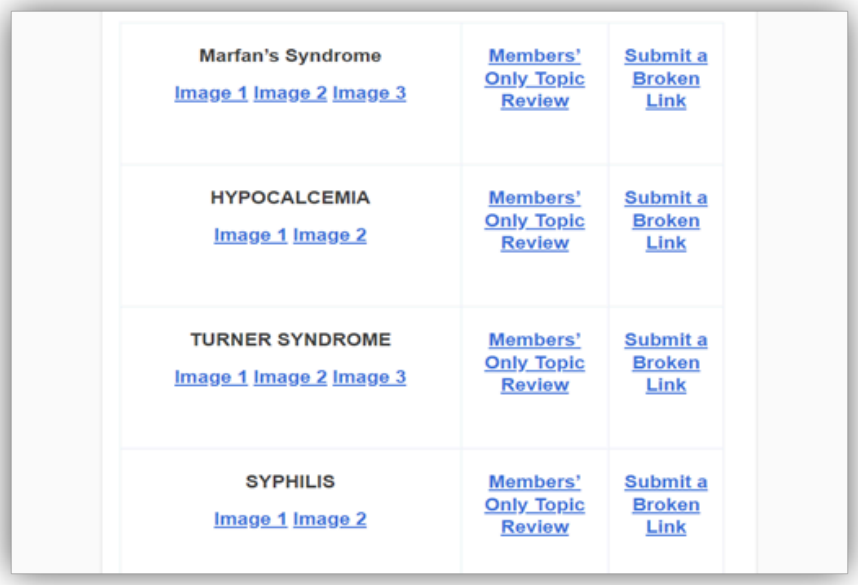


FREQUENTLY ASKED QUESTIONS

"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:

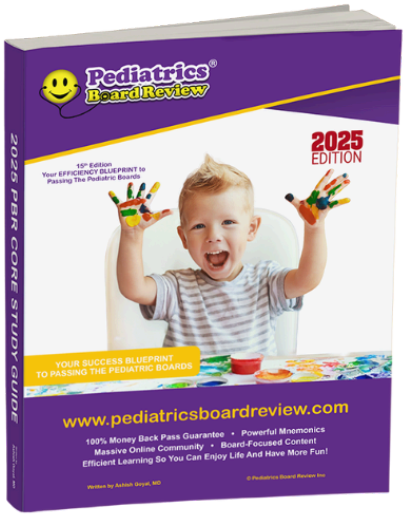
1. **IT'S FRUSTRATING TO HAVE AN OLDER BOOK. WATCH!**
    - You will see in this guide that many submissions will reference specific page numbers and specific lines within a paragraph. This happens all year long, especially in our private forum. This is NOT the time to be spending your energy cross-checking 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 **2025 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 MEDICAL UPDATES and TRUE ERRORS that will be addressed in our next edition.

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

CORRECTIONS FOR 2025 EDITIONS

CH. 11 – GASTROENTEROLOGY

The video notes that celiac disease is a type of food protein-induced enteropathy, which is usually diagnosed from clinical history. However, if celiac disease is suspected, a biopsy is warranted. In patients with failure to thrive and concern for a food protein-induced enteropathy, what additional clues in the history can help determine whether a biopsy for celiac disease is indicated?

Celiac disease should not be diagnosed solely based on clinical history. Though clinical suspicion may be raised by symptoms such as persistent diarrhea, poor weight gain, anemia, short stature, amenorrhea, or alopecia, the gold standard for diagnosis is a small intestinal biopsy. Serologic testing (e.g., tissue transglutaminase IgA) is supportive but not definitive. Biopsy should be performed before initiating a gluten-free diet, as dietary changes can normalize mucosal appearance and result in a false-negative. Dermatitis herpetiformis is a special case—if confirmed by skin biopsy, no intestinal biopsy is required. The Core Study Guide is correct; we will update the video to reflect this as well.

CH. 12 – PHARMACOLOGY & DRUG PEARLS

The pharmacology chapter video states diazepam is contraindicated during pregnancy. Is that correct?

The FDA has phased out its old letter-based pregnancy categories. Under that old system, a lot of the benzodiazepines were categorized as category D, indicating positive evidence of human fetal risk, but the potential benefits might warrant use in pregnant women. Even diazepam's product label advises against use during pregnancy unless the benefits outweigh the potential harm to the fetus. Older studies showed a potential increased risk of cleft lip and cleft palate if diazepam was used in the first trimester. But recent larger and better design studies haven't really confirmed that association. We will update the video to say it's not an absolute contraindication, but avoid it if you can.

CH. 16 – INFECTIOUS DISEASES

In the Infectious Diseases chapter, the Core Study Guide says invasive aspergillus is treated with voriconazole, but the Pulmonary chapter says to treat invasive aspergillus with amphotericin. Can this be clarified, please?

Voriconazole would be first line and amphotericin B would be used if voriconazole isn't working. Thank you for bringing this up, and we'll be making modifications. Here's what the revised topic may look like for next year's book:

(DOUBLE TAKE) ASPERGILLUS

Aspergillus can present as **allergic bronchopulmonary aspergillosis (ABPA)** in asthmatics and patients with cystic fibrosis, characterized by recurrent exacerbations, eosinophilia, very high

total IgE, Aspergillus-specific IgE/IgG (precipitins), and central bronchiectasis on CT. ABPA is **steroid-responsive**, and adding an oral triazole (e.g., itraconazole) as an adjunct can reduce steroid exposure and relapses. In immunocompromised hosts, **invasive aspergillosis (IA)** causes pulmonary nodules often with a halo sign and may cavitate (air-crescent sign during recovery). Treat IA with **voriconazole as first-line**. Use lipid amphotericin B when triazoles are contraindicated, not tolerated, ineffective, or when local patterns or species suggest reduced azole activity. Protective environments (e.g., HEPA-filtered positive-pressure rooms) are used to protect profoundly immunocompromised patients at risk of acquiring molds, not to prevent transmission, since Aspergillus is ubiquitous.

CH. 17 – VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

Are there any changes that haven't been reflected in the Core Study Guide?

Prevnar 20 has replaced Prevnar 13 with the same schedule. For measles post-exposure prophylaxis, all severely immunocompromised patients should receive IVIG, not IGIM, even if the child is <12 months old. For children over 12 months old exposed to measles, a second dose of MMR should be given as long as it's been at least 28 days since MMR #1 even if they're not 4 years old yet. We've updated the CSG with these changes.

CH. 23 – NEUROLOGY

Can you please go over what Benign Paroxysmal Vertigo of Childhood (BPVC) is? How do they present, what is the treatment plan, and is diphenhydramine indicated?

For anyone who attended the webinar, we mixed up benign paroxysmal vertigo of childhood (BPVC) and benign paroxysmal positional vertigo (BPPV). The "BPV" topic has been removed from the Core Study Guide and replaced with BPVC. BPVC is considered to be a migraine variant in children. Vertigo lasts seconds to minutes. Kids can be unsteady, frightened, and fall. It's usually very self-limited and doesn't require any medications.

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

CLARIFICATIONS FOR 2025 EDITIONS

CH. 1 – ADOLESCENT MEDICINE

Could you please review refeeding syndrome and the role of thiamine/thiamine deficiency?

Refeeding syndrome is a potentially life-threatening condition that occurs when nutrition is reintroduced after a period of starvation or severe malnutrition, such as in anorexia. The hallmark is hypophosphatemia, but it also involves hypokalemia, hypomagnesemia, fluid shifts, and glucose metabolism dysregulation, all triggered by a sudden surge in insulin. As cells shift from catabolism to anabolism, they rapidly consume intracellular electrolytes and nutrients, leading to organ dysfunction due to electrolyte shifts. Clinical features may include arrhythmias, heart failure, respiratory failure, seizures, and delirium.

Thiamine deficiency is a key contributor to the severity of refeeding syndrome. During starvation, thiamine stores are quickly depleted. Once feeding resumes, especially with carbohydrates, the demand for thiamine surges because it is a critical cofactor for enzymes involved in carbohydrate metabolism (e.g., pyruvate dehydrogenase). Without sufficient thiamine, glucose metabolism becomes impaired, leading to lactic acidosis, Wernicke encephalopathy, and even cardiac failure. This is why thiamine supplementation before and during refeeding is essential—it helps restore safe metabolic function, supports ATP production, and reduces the risk of neurologic and cardiovascular collapse.

