Pediatrics Board Review

2022 Corrections and Clarifications Guide

12th Edition
Your EFFICIENCY BLUEPRINT to Passing The Pediatric Boards

2022 EDITION

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Written by Ashish Goyal, MD

PBR'S ANNUAL CORRECTIONS AND CLARIFICATIONS GUIDE

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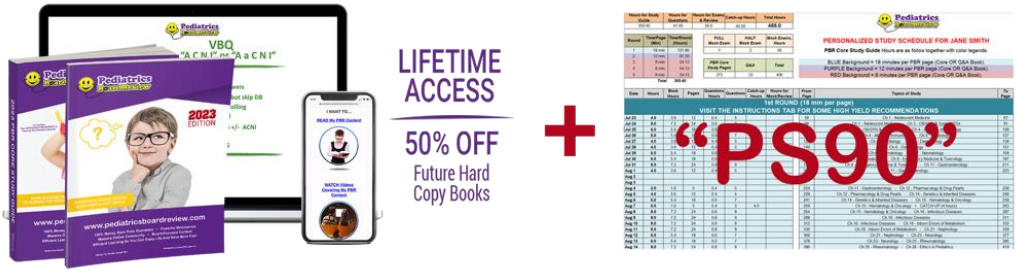

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A FEW WORDS OF THANKS TO THE PBR COMMUNITY

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

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

THANKS TO YOU!

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

   www.pediatricsboardreview.com/error

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

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

   www.pediatricsboardreview.com/badlink

3. An absolutely MASSIVE THANKS TO DR. 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. Marie Jose Moubarak
   - 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 Facebook CREW!** Yes… We kind of "stalk" the group and if I see something comes up that might warrant a correction in the PBR. I set it aside for this time of year to review.
4. **Requests for content clarification through the portal or "The CREW"**. In general, the "PBR Facebook CREW!" is meant to help you get the help you need to understand a topic. BUT, if I see that there's a topic that could be explained *better* based on CREW conversation, I make a note of it and try to polish it up for the next edition and address the issue in this guide.

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

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

ALL study guides have errors! I'm simply the only author who is crazy enough, and passionate enough, to take on something like this prior 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 ACTIVE discussion within the members' only PBR Facebook CREW! about board review topics that I THOUGHT were explained well within the PBR. That

discussion leads me to believe that I can be EVEN MORE clear in future editions. There will be many additional clarifications and updates in the next edition.

4. **COST (No... I'm not just talking about money!)**
    - By cost, I mean money and opportunity cost. The cost of a new book is minimal compared to the hard **financial cost** and **opportunity cost** of FAILING the boards. The financial cost of FAILING includes over $2000 for your board fees, plus the cost of taking time off of work to study again next year (THOUSANDS of dollars of lost income). You also must include the stress and the time away from loved ones as a tremendous unmeasurable cost.
    - If you're planning on using the older version due to financial concerns, that's actually pretty silly. As your guide on this journey, I feel that it's important that I be blunt when it comes this point. I have such a passion for efficiency and QUALITY USE OF TIME that it really **pains** me to hear about physicians that are trying to go back and forth between the corrections guide and their old study guide in order to save a few dollars. Plus, having a NEW and CLEAN book that you can start going through with my highlighter trick is a much better means of achieving DEEP STUDY.
5. **REFERENCES TO PBR IN THE CREW!**
    - The PBR Facebook CREW! comes alive with discussion as the boards approach. Many PBR alumni have said that the Facebook CREW! heavily contributed to their success on the boards. When your peers in "The CREW" are referring to a topic on a certain page, do you really want to (again) waste your precious time fumbling around and trying to find the topic they're referring to?
6. **UPGRADED FORMATS**: Every edition is MUCH better than the previous.
    - **Corrections**
    - **Clarifications**
    - **New image links**
    - **New, Timesaving Innovations**. For example, our links used to be EXTREMELY long. Now we have a system that turns http://upload.wikimedia.org/wikipedia/commons/4/45/Aphthous_ulcer.jpg into something easy like www.pbrlinks.com/aphthous1. **HOW COOL IS THAT! Try typing out the 2 different links and see HOW FAST you get to review images using the new PBR link ☺ - these things get me SO EXCITED!**

DISCLAIMERS/WARNINGS

PLEASE READ THIS BEFORE YOU GET STARTED

- The **page numbers** in this guide refer to the **2022 Editions of the Pediatrics Board Review** books (covers shown below).

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

www.pediatricsboardreview.com/ERROR

LET'S GET STARTED WITH THE CORRECTIONS FIRST!

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

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

ADOLESCENT MEDICINE

For male development, the mnemonic TAP Her says adrenarche occurs before penile enlargement, but per the chart on the first page of the chapter, it seems penile enlargement (SMR 3) occurs before axillary hair(SMR 4)

Thanks for letting us know! In boys, testicular enlargement ALWAYS should come first in pubertal development.. In some males, the second step is penile enlargement, followed by axillary hair development. In others, the second step is axillary hair development, followed by penile enlargement. So the TAP mnemonic doesn't always apply. See below for our latest Core Study Guide content on this topic.

PUBERTY

NOTES: Please note that there is a great deal of overlap and repetition between the puberty section and the Endocrinology section.

* Know conversion from inches to centimeters. 1 inch is about 2.5 cm!

* Sexual Maturity Ratings (SMR) and Tanner Staging begins with ONE. There's NO ZERO. Tanner/SMR 1 = Prepubertal

* **Experts disagree regarding some SMR descriptions.** They definitely can't agree on the age at which Delayed Puberty is diagnosed. Don't stress! Questions on the exam should be fairly clear.

NORMAL PUBERTY TIMELINE

SMR	Girls	Boys
Limits	Delayed Puberty: 13 – 14 yo Precocious Puberty: 2° signs before 8 yo	Delayed Puberty: 14 – 15 yo Precocious Puberty: 2° signs before 9 yo
1	**Basal growth at 5–6 cm/yr**, boyish chest (papilla elevation only), no hair	<4 ml volume or <2.5 cm diameter of testicle, no hair, baby penis, **basal rate of 5–6 cm/yr**, no hair
2	**Accelerated growth at 7–8 cm/yr**, a breast bud is the 1st sign of puberty (palpable, areola enlarges), Hair only along the labia (coarse)	**>4 ml or >2.5 cm** (this is the 1st sign of puberty), hair at base of penis. Penis *may* start to enlarge (usually at SMR 3)

3	**PEAK ht velocity of 8–10 cm/yr**, elevation of breast contour, areola enlarges, curly hair at pubis, axillary hair begins, acne. This stage is similar to a boy's SMR 3 + 4 combined. "Imagine a girl sitting on a 3-LEGGED STOOL crying because she has hair in her armpit and now has acne!"	Accelerated vertical (and penile) growth >12 ml/3.5 cm, Gynecomastia in 50% of boys 10–16 yo, resolve in 3 yrs), CURLY hair at pubis. "Think about the 3 Stooges. They all had funny pubertal voices, and the fat one had BOOBS/GYNECOMASTIA and was named CURLY."
4	**Mound on mound**, enlarged areola. Dense hair, none at the thigh. Menses usually occurs around SMR 3 or 4.	**PEAK height velocity at 10 cm/yr**, no thigh hair, develops **AX**illary hair, acne, and body odor. "Teenage boy with raging hormones is pissed about acne & hair so takes an **AX** to his **4 DOOR CAR** (SMR **4**) which explodes and burns his hair!"
5	Stop growing at about 16 yo, areola recesses to general contour of breast and the **breasts again look like Tanner 3**	>4.5 cm penis, thigh hair, stop growing at ~17 or 18, +facial hair at sides, no more gynecomastia

NORMAL PUBERTY PEARLS

Here are some great pearls and shortcuts about normal puberty.

* Girls have adult-looking breasts in SMR 3 and 5.

* SMR 4 = mound on mound breasts

* SMR 2 to 5 usually lasts about 3 to 5 years in total duration for both sexes.

* MENARCHE usually occurs in SMR 3 or SMR 4 OR, within 2–3 YEARS of the onset of puberty.

* MENSES/HEIGHT: At the onset of menses, girls are probably within 1–2 inches (2.5–5 cm) of their adult height. Why do I say that? Because they're probably in SMR 4 (which occurs **after** the peak height velocity).

* VAGINAL BLEEDING: Bloody vaginal discharge while in SMR 2 shouldn't happen. Consider a foreign body (e.g., toilet paper) in your differential.

HEIGHT

For the test, pre-pubertal basal rate for height in both boys and girls is 5–6 cm/year. The peak is 10 cm/yr. Early puberty results in shorter adult height.

GROWTH SPURTS

Elevated alkaline phosphatase can be normal during growth spurts. Hematocrit increases alongside growth spurts.

THELARCHE, ADRENARCHE THEN MENARCHE

(THELARCHE) Breast development → (ADRENARCHE) Hair development → (MENARCHE) Menses

PEARLS AND MNEMONICS: "Girls are TAMer than boys." "Boys like to TAP Her!"

- Girls are "TAMer" = Thelarche, then Adrenarche, then Menarche = Breast development → Hair development → Menses. Thelarche = first sign of puberty – stage 2. Adrenarche is the same thing

as Pubarche. Breasts: Look most natural at SMR 1, 3, and 5. TAM = “Breasts are higher than Pubic hair which is higher than a Vagina.”

- Boys = “TAP Her” = Testicular enlargement, then Adrenarche, then Phallus/Penile enlargement, THEN Height velocity peaks. Keep in mind that while testicular enlargement is normally the first sign of puberty in males, Penile enlargement may begin to occur before Adrenarche.

Adolescent chapter: CAH

--(p.61) Under the CAH introduction: How does negative feedback result in high levels of ACTH being released from the pituitary glands? Should this read the lack of negative feedback?

Yes! You’re correct, so thanks for pointing this out.

In 21-hydroxylase deficiency, excessive amounts of 17-hydroxyprogesterone are released. The patient has low cortisol and aldosterone levels. ACTH responds to cortisol. If the cortisol level is low, then this is not sufficient to shut off the ACTH, so this is lack of a negative feedback. This causes the ACTH levels to be high. Below is our correction for the next Core Study Guide Edition.

CONGENITAL ADRENAL HYPERPLASIA (CAH) INTRO

In congenital adrenal hyperplasia (CAH), there is a cortisol and aldosterone manufacturing problem in the adrenal glands. The low cortisol levels result in an absence of negative feedback at the level of the pituitary glands, which results in high levels of ACTH being released → Results in an increase in cortisol precursors → Resulting in more ANDROGENS. It is diagnosed by measuring 17-hydroxyprogesterone (expect levels to be HIGH). Additional details on this topic are provided in the ENDOCRINOLOGY chapter under Congenital Adrenal Hyperplasia.

ENDOCRINOLOGY

There were no Endocrinology corrections for 2022!

OB/GYN & SOME STDS

For N gonorrhea tx in the PBR 2022 edition, page 94, second line, it should say DUAL therapy with ceftriaxone IM x 1 and PO azithromycin x 1. As it stands, it does not include the azithromycin part.

The gonorrhea treatment regimen in the Core Study Guide is accurate, but the chlamydia treatment regimen needed updating.

One dose of IM Ceftriaxone is first-line treatment for gonorrhea infection. Second-line treatment is gentamicin PLUS azithromycin.

First-line treatment for chlamydia trachomatis infection is doxycycline for 7 days. Second-line treatment is azithromycin x 1 dose, or levofloxacin for 7 days.

Treat gonorrhea and chlamydia co-infection with one dose of IM ceftriaxone PLUS doxycycline for 7 days. Below is our chlamydia update for your review and our gonorrhea section is also included as a refresher.

(DOUBLE TAKE) CHLAMYDIA TRACHOMATIS

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

* SEXUALLY TRANSMITTED DISEASE (STD): Treat with **DOXYCYCLINE for 7 days**. If the patient is given doxycycline and there is recurrence, treatment failure, a concern for noncompliance, or a doxycycline allergy, treat with AZITHROMYCIN x 1 dose, or LEVOFLOXACIN for 7 days. Test for other STDs including gonorrhea, syphilis, and HIV and treat if positive.

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

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

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

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

NEISSERIA GONORRHEA

Neisseria gonorrhea creates a smelly, greenish discharge. It is USUALLY asymptomatic, so consider this in the differential for any **adolescent patient with arthritis**. Treat uncomplicated infections in adolescents with IM ceftriaxone x 1. If allergic to ceftriaxone, treat with gentamin PLUS azithromycin. Test for other STDs including chlamydia, syphilis, and HIV and treat if positive. If chlamydia co-infection has not been excluded when the patient is diagnosed with gonorrhea, then also treat presumptively for chlamydia with PO doxycycline for 7 days.

* **MNEMONIC**: Gonorrhea is also known as “the CLAP” because on Gram stain the diplococci kind of look like two hands clapping. “C for the Clap, and C for Ceftriaxone x1.” If you are wondering about “GC,” it comes from “GonoCoccus.”

* DISSEMINATED GONORRHEA: Once the disease has disseminated, the local symptoms are no longer present! Look instead for a rash and joint involvement. It can also cause meningitis and endocarditis. This is an **INTRACELLULAR** diplococci. Fitz-Hugh/PID. When suspecting disseminated gonorrhea (e.g., single pustule + swollen knee), culture any pustules as well as all orifices.

* **PEARL**: Treatment regimen for chlamydia *and* gonorrhea co-infection is IM ceftriaxone x 1 plus PO doxycycline for 7 days.

ALLERGY & IMMUNOLOGY

Allergic Rhinitis: For Allergic rhinitis, First line treatment mentioned is oral anti-histamine(mild). But I think its Nasal corticosteroids as first line no matter the severity. I always ask this question as Allergist answers it differently from general pediatrician(as we would commonly use oral zyrtec). I am a first year A/I fellow and we discussed the most recent guidelines and per JACI 2020 publication it also mentioned nasal steroids are first line. But during our board review our other attending told us the same treatment mentioned in the PBR book. If we can clarify this from the peds boards side it would be really helpful. Thanks

You’re right! Nasal steroids are more effective, especially for nasal congestion. Below is our new update for the Core Study Guide:

HAY FEVER/ALLERGIC RHINITIS

Hay fever due to pollen typically takes a few years to develop. Although some young children can start to show signs of allergic rhinitis, if a child younger than 3 years of age presents with rhinitis, consider other diagnoses.

* **R**agweed frequently causes **R**espiratory symptoms along with the usual sneezing, allergic conjunctivitis, rhinitis, and tearing of hay fever.

* Treatment options include intranasal steroids (first-line therapy for symptomatic patients), oral antihistamines and intranasal antihistamines.

CARDIOLOGY

Pg 119: If the SA node only fires at 80 beats/min, how do newborns have normal HR from 110-160?

Excellent point! The SA node firing rate is AGE DEPENDENT. The younger the child is, the higher the SA node firing rate is. In adults, the SA node fires at 80 beats/minute. In adults, bradycardia is < 60 beats/minute, and tachycardia is >100 beats/minute. Please see the below table for your reference as well as the updated topic underneath it.