How should secondary amenorrhea be evaluated in adolescents?

The evaluation of secondary amenorrhea begins with a urine pregnancy test (HCG). If negative, assess for hypothalamic, pituitary, ovarian, or uterine causes by ordering LH, FSH, estradiol, prolactin, and TSH. The LH:FSH ratio is >2 in PCOS. If there is no withdrawal bleeding after a 10-day progesterone challenge, consider estrogen deficiency and evaluate FSH for ovarian failure.

What are the diagnostic criteria and workup for Polycystic Ovary Syndrome (PCOS) in adolescents?

Diagnosis of PCOS in adolescents requires both irregular menses and signs of hyperandrogenism. Clinical signs include hirsutism and severe acne. Biochemical signs include elevated total or free testosterone. Rule out other causes such as non-classic congenital adrenal hyperplasia by checking 17-hydroxyprogesterone. Additional labs include LH, FSH, estradiol, prolactin, and TSH.

What is the significance of primary versus secondary amenorrhea, and when should Turner syndrome be considered?

Secondary amenorrhea is commonly from pregnancy, or consider an eating disorder if the patient has been losing weight rapidly. Primary amenorrhea in a girl with short stature should raise suspicion for Turner syndrome. Initial labs should include FSH and LH. High FSH suggests ovarian failure (hypergonadotropic hypogonadism), as seen in Turner syndrome. If FSH is low, central causes such as hypopituitarism or pituitary tumors should be considered. A karyotype should be ordered if Turner syndrome is suspected.

CH. 2 – ENDOCRINOLOGY

What are the key features of congenital hypothyroidism and how is it diagnosed and managed?

Symptoms of congenital hypothyroidism include lethargy, feeding problems, constipation, hypotonia, dry skin, and prolonged jaundice. Congenital hypothyroidism is screened at birth using TSH and sometimes free T4. Elevated TSH indicates primary hypothyroidism, often due to thyroid dysgenesis or ectopy. Serum TSH and free T4 are needed to confirm the diagnosis. Early treatment with levothyroxine is essential to prevent neurodevelopmental delays. Any midline neck mass warrants assessment for ectopic thyroid before surgical removal to avoid removing the only functioning thyroid tissue.

How do you identify and manage neonatal Graves' disease?

Neonatal Graves is caused by maternal thyroid receptor antibodies (TRAb) crossing the placenta. Diagnose when TFTs show elevated age-appropriate free T4 and/or T3 with suppressed TSH, usually showing this pattern around day 3 to 7. Positive infant or cord TRAb supports the diagnosis, though the results can can take time to return. Start therapy if the infant is symptomatic (resting tachycardia, irritability, poor feeding or weight gain, heart failure, SVT) or if free T4 is markedly high. Use methimazole as first line and add propranolol for significant tachycardia. Reserve short courses of iodine or steroids for severe cases. If TRAb is negative and TFTs are normal, routine observation is reasonable. If TRAb is positive or unknown, recheck TFTs during the first 1 to 2 weeks. Most cases resolve as maternal antibodies clear over weeks to a few months.

How is hyperthyroidism (Graves' disease) diagnosed and treated in children?

Pediatric Graves' disease presents with symptoms such as tachycardia, weight loss, heat intolerance, and sometimes exophthalmos. Diagnosis is confirmed by low TSH, high free T4, and positive TSI or TSH receptor antibodies. Treatment includes beta blockers like atenolol to stabilize cardiac symptoms, followed by methimazole to suppress thyroid hormone production. PTU is avoided due to its risk of liver toxicity.

What causes hypocalcemia in children and how is it evaluated and managed?

Causes include DiGeorge syndrome, maternal diabetes, prematurity, and hypoparathyroidism. Consider pseudohypoparathyroidism when PTH is elevated despite low calcium. Hypocalcemia can cause seizures, tetany, and QT prolongation. Evaluation includes total and ionized calcium, BMP, phosphorus, magnesium, vitamin D, and PTH levels. In acute cases, treatment is IV calcium gluconate.

How is adrenal insufficiency (Addison disease) identified and treated?

Addison disease (primary adrenal insufficiency) can present with fatigue and hyperpigmentation and look like dehydration with hypotension, hyponatremia, hyperkalemia, and hypoglycemia. Diagnosis involves checking cortisol and ACTH levels. Treatment is urgent IV fluids (normal saline) and corticosteroids.

What are the causes and treatment of pediatric hypercalcemia?

Hypercalcemia (calcium > 12) in children is uncommon but may be caused by Williams syndrome, immobilization, vitamin A or D toxicity, or rarely parathyroid adenomas. Treatment begins with IV fluids followed by loop diuretics if needed. If high PTH is noted, consider imaging for a parathyroid adenoma. Malignancy-related hypercalcemia is rare in children.

What is pseudohypoparathyroidism and how is it different from hypoparathyroidism?

Pseudohypoparathyroidism is a genetic disorder that causes PTH resistance. It presents with hypocalcemia, hyperphosphatemia, and elevated PTH. Patients may have Albright hereditary osteodystrophy features such as round facies, short 4th/5th fingers or toes, and developmental delay. This differs from hypoparathyroidism, where PTH is low.

What are the pubertal milestones and how is delayed or precocious puberty evaluated?

First signs of puberty are breast buds (girls) and testicular enlargement (boys). Precocious puberty is when puberty starts before age 8 in girl or 9 in boys. Delayed puberty is defined as no signs of puberty by 13–14 in girls and 14–15 in boys. Evaluation includes bone age, LH, and FSH. LH >0.3 suggests central precocious puberty, which may require brain MRI to exclude tumors. Treatment for central precocious puberty includes GnRH analogues like leuprolide.

When a child presents with early onset of pubic hair, is obtaining a bone age X-ray the appropriate first step in evaluation?

In general, when a child develops pubic hair (pubarche) earlier than the usual age thresholds (i.e., before 8 years in girls and 9 years in boys), the recommended initial evaluation does include obtaining a bone-age X-ray. The bone-age assessment helps clarify whether skeletal maturation is advanced, suggesting a more pathologic process such as central or peripheral precocious puberty.

In cases of isolated pubic hair without other pubertal signs—such as no breast development in girls or no testicular enlargement in boys—premature adrenarche is the most likely benign cause, and bone age is often not accelerated. If bone age is advanced (for example, >2 standard deviations above chronological age), further endocrine evaluation—including hormone panels and possibly GnRH stimulation testing—is warranted to distinguish central vs. peripheral causes.

In cases of suspected precocious puberty, what should be the initial workup?

The initial workup should include a bone age and LH, FSH, and gonadal hormones (estradiol in girls or testosterone in boys). These help determine whether the puberty is gonadotropin-dependent (central) or gonadotropin-independent (peripheral). A basal LH level that is clearly elevated (especially if in the pubertal range) suggests central precocious puberty (CPP), and a GnRH stimulation test can be used for confirmation if needed. Elevated sex steroids with suppressed LH/FSH suggest a peripheral cause. If central precocious puberty is confirmed, a brain MRI is often warranted—especially in children under 6. If the findings suggest a peripheral cause (e.g., congenital adrenal hyperplasia or an ovarian tumor), the lab panel may be expanded accordingly.

In girls, if LH/FSH are elevated—especially in the pubertal range—a diagnosis of central precocious puberty is likely; in such cases, a brain MRI is generally indicated to rule out central lesions, with particularly higher urgency in girls younger than 6 years or those with rapid progression of pubertal signs. Conversely, if gonadotropins remain in the prepubertal range and peripheral sex steroids are elevated, this suggests a peripheral source, and pelvic ultrasound may be useful to evaluate ovarian and uterine morphology for possible tumors or cysts.

In boys, if LH is elevated in the setting of precocious puberty, this again supports central precocious puberty, and a brain MRI is indicated. However, if LH remains normal or low yet testosterone is elevated, a peripheral mechanism such as a β-hCG–secreting tumor or Leydig cell tumor should be suspected, and evaluation should include measurements of β-hCG (and α-fetoprotein), plus relevant imaging—such as testicular and potentially brain imaging—to localize the source.

CH. 3 - OB/GYN & SOME STDS

On page 96 in the "Double Take Syphilis" section, it states that confirmatory treponemal testing (e.g., FTA) is needed due to possible false positives from non-treponemal tests. However, the same paragraph also mentions that treponemal serologic tests like FTA are difficult to interpret in infants and are therefore not recommended. This seems contradictory—could you please clarify this recommendation and explain the reasoning behind it?

I can see how this could be confusing, so I've rewritten the topic. Here it is!

(DOUBLE TAKE) SYPHILIS

Syphilis is caused by TREPONEMA PALLIDUM. Screen with a non-treponemal test (RPR or VDRL) and **confirm with a treponemal assay** such as FTA-ABS or a treponemal EIA since non-treponemal tests may yield false positives. Treponemal tests tell you if infection is present or has **ever been** present, but they do not track disease activity. Non-treponemal titers roughly reflect disease activity and fall with effective treatment, so use them to stage disease and to monitor response. For neonates, maternal IgG crosses the placenta and can make treponemal tests (i.e., FTA-ABS) positive, so rely on a **quantitative non-treponemal serologic test (**RPR or VDRL) drawn from the baby's serum and compare it to the maternal titer. If the infant titer is at least **fourfold** higher than the mother's titer, or if the mother was inadequately treated, treat for congenital syphilis. Also treat if the mother was treated **less than 30 days before delivery or received a non-penicillin regimen such as erythromycin**, which does not reliably prevent congenital infection. If the mother was appropriately treated and the infant's titer is lower than the maternal titer, this is most consistent with passive antibody transfer, and you should follow titers without treatment. Titers should show a decline by 3 months and become nonreactive by 6 months.

* CONGENITAL SYPHILIS: The baby can be born with a maculopapular rash, HSM, generalized lymphadenopathy and **PEELING SKIN**. If left untreated, the baby may later develop a **PERFORATED PALATE, a PERFORATED NASAL SEPTUM**, hearing loss, **HUTCHINSON TEETH** or a host of other complications. Hutchinson teeth are peg-shaped (cone-like) but also have a central notch that is extremely specific for congenital syphilis. Treat with PENICILLIN (PCN).

* CONDYLOMA LATA refers to SECONDARY SYPHILIS, in which white-gray coalescing papules are seen.
* **PEARL**: If the FTA is positive but VDRL is negative, also consider LYME DISEASE (BORRELIA BURGDORFERI).

* **NAME ALERT/MNEMONIC**: 

Condyloma LATA (AKA "condyloma FLATa," are much more FLAT than Condyloma ACUMINATA (which is found with HPV infections).

* **NAME ALERT/MNEMONIC**: 

Peg teeth are also found in patients with Incontinentia Pigmenti (AKA "incontinentia PEGmentia").

* **IMAGE**: (PEG-SHAPED TEETH) – www.pbrlinks.com/PEGTEETH1
* **IMAGE**: (HUTCHINSON TEETH) – www.pbrlinks.com/HUTCHTEETH1

On page 98, the section on vaginal discharge at birth states that pink or white discharge is benign due to maternal estrogen exposure. Later, it says that signs of bleeding or petechiae should raise concern for vitamin K deficiency. However, I was taught that slight vaginal bleeding in newborn girls—particularly in the first two weeks—is also a normal estrogen withdrawal response. Could you please clarify what is meant by "pink" discharge in this context?

Pink or white vaginal discharge in neonates is most often due to estrogen withdrawal and is benign. Slight vaginal bleeding (also from estrogen withdrawal) is common and not concerning. However, vitamin K deficiency should be considered if bleeding is more severe, especially in infants whose parents declined vitamin K prophylaxis. The key is the amount of bleeding and clinical context.

On page 95, the book discusses empiric treatment for gonorrhea and chlamydia. If testing later confirms gonorrhea but excludes chlamydia co-infection, should treatment still cover both organisms, or should antibiotic therapy be tailored based on the final results?

Empiric treatment should include coverage for chlamydia, but treat only for gonorrhea if chlamydia has been excluded, or once it can be excluded. This is supported by the CDC STI guidelines.

I've been struggling to remember the absolute contraindications to combined oral contraceptives. I created a visual memory aid, which includes breast cancer (depicted as a crab on the breast), pregnancy (a glowing belly), liver disease (a liver-shaped hat or hair bow), and breastfeeding within the first 6 weeks postpartum (represented visually as well). Would it be possible to review and confirm that these are correct and comprehensive absolute contraindications?

The listed absolute contraindications—breast cancer, pregnancy, liver disease, and breastfeeding within the first 6 weeks postpartum—are correct but not comprehensive. Other absolute contraindications include stroke, migraines with aura, and uncontrolled hypertension. It's not necessary to memorize the entire list; focus on the high-yield items likely to appear on the boards.

On page 91, in the GBS section about infants less than 34 weeks gestation, the first two bullet points appear to be identical. Could you please review the appropriate treatment approach for

these infants and clarify if there is a difference between these two bullet points or if this is a duplication error?

Thanks for bringing this up. There is definitely a bit of redundancy between the two bullet points regarding management of infants ≤34 weeks gestation at risk for GBS infection. We've consolidated the two points in the CSG to be clearer that infants ≤34 weeks gestation should get a blood culture + antibiotics (and consider LP for sick/high-risk infants) if they meet any of the following criteria: preterm labor OR prelabor ROM OR induced labor with inadequate IAP OR any concern for intraamniotic infection OR respiratory or cardiac instability.

CH. 4 – ALLERGY & IMMUNOLOGY

Could you review the key points we should know about rebound anaphylaxis? How do we distinguish symptoms of rebound from those of the initial anaphylactic reaction? Is it sufficient to monitor the patient for 4 hours in the ED instead of 6? And should we treat a rebound reaction by continuing to administer the EpiPen?

Rebound anaphylaxis, also known as biphasic anaphylaxis, is defined by the recurrence of symptoms after resolution of the initial episode. It is more likely when there's a delay in initial epinephrine administration. To distinguish it from a refractory reaction, look for complete resolution followed by recurrence. Historically, patients were monitored for 6 hours, but recent updates suggest shared decision-making is acceptable in some cases, especially considering the cost and burden of ED visits. There is no consensus yet on exactly what observation period is appropriate. The biphasic reaction typically occurs within 4–8 hours. Epinephrine should be administered again if symptoms return.

Could you comment on X-linked agammaglobulinemia? I don't see it mentioned in the core study guide.

X-linked agammaglobulinemia is covered in the Allergy and Immunology chapter of the Core Study Guide (also referred to as Bruton's agammaglobulinemia). It's a disorder of B-cell development, leading to undetectable levels of immunoglobulins (IgG, IgA, IgM, IgE). Clinical manifestations begin around six months of age when maternal IgG wanes, with recurrent bacterial infections (e.g., otitis, pneumonia, sepsis). Physical exam may reveal absent tonsils and lymph nodes due to lack of germinal centers. T-cell levels may be elevated to compensate. Complement deficiency may present similarly but will show normal immunoglobulin levels and low CH50.

I've included the CSG text on X-linked agammaglobulinemia below:

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

Agammaglobulinemia (AKA X-linked agammaglobulinemia or Bruton's agammaglobulinemia) is X-linked, so it is seen in **B**oys. There is a total absence of **B** cells, which means there are **NO IMMUNOGLOBULINS. NO Igs**! Labs may show **high T-cell counts**. Patients have **tiny or absent tonsils and no palpable lymph nodes**. It results in recurrent **B**acterial infections and presents around 6 months of age. You might be presented with a child who has a history of "many antibiotic courses." This could refer to recurrent infections with enCAPSulated organisms, especially Pseudomonas, Streptococcus pneumonia, and Haemophilus influenza. Look for

sepsis, meningitis, and recurrent pneumonia. Pneumocystis pneumonia (AKA PCP) does NOT occur in this disorder. If you see PCP, think Hyper-IgM or SCID! TREAT with IVIG for life and give prophylactic antibiotics. BMT is curative.
MNEMONIC: The age of presentation (6 months) happens to be around the same age when the mother's immunoglobulins/antibodies begin to wane!
PEARL: C1-C4 complement deficiency also results in bacterial infections, but not as severe. CH50 is a good lab test to differentiate these two entities. CH50 is low in C1-C4 complement deficiency and normal is Bruton's agammaglobulinemia. When CH50 is normal, it means ALL of the complement pathways are okay (C1–C9).