HEART RATE

AGE	RESTING (AWAKE)	RESTING (SLEEPING)
NEWBORN	100-180	80-160
1WK - 3MO	100-220	80-200
3MO - 2YR	80-150	70-120
2YR - 10YR	70-110	60-90
10YR - ADULT	55-90	50-90

SINOATRIAL NODE (SA NODE), ATRIOVENTRICULAR NODE (AV NODE) and VENTRICULAR INTRINSIC RATES

* The SA node firing rate is AGE DEPENDENT. The sinoatrial node intrinsically fires at about 80 beats/minute in adults, but the firing rate is higher the younger the child is.

MNEMONIC:

* The atrioventricular node (AV node) intrinsically fires at 60 beats/minute.

* The ventricle intrinsically fires at 40 beats/minute (so you could see a rate of 40 in a patient with 3rd degree AV Block).

MNEMONIC: The "S" of SA node looks like the "8" of 80 beats/minute: "8A node." From higher up to lower (SA to AV to Ventricle), the intrinsic rates decrease by 20 from 80 to 60 to 40 beats/minute. Keep "8A Node beating at 80 beats/minute" in mind, and you should be fine.

DERMATOLOGY

Dermatology Page 149

The links to images under dermoid cysts are incorrect. They are epidermal cysts and not dermoid cyts.

We checked, and you are correct. We will update our image links for these two topics in the next edition of the Core Study Guide. Here are 2 images for your quick review!

DERMOID CYSTS

Dermoid cysts are saclike growths present at birth. They are like teratomas in that they can contain hair and teeth. They are often associated with tufts or sinuses. They grow slowly and can get infected, so most of them should be REMOVED. Especially those in sensitive areas, including the face or nasal area. They will require imaging before removal.

IMAGE 1
IMAGE 2

NEONATOLOGY

Meds during breastfeeding – I'd like to double check if Diazepam is actually a contraindication b/c lactmed says to use diazepam cautiously during breastfeeding, but does not state it is a contraindication.

Same with metronidazole--just wanted to confirm it is NOT a contraindication to breastfeeding.

Yes! You are correct about both. Although diazepam is a longer-acting medication that some experts recommend using the "pump and dump" method with, it's not a contraindication. This will be corrected (we'll share the new version below). For metronidazole, it's also safe to use during breastfeeding. This will be filled under the CONTRAINDICATIONS TO BREASTFEEDING section on page 167.

DIAZEPAM

Diazepam is NOT contraindicated in breastfeeding, but it is used with caution. Diazepam gets secreted into breastmilk and can potentially cause babies to experience withdrawal or sedation.

METRONIDAZOLE

Metronidazole is active against parasites and anaerobic infections. It is often used for intra-abdominal infections. Some of the commonly treated organism include **G**iardia, **E**ntamoeba, **T**richomonas, **B**acteroides, **C**lostridium, and **G**ardnerella.

PEARL: For Trichomonas, it's a one-time dose. Nursing mothers should stop breastfeeding for 24 hours.

* CONTRAINDICATIONS TO BREASTFEEDING include herpes simplex virus (HSV) with lesions on the breast; HIV; active, untreated tuberculosis (TB); most recreational drugs; chemotherapy; sulfa drugs (in the first month of life); tetracycline; some psychotropic drugs; a number of other drugs. Breastfeeding is also usually contraindicated if the baby has classic galactose deficiency and may need to be modified if it has certain other **INBORN ERRORS OF METABOLISM**. An inverted nipple may be a contraindication depending on the degree of inversion. Nipple shields or breast shells may be needed.

DEVELOPMENTAL MILESTONES

Latest developmental milestones

I am a bit worried about the recent publication from Aap in feb 2022 with changes in cdc checklist for milestones. Like for 2 years old they do not mention the number of words or for a 4 year old they dont mention drawing cross or square anymore. Or raking at 9 month rather then 6 month. Do you think with these recent changes will they be testing us of these topics and what resource should we stick to. Please give ur input on this.

The AAP has made MANY of changes to the developmental milestones checklists as of this year, which means everyone who is taking the exam is in the same boat. PBR will need to do a deep dive into these changes in order to address modifications for 2023 and beyond. Note that because the info is so new, it's likely that the questions on the boards this year will not have been updated to include the latest

recommendations and we recommend that you continue to focus on the information that you have in your PBR materials.

EMERGENCY MEDICINE & TOXICOLOGY

SHARP OBJECT INGESTION - Page 209: Need to fix formatting of "SHARP OBJECT INGESTION" and make it clear that it's a different topic from FOREIGN BODY INGESTION.

Thank you! We fixed the formatting error for the next edition for the Core Study Guide and have moved SHARP OBJECT INGESTION down to its own line.

SHARP OBJECT INGESTION

Sharp objects noted to be in the stomach or proximal duodenum should be removed with a flexible endoscope immediately. Once the object is beyond the reach of a flexible endoscope, SURGICAL removal is indicated for SYMPTOMS (pain, vomiting, fever or evidence of bleeding) or if the object FAILS TO PROGRESS on imaging over 3 consecutive days.

VITAMIN & NUTRITIONAL DISORDERS

There were no Vitamin & Nutritional Disorders corrections for 2022!

GASTROENTEROLOGY

Hypokalemia in PS: PBR Core Study Guide states that hypokalemia results in the wasting of H+ to hold on to K ions. I think it is supposed to be the other way around. K+ is lost because the kidneys are trying to hold onto the H+ ions.

Thanks for bringing this to our attention! We did a deeper dive into this topic and it's pretty complicated. Our "SIDE NOTE" was wrong, but this is so complicated and variable depending on whether it's in the earlier or later course of illness that we think this is low yield, and we're removing the "SIDE NOTE" from that page. If you're curious about the complexities, **this page** may be a good read.

Here's the updated topic.

PYLORIC STENOSIS

Pyloric stenosis results from a gastric outlet obstruction due to a thickening or elongation of the pylorus. Look for **NON**-bilious, projectile emesis in a HUNGRY child. Labs may reveal a hypochloremic **hypOkalemic** metabolic alkalosis and possibly an elevated indirect bilirubin. An upper GI series may show the "string sign" or "railroad track "or "double track" sign. The railroad track sign is due to two

lines of contrast created by thick muscle, with a connection due to contrast in rugae. Diagnosis is made by ultrasound showing a pylorus that is **> 14 mm long** or **> 4 mm thick**.

* **PEARLS:** If you see a normal potassium level in a patient with pyloric stenosis, know that the total body potassium is still low. If the serum pH is normal or acidotic, it is NOT pyloric stenosis. Pyloric stenosis occurs in boys > girls.

* **PEARLS:** Erythromycin and azithromycin are BOTH associated with an increased risk of developing pyloric stenosis, but the risk is HIGHER with erythromycin (especially during the first two weeks of life). Give azithromycin if the baby is less than 6 weeks old**.**

* **IMAGE**: (Railroad Track) www.pbrlinks.com/PYLORIC1

* **IMAGE**: (String Sign) www.pbrlinks.com/PYLORIC2

* **MNEMONIC**: **4**yloric stenosis, 1**4** mm, and **4** mm. Remembering the diagnostic criteria can be tough. Use "**4**yloric stenosis" to help you.

Does Volvulus also show double bubble sign? Or is it mostly duodenal atresia? Also, the book says a double bubble can be seen with antral webs. Is that correct?

Yes, the double bubble sign is seen in both conditions. The double bubble sign indicates proximal intestinal obstruction. To answer a board question correctly, they will need to provide additional clinical information (besides the double bubble sign) to choose the correct diagnosis. Also, on further review, we found that antral webs are NOT associated with the double bubble sign. Below is our updated information on this topic in the Core Study Guide.

DOUBLE BUBBLE

A "double bubble" refers to the radiologic sign noted when there is a small bowel obstruction. There will be a large "bubble" and a small "bubble." These represent a dilated stomach and a dilated duodenum, respectively. Most commonly, the double bubble sign is seen in duodenal atresia, followed by intestinal malrotation. Other associated conditions include **duodenal webs, annular pancreas and choledocal cyst. With duodenal atresia, NO DISTAL GAS WILL BE SEEN. With malrotation or perforated duodenal webs, DISTAL GAS WILL BE SEEN.**

IMAGE: www.pbrlinks.com/DOUBLEBUBBLE1

PHARMACOLOGY & DRUG PEARLS

Diazepam question: p. 239 I believe what you have about diazepam being a contraindication to BF is incorrect, lactmed says to use diazepam cautiously during breastfeeding, but it is NOT a contraindication.

Thanks for letting us know! Please see our update for the next edition of the Core Study Guide below.

DIAZEPAM

Diazepam is NOT contraindicated in breastfeeding, but it is used with caution. Diazepam gets secreted into breastmilk and can potentially cause babies to experience withdrawal or sedation.

OPHTHALMOLOGY

There were no Ophthalmology corrections for 2022!

GENETICS & INHERITED DISEASES

There were no Genetics and Inherited Diseases corrections for 2022!

HEMATOLOGY & ONCOLOGY

There were no Hematology & Oncology corrections for 2022!

INFECTIOUS DISEASES

On page 292, it says common transmission-based precautions for measles are airborne and on page 309, it says that the measles virus is transmitted via droplets. Please clarify. Thanks.

Great catch! Measles transmission is airborne and NOT via droplets. Below is our update for the Core Study Guide.

MEASLES (AKA RUBEOLA)

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

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p. 331 states C. trachomatis commonly affects neonates (ages 2-19 weeks) while C. pneumoniae affects school aged children aged 5-15yo, whereas on p. 300 it states C. pneumoniae often affects children less than 2 months of age. Which is correct?

Thanks for pointing out this discrepancy. Chlamydia pneumonia typically affects older kids. Below is our correction for the Core Study Guide.

CHLAMYDIA PNEUMONIAE

Chlamydia pneumoniae causes an atypical pneumonia. It is often noted in school-age children who are 5-15 years of age. The patient may have tachypnea, a staccato cough ± eye discharge. For the boards, the patient may be AFEBRILE. Look for intracytoplasmic inclusion bodies and a gram-negative organism. You can also do a PCR. First-line therapy for children of all ages is a macrolide (erythromycin or azithromycin). For a child > 8 years of age, doxycycline or a fluoroquinolone would be acceptable, but as a second-line treatment.

PEARL: If you see the phrase "staccato cough," PICK THIS!

PEARL: Both erythromycin and azithromycin are associated with increased risk of infantile hypertrophic pyloric stenosis (IHPS), particularly in infants younger than two weeks of age. However, treat Chlamydia pneumoniae infections in a child less than 6 weeks old with azithromycin because the benefits outweigh the risks in this case.

What is the BCG vaccine? If a patient received BCG vaccine, then can we still use a ppd test, or would we have to do Quantiferon gold test instead?

The BCG vaccine is given in TB-endemic areas and protects against TB. If the BCG vaccine was given and there is a positive PPD test, obtain a Quantiferon Gold test. If the Quantiferon Gold test is negative, the patient does not have TB. If the Quantiferon Gold test is positive, obtain a CXR and cultures. Please note that the Quantiferon test may not be reliable test for children under the age of 2 years due to lack of research/data.

MYCOBACTERIUM TUBERCULOSIS (AKA MTB or TB)

Myco**bacterium** tuberculosis (AKA MTB or TB) is a bacterium (not a fungus) that does not allow for Gram staining (that's why acid-fast detection is used). It's presented here due to overlapping symptoms with the fungal infections. For newborns and young children, look for a prolonged illness with fever and cough. For older kids, look for fevers, chills, night sweats, weight loss, immigrant status, travel to an endemic area, hilar lymphadenopathy, an apical infiltrate, a pleural effusion, and/or a **supraclavicular lymph node**. Diagnose by AFB smears of sputum/secretions, a positive PPD, or a Quantiferon Gold. The BCG vaccine protects against TB, is given in TB-endemic areas and can cause a false positive PPD test. If TB is suspected in a child who received the BCG vaccine and now has a positive PPD, obtain a Quantiferon Test. Note that if a child has previously had TB then a positive Quantiferon result may be due to a false positive or latent TB. Regarding PPD READINGS, see below:

* **PEARL**: The PPD and treatment information is a little overwhelming. I have tried to simplify it, but if it's too much, MOVE ON. At the most, it would be worth one question. Other areas are MUCH more high-yield.

* < 5 MM INDURATION: Negative for MTB

  - **PEARL**: If +induration and < 5 mm, consider an infection with ATYPICAL mycobacteria.

* 5–9 MM INDURATION: Positive **IF** there are X-ray findings or there is a history of "close contact" with someone who recently got diagnosed with TB (newly +PPD), or the patient is immunocompromised. If none of the above conditions exist, a 5–9 mm induration in an otherwise healthy child is considered NEGATIVE for tuberculosis.

* 10-14 MM INDURATION: POSITIVE if there is ANY risk factor.

* ≥ 15 MM is used as a POSITIVE only for the lowest-risk patients with ZERO risk factors.

* TREATMENT AFTER A +PPD SCREEN:

  - NORMAL CHEST X-RAY: Single therapy with INH for 9 months.
  - POSITIVE CHEST X-RAY: Positive means the presence of hilar lymphadenopathy, an infiltrate, or a pleural effusion. The patient gets 3- to 4-drug therapy.
    - TRIPLE THERAPY: Rifampin, isoniazid, and pyrazinamide.

      MNEMONIC: You must treat, or the patient will have to **R**est **I**n **P**eace!
    - QUADRUPLE THERAPY: Ethambutol is added to the regimen. The ethambutol is stopped after 2 months.

* **TREATMENT** FOR NEWBORNS WITH **+PPD MOMS**

  - If mom is asymptomatic **AND** has a negative chest X-ray, check a PPD in the baby every 3 months. If it turns positive, the baby will need treatment with INH for a year if the chest X-ray is negative, and triple to quadruple therapy if it is positive.
  - If mom has active disease **OR** a positive chest X-ray, the baby is considered positive as well and gets full treatment (triple to quadruple drug regimen).

* **TREATMENT** FOR OLDER KIDS WITH **ACTIVE TB IN A HOUSEHOLD CONTACT**

  - PPD NEGATIVE & CXR NEGATIVE CHILD = INH prophylaxis for 12 weeks.
  - PPD POSITIVE & CXR NEGATIVE CHILD = INH prophylaxis for 9 months.
  - PPD POSITIVE & CXR POSITIVE CHILD = TRIPLE or QUADRUPLE therapy
  - TREATMENT FOR TB MENINGITIS: Same meds + STEROIDS + STREPTOMYCIN

* **PEARLS:** The right supraclavicular nodes drain the mediastinum and the lungs and are therefore more likely to show adenopathy in lung infections. The left side drains the thorax and the abdomen (lymphadenopathy is more likely to be lymphoma on the exam).