This may or may not be high-yield for the boards, but could you help differentiate serum sickness from drug hypersensitivity reactions? Both can present with fever, rash, and lymphadenopathy, but are there specific clinical features such as timing, joint involvement, or visceral symptoms that help distinguish between the two?

Serum sickness typically presents 7–14 days after exposure and is characterized by fever, rash, lymphadenopathy, and more prominent joint involvement—but generally lacks mucosal or visceral organ involvement. In contrast, severe drug hypersensitivity reactions such as DRESS, Stevens-Johnson, or TEN tend to occur sooner (within 7 days) and often include mucosal involvement and renal or hepatic dysfunction. It's critical to get a thorough medication history and look at timing of symptoms. There is a lot of overlap, and it can be a little challenging to differentiate them.

If on the exam they give a question about a child with possible milk and peanut allergies and after performing allergy testing, RAST test is positive, what is the next best step in management? What if the child has had no clear reaction?

If a child has a clinical reaction suggestive of a food allergy and a positive RAST/IgE test, the next best step is to prescribe epinephrine, instruct the family to avoid the suspected food, and refer to an allergist. However, if there has been no clinical reaction and the food is being tolerated, even with a positive IgE or RAST test, this reflects sensitization—not a true allergy—and the food should remain in the diet. Elevated IgE levels alone, especially in children with eczema, are not diagnostic of food allergy.

When trying to identify an immunodeficiency in a question stem, how do you approach being told what infections a child has had—such as pneumonias and sinusitis? For example, fungal infections would point you toward a T-cell deficiency, but does a general infection history help, or do you keep reading for more specific symptoms like eczema or tiny lymph nodes?

Recurrent sinopulmonary infections (sinusitis, otitis media, pneumonia) suggest a B-cell or complement deficiency. Fungal infections, viral warts, and pneumocystis point toward a T-cell deficiency. Further physical findings such as absent tonsils or lymph nodes support a B-cell deficiency. The presence or absence of immunoglobulins, lymphopenia, and CH50 levels can further differentiate these disorders. The CSG chart on page 107 is helpful for differentiating between types of immunodeficiencies.

Could you please discuss the difference between food protein–induced enteropathy and food protein–induced proctitis/colitis?

Food protein–induced proctitis/colitis usually presents in otherwise healthy infants with bloody stools and is generally benign and self-limited. In contrast, food protein–induced enteropathy causes malabsorption and failure to thrive, resembling celiac disease but without the autoimmune component.

CH. 5 – CARDIOLOGY

If an asymptomatic teenager comes to the office and his BP is 140/90, what should my next step be? Should I repeat the BP in 1 week? What are the guidelines for managing hypertension based on severity?

For teenagers (13+ years), a BP of 140/90 qualifies as stage 2 hypertension. The next step is to repeat the BP within one week. If it remains in the stage 2 range, the patient should undergo ambulatory blood pressure monitoring and be referred to a specialist. Always consider age, sex, and height percentiles (www.pbrlinks.com/BPGUIDE) when assessing BP in pediatric patients.

On pages 130–131, only indomethacin is mentioned as medical treatment for a patent ductus arteriosus (PDA). Does this imply that indomethacin should be preferred over acetaminophen for PDA closure? How does the ABP view the use of acetaminophen compared to indomethacin for this indication?

Indomethacin and acetaminophen are both used for PDA closure, but there's no consensus on which to use. Indomethacin was preferred in the past, but acetaminophen is being used more frequently now because of fewer side effects. Either one is fine for the boards unless there's a contraindication. The CSG has been updated to include acetaminophen as an option for PDA closure.

Could someone clarify the mnemonic for ventricular septal defects (VSDs) on page 125? It describes a "SEPTAL defect" using the image of a 3-year-old's home, a roll of perforated stamps (SEPTATED), TRIcycles, and a CAT licking the stamps. Is the "TRIcycle" meant to help recall the association with trisomies? And does the "CAT" refer to cri-du-chat syndrome?

Yes, "TRIcycles" refer to trisomies 13, 18, and 21, which are associated with VSDs. "CAT" refers to cri-du-chat syndrome, which is also associated with VSD. Note that AV canal defects are more characteristic of Down syndrome than VSDs alone, though both can be present.

What clinical features in a patient with a large facial hemangioma should prompt an echocardiogram to evaluate for PHACE syndrome?

Large facial hemangiomas can be the first visible sign of PHACE syndrome, which includes posterior fossa malformations, arterial anomalies, cardiac defects (especially coarctation of the aorta), and eye abnormalities. The cutaneous red flag is a large (>5 cm), segmental hemangioma of the face or scalp. An echocardiogram should be obtained in any infant with a large segmental facial hemangioma, regardless of cardiac symptoms.

How is the QT interval measured on an EKG, and how do we accurately calculate the corrected QT interval (QTc)?

On an EKG, the QT interval is measured from the beginning of the QRS complex to the end of the T wave. It's typically measured in lead II or V5/V6, where the T wave is most clearly visible.

To correct for heart rate variability, the corrected QT interval (QTc) is calculated using Bazett's formula ($QTc = QT / \sqrt{RR}$), where QT and RR are both measured in seconds.

Could you review the management of supraventricular tachycardia (SVT) in both acute and chronic settings? I'm confused about when beta blockers are indicated versus when ablation therapy should be pursued.

Acute SVT Management (Stable Patients)

- Start with vagal maneuvers (e.g., Valsalva, carotid sinus massage)—often effective for AVNRT/AVRT if hemodynamically stable.
- If unsuccessful, adenosine is first-line due to its rapid action and short half-life (~10 seconds).
- Failing adenosine (or if contraindicated), proceed with IV calcium channel blockers (verapamil, diltiazem) or beta-blockers (e.g., metoprolol or esmolol) for AV nodal–dependent SVT.
- For pre-excited AF/WPW, AV-nodal blocking agents (including adenosine, calcium blockers, beta-blockers, digoxin) are contraindicated due to risk of accelerating conduction via the accessory pathway. Use IV procainamide if stable or electrical cardioversion if unstable.

Chronic SVT Management (Long-Term Control)

- Catheter ablation (usually radiofrequency) is recommended as first-line for symptomatic, recurrent SVT (e.g., AVNRT, AVRT, focal atrial tachycardia), with success rates over 90% and recurrence <5%.
- If ablation is not feasible or the patient prefers medical therapy: Use beta-blockers or non-dihydropyridine calcium-channel blockers for AVNRT/AVRT (Class IIa recommendation per ESC). For recurrent focal atrial tachycardia, ablation is preferred, but beta-blockers can be used if ablation is not an option.

Can you explain when subacute bacterial endocarditis (SBE) prophylaxis is recommended? I'm finding the concept challenging, especially when answering board-style questions.

The current guidelines recommend antibiotic prophylaxis for SBE (infective endocarditis) only in a small subset of patients with the highest-risk cardiac conditions who are undergoing specific invasive procedures.

High-risk cardiac conditions include:

- Prosthetic cardiac valves (including transcatheter implants) or valve repairs with prosthetic materials (e.g., annuloplasty rings, chords)
- A history of prior infective endocarditis
- Certain congenital heart diseases (CHD): unrepaired cyanotic CHD, or repaired CHD with residual shunts or valvular regurgitation at or adjacent to the site of a prosthetic patch/device—or repair within the past six months
- Cardiac transplant recipients who develop structurally abnormal valves or regurgitation

Prophylaxis is generally reserved for high-risk patients undergoing procedures that are likely to cause bacteremia, especially:

- Dental procedures involving manipulation of gingival tissue, periapical regions, or perforation of the oral mucosa (e.g., extraction, scaling, root canal)
- In certain high-risk individuals, invasive respiratory tract procedures involving mucosal incision or biopsy (e.g., tonsillectomy, adenoidectomy), or procedures involving infected skin, skin structures, or musculoskeletal tissue may warrant prophylaxis

Prophylaxis is not recommended for routine dental anesthetic injections, dental X-rays, placement or adjustment of orthodontic appliances, shedding of deciduous teeth, lip/mucosal trauma, or for gastrointestinal or genitourinary procedures, even in high-risk patients.

CH. 6 – DERMATOLOGY

Page 145 of the 2025 Core Study Guide notes that guttate psoriasis is preceded by a group A strep infection. Is the group A strep a catalyst or does it provoke the expression of psoriasis, which I thought was a hereditary condition?

Psoriasis is a hereditary condition, but a group A strep infection can act as a trigger, provoking the expression of psoriasis in genetically predisposed individuals. Other triggers include COVID, other infections, and vaccinations.

Whenever I get a question that describes a "scaly rash," my mind always goes to psoriasis and I forget about fungal causes. Are there any other typical causes of scaly rash that I should keep in mind for my differential diagnosis? What are the key differences between them?

Three causes of scaly rash I think about frequently are psoriasis, fungal infections, and eczema. Psoriasis is thick and silvery, symmetric, and rarely itchy. Fungus is annular, with a clear central area, and the peripheral ring is red and scaly, itchy and asymmetric. Eczema can have light scale but it's poorly demarcated, unlike the demarcation in psoriasis. Also think about pityriasis rosea, a post-viral rash with a herald patch and powdery scale.

Which comes first in psoriasis, itch or rash?

Psoriasis typically begins with the rash, not itch. While some patients may experience itchiness—especially on the scalp—the rash usually precedes the symptoms. Scratching can induce new lesions due to the Koebner phenomenon, but this is not the origin of the initial plaques.

Please explain the difference between incontinentia pigmenti and hypohidrotic ectodermal dysplasia.

Incontinentia pigmenti is an X-linked dominant disorder that typically presents in females, who show characteristic cutaneous stages: a neonatal blistering rash, wart-like lesions, swirling hyperpigmentation,

and later hypopigmented, hairless streaks. Other affected tissues include hair, teeth, nails, eyes (with risk of retinal detachment), and occasionally the central nervous system with possible developmental delays or seizures. The condition is usually prenatally lethal in males.

Hypohidrotic ectodermal dysplasia is typically inherited in an X-linked recessive manner (though autosomal dominant/recessive forms exist) and is characterized by hypohidrosis (reduced or absent sweating), sparse or brittle hair, and hypodontia or malformed teeth. It does not feature the staged skin lesions seen in IP or CNS involvement.

Is the treatment of choice for cradle cap reassurance or treat?

It can resolve without intervention, but a mild steroid or topical antifungal such as ketoconazole shampoo can also be used.

With erythema multiforme, would the vignette almost always include involvement of the feet and hands?

Erythema multiforme is typically characterized by involvement of the hands and feet, but there are atypical cases where these areas might not be affected initially or at all. For the boards, though, the hands and feet are likely to be mentioned in the vignette since they'll focus on more classic presentations.

Does you have a helpful mnemonic or memory aid for recalling the key clinical features of McCune-Albright Syndrome?

You can thank PBR's AI "Memory Buddy" for this!

Mnemonic: "MACule Albright's PAC"

M for Macules: Think of irregular café-au-lait macules spreading like confetti.
A for Advanced Adolescence: Imagine Albright hosting an early teenage party (Precocious Puberty).
C for Cracked Bones: Picture bones cracking like party poppers, representing fibrous dysplasia.
Breakdown:

Macules: Relate "MAC" to the unique skin patterns.
Advanced Adolescence: Connect "A" to early puberty.
Cracked Bones: Tie "C" to the bone fractures and deformities.

CH. 7 – NEONATOLOGY

Page 170 says term infants are 37–42 weeks gestation and post-term infants are >42 weeks, but page 173 says that full-term infants are 37 weeks to 41 weeks and 6 days gestation. Which is correct?

Full-term is up to 41 weeks and 6 days (41 6/7), and ≥42 weeks is considered post-term. Use 42 weeks as a simple cutoff to define post-term infants. There may have been a discrepancy in an older edition of the study guide, but the current CSG is correct.

Could you please explain the mechanisms behind Rh and ABO incompatibility and how they contribute to neonatal jaundice? Additionally, could you clarify the role of the direct antiglobulin test (DAT) in evaluating these conditions?

ABO: When an O blood type mother has a fetus with A or B blood type, naturally occurring antibodies can attack fetal red blood cells, causing jaundice—even in the first pregnancy.

Rh: When an Rh-negative mother has an Rh-positive baby (from an Rh-positive father), the first pregnancy is typically unaffected. During delivery, fetal RBCs enter the maternal circulation, sensitizing the mother. In subsequent pregnancies, maternal anti-Rh antibodies can attack fetal RBCs, causing hemolysis and jaundice.

DAT: The direct Coombs test detects antibody-coated RBCs in the baby, which are present in Rh and ABO incompatibility. A positive DAT indicates a higher risk of hyperbilirubinemia, but a negative DAT does not rule it out.

Can we talk about when to start phototherapy depending on the age of child and risk factors with different levels of hyperbilirubinemia?

Phototherapy should be considered if bilirubin is >8 mg/dL within the first 24 hours. A cord bilirubin >5 mg/dL can also signal risk. Use the Bhutani curve and bilirubin nomograms, which account for age, hemolytic disease, sepsis, and clinical instability.

CH. 8 – DEVELOPMENTAL MILESTONES

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, would this count as a specific name? Or would they actually have to be using the word bottle? I’m having trouble with a question about whether an infant who says “mama”, “dada”, and “baba” (for bottle) is more likely to be 12 months old or 15 months old.

Yes, “baba” for “bottle” counts as a specific word at 12 months, as long as the child uses it consistently to refer to the same object. At this age, perfect pronunciation is not expected. A 12-month-old should be able to say “mama,” “dada,” and one other specific word. So if a child is saying “mama,” “dada,” and

“baba,” that fits the 12-month milestone. At 15 months, we expect one to two additional words beyond “mama” and “dada,” so this child is more likely to be 12 months old.

Have you received any feedback confirming whether the updated developmental milestones were included on the last pediatric board exam? And do you know which question banks are the most up to date? It's been incredibly frustrating to have dedicated so much energy into unlearning the old milestones and learning the new ones but get questions wrong because they're not updated. Often, the correct answer is still in gray areas according to previous milestones. Do you have any advice on how to attack these kinds of questions?

Great questions, and I completely understand the frustration. I don’t have specific feedback, but I’d be shocked if the ABP isn’t using the new milestone updates by now. In terms of which question banks are most updated, you'd have to ask them and take their answers with a grain of salt. Even if they say they update the questions every year, they may only do a small subset. The 2022 CDC updates were designed to reflect milestones that at least 75 percent of children are expected to achieve at a given age.

I’ve noticed some inconsistencies between question banks and the PBR book when it comes to the age cutoffs for developmental milestones. I’m planning to stick with the book’s milestones and move on. What’s your take on that approach?

That’s an EXCELLENT plan. As you’ve probably seen, there’s variation across resources because different tools and question banks often rely on different OUTDATED data sets. The PBR materials were updated aggressively and are aligned with the updated 2022 CDC and AAP surveillance milestones. Sticking to the one trusted source will help you stay consistent, reduce confusion, and get more questions on the actual BOARDS correct! PEARL: If you ever come across a question that feels like it’s testing the “old way,” just anchor your age to the milestone that reflects the most advanced skill listed and reason backward from there.

When it comes to speech and language development, does a child need to meet the milestone exactly? For instance, if a 4-year-old is 97 percent intelligible instead of 100 percent, should that be considered a speech delay requiring referral?

Not necessarily. Milestone expectations aren’t absolute cutoffs but rather surveillance guidelines. At 4 years, a child is expected to be 100 percent intelligible to strangers, but if a child is speaking clearly enough to be understood 97 percent of the time, that’s generally within a normal range unless there are other concerns. But context matters. If the child is also meeting other language and social milestones, there may not be a need for immediate referral. However, if there are additional red flags (such as frustration with communication, regression, or issues with social interaction), then further screening or referral would be appropriate.

How should milestone clues be interpreted when they suggest both lower and higher age-level skills in a vignette?

When a child in a vignette demonstrates some age-appropriate milestones and one more advanced milestone, consider choosing the older age and interpreting the rest of the information as red flags or developmental delay. The ABP is more likely testing whether you can recognize a delay than whether you

can spot precocious development. This reasoning strategy can help you choose the best answer in cases with mixed developmental signals.

CH. 9 – EMERGENCY MEDICINE & TOXICOLOGY

How can learners better approach mastering toxicology for the boards?

The key to mastering toxicology is memorization. Focus on:
- Sympathetic vs parasympathetic signs
- Vital signs, pupil response, and mental status changes by drug class
- Knowing antidotes and treatment cutoffs

These questions are straightforward if you know the patterns cold.