* **PEARL**: To check for how infectious a patient is, do NOT get a chest X-ray. Obtain sputum samples or gastric aspirates for AFB smear.

PEARL: Don't worry about the duration of treatment since it varies based on the type of TB they have (e.g., pulmonary, osteoarticular, meningitis, etc.).

VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

There were no Vaccines, Immunizations and Contraindications corrections for 2022!

INBORN ERRORS OF METABOLISM AND METABOLIC DISORDERS

There were no Inborn Errors of Metabolism and Metabolic Disorders corrections for 2022!

ACID-BASE DISORDERS AND ABGS

There were no Acid-Base Disorders and ABGs corrections for 2022!

FLUIDS & ELECTROLYTES

There were no Fluids & Electrolytes corrections for 2022!

NEPHROLOGY

late question too (sorry) regarding proteinuria workup: for 2+ proteinuria, do you first obtain an am void urine protein/creatine ratio?

Most commonly, proteinuria in asymptomatic children is caused by transient or orthostatic proteinuria. After a positive urine dipstick for 2+ proteinuria, the next step in the workup is two tests. Yes, obtain a first AM void sample at home to check a spot protein:creatinine ratio. Also, obtain a second urine sample in the office to send for urinalysis. Any next steps in the workup depend on the results of these two tests.

Below are the updated recommendations for our next edition of the Core Study Guide.

PROTEINURIA

Proteinuria is often benign and due to **orthostasis** (meaning children get it when they have been upright for a while). They may also get **transient proteinuria** from general medical illnesses and dehydration. If it's said to be 1+, just repeat in 2 weeks. If it's 2+ or greater, you HAVE to do a workup. First, order one urinalysis on an AM void and a second urinalysis on an additional urine specimen obtained in the office. If there is **NO PROTEIN** noted on the AM void and a normal urinalysis,it's probably due to transient proteinuria (which CAN indeed cause 2+ proteinuria). If there is NO PROTEIN noted on the AM void and proteinuria on the office sample, it's likely due to orthostasis. **If there is proteinuria** on the AM void and proteinuria on the second urine sample, this indicates persistent proteinuria. Obtain additional urine samples for urinalysis with microscopy, order bloodwork (BUN, creatinine, serum electrolytes, cholesterol, albumin and total protein) and obtain a blood pressure. If the proteinuria continues to persist, or if any of the labs are abnormal, obtain a 24-hour urine protein collection.

PEARLS: Urine **protein**:creatinine cut-off is 0.2. Urine **calcium**:creatinine cut off is 0.25.

STATISTICS

There were no Statistics corrections for 2022!

NEUROLOGY

There were no Neurology corrections for 2022!

ORTHOPEDICS & SPORTS MEDICINE

There were no Orthopedics & Sports Medicine corrections for 2022!

RHEUMATOLOGY

In the PBR book on page 414, it says that NSAIDs are not to be used as monotherapy for JIA, but I had two questions from Med Study and True Learn and they both said it can be used as initial monotherapy. Can you provide some clarity? Thanks!

You are correct! Usually in clinical practice, you can start with an NSAID trial if the disease is not severe, but typically this will only work for a few months. Then another agent will need to be added or the patient will be switched to more advanced treatments.

JUVENILE IDIOPATHIC ARTHRITIS (JRA, JIA)

KNOW JUVENILE IDIOPATHIC ARTHRITIS (AKA JUVENILE RHEUMATOID ARTHRITIS or JRA) WELL! The diagnosis requires knowing quite a few details. The child should have been under the age of 16 at the time of **symptom onset**. Symptoms must be present for at least **6 WEEKS** before the diagnosis can be made. In children, if arthritis is present it is more common in the **LARGE joints** and rheumatoid nodules are much less common when compared to adults. A positive rheumatoid factor indicates a worse **prognosis**. Do NOT order a rheumatoid factor for diagnostic purposes. It can **help with prognosis/subtyping, but a negative RF does NOT rule out RA**.

***** **O**LIG**O**ARTICULAR JIA (OLIGOARTHRITIS AKA PAUCIARTICULAR JUVENILE IDIOPATHIC ARTHRITIS): This refers to JRA that affects **4 OR FEWER JOINTS**, and is the more common type of JRA (>50%). ANA is often present but other markers such as RF are usually negative. It's more common in younger girls and is associated with chronic uveitis. Since **visual complaints may be absent,** patients need to have **regular eye exams**. Boys have a better prognosis.

- **MNEMONIC:** The **O's** for **O**lig**O** look like EYES and need regular eye exams because it is the more serious subtype.

***** POLYARTICULAR JIA (POLYARTHRITIS): This refers to JIA that affects **5 OR MORE JOINTS**. This is also more common in young girls. Systemic symptoms outside of the joints are not common.

***** SYSTEMIC (AKA STILL'S DISEASE): This is equally common in boys and girls. There are many classic symptoms and findings of which to be aware, including an episodic, salmon-colored "**EVANESCENT RASH.**" Patients may also have an **extremely high LEUKOCYTOSIS** (> 30K), with **spiking fevers, lymph-adenopathy,** and **possible hepatosplenomegaly**. Patients may also have **pleurisy**, **pericarditis,** and the **Koebner phenomenon** (linear skin lesions appearing along a site of injury, rubbing, or scratching). Serum **markers are NEGATIVE**.

- **PEARL: If everything else fits and the patient doesn't have an arthritis, go ahead and pick this diagnosis!** The other symptoms are commonly present well before the arthritis component kicks in.
- **PEARL:** This can be a difficult diagnosis to make and is often missed in clinical practice and on the pediatric board exam. **Please be VERY, VERY comfortable with this topic.**
- **PEARLS:** In comparison to leukemia, pain is in the morning (not at night), pain is in the joints (not the bone), mild hematologic anomalies (not severe), symptoms wax and wane (not persistent/ worsening), symptoms are insidious in onset (not acute), and JIA may have a rash. **BOTH can have lymphadenopathy and hepatosplenomegaly**. In comparison to septic arthritis, remember the insidious onset of symptoms for JIA (not acute).

* Treatment should be directed towards underlying synovitis and inflammation. Initial treatment for polyarthritis should start with disease-modifying antirheumatic drugs (DMARDs) such as methotrexate or a tumor necrosis factor (TNF) inhibitor in addition to methotrexate for more severe disease. Start with an NSAID monotherapy trial for 1-2 weeks for mild-moderate oligoarticular JIA or systemic JIA, If there is no improvement, move on to the other treatment options.

- **PEARL:** Children receiving methotrexate should be supplementing with folic acid or leucovorin (folinic acid).
- **PEARL:** Long term, high dose use of steroids should be avoided for patients with JIA. Short term, low dose steroids may be helpful in some patients, but DMARDs such as anti-TNF agents are the preferred treatment for children with JIA.

PULMONOLOGY

There were no Pulmonology corrections for 2022!

PSYCHIATRY & SOME SOCIAL ISSUES

There were no Psychiatry & Some Social Issues corrections for 2022!

ETHICS IN PEDIATRICS

There were no Ethics in Pediatrics corrections for 2022!

PATIENT SAFETY AND QUALITY IMPROVEMENT

There were no Patient Safety and Quality Improvement corrections for 2022!

PEDIATRIC LAB VALUES

There were no Pediatric Lab Values corrections for 2022!

PEDIATRIC VITAL SIGNS

There were no Pediatric Vital Signs corrections for 2022!

QUESTIONS & ANSWERS BOOK

There were no Questions & Answers Book corrections for 2022!

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

NOW LET'S GO OVER THE CLARIFICATION REQUESTS!

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

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

ADOLESCENT MEDICINE

On page 59, an adolescent female with breasts but no pubic hair has an XY genotype and the diagnosis is androgen insensitivity. My understanding for one to develop breast there needs to be estrogen present from ovaries. How is an XY person developing breasts? Thanks!

These patients are genotypic males, so there are no ovaries present to produce estrogen. However, they can produce estrogen from aromatization of testosterone. This causes breast development. Here's a review of the topic:

ANDROGEN INSENSITIVITY SYNDROME (AKA TESTICULAR FEMINIZATION)

ANDROGEN INSENSITIVITY SYNDROME (AKA testicular **feminization)** = XY karyotype = X-LINKED RECESSIVE = **"**te**X**ticular feminization" = Receptor insensitivity to androgens so no male external genitalia develop even though testes are present. Look for a blind ending vagina, lack of uterus, and lack of ovaries in what may appear to be a phenotypic female. Testes may be found in the inguinal canal. This could even present as PRIMARY AMENORRHEA in a phenotypically female person with BREASTS but no pubic hair!

PEARLS:

* If the test mentions a family history of MATERNAL "AUNTS" who are STERILE, they are probably XY TE**X**TICULAR FEMINIZED **UNCLES**.

* Patients are XY, so MIH IS PRESENT. Therefore, there are NO **INTERNAL** FEMALE STRUCTURES

* Androgens are also present, but the receptors are insensitive. Therefore, the default programming kicks in, and **EXTERNAL** female genitalia (blind vagina) are formed.

* Since patients are not sensitive to androgens, there is NO ADRENARCHE/PUBARCHE. Estrogen receptors work, and some estrogen is present due to conversion from testosterone, so the phenotypical female WILL DEVELOP BREASTS. + Breasts, NO hair, NO menses.

What is the difference between peripheral precocious puberty vs gonadotropic independent precocious puberty? Thank you for the clarification.

In central precocious puberty (CPP), there is an elevated basal LH level. On the GnRH stimulation test, the LH level and LH:FSH ratio will also be elevated. All of these levels will be low or normal in peripheral precocious puberty (PPP). Both disorders have an advanced bone age. In CPP, the stages of pubertal development will be in the same order as normal puberty. In PPP, the sequence of pubertal development may be abnormal.

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pg 69 in 2022 book - For PCOS, besides High LH and Low FSH, what are the levels for the other labs -- testosterone, DHEA-S, Estrogen, Progesterone, etc. ? Also, do you have to have ultrasound showing multiple cysts in the ovaries?

PCOS diagnostic criteria differs for adults and adolescents. Per the 2020 International Diagnostic Criteria for PCOS in Adolescents, the serum free or total testosterone level will be elevated. The other hormones (DHEA-S, estrogen, progesterone) are not part of the diagnostic criteria. Ultrasound is not part of the diagnostic criteria either since polycystic ovaries can be normal in this age group. Please see our update for the Core Study Guide below.

POLYCYSTIC OVARIAN SYNDROME (PCOS) AS A CAUSE OF AMENORRHEA

Polycystic ovarian syndrome is a **clinical** diagnosis. Labs are supportive. Look for an LH:FSH ratio > 2 and an elevated free or total testosterone level in a patient with acne, irregular menses, excess hair on her body, and/or signs of insulin resistance. This can be caused by anything increasing androgens, including Cushing's syndrome and exogenous steroids.

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pg 66 of 2022 book - In Klinefelter syndrome, do they have Low Testosterone levels? Can you then treat them with exogenous testosterone?

These patients can have low or normal testosterone levels. The main markers are elevated FSH and LH, but the FSH will be more elevated from its baseline than the LH. Treating with testosterone will not make the testes larger in size or prevent infertility, but will mitigate any metabolic effects from a testosterone deficit.

On page 63, under Constitutional delay of puberty. It mentions using testosterone as an option. What are the indications to treat vs reassure and observe?

If the bone age is delayed and there is normal linear (pre-pubertal) growth, just reassure the family and observe. If the bone age is advanced and linear (pre-pubertal) growth is not normal, the patient needs an additional workup. If the patient is around 14 years old and has Tanner II staging, can give a 4-6 month course of testosterone to help pubertal and height development advance.

Page 59: What is the difference (if any) between the last two pearls: Androgen excess versus Premature Adrenarche

Great question! Premature adrenarche can be a normal variant that can be observed, but androgen excess is typically not. Androgen excess can have other signs, such as virilization, advanced skeletal maturity and amenorrhea. Below is our update to the PEARLS AND MNEMONICS section to provide some clarity.

NORMAL PUBERTY PEARLS

Here are some great pearls and shortcuts about normal puberty.

* Girls have adult-looking breasts in SMR 3 and 5.

* SMR 4 = mound on mound breasts

* SMR 2 to 5 usually lasts about 3 to 5 years in total duration for both sexes.

* MENARCHE usually occurs in SMR 3 or SMR 4 OR, within 2–3 YEARS of the onset of puberty.

* MENSES/HEIGHT: At the onset of menses, girls are probably within 1–2 inches (2.5–5 cm) of their adult height. Why do I say that? Because they're probably in SMR 4 (which occurs **after** the peak height velocity).

* VAGINAL BLEEDING: Bloody vaginal discharge while in SMR 2 shouldn't happen. Consider a foreign body (e.g., toilet paper) in your differential.

HEIGHT

For the test, pre-pubertal basal rate for height in both boys and girls is 5–6 cm/year. The peak is 10 cm/yr. Early puberty results in shorter adult height.

GROWTH SPURTS

Elevated alkaline phosphatase can be normal during growth spurts. Hematocrit increases alongside growth spurts.

THELARCHE, ADRENARCHE THEN MENARCHE

(THELARCHE) Breast development → (ADRENARCHE) Hair development → (MENARCHE) Menses

PEARLS AND MNEMONICS: "Girls are TAMer than boys." "Boys like to TAP Her!"

- Girls are "TAMer" = Thelarche, then Adrenarche, then Menarche = Breast development → Hair development → Menses. Thelarche = first sign of puberty – stage 2. Adrenarche is the same thing as Pubarche. Breasts: Look most natural at SMR 1, 3, and 5. TAM = "Breasts are higher than Pubic hair which is higher than a Vagina."
- Boys = "TAP Her" = Testicular enlargement, then Adrenarche, then Phallus/Penile enlargement, THEN Height velocity peaks. Keep in mind that while testicular enlargement is normally the first sign of puberty in males, Penile enlargement may begin to occur before Adrenarche.

ENDOCRINOLOGY

Addison's Disease

1) What is the electrolyte (Na+, K+) composition of urine in Addison's? Is the urine sodium concentration elevated?
2) What is the volume status in Addison's? Are patients with Addison's typically volume depleted?

The main problem with Addison's disease is cortisol deficiency. Often there is also aldosterone deficiency. These patients will be hyponatremic and hyperkalemic. Urine electrolytes have no role in diagnosing or monitoring these patients. The volume status is critical. They can present to the ED with hypotension, hypoglycemia and hyponatremia, which means they are volume-depleted. Fluid resuscitate with saline boluses and give glucose.

PTH question: In reference to p. 216--Why is PTH and Calcium normal in familial hypophosphatemic rickets? Wouldn't PTH want to respond to the body's low phos? Also, why is phos high in hypoparathyroidism and pseudohypoparathyroidism?

Finding a great explanation for your first question has been a bit challenging, and the mechanisms are not going to be tested on the exam. We've double checked the content in the Core Study Guide and this article supports what we have written, so hopefully it will give you some comfort.

For your second question, PTH acts to increase calcium levels but inhibits phosphorus reabsorption in the kidney. When PTH is low or PTH resistance is high (pseudohypoparathyroidism), this will cause high phosphorus levels.

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pg 216 of 2022 book. Rickets lab pattern "Normal Calcium and Low Phosphorus" Initial vitamin D depletion, Low Vitamin D results in Low phosphorus reabsorption. Why is there a compensatory increased PTH that temporarily normalizes calcium? How does that occur?

If the vitamin D level is low, then calcium is not being optimally added to the bones, causing the calcium level to rise. This will trigger PTH to increase in order to drive the calcium into the bones. This will transiently normalize the calcium level

	LOW CALCIUM	NORMAL CALCIUM
LOW PHOSPHORUS	Severe Vitamin D deficiency	Familial hypophosphatemic rickets (calcium *can* be low) Early vitamin D deficiency
HIGH PHOSPHORUS	Hypoparathyroidism Phosphorus overload Pseudohypoparathyroidism	Renal disease Growth hormone excess High phosphorus diet

Hyperthyroidism - On page 77: Under Graves disease.
PEARL: PTU is quite toxic. My question: To my knowledge, pregnant women with hyperthyroidism are usually on PTU for the first trimester then methimazole for the remainder of the pregnancy. If PTU is that toxic then shouldn't it be avoided in the 1st trimester ?

Both meds are potentially teratogenic, but with methimazole the risk is higher for severity and frequency of birth defects so PTU is used during the first trimester to avoid those birth defects. PTU has risks of hepatotoxicity. Here's an updated version of this topic:

NEONATAL THYROTOXICOSIS (AKA NEONATAL GRAVES DISEASE)

Neonatal Thyrotoxicosis (AKA Neonatal Graves Disease) occurs when **maternal** thyroid-stimulating antibodies cross over and cause symptoms in the immediate **newborn period.** Symptoms include tremors, tachycardia, and SVT. This occurs in fewer than 10% of babies born to moms with Graves Disease. For a pregnant woman with Graves Disease, give **P**TU during the first trimester of **P**regnancy. Methimazole is not given in the first trimester because of the risks of birth defects during this time period. After the first trimester, switch to methimazole in order to avoid PTU's hepatotoxicity risk.

PEARL: If a patient has symptoms that are suggestive of both Neonatal Thyrotoxicosis and an Inborn Error of Metabolism (IEM), look at the age of onset! IEMs do not result in symptoms within the immediate newborn period.

Why does secondary adrenal insufficiency not have hyperkalemia or hyponatremia? Is aldosterone still being made?

Sodium and potassium levels are okay in secondary adrenal insufficiency because the Renin-Angiotensin (R-A) System is fine (since the adrenal glands are NORMAL and aldosterone is being produced).

Here's a topic review:

ADDISON DISEASE (AKA ADDISON'S DISEASE)

ADDISON DISEASE (AKA **AD**DISON'S) = **AD**RENAL INSUFFICIENCY = Electrolyte shifts (hyperkalemia or hyponatremia) can result in weakness, myalgias, malaise, nausea, and vomiting. HypOglycemia occurs from a lack of cortisol. ACTH levels are **HIGH** and can result in hyperpigmentation. When an ACTH stimulation test (Cosyntropin) is performed, a normal response (a rise in cortisol levels) **does not** occur. Patients may have elevated levels of ADH. This is an appropriate elevation and should NOT be diagnosed as SIADH.

* **MNEMONICS and PEARLS**:

- Keeping track of which eponym refers to cortisol deficiency or excess can be tough. Instead of ADrenal Insufficiency, call it "**AD**renal **D**eficiency." So **AD**dison's **D**isease = "**AD**renal **D**eficiency" = **AD D** and **AD D** = **ADD**ison's!!!

- It also means Aldosterone **D**eficiency. Aldosterone helps with sodium retention and potassium excretion. In "AD D," it is deficient, resulting in **hyperkalemia and hyponatremia**. If you ever see this combination of electrolytes on a chemistry panel, have a HIGH suspicion for some type of aldosterone deficiency.
- If they talk about a patient having a really good "tan," they may be referring to Addison's-related hyperpigmentation.
- "Think of the rise in ADH levels as an appropriate effort to retain water due to insufficient mineralo-corticoid (aldosterone)!"

* **PRIMARY ADDISON'S DISEASE** is the most common reason for adrenal insufficiency in children. It results in slow autoimmune destruction of the adrenal gland.

* **ADRENAL INSUFFICIENCY** can also be due to infection, adrenal hemorrhage (which results in very abrupt signs of Adrenal Insufficiency), or can be idiopathic.

* **SECONDARY ADRENAL INSUFFICIENCY** = A pituitary issue = **LOW** ACTH. There is **NO** hyperkalemia or hyponatremia. ACTH stimulation with Cosyntropin **does** result in improved cortisol levels. Patients sometimes have other midline defects.

  - **PEARL**: Sodium and potassium levels are okay in secondary adrenal insufficiency because the **Renin-Angiotensin (R-A) System is fine** (since the adrenal glands are NORMAL and aldosterone **is** being produced).
  - **MNEMONIC**: Secondary adrenal insufficiency is sometimes associated with midline defects. "This makes sense since the pituitary is also a midline structure!"

* **TREATMENT**

  - Maintenance therapy for Primary Adrenal Insufficiency is Hydrocortisone (to replace cortisol) and Fludrocortisone (to replace aldosterone).
  - Maintenance therapy for Secondary Adrenal Insufficiency is **just** hydrocortisone (to replace cortisol). Since the R-A System is intact, fludrocortisone is not needed.
  - Give stress dose steroids for fever, acute illness, surgery, or trauma. A mild illness (like a URI without fever) does not require stress dose steroids. The stress dose is usually double, or triple, the maintenance dose.
  - SALINE + GLUCOSE + IV HYDROCORTISONE for **ADRENAL CRISIS**. Signs, symptoms, and labs in an adrenal crisis can include nausea, vomiting, malaise, hyperkalemia, and hyponatremia.

How can a patient with renal disease make calcium at a normal level but not be able to excrete phosphorus? Why do they have Normal calcium and High phosphorus?

Hydroxylation of vitamin D occurs in the liver, so it can help maintain a normal calcium level. As phosphorus is metabolized in the kidney, renal disease will adversely affect the phosphorus level.

Why does 17 hydroxylase deficiency result in HTN if there is and hypokalemia if you have aldosterone being made?

In 17-hydroxylase deficiency, there will increased synthesis of deoxycorticosterone from progesterone, which then leads to increased levels of corticosterone. Corticosterone is a potent mineralocorticoid, which can cause severe hypertension.

Why does Mullerian Inhibitor Hormone deficiency result in both testes/penis being made along with uterus and fallopian tube being present when on page 84 of 2022 PBR book it said that the Mullerian ducts must regress for the male internal phenotype to occur?

As mentioned in the PBR, the INTERNAL male phenotype development requires the regression of the Mullerian ducts. Therefore, MIH does not have affect the external phenotype. Here's an updated and more concise version of the topic:

MULLERIAN INHIBITOR HORMONE DEFICIENCY (AKA MIH RECEPTOR DEFECT)

An XY child with Mullerian Inhibitor Hormone deficiency (MIH receptor defect) is born with normal testes/penis and **also** a rudimentary uterus and fallopian tubes. MIH would normally block the development of female internal organs, but this does not occur in MIH deficiency. Male external organs develop due to androgens.

Page 82: I understand the difference between primary and secondary adrenal insufficiency. But what is the clinical presentation or an example of a vignette for secondary adrenal insufficiency?

The patient will have weakness, fatigue, poor growth and mild nausea/vomiting/diarrhea.

OB/GYN & SOME STDS

Syphilis tests - p. 95: Are the non-treponemal tests (RPR and VDRL) essentially screening tests? So they are more sensitive? The treponemal tests (FTA) is confirmatory so thus more specific?

You've got it! The non-treponemal screening tests (RPR, VDRL) are more sensitive. The confirmatory test (FTA) is more specific.

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pg 93 of 2022 book - What is staccato cough? How is that different than other types of cough?

Staccato cough is seen in neonates with chlamydia infection. A staccato cough is described as a machine gun type of cough, meaning a series of burst of coughs, with inspirations in between. It is repetitive.

pg 89 to 90 of 2022 book - Could you please review the clinical scenarios for GBS prophylaxis and why these are necessary?

This is an important topic! Remember that the goal of GBS prophylaxis with antibiotics is to protect neonates from developing GBS meningitis.

If a pregnant mother had a prior baby with invasive GBS infection, go ahead and treat during the new baby's delivery. Also, treat if the mother has a positive rectovaginal swab around 36 weeks gestation. If the mother had GBS in the urine at any time during this current pregnancy, then treat at delivery. If the GBS status is unknown PLUS ROM >18 hrs, temp is at least 100.4 F, gestation is less than 37 weeks or positive intrapartum NAAT testing, then treat.

Treat with PCN. Treatment is most effective when given at least four hours prior to delivery. An alternative treatment is cefazolin. Here's a very important topic review:

(DOUBLE TAKE) GBS SCREENING AND PROPHYLAXIS MADE EASY!

Here are some key points and **PEARLS** about GBS screening and prophylaxis.

* RECTOVAGINAL GROUP B BETA HEMOLYTIC STREPTOCOCCUS (GBS) SCREENING CULTURES: These are obtained at **36 to 37 6/7 weeks gestation**.

* INTRAPARTUM ANTIBIOTIC PROPHYLAXIS (IAP): Intrapartum Antibiotic Prophylaxis (IAP) refers to antibiotics given to mom when she presents for delivery. If indicated, give IV penicillin, ampicillin or cefazolin at least 4 hours prior to delivery. The latest guidelines from the CDC may be viewed for some "light reading" by visiting www.pbrlinks.com/gbsprophylaxis.

* TO GIVE OR NOT TO GIVE INTRAPARTUM ANTIBIOTIC PROPHYLAXIS? IAP is indicated for any of the following scenarios:

  - Invasive GBS disease was present in a **previous** infant
  - Positive GBS was noted in the URINE at ANYTIME during **THIS** pregnancy (regardless of treatment and subsequent cultures)
  - Positive rectovaginal GBS screening culture noted 36 to 37 6/7 weeks in **THIS** pregnancy
  - Unknown GBS status PLUS any of these:
    - < 37 weeks gestation
    - ROM ≥ 18 hours
    - Intrapartum fever of ≥ 100.4 F
    - Positive intrapartum NAAT testing

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

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

If allergic to PCN, how to tx GBS?

Most PCN-allergic mothers can be treated with cefazolin (depending on the severity of the allergic reaction). Other alternatives are clindamycin and vancomycin.

ALLERGY & IMMUNOLOGY

Repeat Content: Repetition on Anaphylaxis on page 101 and 104. Thank you!

Yup! This was done on purpose, which is why we labelled it as a "Double Take." This makes it clear that we are repeating the same information in other relevant parts of the Core Study Guide for your studying convenience.

Blood Disorders: Under SCID, PCP is not a viral pneumonitis, it's fungal

Sorry for the confusion. The referenced sentence is factually correct. The sentence is listing three different infections that can present in children with SCID.

SEVERE COMBINED IMMUNODEFICIENCY (SCID)

Severe combined immunodeficiency (SCID) is a **T AND B CELL DEFICIENCY** (hence the word COMBINED). With T and B cells reduced, the hallmark is a low lymphocyte count (LYMPHopenia). **Look for an Absolute Lymphocyte Count (ALC) that is well below normal, remembering that normal lymphocyte counts below 2 years old are much higher (over 3000/mm^3) than in older children and adults.** These patients do NOT have **LYMPH**adenopathy, but they DO have a small thymus. The presence of a thymus means a bone marrow transplant **is** a viable treatment option. These patients get **all kinds of infections** (viral, bacterial, fungal, and opportunistic). The patient usually presents in the **first 3–6 months with otitis media (OM), thrush, diarrhea, and dermatitis.** There is **complete absence of T-cell function** (on fluorometric analysis of T, B, and NK cells). **This condition usually presents closer to 3 months**. In contrast, a pure B-cell deficiency presents around 6 months of age.

* **Possible presentations: PCP**, a viral pneumonitis that doesn't resolve or recurrent candidiasis that seems to be refractory to treatment. For PCP patients, always consider HIV in the differential, too.

* LIVE VACCINES: Do not give SCID patients live vaccines!
* CURE: Bone Marrow Transplant (BMT)

Allergy/Immuno Question: Pg 99 in 2022 - Why is RAST testing not affected by taking anti-histamine? How is RAST testing different from skin testing (which does get affected by antihistamines or antidepressants)?

Great question! This is always a good topic to review. RAST testing is checking serum-specific IgE testing. Therefore, antihistamines do not affect this type of testing.

Skin testing is measuring mast cell degranulation when the IgE is crosslinking with the allergen. Antihistamines will block the H1 or H2 receptors on the mast cells, therefore affecting skin testing results.

Topic review:

VASOMOTOR RHINITIS

Triggers for vasomotor rhinitis may include emotions, cold wind, change in temperature (or humidity), and pollution. Typical environmental allergens are NOT triggers.

SKIN TESTING

SKIN TESTING = AEROALLERGEN TESTING

* FALSE **negatives** can occur when patients are on antihistamines or antidepressants!

* NEGATIVE PREDICTIVE VALUE: NPV of skin testing for foods or inhalants is excellent.

* POSITIVE PREDICTIVE VALUE: PPV is good for inhalants but not good for foods.

PEARL/SHORTCUT: Skin-testing results are considered fairly reliable, regardless of whether they are positive or negative for airborne substances (e.g. pollen, pet dander, and dust mites). Skin testing can be helpful to diagnose food allergies, but given their complexity, additional tests may be needed.

PEARL: Oral food challenge (OFC) under the supervision of a clinician is the gold standard for diagnosing a food allergy. Initially, the potential food allergen is eliminated from the diet (called an "elimination diet"). Chronic symptoms may improve and a provisional food allergy diagnosis is given until an OFC can be done to confirm the diagnosis.

IMMUNOTHERAPY

About 50% of patients with hay fever respond to immunotherapy. There's a 0.5% chance (1 in 200) of having a severe reaction during therapy. If it happens, it will usually occur within 30 minutes, and will usually occur during peak pollen seasons or within the first year of immunotherapy (the rapid build-up phase).

PEARL: Contraindications for immunotherapy include poorly controlled asthma, beta blocker use, and history of repeated episodes of anaphylaxis with allergy shots.

RADIOALLERGOSORBENT TESTING (AKA RAST)

Since radioallergosorbent testing (RAST) is IN VITRO (blood) testing, it is NOT affected by taking anti-histamines.