CH. 10 – VITAMIN & NUTRITIONAL DISORDERS

What are common presentations and lab clues for diagnosing different types of vitamin D deficiency?

Vitamin D deficiency can result from gut malabsorption, liver dysfunction, or renal disease. Some key exam clues include rachitic rosary, delayed fontanelle closure, and altered calcium and phosphorus levels. Gut-related issues affect 25-hydroxy vitamin D, while kidney-related problems affect 1,25-dihydroxy vitamin D. Memorizing the expected lab patterns for each cause is essential for board questions.

How does zinc deficiency typically present, and what clinical settings increase risk?

Zinc deficiency may mimic eczema or atopic dermatitis but often localizes around the mouth and diaper area. It's commonly seen in children receiving TPN without adequate zinc supplementation. The inherited form, acrodermatitis enteropathica, is rare. Key clues on boards include periorificial rash and history of TPN or malnutrition.

What are distinguishing features between kwashiorkor and marasmus?

The key distinction between kwashiorkor and marasmus is the presence of edema, which is characteristic of kwashiorkor. Marasmus presents with severe wasting but no edema. This is a classic board topic.

What clinical features point toward essential fatty acid deficiency?

Essential fatty acid deficiency often presents with scaly skin rashes resembling atopic dermatitis and is frequently seen in infants or children with short gut syndrome or prolonged TPN use. The hallmark distinguishing feature is thrombocytopenia. This combination is a high-yield clue for boards.

CH. 11 – GASTROENTEROLOGY

How do you differentiate between gallbladder hydrops and acalculous cholecystitis?

Gallbladder hydrops and acalculous cholecystitis can present with similar clinical symptoms—right upper quadrant pain, tenderness, nausea, and vomiting. The key differentiator lies in imaging. Acalculous cholecystitis, by definition, includes inflammation, which appears as gallbladder wall thickening on ultrasound. Hydrops lacks this inflammatory change and is more commonly seen in conditions like Kawasaki disease or ITP. Ultrasound findings are essential for distinguishing between the two.

CH. 12 – PHARMACOLOGY & DRUG PEARLS

Can you explain zero vs first-order kinetics, using a drug example?

For the boards, this feels too deep and is unlikely to be tested. We recommend not focusing on this area if it's going to take time away from other, higher-yield topics.

Any suggestions for how to remember contraindicated drugs for breastfeeding mothers?

This topic is relatively low yield, but it comes down to rote memorization. Creating a list to memorize or using PBR's "Memory Buddy" mnemonic creator are some ideas. Hope that helps!

Is diazepam safe during breastfeeding?

Diazepam is not contraindicated during breastfeeding but should be used with caution. Because it crosses into breast milk, it can lead to excessive sedation, respiratory depression, and lethargy in infants. Pediatricians should suspect medication exposure if a previously alert newborn suddenly becomes lethargic or hypoxic after breastfeeding from a mother taking diazepam.

CH. 13 – OPHTHALMOLOGY

How would you differentiate between cataracts and glaucoma on an eye exam?

Glaucoma and cataract can both cause vision problems, but the key to telling them apart is focusing on their differences. While they may share symptoms like poor visual tracking or an abnormal red reflex, glaucoma in infants often presents with excessive tearing, light sensitivity (photophobia), frequent blinking, and enlarged or cloudy corneas due to increased eye pressure. In contrast, cataracts typically cause a white or opaque red reflex and lack of visual fixation, but without tearing or photophobia. Cataracts don't affect eye pressure or corneal clarity. So, when faced with overlapping symptoms, think about eye pressure signs (glaucoma) versus lens opacity without tearing (cataract).

CH. 14 – GENETICS & INHERITED DISEASES

What are some easy ways that we can differentiate between X-linked dominant and X-linked recessive inheritance patterns?

Anytime you see the word “dominant,” it should be present in all generations. In X-linked dominant patterns, affected fathers pass it on to all daughters but not sons, making it more common in females. In contrast, X-linked recessive (XLR) is usually passed by a carrier mother to sons, while an affected father cannot pass it to his sons. This makes XLR more common in males and allows it to skip generations. Use a pedigree to assess generational patterns—if it affects all generations, it's likely dominant.

I thought the most common type of Alport syndrome is X-linked recessive, but page 258 of the core study guide says it is X-linked dominant. Could you clarify which is correct?

X-linked dominant is probably the most common, but since there are many different inheritance patterns for Alport syndrome, the board exams are unlikely to test this beyond the fact that the most common form is X-linked. Focus on recognizing the X-linked inheritance pattern, particularly in affected males presenting with hearing loss and renal symptoms.

CH. 16 – INFECTIOUS DISEASES

If a child develops erythema multiforme after receiving a vaccine, does this contraindicate completing the vaccine series? Specifically, if the reaction occurred after the first dose of the MMR vaccine and the child lives in a community with increased measles risk, how should this affect the decision?

Erythema multiforme is not listed in the Red Book as a side effect of the MMR vaccine and is not considered an absolute contraindication to continuing the vaccine series. Even if it occurred after a prior dose, the vaccine series should be completed, particularly in a high-risk community. This reaction is not likely to appear on the pediatric boards.

What are the key features and diagnostic methods for West Nile Virus infection?

West Nile Virus infection is asymptomatic in about 80% of cases. Transmission is primarily via mosquito bites, with rare cases via organ transplantation or blood transfusion. The most reliable method for diagnosing encephalitis due to West Nile Virus is detecting IgM in the CSF. Serum IgM may support the diagnosis but is less definitive.

What other infectious or non-infectious causes of draining lymphadenopathy should be considered beyond Bartonella, Brucellosis, and Yersinia pestis?

In addition to Bartonella, Brucellosis, and Yersinia pestis, other infectious causes of draining or suppurative lymphadenopathy include:

- **Staphylococcus aureus and Group A Streptococcus** – These can cause local skin infections leading to reactive and sometimes suppurative regional nodes.

- **Mycobacterium tuberculosis** – Can cause scrofula, especially in the cervical nodes, with caseating granulomas and possible drainage.
- **Atypical mycobacteria (especially Mycobacterium avium complex)** – More commonly seen in young children and immunocompromised individuals.
- **Francisella tularensis (Tularemia)** – Often causes ulceroglandular disease with painful lymphadenopathy that can become suppurative.
- **Sporothrix schenckii (Sporotrichosis)** – A fungal infection that follows lymphatic channels and may present with ulcerated lesions and draining lymph nodes.
- **Actinomyces israelii** – Causes chronic granulomatous infection that can lead to abscesses with sinus tract formation (e.g., in cervicofacial disease).
- **Chlamydia trachomatis (LGV serovars)** – In sexually transmitted lymphogranuloma venereum, painful inguinal nodes may coalesce and rupture.

While viral causes (e.g., EBV, CMV) can cause significant lymphadenopathy, they do not typically drain. Drainage usually implies suppuration, which points more toward bacterial, fungal, or mycobacterial causes.

CH. 17 – VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

If a bat is found in a room with a child who has no visible bite or scratch marks, and the family is vacationing in Hawaii, should rabies immune globulin and the rabies vaccine series be administered? Additionally, if the child in Hawaii has visible bat bite or scratch marks, would post-exposure prophylaxis be indicated in that case?

Rabies prophylaxis decisions can be tricky, especially when geographic factors like being in Hawaii are involved.

Hawaii is officially rabies-free, and indigenous bats do not carry the rabies virus. Therefore, in the absence of any bite or scratch marks, and if the bat exposure occurred exclusively within Hawaii, you would not administer rabies immune globulin or the vaccine series, per CDC and Hawaii Department of Health guidelines.

However, if there are visible bite or scratch marks, even in Hawaii, the decision becomes more nuanced. While Hawaii remains rabies-free, any potential exposure through skin breaches (e.g., from a bat of uncertain origin) may warrant treatment depending on the clinical context and whether there's concern the bat may have entered the state via transport or cargo. In practice, many clinicians would still consult local health authorities before initiating rabies post-exposure prophylaxis in such cases, but err on the side of caution and treat if there's any uncertainty or high-risk exposure.

In contrast, if the same scenario took place in the continental U.S., PEP would be strongly recommended, even without bite marks, due to the risk of unrecognized bat bites and the near-universally fatal nature of rabies once symptoms appear.

What are the current recommendations regarding vaccines that contain gelatin for patients who observe halal or kosher dietary practices?

In brief, gelatin used in vaccines—typically of porcine origin—is highly purified and hydrolyzed, transforming its chemical properties significantly—so much so that some religious authorities consider it permissible when administered as a non-oral form, such as injection or nasal spray.

The UK Health Security Agency (UKHSA), having consulted with Jewish halachic experts, notes there is no issue under Jewish law with porcine-derived ingredients in non-oral products like vaccines. Similarly, some Muslim scholars have ruled that such highly processed gelatin does not break dietary laws, considering both its purification and route of administration.

However, it is important to acknowledge that views differ within both communities: while many accept these vaccines, others may opt to avoid them unless no safe alternative exists. When available, acceptable alternatives should be offered—for example, injectable flu vaccines (gelatin-free) instead of nasal spray.

Can you please review post-exposure prophylaxis and when it is and isn't indicated? Do you have any ways to help us remember this a little better?

PEP is indicated only when there's a risk of serious infection and a method to prevent it exists. In pediatrics, the main organisms you should focus on are:

- N. meningitidis – Rifampin or ceftriaxone for close/intimate contacts, even after just one confirmed case.
- H. influenzae type B (Hib) – Rifampin for household contacts only if there's a child <4 who is not fully immunized, or someone is immunocompromised. For school/daycare, wait until two confirmed cases.
- Pertussis – Macrolide for all close contacts, especially high-risk groups (infants, pregnant women, people with chronic disease)
- Hepatitis A – Give the vaccine (or immunoglobulin for <12 months or immunocompromised) within 2 weeks of exposure.
- Hepatitis B – Depends on immunization status. If unvaccinated or unknown immunity, give HBIG + start the vaccine series.
- Varicella – Give varicella immunoglobulin to high-risk exposed individuals (e.g., pregnant, immunocompromised, certain infants).
- Measles
    - <6 months: give IGIM.
    - 6–11 months: IGIM or MMR.
    - ≥12 months: MMR if not yet given.
    - Pregnant or immunocompromised: IVIG if not immune.

Memory tip:

Think "HIP MVM" (Hib, Influenzae, Pertussis, Meningococcus, Varicella, Measles) — these are your core pathogens. For each, ask:

- Is the exposed person at risk?
- Is there something we can do to prevent disease (vaccine or IG)?

On page 340, is the adenovirus vaccine considered a live vaccine? Also, could you provide a comprehensive list of live vaccines—both currently used and those historically administered—including MMR, varicella, yellow fever, intranasal influenza, rotavirus, oral polio vaccine (OPV), and adenovirus?

Yes, the adenovirus vaccine is considered a live vaccine, but it's important to note that it is only administered to military personnel and is not part of the routine pediatric schedule. It's typically not tested on pediatric board exams unless you're specifically told it's a military patient.

Here's a comprehensive list of live vaccines, broken into currently used and historically used categories:

Currently Used Live Vaccines (U.S.)

- MMR (Measles, Mumps, Rubella)
- Varicella (VZV)
- Rotavirus (oral)
- Intranasal Influenza (LAIV, aka FluMist)
- Yellow Fever (used in travel medicine, not routine for all children)

Live Vaccines with Limited/Specific Use

- Adenovirus (oral, for military use only)

Historically Used Live Vaccines (No Longer Routine in the U.S.)

- Oral Polio Vaccine (OPV / Sabin vaccine) – replaced by inactivated polio vaccine (IPV) due to risk of vaccine-associated paralytic polio.

Memory Aid: Use the mnemonic "MR. FARM-SPY is ALIVE" to remember the key live vaccines:

- Measles
- Rubella
- Flu (intranasal)
- Adenovirus
- Rotavirus
- Mumps
- Sabin (OPV)
- Pox viruses (Varicella)
- Yellow Fever

Keep in mind that live vaccines are contraindicated in pregnancy and severely immunocompromised patients.

CH. 18 – INBORN ERRORS OF METABOLISM

Could you clarify what the tissue levels of copper are in Wilson disease and Menkes syndrome (page 361)?

In Wilson disease, tissue copper levels are elevated due to a defect in the ATP7B gene, which prevents proper copper excretion through bile. As a result, copper accumulates in the liver, brain, and cornea, leading to progressive hepatic and neurologic damage. In contrast, Menkes syndrome involves a defect in the ATP7A gene, which impairs copper transport from the intestines into circulation. This leads to high tissue copper levels in certain cells, but because copper cannot reach where it's needed (e.g., brain, enzymes), the body experiences a functional copper deficiency. While both conditions show low serum copper and ceruloplasmin, Wilson reflects toxic overload, whereas Menkes reflects inaccessible copper, resulting in systemic deficiency.

What kinds of board-style clues help differentiate Wilson disease from Menkes syndrome?

Wilson disease clues include signs of hepatic failure (perhaps esophageal varices might be mentioned instead of hepatomegaly) or Kayser-Fleischer rings, while Menkes clues may involve a male-only presentation or pedigree suggesting X-linked inheritance. Serum copper and ceruloplasmin are low in both; tissue copper is elevated but due to different mechanisms.

Could you explain the role of carnitine in the management of organic acidemias?

Carnitine is used in organic acidemias because it binds to toxic organic acids and forms water-soluble acylcarnitines that can be excreted in the urine. This helps reduce the acid burden in the body and prevents further metabolic decompensation. Patients often become carnitine-deficient due to rapid loss in the urine, so supplementation is essential. It's found naturally in red meat and dairy, but is also given as L-carnitine supplements in medical treatment.

What is the importance of the chart at the beginning of the Inborn Errors of Metabolism chapter?

The chart at the beginning of the chapter is critical. Feedback on the importance of that chart has been shared by PBR members year after year. It summarizes key features of inborn errors (lab patterns, treatment, clues, etc.) and is crucial. Mastery of this chart allows rapid recognition of high-yield conditions and families of conditions.

CH. 19 – ACID-BASE DISORDERS

I'm having some difficulty understanding the example in the Core Manual regarding how to adjust the anion gap based on albumin levels. It states that the upper limit of normal for the anion gap decreases by 2.5 mEq/L for every 1 g/dL drop in albumin below 4 g/dL. Could you walk us through what this means using a patient example—for instance, if the albumin is 2.0 g/dL? That would really help clarify the concept.

Sure! A normal anion gap is 12. A normal albumin level is 4 g/dL. The adjustment rule is to subtract 2.5 from that normal value for every 1 g/dL decrease in albumin below 4.

Assume a patient's albumin is 2 g/dL. That's 2 g/dL below normal. So, we multiply 2 by 2.5, which gives

us 5. The normal upper limit of the anion gap is normally 12, but we reduce it by 5. This means an anion gap of 8 or higher (not 13 or higher) now represents an anion gap metabolic acidosis.

If a patient has an overarching respiratory alkalosis, how do we calculate the expected metabolic compensation? Also, if there are any metabolic issues, how do we identify those?

If the overarching acid-base disorder is a respiratory alkalosis, the first thing we want to do is calculate the expected metabolic compensation. This helps us figure out whether the bicarbonate level we're seeing is appropriate or if there might be another acid-base process happening.

We start by assuming a normal pCO_2 is 40 mmHg and a normal bicarbonate (HCO_3^-) is 24 mEq/L. Then, based on how much the pCO_2 has decreased from the baseline, we apply the compensation rules from the table.

Acute	R. Acidosis (**A**cid)	For every 10	↑	of pCO2	↑	HCO3 by	1
	R. Alkalosis (Base)	For every 10	↓	of pCO2	↓	HCO3 by	2
Chronic	R. Acidosis (Acid)	For every 10	↑	of pCO2	↑	HCO3 by	**3.5**
	R. Alkalosis (Base)	For every 10	↓	of pCO2	↓	HCO3 by	**5**

We can use an acute respiratory alkalosis for this example, which has the following rule: For every 10 mmHg drop in pCO_2, the HCO_3^- should decrease by 2 mEq/L.

- If pCO_2 drops from 40 to 30, that's a 10-point drop.
  → Bicarb should fall by 2, so expected HCO_3^- = 24 − 2 = 22.

- If pCO_2 drops from 40 to 25, that's a 15-point drop.
  → That's 1.5 intervals of 10, so 1.5 × 2 = 3.
  → Expected HCO_3^- = 24 − 3 = 21.