Allergy/Immuno Question

Pg 102 to 103 in 2022 book. Why are the following NOT IgE mediated: 1) food protein induced enteropathy, 2)food protein induced proctitis/colitis, 3)food protein induced enterocolitis syndrome (fpies)? If not via IgE, then how do these occur? Are these the same type of illness, just on the spectrum of severity of illness? How are they different?

The exact mechanism for each of these is still yet to be figured out. What you should know for the boards is the clinical presentation.

ENTERopathy = ENTERING the tissue of the bowel wall and as a result you'll see symptoms related to disruption of the bowel wall.

PROCTITIS/COLITIS = More typically, you'll have a baby who is reacting to cow's milk protein, and possibly to soy too. These babies will have blood in the stool. Typically these children will outgrow their symptoms.

FPIES: These kids will get sick reproducibly 1-3 hours after ingesting certain foods. Some kids will look somewhat septic.

Page 105 of 2022 book. What is the difference between serum sickness vs. serum sickness-like-reaction? Are these treated the same way?

To differentiate between these two reactions, look at what the trigger is. In serum sickness, there is a serum protein based reaction. In a serum sickness-like reaction, there will be a different trigger like illness, antibiotics and infection. Treatment is generally the same.

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pg 107 of 2022 book. What is "Anergy"? "Skin testing with Candida, Mumps, Tetanus, and PPD can all be used to diagnose delayed type hypersensitivity anergy." How does this check for T cell defects?

Anergy is when the reaction you would expect is not happening. There is a lack of response in a situation where you would expect a response. For example, we're all exposed to candida and if exposed with a skin test, you SHOULD get a small area of induration. If you do not, there's something wrong. Maybe the T-cells are deficient, not functioning properly, etc. If you're not responding, that means the lack of response to a PPD does NOT mean that you have not been exposed to TB. So, in this example it helps to check for T-cell function and the lack of a response is referred to as anergy.

1.- To test for cell mediated immunity:

- PPD (looks for induration? Like when looking for TB or just redness, and if no reaction does that means abnormal cell mediated immunity or just not exposed to mycobacterium.

- also Cándida skin testing, MUMPS or tetanus, are done by the allergist or is something PCP's should do before referral

2.- To test for humoral immunity

- We first do antibody titers (for tetanus for example), and if normal IgG levels ? Or both.

If you do have cell-mediated immunity and your T-cells are working, you should have an induration. If you don't have functional T-cells, a PPD is not a good test for you. For example, if HIV, you may use something else like Quantiferon.

These types of tests are typically done by an allergist. When evaluating any patient for an immune deficiency, what is the clinical scenario? That will guide you more towards the type of testing that you need. If you are thinking humoral immunity (recurrent sinopulmonary infections), you test accordingly.

Immunoglobulin testing falls into two categories: qualitative and quantitative.

- Quantitative: Levels of immunoglobulins
- Qualitative: If low IgE, is it working? Check for IgG antibody titers to see if your body had good memory to things it should (like tetanus).

Question about Humoral Defects: The bottom of p. 116 mentions checking for antibody titers as a way to check for a B-cell (humoral) deficiency, whereas the bottom of p. 105 mentions "isohemagglutinins and/or serum IgG (or Ig subclasses from previous vaccinations)" as a way to check for humoral defects. Are these talking about the same thing? If not, how exactly are they different?

Yes, these are checking for the same thing. Checking for IgG antibody titers to something such as tetanus, for which you should have plenty of antibodies after vaccination, is one way to test for humoral defects.

For Hymenoptera/insect sting do we check IgE Antibodies in blood (Is this same as RAST) or is there any other specific test to find out?

There are two ways to test this: skin and blood (RAST) testing. SKIN testing assesses for venom allergy, but ONLY in kids that have symptoms suggestive of a systemic reaction. Skin and blood testing have a high rate of false positive. The benefit of testing is to know if it makes sense to start allergy shots. RAST testing is available, but skin testing is preferred.

FPIES Question: How would you differentiate FPIES from anaphylaxis? Seeing as how FPIES can cause vomiting, diarrhea, lethargy, shock? Is it related to timing of presentation?

FPIES symptoms occur around 1-3 hours after ingestion. There is no itching, urticaria, angioedema or wheezing. Symptoms are related to GI output and hypovolemia

Anaphylaxis occurs immediately (most often within 30 minutes of ingestion). Mast cell mediated symptoms develop. Symptoms include pruritis, hives, angioedema, flushing, wheezing, chest tightness, throat swelling, vomiting, LOC and anaphylaxis.

How do we know young children(<6mo) have an egg allergy if there is no exposure to egg in the first 6 months and none of the vaccines in this period contain egg?

We would not know that someone is egg allergic unless they had tried egg orally and reacted or perhaps if someone had allergy testing done early and had a very large skin test reaction (or high serum specific IgE level).

CARDIOLOGY

Pg 119-120: Could you explain the difference between AVNRT & AVRT, and which one is present in SVT?

AVRT and AVNRT are accessory pathway reentrant tachycardias that are also types of SVT. The only difference between the two is where the reentrant pathway lies. In AVNRT, the reentrant pathway is near the AV node. This means that ablation is NOT a treatment option due to the risk of causing complete heart block by accidentally ablating the node. In AVRT, the reentrant pathway is far from the AV node (closer to the LV wall). This means that it can be treated with ablation. Keep in mind that this question is beyond the boards. The level of knowledge that you need to answer Board questions are explained well in the Core Study Guide. Please see our updates for the Core Study Guide below.

WOLFF-PARKINSON-WHITE SYNDROME (WPW) AND AV REENTRANT TACHYCARDIA (AVRT)

In Wolff-Parkinson-White Syndrome, an extra (accessory) pathway bypasses the AV node and connects the atrium directly to the ventricle. While a **normal** AV node would conduct slowly, providing the short delay between atrial and ventricular depolarization, the accessory pathway conducts more rapidly and "short-circuits" the delay. This "pre-excitation" through the accessory pathway results in a wave of depolarization that bypasses the AV node. The result is a short PR (less than 0.12s) and a widened QRS (greater than 0.10s). A DELTA WAVE is present due to fusion of the QRS and pre-excitation wave.

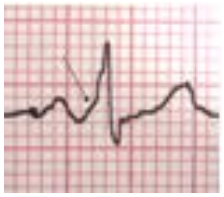
Children with WPW and similar disorders with accessory pathways may be prone to develop AV REENTRANT TACHYCARDIA (AVRT). Note that *WPW* refers to a static condition of having an accessory pathway, while AVRT refers to the result (tachycardia) that **can** occur. The reentrant tachycardia

happens when the depolarization wave passes from the atria to the ventricles along one pathway but then continues back from ventricles to atria via the other pathway. Usually, the circle is “orthodromic” (“straight-racing” with a narrow complex tachycardia), with forward conduction through the AV node then retrograde from ventricle to atrium via the accessory pathway. The goal of treatment is to block conduction somewhere (usually the AV node) long enough to stop the cycle. If the circle is “antidromic” (rare), forward conduction is down the accessory pathway and then up the AV node and results in a wide complex tachycardia (low-yield for the boards).

Narrow-complex tachycardia in WPW is treated the same as any SVT, beginning with vagal maneuvers and adenosine. CALCIUM CHANNEL BLOCKERS and BETA BLOCKERS should be “avoided.” Furthermore, if a WPW patient has ATRIAL FIBRILLATION OR FLUTTER then AV NODAL AGENTS (BETA BLOCKERS, DIGOXIN, and VERAPAMIL) are **CONTRAINDICATED** because they increase the refractory period of the AV node and make forward conduction through the accessory pathway more likely. This can worsen the tachycardia because accessory pathways typically have a shorter refractory period than the AV node. Also, AV nodal agents do not affect the refractory period of the accessory pathways. This can lead to deterioration of the arrhythmia into ventricular fibrillation and death. For atrial fibrillation or flutter, use IBUTILIDE or PROCAINAMIDE (these increase the refractory period of the accessory pathway and may result in restoration of sinus rhythm) and prepare for possible electrical cardioversion or defibrillation if the arrhythmia does not terminate. If shown an EKG with delta waves, pick WPW and move on. Note, however, that the delta wave is *not* seen during episodes of reentrant tachycardia.

For the chronic treatment of WPW, options include beta-blockers, calcium channel blockers, and possibly a cardiac ablation.

IMAGE: www.pbrlinks.com/deltawave1



* NAME ALERT: The name alert is for AV NODE REENTRANT TACHYCARDIA (AVNRT) with “NODE” in the name.

AV NODE REENTRANT TACHYCARDIA (AVNRT) 

In AV **node** reentrant tachycardia, the tachycardia originates at or near the node itself and is due to a fast and a slow conduction pathway around or in the AV node. In typical AVNRT, a “slow fast” AVNRT results in which there is conduction down the slow pathway and up the fast pathway. EKG findings (look at V1) may include retrograde P waves fused towards the end of QRS complexes, or a small R’ that is similar to that of a right bundle branch block (but with a normal QRS duration of < 120ms). AVNRT as a cause of supraventricular tachycardia (SVT) is common and **will** respond to vagal maneuvers and adenosine.

* NAME ALERT: The name alert is for AV REENTRANT TACHYCARDIA (AVRT) without “NODE.”

ADENOSINE AND VAGAL MANEUVERS

Adenosine and vagal maneuvers increase the refractory period, or temporarily block, the AV node (AVN).

ATRIAL TACHYCARDIAS

Atrial tachycardias do NOT respond to adenosine or vagal maneuvers. This includes atrial fibrillation and atrial flutter.

MNEMONIC: If signals from the atria are firing away on their own, they will continue to do so even if a signal that is further down the pathway (AVN) is blocked.

ATRIAL FIBRILLATION & ATRIAL FLUTTER

Atrial fibrillation and atrial flutter are most commonly seen in children with repaired structural heart disease, **not** in normal children. Also look for a cause of atrial dilation or tachycardia (e.g., hyperthyroidism). You may use adenosine to slow the rate for diagnostic purposes, but not for treatment. Treatment options include beta blockade, calcium channel blockers, digoxin, cardioversion, and ablation. These do not slow the atrial rate, but they do slow the ventricular rate by AVN blockade. See the caution above about atrial fibrillation and flutter in patients with WPW.

VENTRICULAR TACHYCARDIA (VT OR VTACH)

Etiologies of ventricular tachycardia include digoxin **toxicity**, hyperkalemia, and prolonged QT interval. In the absence of prolonged QT, treat with amiodarone. You may also use lidocaine or cardioversion.

Pg 120: Why don't WPW waves show up in an SVT pt necessitating a rpt EKG?

If WPW causes a widened QRS, isn't every WPW a wide complex tachycardia? If so, how is narrow complex even possible if by definition, WPW causes prolonged QRS?

WPW does not cause wide complex tachycardia. There are many types of SVT, and WPW causes a narrow complex SVT. For the purposes of the exam, let's simplify things.

A wave is supposed to go from the SA node to the AV node and down into the ventricles. This process repeats normally.

In WPW, the wave goes from the top of the atrium and SPLITS into 2 waves as it goes down. One goes to the AVN and one goes down a separate accessory pathway and cause some preexcitation. That preexcitation creates the delta wave. When SVT occurs, there is only pathway that is cycling again and again, so there is no longer any "pre" excitation, and therefore there is no delta wave during SVT.

The 2nd paragraph states "goal of treatment is to block conduction somewhere(usually the AV node)". However, below that, the paragraph is basically stating don't use AVN blockers as "AV nodal agents do not affect the refractory period of the accessory pathways". And by that logic, isn't Adenosine also an AVN blocker?

When SVT occurs, waves travel through the AV node and come back up through the accessory pathway and then goes back down the AVN again. The goal of treatment is to block the conduction at the level of the AVN, which stops the SVT from occurring. The blocking agent used is adenosine, which blocks the cycle by abruptly having the heart briefly go into cardiac arrest. This resets the heart. The hope at that time is that (1) the heart will restart on its own and (2) that it will restart with normal conduction.

Increasing vagal tone with vagal maneuvers also breaks the cycle. Be ready to do CPR if the heart arrests for a longer period of time than desired. Please refer to the above information from the Core Study Guide on this topic.

Why does a paradoxical split occur w/ an Aortic Stenosis?

The normal heart sounds are S1 and S2. S1 indicates closure of the mitral and tricuspid valves. S2 indicates closure of the atrial and pulmonic valves. In inspiration, there is a physiologic split (S1 single, S2 is split) heard. A physiologically split S2 means that the AV closes earlier than the PV.

In aortic stenosis there is a paradoxical split. In aortic stenosis the S1 is single, but the PV closes sooner than the AV. This is because there is delayed closure of the stenotic AV, as it takes longer for the LV to fully eject the blood from its chamber.

Is there a way to quickly think through what a clear chest XR versus a congested heart failure CXR means in terms of which CHD it could be? I'm still confused about how to reason through expected CXR findings.

We suggest approaching this topic by considering the pathophysiology. If the CXR demonstrates CHF, this means that there is pulmonary edema. Causes of pulmonary edema include excessive blood flood into the lungs (pulmonary over circulation due to ASD or VSD) or increased pulmonary congestion (drainage issues caused by LV failure, meaning that there is back pressure causing pulmonary congestion). This a good starting point to begin determining the cause of CHF seen on CXR.

Can you please explain newborn pulse oximetry screening results that indicate CHD vs. PPHTN vs. normal?

Great questions! The newborn pulse oximetry screening for CCHD is done in the upper and lower extremities prior to discharge from the nursery. It is a valid screen in neonates older than 24 hours of life.

If the pulse oximeter reading is less than 95% in either extremity or if there is greater than a 3% differential between the two extremities' readings, then this is a failed CCHD screening. This necessitates a pediatric cardiology evaluation.

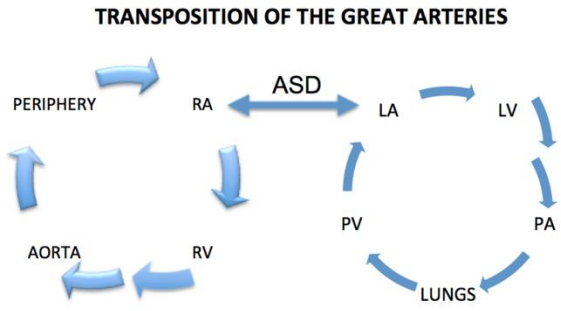
I am confused about this reverse cyanosis and differential cyanosis. Please reference page 130 in the 2022 edition. Thank you!

In differential cyanosis, the PA-aorta connection is the PDA. If the PA pressure is high, deoxygenated blood shunts to aorta and lower extremities. Therefore the oxygenation is higher in upper extremities and lower in the lower extremities. Differential cyanosis is where there is greater than a 3% pulse oximeter readings discrepancy between the upper and lower extremities. One cause of differential cyanosis is PPHTN.