If your patient has a bicarb level that's lower than expected, it suggests there's an additional metabolic acidosis present. If the bicarb is higher than expected, there could be a concurrent metabolic alkalosis.

So, in summary, calculate expected bicarb using the compensation rules, then compare that to what you see on the labs. If the numbers don't match, there's a second acid-base disturbance hiding underneath. Keep in mind that if there's an additional metabolic acidosis present, you will not know if it's a gap or non-gap metabolic acidosis until you calculate the gap. ALWAYS calculate the gap!

In terms of looking for other metabolic disorders, look for incomplete or overcompensation, look for an anion gap, and memorize the shortcuts in the book to help you quickly identify other acid-base disorders.

CH. 21 – NEPHROLOGY

Can you explain the management of the first febrile UTI in a newborn or children younger than 2 years of age?

In children aged 2 months to 2 years with a first febrile UTI, treatment should include a 5-day course of a first-generation cephalosporin (e.g., cephalexin) and a renal/bladder ultrasound to assess for structural problems or hydronephrosis. A VCUG is not indicated after the first UTI unless imaging reveals structural abnormalities or if the UTI recurs.

CH. 23 – NEUROLOGY

Is the word "benign" in benign childhood epilepsy with centrotemporal spikes a misnomer?

While it's benign for most children in that they do not have long-lasting complications, it's associated with cognitive deficits, language impairment, behavioral problems, and even rare sudden unexpected death. So yes, it does feel like a potential misnomer.

How do you differentiate spinal muscular dystrophy and Duchenne muscular dystrophy?

To differentiate Duchenne muscular dystrophy (DMD) and spinal muscular atrophy (SMA), focus on CK levels (markedly elevated in DMD, normal/slightly elevated in SMA), age of onset (DMD: 2–5 years; SMA: often <1 year), and key clinical findings (Gower's maneuver and calf pseudohypertrophy in DMD vs. tongue fasciculations and hypotonia in SMA). Also, DMD has a longer course with progressive loss of ambulation, while most infants with SMA don't live past 2 years.

Can you summarize how to diagnose migraine headaches?

Migraine is a diagnosis of exclusion. It comes down to history and physical exam. Start with a migraine diary. Look for key triggers and typical features such as unilateral pain, photophobia, phonophobia, nausea, vomiting, auras, and relief with darkness or sleep. It can last hours or even days. If there's anything questionable in the history or focal neuro deficits, start with a head CT. Triptans are effective for moderate to severe attacks, after trying acetaminophen or NSAIDs.

What are neonatal seizures and how are they evaluated?

Neonatal seizures can be subtle, with brief inactivity or odd facial movements, like lip smacking or staring. If it happens within the first 24 hours of birth, it's usually due to some asphyxia event, but in that age group, a good blood and urine workup to check for a metabolic disease or abnormality such as hypoglycemia or hypocalcemia. EEG is also an important part of the workup.

CH. 24 – ORTHOPEDICS & SPORTS MEDICINE

Our book emphasizes the importance of always performing joint aspiration to avoid missing a septic joint. However, in a 2025 AAP PREP question, the correct answer was prescribing a nonsteroidal anti-inflammatory medication rather than consulting orthopedic surgery for joint aspiration and fluid analysis. Could you please clarify the reasoning and provide guidance on how we should approach similar questions in the future? How should we decide when joint aspiration is essential versus when conservative management is appropriate?

While transient synovitis may be managed conservatively with NSAIDs, septic arthritis is a dangerous condition that requires prompt diagnosis via joint aspiration. Since the details of the question stem weren't provided, the assumption is that it described a case of transient synovitis (e.g., afebrile child, weight-bearing, normal labs). The Kocher criteria (non–weight-bearing, temp >38.5°C, ESR >40, WBC >12K) help differentiate septic arthritis from benign conditions. The more Kocher criteria are met, the more likely that septic arthritis is, and joint aspiration is the correct board answer. Conservative management (NSAIDs) is only appropriate once septic arthritis has been reasonably excluded. When in doubt, aspirate.

Could you clarify the guidance on imaging for developmental dysplasia of the hip (DDH)? The teaching point mentioned using ultrasound from 6 weeks to 6 months and X-ray after 4 months. What is the preferred imaging modality for infants between 4 and 6 months of age—ultrasound or X-ray?

By about 4 to 5 or 6 months, the femoral head begins to ossify, making X-ray the most appropriate diagnostic tool. The American College of Radiology Appropriateness Criteria states that for infants between 4 to 6 months with suspected DDH, pelvic radiographs are "usually appropriate", while hip ultrasound "may be appropriate" but is not the preferred initial study. So between 4 and 6 months of age, if DDH is suspected, X-ray is the preferred imaging modality, but ultrasound still has a limited role.

CH. 26 – PULMONOLOGY

Could you please provide a review of asthma management in preparation for our upcoming exam? In particular, I would appreciate a detailed explanation of SMART (Single Maintenance and Reliever Therapy). I'm looking to understand which combination of inhaled corticosteroid (ICS) and long-acting beta agonist (LABA) is used in SMART, what ICS dosing level (low, medium, or high) is typically recommended, which patient populations are considered eligible for this approach, and what age groups are currently approved for SMART therapy.

The 2025 PBR chapter on Pulmonology includes updated asthma management guidance aligned with the 2020 asthma treatment guidelines, and it does recommend the use of a single ICS/LABA inhaler for moderate persistent asthma. This is the same regimen now referred to in guidelines and clinical practice as SMART therapy (Single Maintenance and Reliever Therapy). The chapter currently does not include the term "SMART," but the strategy itself is correctly described.

SMART therapy involves using budesonide-formoterol as both the daily controller and as-needed reliever inhaler. Formoterol is the only LABA with a rapid onset of action, which allows it to double as a

reliever—making it unique among LABAs. In SMART, a low to medium dose of ICS is typically used; high doses are not the standard due to increased side effects with limited added benefit. This approach is recommended for patients with moderate to severe asthma (generally GINA step 3 or higher) who are not well-controlled on low-dose ICS or ICS-LABA therapy alone.

As of this writing, SMART is FDA-approved for patients aged 12 and up in the United States, though some global guidelines and regulatory agencies may support its use in children as young as 4.

We've updated the CSG with this additional information.

CH. 30 – PEDIATRIC LAB VALUES

Will we be expected to know the normal ranges for urine studies on the pediatric board exam?

According to the ABP, "most exam questions are preceded by a clinical stem that provides information about a patient, including laboratory and diagnostic findings." While they expect familiarity with common lab values, for less common labs, they typically provide either a qualitative or quantitative reference to indicate whether a value is low, normal, or high.

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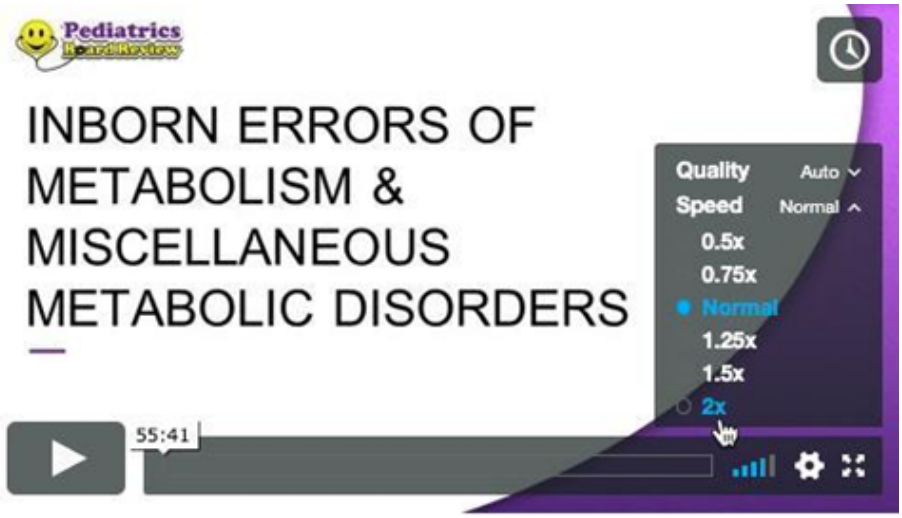

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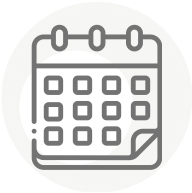

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