In reverse differential cyanosis, the aortic pressure is greater than the RA pressure. The aorta has oxygen-desaturated blood which is sent to the brain and upper extremities. Highly oxygen-saturated blood is sent to the lungs, going from the PA to the aorta to the descending aorta via the PDA. Therefore, the upper extremities' pulse oximeter reading is lower than the lower extremities' oxygen saturation. This phenomenon typically happens in TGA, unless proven otherwise. Refer to cardiology.

TRANSPOSITION OF THE GREAT ARTERIES (TGA/TOGA)

The "great arteries" are the AORTA and the PULMONARY ARTERY. In Transposition of the Great Arteries, the LV leads to the PA, and the RV leads to the Aorta. This is the most common cardiac cause for cyanosis on **DOL 1**, and usually presents **within hours**. The EKG shows RVH. The two circuits do not connect and are "running in parallel" (see image). Mixing needs to occur in order to support life. Often a VSD is present, but if not, then a septal "defect" needs to be created. To treat, **create an ASD** to allow mixing (Rashkind procedure, balloon atrial septostomy done in cath lab or at beside in NICU). Mixing at the PDA also helps (though not as much), so create the presence of BOTH (**ASD and the PDA)** by also giving **PGE**. The ASD (or existing VSD) allows a RIGHT to LEFT shunt (deoxygenated circuit to oxygenated circuit) to be created. CXR shows an **EGG SHAPED and vascular congestion** (due to blood flow from the LV to the PA). There is no associated murmur. In the image below, note the circuits running in parallel. Treatment is an ASD (represented by the crossed arrows).



PEARL: If you suspect a cardiac cause for cyanosis on DOL 1, TOGA is probably your answer!

DERMATOLOGY

Clarification: On page 144, it states, "Rashes that Spare Inguinal folds" and it also states eczema and contact dermatitis should be high on differential. However, on the previous page 143 under cutaneous candiadiasis, a diaper dermatitis: "a diaper dermatitis, can occur secondary to a contact dermatitis or recent antibiotic use...this rash goes into inguinal folds (bolded). " The explanations seem to contradict one another or I am missing something?

Diaper dermatitis is a very common issue on the boards and in clinic. Common causes of diaper dermatitis include contact dermatitis, cutaneous candidiasis and atopic dermatitis.

Contact dermatitis is the most common diagnosis. This is a red rash in reaction contact with urine/fecal matter. There is NOT inguinal folds involvement because they are protected by the overlying skin-to-skin contact. Treat with barrier ointments.

Cutaneous candidiasis does have inguinal folds involvement, as well as satellite lesions. Other areas are nail folds (may present as chronic paronychia). Treat with nystatin (anti-candidal agent) or clotramizole (antifungal agent). Apply zinc oxide over the medication to "pat" down the treatment and to protect against contact dermatitis.

Atopic dermatitis has NO inguinal folds involvement, but the rash itself differs from contact dermatitis. The baby gets itchy, scratches the area and THEN develops the rash (thick, dry, flaky and lichenification). Lichenification is prominence of the resting lines that occurs in response to the scratching. Treat with topical steroids to decrease the inflammation and topical emollients to help heal the skin barrier. Alternatives to topical steroids are calcineurin inhibitors (tacrolimus and pimecrolimus).

Any time the skin barrier is broken (like from contact dermatitis or atopic dermatitis), the overlying biofilm organism can cause a secondary rash. Below the waist, the prominent organism on the skin is candida. Above the waist the predominant organism on the skin is Staph.

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pg 144 of 2022 book - Why is Langerhans cell Histiocytosis associated with Diabetes Insipidus? How would these patient's present? Would the skin lesions show up before the bone lesions or do they occur simultaneously? Are the Histiocyte cells depositing into certain areas of the body to manifest symptoms?

Langerhans Cell Histiocytosis is a rare diagnosis that is part of the diaper dermatitis differential.
Key to diagnosis is petechial rash/bloody crusty erosions ascending above the suprapubic area. LCH is an umbrella term encompassing multiple syndromes. One possibly tested syndrome within LCH is eosinophilic granulomatosis (AKA Hand-Schuller-Christian disease). These kids have diabetes insipidus, osteolytic bone lesions and petechial rash in the diaper dermatitis area. Abnormal histocytes are proliferating and then invade other areas. Once they get into the bloodstream they infiltrate the bone, causing osteolytic bone lesions. They can also invade the hypothalamus and cause DI (very rare finding).

Is propranolol topical or oral?

It's given orally. If the hemangioma is not involuting enough or it's in a dangerous area that could start to obstruct the function of an organ (eye or nose), treat with propranolol(first-line). Side effects include hypoglycemia and bradycardia. So give the medicine with feeds.

How to differentiate pityriasis alba vs pityriasis versicolor?

Kids with pityriasis alba present with a history of atopy, (rhinitis, food allergy, asthma and eczema), and it is a subtype of eczema. The eczema can get out of control, causing white patches on the cheeks. Lichenification, itching and thickness of the skin will persist.

Pityriasis versicolor presents in teens and adults. It affects the head, face, neck, upper chest, upper back and upper arms. When the sebaceous glands get activated in puberty. yeast will "eat" there. Salicylic acid gets released in the process, which decreases pigmentation in these areas (very branching views, lots of circles).

Is Tinea versicolor same as pityriasis versicolor? For hypopigmentated patch on face it is confusing to answer what it could it be? I would appreciate your explanation on hypopigmentation patches.

Yes! Tinea versicolor and pityriasis versicolor are the same. In a patient with darker skin complexion, there will be hypopigmentation (large area, branching, symmetrical). In a patient with lighter skin complexion, the affected areas can be light pink in color or hypopigmented. In contrast, pityriasis alba will present with more isolated and a smaller number of patches.

Is there any difference between the treatment of congenital Vs infantile hemangioma ?

Congenital hemangiomas are NOT responsive to propranolol, but infantile hemangiomas are. Congenital hemangiomas do not rapidly proliferate, whereas infantile hemangiomas do rapidly proliferate.

For scabies and pediculosis capitis- Is the treatment of choice 5% or 1% Permethrin?

Treatment choice is the 5% strength. Treat the patient and household contacts and then repeat the dose after one week.

The Mazzotti reaction can occur within 1 week of the treatment. It is an inflammatory response (fever, chills and myalgias) because the body is responding to what is getting released from the dead parasites. Treatment is supportive care.

Keep in mind that the boards will not ask about percentage, doses, duration and frequency of meds.

NEONATOLOGY

Microcephaly:
When to order MRI, when to observe, when to refer?
- When crossing more than two percentile lines?
- When HC above 95%?
- When having delays?

In the newborn period, if there is microcephaly PLUS any other symptoms (developmental delays, seizures, feeding intolerance, abnormal examination, dysmorphism etc.), obtain an MRI. If the HC is <5th percentile, order a urine CMV. In an older child, if there is crossing of percentiles, HC >95th percentile, vomiting, drooping eyelids etc, you would be concerned about an intracranial process. This would warrant imaging.

When is consider familial macrocephaly?
When parents have more than 55cm HC, or 60cm HC, how do you decide is familial?

There doesn't seem to be any cut off. This is a diagnosis of exclusion, so if there's no pathology PLUS a history of macrocephaly in the parents, it's Benign Familial Macrocephaly. Practically speaking, just ask the parents if they have been told that they have large heads, their other children have large heads etc.

Topic review:

HEAD CIRCUMFERENCE – MACROCEPHALY, HYDROCEPHALY, AND MICROCEPHALY

To know if you're dealing with possible macrocephaly, hydrocephaly, or microcephaly, you first have to know what a baby's head circumference should be. A normal newborn's head circumference is approximately 35 cm. After that, expect the head circumference to increase by 1 cm/month for 6 months and then ½ cm/month from 6–12 months.

PEARL: 50th Percentile for HC at birth is 35 cm. For a child born with a head circumference of 35 cm, head circumference should increase to 44 cm at one year of age.

* MACROCEPHALY: > 97 percentile for age, though the history will usually include a normal HC at birth. If the child's development is in fact normal, just measure the parents' heads to see if it is FAMILIAL MACROCEPHALY!

  - **PEARL**: If there are developmental issues or other issues with the child, HYDROcephaly is likely the main problem.

* HYDROCEPHALY: Look for a **BIG HEAD AT BIRTH** + NEUROLOGIC ISSUES.

  - **PEARL**: Large anterior fontanelles are found in both macrocephaly and hydrocephaly. The finding is very nonspecific. If the ABP mentions a **BULGING FONTANELLE**, think HYDROCEPHALY.
  - **PEARL**: Neither macrocephaly nor hydrocephaly result in papilledema. Both of these conditions are usually due to slow processes, and papilledema is usually the result of an ACUTE problem.

* MICROCEPHALY: HC is usually **normal at birth** (~35 cm) and then falls off the curve.

What is the difference between IUGR and SGA? Why can a neonate be one and not the other?

IUGR reflects the FETAL status and is the end result of fetal distress, leading to size issues. It is ALWAYS pathological (placental problem, genetic condition etc). It is important to determine the cause of the IUGR. SGA is a term used to reflect small weight POST delivery. SGA can be a “normal variant” (small parental size, genetics etc.).

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pg 166 of 2022 book - what is the difference between Macrocephaly vs. Hydrocephaly?

Both conditions cause a large head circumference. Hydrocephalus is due to enlarged ventricular size. Macrocephaly can have different causes (familial, increased brain volume, cysts, tumors, dilated vessels etc.).

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pg 166 of 2022 book - why do premature babies have higher protein requirement?

For a preterm baby, you want to support the anabolic state. Higher protein intake helps avoid a negative nitrogen balance because that would result in a catabolic state. Plus, these children need more calories for growth and tissue formation.

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pg 168 of 2022 book - why does well water need to be sterilized? what chemicals/metals/ions does it contain?

Great question!

Boil/sterilize well water to kill germs such as Giardia, Cryptosporidium and E. coli.

Well water may contain toxic heavy metals (lead, arsenic, mercury etc.), nitrates and Volatile Organic Compounds (VOCs). Increased nitrates exposure increases a baby’s risk of developing methemoglobinemia.

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pg 179 in 2022 book - Why is the risk of malignancy increased for BOTH testicles, even if only one testicle is undescended? How are both affected when only one is mal-positioned? Why would risk persist even after orchiopexy?

I wish we could give you a great answer to this, but the reason is not well understood.

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pg 175 to 176 in 2022 book - What is the anatomical and/or physiological reasoning to have differences in the septic work up based on neonate/infant age? Why does work up slightly change from 0 to 7 days old, 8 to 21 days old, 22 to 28 days old, 29 to 60 days old, 60 to 90 days old??

In general, the older the baby is, the stronger it is. Older babies will have a greater ability to fight off infections and develop antibodies. Early-onset sepsis occurs in babies from DOL 0-7. Late-onset sepsis occurs in neonates above the age of 7 days. The septic workup can differ in these two age groups because different organisms affect them. Another reason the workup is different is because the new guidelines were recently released and we found that doing LPs on all babies less than 28 DOL was unnecessary. Therefore the workup is further stratified based on the age of the neonate.

Important topic review:

(DOUBLE TAKE) FEBRILE INFANT AND SEPTIC WORKUP

For babies with a temperature of at least 38 degrees C° rectally, workup and treatment depend on the infant's age. The most common causes of neonatal sepsis are E. coli and group B strep, with E. coli being more common because of GBS screening and treatment. Risk factors for invasive bacterial illness (IBI) include temperature $\geq$ 38.6 C° (101.5 F°), prematurity, congenital defects, indwelling catheters, recent antibiotic use (in the last 1 week), and maternal factors (fever, PROM, and +GBS status). The risk of invasive bacterial illness (IBI) decreases with age. Consider HSV in babies if the mother has risk factors, if the CSF shows pleocytosis, or if the baby has suggestive lesions or seizures. For most of the babies admitted to the hospital due to a fever, they can be discharged after 36 hours if they are fever free and all cultures are negative. Babies 0-7 days old should be discharged after 48 hours. Please note that the decision tree can be complicated, and the AAP allows for variations based on the individual circumstances.

* **0-7 DAYS OLD**: These babies are at **high risk for early-onset bacterial infection**. Obtain CBC, blood cultures, tracheal aspirates (if intubated), and lumbar puncture. A urinalysis is not needed since a positive urine culture in this age range is usually due to severe bacteremia. Obtain a CXR and other imaging only if symptoms warrant it. Inflammatory markers (procalcitonin and/or C-reactive protein PLUS ANC) are not required but may be obtained. Start empiric IV antibiotics immediately (do not delay for an LP). Treat with ampicillin PLUS gentamicin or use ampicillin PLUS an expanded-cephalosporin (ceftazidime, cefepime, or cefotaxime). Add acyclovir if HSV prophylaxis is indicated. Add vancomycin if MRSA infection rates are high (>10% of S. aureus infections) or when treating for meningitis (helps to cover resistant S. pneumoniae).
* **8-21 DAYS OLD**: Same workup and treatment as the previous age range, but also **obtain a urinalysis** and culture. Inflammatory markers may be obtained. If needed, obtain additional cultures based on history and exam. Obtain CXR and other imaging only if symptoms warrant it. Treat with the same antibiotics mentioned for the previous age range.
* **22-28 DAYS OLD**: Same workup and treatment as the previous age range, but inflammatory markers **should** be obtained. Obtain CXR and other imaging only if symptoms warrant it. Treat with the same antibiotics mentioned for the previous age range.
* **29-60 DAYS OLD:** Workup is similar to the previous age range, but only obtain an LP if the inflammatory markers are elevated or if symptoms warrant it. Treatment depends on whether the child is ill-appearing or well-appearing, and also on whether risk factors IBI are present. Can monitor on an outpatient basis

without antibiotics if the child is well-appearing, there are no IBI risk factors, and the workup is negative. If treating, do so with ceftriaxone or cefotaxime PLUS ampicillin. Add acyclovir and vancomycin if indicated.

* **60-90 DAYS OLD**: The workup is more focused. Obtain a urinalysis and urine culture. Consider getting a CBC and a CXR if indicated. Consider seasonal viruses in your differential and send viral tests if appropriate. Treatment depends on whether the child is ill-appearing or well-appearing. Can monitor on an outpatient basis without antibiotics if the child is well-appearing and the workup is negative. Monitor on an outpatient basis with oral antibiotics if the workup suggests a UTI. If the child is septic and needs to be admitted, treat with ceftriaxone or cefotaxime PLUS ampicillin. Add acyclovir and vancomycin if indicated.

GBS ppx: Can you review the reasons for the specific antibiotics used in different types of GBS disease? For example, why is Nafcillin used for septic arthritis and cellulitis? I've never seen Nafcillin used in practice. Thanks!

Reasons can include sensitivities, penetration, etc. We've decided not to look at all possible scenarios and give reasons for each because we don't think that would be a good use of your time to review. But, reviewing this key topic would be!

Treating Newborns with GBS Disease

In general, ampicillin and gentamicin are good empiric treatments when GBS is suspected but not confirmed. If meningitis is suspected, add on an expanded-spectrum cephalosporin (eg, cefotaxime, ceftazidime, or cefepime). If the culture results are known, the definitive treatment is penicillin G since GBS is uniformly sensitive to penicillin and ampicillin. For specific antibiotic regimens in situations where culture results are not yet available, empiric treatment is based on the site of the infection and whether the infection is early onset" (birth to DOL 6) or late onset (DOL 6+):

- EARLY ONSET
  - Bacteremia, sepsis or pneumonia: ampicillin IV + gentamicin IV
  - Meningitis: ampicillin + gentamicin + cefotaxime
- LATE ONSET
  - Bacteremia: ampicillin (or vancomycin) + gentamicin (or cefotaxime)
  - Meningitis: ampicillin (or vancomycin) + gentamicin + cefotaxime
  - Cellulitis/adenitis: nafcillin (or vancomycin) + gentamicin (or cefotaxime)
  - UTI: ampicillin (or vancomycin) + gentamicin (or cefotaxime)
  - Septic arthritis or osteomyelitis: nafcillin (or vancomycin) + cefotaxime

Page 169 indicates Premature caloric nutrition as 24 kcal/oz. Does every premature less than 37 weeks has to be on 24 kcal/oz? Or Does it depends on VLBW and LBW - for example VLBW - 24kcal/oz ad LBW - 22kcal/oz. Thank you.

The number of calories for premature infants can vary, but for the boards, know that if you have a premature baby s/he will likely need something > 20 kcal/oz. If the neonate is < 34 weeks at birth, then 24 kcal/oz is likely the answer.

So for the boards do we still do LP on < 28 d old neonate as part of a septic workup?

Yes. As stated in Core Study Guide, you would still perform an LP as part of the septic workup for this age.

What do you recommend we know about TPN for the boards?

The PREMATURE INFANT NUTRITION section in the Core Study Guide is good to review per our Neonatologist. He also mentioned that prematurity can lead to hypoglycemia, so higher dextrose is needed in TPN.

On page 174 - Early onset vs late onset sepsis. Is Bacteremia most common reason in both cases? In early onset sepsis - Is Bacteremia followed by Meningitis and pneumonia or is it Bacteremia followed by pneumonia and Meningitis - The order on page 174 for Early onset and Late onset is little confusing.

Sorry for the confusion. The order of the bullet points was not meant to suggest a specific order based on commonality.

The book (on page 169) states Full term is 37 to 41&6/7, but i thought full term is 39-41&6/7, and term refers to the first range.

We've looked into this and discussed it with a neonatologist. He agrees that the content is correct in the Core Study Guide.

FULL TERM

For the boards, assume 37–41 + 6/7 weeks gestation is full-term.

NEONATE

The term "neonate" applies from birth to 1 month of age.

INFANT

The term "infant" applies from 1 month to 12 months of age.

Is e. coli or strep pneumonia the most common for sepsis/bacteremia. The guide lists both

E. coli is the most common cause of sepsis in neonates.

DEVELOPMENTAL MILESTONES

pg 194 of 2022 book - What is cognitive reasoning? What is the difference between Concrete thinking vs. Abstract thinking?

Concrete thinking is also known as literal thinking. It is a type of reasoning that is based on literal interpretations and what a person is able to experience through the senses (sight, vision, touch). This is a "black and white" thought process.

Abstract thinking (AKA cognitive reasoning) is the exact opposite of concrete thinking, where there are "shades of gray". It is a more complex type of mental processing, where something can be analyzed in multiple different ways, using analogies and metaphors.

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stranger anxiety
separation anxiety
age of presentation:

Separation anxiety typically begins at age 8 months and intensifies at age 10-18 months. Stranger anxiety beings at age 8-9 months. Both resolve by age 24 months.

EMERGENCY MEDICINE & TOXICOLOGY

Emergency/Toxicology

pg 203 of 2022 book - Why and how does Marijuana cause gynecomastia? Does it affect steroid hormone pathway? Does Marijuana decrease sperm count or activity, or affect fertility in any way?

Marijuana increases estrogen and decreases testosterone, causing gynecomastia in males. There is mixed data regarding whether or not it affects the steroid hormone pathway or fertility. Studies have shown that MJ can increase sperm count but decrease sperm activity. Keep in mind that the last two questions are beyond the scope of the boards.

Can you go over the different ways to manage a Tylenol Overdose(ie symptomatic vs asymptomatic, came in before 4 hours vs after 4 hours). Sometimes you treat empirically, other times you collect labs first. I've seen a lot of different formulations on questions and I always seem to get tripped up on it. Thanks.

If the patient is able to tell you how much and when the Tylenol was taken, then you can plot it out on the **nomogram**. If this is not available, then do not wait the 4 hours, and just empirically treat. Remember that the 2 hour level cannot be used as a 4 hour level.

Why are the symptoms for hydrocarbon inhalation so much different than ingestion?

Great question! With hydrocarbon ingestion, this enters the digestive system, so kids can experience nausea and vomiting. There is also a potential aspiration risk, which allows the hydrocarbons to enter the lungs, thereby causing ARDS/hypoxia. With hydrocarbon inhalation, the toxin goes from lungs straight into bloodstream. This causes more severe toxicity than ingestion, including neurologic symptoms such as ataxia.

VITAMIN & NUTRITIONAL DISORDERS

Vitamin question: how to differentiate between zinc deficiency and vitamin b2 (is this testable)?
Zinc deficiency causes dermatitis of the perioral and perianal areas, as well as the extensor surfaces of the limbs. Vitamin B2 deficiency causes cheilitis, glossitis and stomatitis with seborrheic dermatitis anywhere on body.

(DOUBLE TAKE) ZINC DEFICIENCY

Breastfeeding helps with zinc absorption. If a child begins having medical problems once weaned from breast milk, consider zinc deficiency in your differential. Zinc deficiency causes a **SCALY and EXTREMELY ERYTHEMATOUS** dermatitis in the perioral and perianal area (**around the natural orifices**) that can DESQUAMATE. The rash is sometimes described as erosive and eczematous. It can also be associated with ALOPECIA and poor taste.

* **MNEMONIC**: Poor taste, huh? Have you ever had Zinc lozenges? They are disgusting! It's probably a good thing that you have hypogeusia when you are eating Zinc lozenges!
* **IMAGE**: www.pbrlinks.com/ZINC1
* **IMAGE**: www.pbrlinks.com/ZINC2
* **IMAGE**: www.pbrlinks.com/ZINC3
* **IMAGE**: www.pbrlinks.com/ZINC4

*** PEARLS:**

- CROHN'S DISEASE: If a Crohn's patient is suffering from diarrhea, they may have zinc deficiency since Zn is lost in the stool.

- (DOUBLE TAKE) STRICT VEGETARIANS AND VEGANS may be susceptible to multiple nutritional deficiencies, including deficiencies in IRON, ZINC, CALCIUM, and VITAMIN B12. Vegans avoid all animal-derived products (including milk and eggs). B12 deficiency can result in megaloblastic anemia, vitiligo, peripheral neuropathy, and even regression of milestones.
- **MNEMONIC**: Did you know giraffes are vegetarian? Imagine a giraffe standing in Times Square reaching its long neck into the sunroof of a FUZZY CAB that has green, grass-like seats and fuzzy floor mats. FUZZY CAB = FeZi CaB12!

RIBOFLAVIN (B2) DEFICIENCY

Riboflavin (Vitamin B2) deficiency can result in anemia, angular stomatitis=cheilosis, glossitis ("tongue = riboFLAVor"), and seborrheic dermatitis.

PEARL: Phototherapy can result in decreased riboflavin (B2) levels. Keep this in mind with premature hyperbilirubinemic children.

MNEMONIC: Imagine that the drawing below represents a premature baby with protective glasses on (for phototherapy). The lenses have "2" written on them to remind you of the B2 deficiency. There is a rash at the scalp (seborrheic dermatitis), and there are steep angles at the edges of the lips to remind you of angular stomatitis. He's bleeding from the angles of his mouth to help you remember the possible anemia.

Is there a mnemonic for what gets absorbed in what part of the colon?

Nope! I'd recommend breaking up the deficiencies by location. Iron gets absorbed in the duodenum. Bile salts and fat-soluble vitamins get absorbed in the mid-gut (terminal ileum). Water and electrolytes get absorbed in the colon.

What kind of anemia does B2 causes - is it microcytic or normocytic?

B2 deficiency causes a normocytic, normochromic anemia. It rarely is an isolated deficiency and is often associated with other nutritional deficiencies. It may also be associated with other types of anemia, including iron deficiency.

GASTROENTEROLOGY

GI question: Pg 232 to 234 of 2022 book. Why is it that for Pyloric stenosis the diagnosis is made by abdominal ultrasound vs. for Volvulus the gold standard study is Upper GI series? How do these testing modalities differ? Is upper gi series the same as "upper gi with small bowel follow through"?

Abdominal ultrasound is assessing the length and thickness of the pylorus muscle.

UGI series is used to look for volvulus. This evaluates the lumen, which is lost when the intestine is twisted in a volvulus.
UGI series can assess the esophagus, stomach and the proximal ⅓ of small intestine. The SBFT evaluation assesses the distal small bowel 1-3 hours later. SBFT is not performed too often anymore (need clear indication to order it).

Jejunal Atresia: Does jejunal atresia also present with double bubble sign?

This is a rare finding in jejunal atresia more typically seen on ultrasound (versus XR). It is more common to have multiple double bubble signs, or none at all. This probably won't be on the boards unless they give more clues that this is a more distal finding to the duodenum, as the double bubble sign is typically seen in the duodenum and the stomach.

NAFLD: What is the AST/ALT level to make a diagnosis of NAFLD, to order ultrasound, and to refer to GI?

NAFLD does not cause a high elevation of ALT. NAFLD is a diagnosis of exclusion, and an elevated AST/ALT is a nonspecific finding. If the AST/ALT levels are elevated, repeat testing in 2 weeks.
If the ALT is very high (in 400s), this will not occur in isolation and will not likely be caused by NAFLD. In this scenario, consider hemochromatosis or Wilson's disease.

if the patient is cholestatic, obtain an ultrasound to look at the gall bladder.

Do not pick liver biopsy as an answer choice. Typically the answer on the boards will be to order a full liver panel.

If the albumin is low, consider synthetic failure.

if there is conjugated hyperbilirubinemia, you need to rule out obstructive conditions.

These above finding increase the urgency for evaluation by a GI specialist.

H pylori: is the diagnosis by stool or blood?

Diagnose H. pylori by biopsy of the gastric tissue. In children, stool testing and the urea breath test are considered supportive, but not diagnostic. Bloodwork is not recommended.

If given a case scenario of watery diarrhea how to know if it is by giardia or cryptosporidium, any tips?

Clinically Giardia presents as a gassy kid (going into the weeds on this one on for the boards).
They both cause watery diarrhea that can be chronic. The only way to differentiate between the two is testing: Giardia EIA Ag and looking for cryptosporidium in the stool.

Any tips on when to use stool o and P, DFA or Elisa or stool culture on case scenarios , it is sometimes difficult when they give you both options such as stool O and P as well as stool culture? Thank you.

You need to know how to ID different diseases based on the clinical presentation on the boards. Giardia has a separate antigen test (although you should be able to see on stool O & P). Shigella is diagnosed by stool culture. Test a stool sample for cryptosporidium, which is a separate test than stool O & P.

What diagnostic modalities do you use for H. pylori? Is a biopsy required prior to treatment?

Yes, according to the guidelines, you need a biopsy to diagnose it. However, the urea breath testing and stool antigen testing have a high sensitivity.

Is there a clinical difference between Gilbert Syndrome Crigler-Najjar syndrome(especially Type 2)? how would you distinguish between Gilbert vs type 2 Crigler najjar - is there any age presentation difference?

Both cause indirect hyperbilirubinemia, but it is intermittent in Gilbert syndrome and persistent in Crigler-Najjar syndrome. Kids with Crigler-Najjar are sicker, whereas kids with Gilbert syndrome have intermittent symptoms.

What would the actual gut tissue look like when opened for malrotation? I remember a practice question discussing this.

In malrotation, you will see an ischemic gut. Early on, the tissue will be pale. Then it will become gangrenous. This is not highly board relevant, however.

If we are given Ultrasound and CT abdomen in the option for Pancreatitis which one we should go for and is it the same imaging modality of choice for chronic pancreatitis?

Ultrasound is first-line unless the patient presents with severe pancreatitis or hypoglycemia. In these cases, get an abdominal CT for more specific findings to see if surrounding organs are affected or there are any signs of necrosis. Typically these kids will require PICU-level care. If the patient has nausea/vomiting, obtain an ultrasound.

Obtain an MRCP for chronic pancreatitis, which will provide detailed images of the anatomy of the biliary tree. An abdominal CT does not get as detailed a view of the biliary tree, which is key if there is cholestasis or hyperbilirubinemia

Choose ERCP on the boards if there is any indication for a stone somewhere in the biliary tree, as this procedure can resolve the problem during the procedure. MRCP is noninvasive, so one cannot remove a stone with this procedure.

When the clinical stem is asking about further evaluation of a patient with dysphagia, is it safe to say that we should get an upper GI series over an EGD? When would we do an EGD first?

Yes! Obtain an upper GI series to look for obstruction. However, if the patient presents with dysphagia with feeding difficulties, obtain an EGD to rule out obstruction (web, sling), basically anything that is compromising the lumen of the esophagus.

Is upper GI series the same thing as esophagram?

No, they are different studies. During an esophagram, the radiologist is evaluating swallowing in the esophagus. An UGI series looks at the anatomy all the way up to the ligament of Treitz.

Is upper GI series vs upper GI barium series and upper GI contrast study are all same?

Yes! They are all the same.

Is a swallow study the same as an esophagram, and how is that different from a Barium contrast study?

Good question! All of these studies use barium. The modified barium swallow study evaluates a patient ability to swallow in the oral, pharyngeal and upper esophageal phases. A speech therapist will be present when this study is being conducted. A barium swallow study (AKA esophagram) assesses the rest of the esophagus. A barium contrast study will typically refer to an upper GI series or lower GI series.

(when discussing for suspected NAFLD) Just wondering if present on the exam, would it be best to pick US vs MRCP, if referral is not an option?

On the boards, choose ultrasound as the diagnostic test of choice for NAFLD. MRCP does not play a role in diagnosing NAFLD.

When do we do upper Gi series as a first line test?

Order an Upper GI series when you are worried about malrotation.

On page 223 there is a description of PFIC types. However, there isn't enough distinguishing information between type 1 and 2. How would they be tested on the exam? For PFIC3, at least the distinguishing factor is an elevated GGT, are there any other distinguishing factors?
Thank you

Typically, the boards wouldn't ask you to distinguish between the different types of PFIC. Type 2 tends to be worse clinically with liver related disease only and can lead to hepatocellular carcinoma. Type 1 patients have short stature, pancreatitis and liver failure late in life.

I think the risk factors on p. 171 of core and p. 28 of Q&A in 2022 PBR edition need to be clarified/corrected. I believe the thresholds for starting phototherapy are based on the NEUROTOXIC risk factors, not the HYPERBILIRUBINEMIA risk factors. Neurotox risk factors include G6PD, asphyxia, temp instability, sepsis, acidosis, albumin <3 but DOES NOT include ABO incompatibility, Rh dx, sibling w/ hx of jaundice, etc. So the paragraph is misleading b/c it suggests that all risk factors (whether neurotox or hyperbili) would mean you should use the high risk bilirubin thresholds for starting phototherapy when it fact its ONLY based on the neurotoxicity risk factors. You would use the hyperbilirubinemia risk factor to help you risk stratify and decide when baby should follow up. Please correct me if I am wrong about this. https://bilitool.org/

Thanks for reaching out! The Core Study Guide is correct in identifying the risk factors for hyperbilirubinemia. The threshold for starting phototherapy is based on the total level of bilirubin rise, regardless of cause or risk factors.

PHARMACOLOGY & DRUG PEARLS

How does anesthesia cause malignant hyperthermia? How do the treatments for malignant hyperthermia work/help?

Malignant hyperthermia is a known side effects of meds used for anesthesia. Kids with certain genetic conditions, like muscular dystrophy, are at higher risk of developing malignant hyperthermia. This is low-yield for the boards, so don’t worry about learning the mechanism of action for this condition.

What is the difference between Hepatic Inducers vs. Hepatic Inhibitors? Why are these facts important?

Hepatic Inducers are meds that work to increase metabolism of certain meds, which speed up how quickly these meds are being cleared. Hepatic Inducers decrease the efficacy of these meds. One type of medicine affected by hepatic inducers are OCPs.

Hepatic Inhibitors are meds that work to decrease the metabolism of certain meds, which decreases how quickly these meds are being cleared from the body. These meds inhibit the cytochrome P450 system.

Thanks to our PBR member who helped improve our mnemonic! Our racist trucker now drinks grapefruit juice!

HEPATIC INDUCERS

Hepatic inducers are medications that work to increase the metabolism of certain medications and thus LOWER their effectiveness. Examples include Carbamazepine, Phenobarbital, Phenytoin, Rifampin, and Saint John's wort.

PEARL: OCPs' effectiveness is DECREASED with these medications, so recommend a backup method of contraception.

HEPATIC INHIBITORS

Fluconazole (and other -azole), Isoniazid, Sulfonamides, and H2 Blockers (especially cimetidine) are hepatic inhibitors. So are grapefruit juice and erythromycin. Focus on remembering these P450 INHIBITORS more than the inducers.

MNEMONIC: (It's PG-13) Imagine a racist trucker sitting in a diner drinking grapefruit juice. He sees a THAI DINEr sitting in a booth talking very loudly on her SUL-FONE. He gets pissed! He gets so angry that he turns as cold as ICE. He then climbs up onto a BLOCK of wood, bends over, and starts shooting cubes/BLOCKS of ICE out of his AZOLE. His aim is pretty good, and he completely knocks the SUL-FONE out of the THAI DINEr's hand!

* **KEY**:

- THAI DINEr should help you think of "ti-dine," as in cimeTIDINE
- SUL-FONE (cell phone) should remind you of SULFONamides.
- BLOCK might help you remember that this mnemonic deals with BLOCKING of Cytochrome P450.
- ICE should help you remember ICE-oniazid/isoniazid.
- AZOLE should help you remember fluconAZOLE and itraconAZOLE.

OPHTHALMOLOGY

pg 244 in 2022 book - What is the difference between Hordeolum and Chalazion? Do they both affect the meibomian gland? How can we tell which is which based on the image?

Hordeola tend to come on faster and they look “angrier” (more erythematous) because they are due to **inflammation** of an oil gland. The oil gland can be meibomian gland, but it’s much more commonly the one of the “glands of Zeis.” Hordeola can be sterile or infected (typically with Staph).

Chalazions come on more slowly, are less painful (or painless), are larger and can have no erythema. They usually occur in the upper eyelid. They are due to **blocked** meibomian glands. Below is our update for the Core Study Guide.

HORDEOLUM (AKA STYE)

A hordeolum (AKA stye) is a **red** and **painful** eye lesion noted at the rim of the eyelid (near the eyelashes) due to **inflammation** of the meibomian gland. They can be sterile or infected (typically with Staph). Prescribe warm compresses and possibly topical antibiotics as well.

PEARL: Do NOT prescribe oral antibiotics.

MNEMONIC: ho-**RED-**e-**OWE**-lum should remind you that this is a RED and PAINFUL eye lesion.

IMAGE: www.pbrlinks.com/HORDEOLUM1

CHALAZION

A chalazion is a much slower-growing, painless lesion that results from **blockage** of the meibomian gland. There can be erythema as well, but it is much less painful. It resolves on its own, and warm compresses can help.

PEARL: Ophthalmology should be involved if it is chronic or interferes with a patient’s vision.

MNEMONIC: “che-LAZY-on” the eye. This is the slow-growing, LAZY eye lesion.

IMAGE: www.pbrlinks.com/CHELAZION1

Why does the "red reflex test" look red for normal, but white for abnormal? Does it have to do with visualizing the blood vessels?

Light reflects off of the retina and gives a red/orange glow (like in nighttime pictures with a flash). That is normal. If that’s not present because something is obstructing the light on its way to the retina, that’s NOT normal and we get a white reflex. That’s bad (possible retinoblastoma getting in the way).

GENETICS & INHERITED DISEASES

pg 261 to 262 of 2022 book - What are the three ways to get Angelman syndrome vs Prader-Willi Syndrome?

Maternal imprinting occurs in Angelman syndrome. These kids have seizure disorders, spasticity, inappropriate laughter, severe mental retardation and absent speech. **P**aternal imprinting occurs in **P**rader-Willi syndrome. Please see below for the ways to get Angelman syndrome.

ANGELMAN SYNDROME (AKA ANGELMAN'S SYNDROME)

Angelman syndrome (AKA Angelman's syndrome) patients can be MALE or FEMALE so don't let the name fool you! These patients tend to be happy, "angelic" and laugh frequently. They are developmentally delayed, and have delayed speech. They are easily excited, can have seizures, and are ataxic with what is referred to as a "puppet gait." The disorder results from the absence or the dysfunction a gene on the MATERNAL copy on chromosome 15. It's a special type of gene. In normal children, both maternal and paternal copies function but they have DIFFERENT effects. If the maternal copy is MISSING, or dysfunctional, or if it has a mutation that makes it behave like the PATERNAL gene (called paternal imprinting), then Angelman Syndrome develops. Also, if the child gets TWO paternal copies of chromosome 15 (paternal disomy) and NO maternal copies of chromosome 15, then Angelman Syndrome develops. The gene location is probably not important for the test, but the fact that it is KNOWN means that this disorder can be diagnosed by FISH.

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

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

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

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

PRADER-WILLI SYNDROME (AKA PRADER WILLI SYNDROME)

Prader-Willi syndrome (AKA Prader Willi syndrome) patients can have hypotonia (floppy baby), mild intellectual disability, almond-shaped eyes (often with mild strabismus), small hands, a HUGE appetite, obesity, and small testicles/penis in boys.

* **PEARLS:** Like Angelman's, it can be found in BOYS or GIRLS. This mechanism of this disorder is the mirror image of that of Angelman's. It occurs due to the absence or dysfunction of the PATERNAL copy of a gene in the same region of chromosome 15 as in Angelman syndrome. It also occurs when two maternal copies of chromosome 15 were received and no paternal copies (maternal disomy), and also

when a mutation of the paternal gene makes it behave like the MATERNAL gene (called maternal imprinting). Symptoms are much milder in females. The gene location is probably not important for the test, but the fact that it is KNOWN (15q11-13) means that this disorder can be diagnosed by FISH.

* **IMAGE**: www.pbrlinks.com/PRADERWILLI1

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

* **MNEMONIC:** Imagine a FAT Will Smith with TINY HANDS shoving tons of ALMONDS in his mouth. He's wearing a t-shirt that says, "MOMMY'S LITTLE FATTY."

    - **KEY:** ALMONDS represent the shape of the eyes, and the t-shirt represents maternal imprinting.

* **MNEMONIC:** Sorry, this is a good mnemonic but not politically correct. Imagine a HUGE, OBESE, and DUMB FISH/WHALE named FREE WILLY with such a SMALL PENIS that you can hardly see it. It's so DUMB that it tried to jump over a dock, but ended up landing on it instead. Now this big DUMB FISH is stuck on the dock. He's HUNGRY. He's thrashing back and forth, and he's FLOPPING his tiny WILLY all around.

* **MNEMONIC IMAGE**: www.pbrlinks.com/PRADERWILLI3

    - **KEY:** FISH represents the mode of diagnosis, HUGE/OBESE represents obesity, DUMB represents intellectual disability, HUNGRY represents the insatiable appetite, and FLOPPING represents the hypotonia. In this mnemonic, you could also make Willy's eyes almond shaped and imagine that he has small fins.

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pg 256 of 2022 book - How and Why do Heinz bodies form in G6PD Deficiency?

Heinz bodies are clumps of denatured Hgb that are attached to cell membranes. They form when there is oxidative stress (exposure to medications, stressors, etc.).

Down syndrome :
Mom normal, dad normal.
If the risk of recurrence "1%+ age-related risk
What is that in a number?
How you measure the age-related risk?
If a mother has a child with Down syndrome, it sounds like the risk of recurrence is 1% plus the age-related risk. How do you measure the age-related risk?

I think we are talking about 2 different "risk categories" here. With increasing maternal **OR** paternal age, the risk of Down syndrome increases.

If a child has Down syndrome, you need to do a chromosomal analysis of both parents. Based on what "mutation" is found, the risk of SUBSEQUENT pregnancy is as follows:

- If the mother is a 'balanced translocation' carrier to another chromosome (usually 13, 14, 15, 22), then the recurrence risk is about 1 in 8.

- If the father is a 'balanced translocation' carrier to another chromosome (usually 13, 14, 15, 22), then the recurrence risk is about 1 in 40.
- If either parent is a 'balanced translocation' carrier to chromosome 21 (21/21) then the recurrence risk is 100%.
- If the chromosomes of both parents are OK, then the recurrence risk is about 1% if the mother is under 40 years of age and about twice that for a mother who is 40 and over. [Selikowitz, M Down Syndrome: The facts, 2nd Ed., 1997, p. 177]

I would recommend focusing on the below information in the Core Study Guide. This should be sufficient to answer any board questions on this topic.

DOWN SYNDROME (AKA DOWN'S SYNDROME)

The term Down Syndrome (AKA Down's Syndrome) specifically refers to having three Chromosome #21s but does NOT differentiate between someone having a TRISOMY 21 Down's (due to a nondisjunction during gametogenesis) versus a Translocation Down's. Down Syndrome is the most common cause of intellectual disability. Fragile X is the most common **inherited** cause of intellectual disability.

* **CLASSIC FINDINGS include** a rounded face, almond-shaped eyes with upslanting palpebral fissures, flat nasal bridge, macroglossia, a single transverse palmar crease (AKA "simian crease"), poor muscle tone (hypotonia), and a wide space between the first and second toes (AKA "sandal toe").

- **IMAGE**: www.pbrlinks.com/DOWNSYNDROME1
- **IMAGE**: www.pbrlinks.com/DOWNSYNDROME2

* **OTHER POSSIBLE FINDINGS**

The patient may also present with AV canal defect, VSD (**AV canal** >> VSD), atlantoaxial instability, small phallus (similar to Prader Willi), excess nuchal skin, clinodactyly (incurved pinkie finger), obstructive sleep apnea with right-sided heart failure, and mild intellectual disability. Patients can usually live independently. Males are typically infertile, while females are not. All patients have an increased risk of leukemia.

- **IMAGE**: (Brushfield Spots) www.pbrlinks.com/DOWNSYNDROME3
- **PEARL**: **Knowing the etiology of a patient's Down's is extremely important for the purposes of counseling**. Since this is such a confusing topic, if all else fails, just choose to do a karyotype on the baby or the parents and MOVE ON TO THE NEXT QUESTION.

* **TRISOMY 21 DOWN'S**: This is the most common form (95%) and is due to nondisjunction of chromosome 21. A karyotype of Mom and Dad's chromosomes is NOT needed because they would show normal chromosomes. The risk for recurrence of TRISOMY DOWN'S in future pregnancies is equal to the mother's age-related risk plus 1%.

* **TRANSLOCATION DOWN'S**: This is very confusing. Parental karyotyping IS needed. Please start by looking at the shortcut below. If that is enough for you, MOVE ON. Essentially, if the child's karyotype shows a translocation, get karyotyping of both parents' chromosomes. The carrier parent will have either the main part of chromosome 21 attached to a different numbered chromosome (usually chromosome 14, in which case this is a partial translocation), or the main part of one chromosome 21 will be attached to the main part of **another** chromosome 21. The second possibility may be shown as t(21q;21q) and carries a 100% risk of future Translocation Down Syndrome babies because that parent

only has that chromosome (21 attached to 21) to donate. The first possibility (in which 21 is attached to 14) carries about a 10-15% chance of future Down's pregnancies if the MOM is the carrier, and about a 5% if the DAD is the carrier. If you take the time to work out the actual inheritance pattern, it's theoretically 33%. But nature doesn't seem to care. In any translocation carrier, that person's siblings should also be given the opportunity to be screened.

* **SHORTCUT**:

Trisomy 21: Age related risk + 1% is the risk of future Down's pregnancies.

Translocation in which the parent is carrying a 21 attached to 21: 100% risk (all future children will have Down's).

Anything else: < 15% Risk

LINK: www.pbrlinks.com/DOWNSYNDROME4

SUMMARY TABLE:

	DAD	MOM	Risk of Recurrence
Trisomy 21	Normal	Normal	1% + Age-related risk
21/14	Normal	Carrier	10-15%
	Carrier	Normal	5%
21/21	Normal	Carrier	100%
	Carrier	Normal	100%

** DOWN SYNDROME HEALTH SUPERVISION*

Children with Down syndrome often have, or acquire, multiple health issues that can include vision problems, thyroid disease, diabetes, celiac disease, seizures, heart disease, hearing loss, sleep apnea, hematologic disease, atlantoaxial instability, and others. Keep an eye out anything in the history of a Down Syndrome child that might warrant further workup in a question asking you about the best "next step" in management.

** ATLANTOAXIAL INSTABILITY IN DOWN SYNDROME*

Suspect this in any Down syndrome patient with a history of a gait disturbance or any other neurologic signs. Reflexes may be brisk. There is no role for routine screening with X-rays in asymptomatic Down Syndrome patients. This diagnosis can be a tricky to make because there is atlantoaxial INSTABILITY between C1 and C2, so the X-ray findings can change over time from normal to abnormal and vice versa. Therefore, if you suspect the diagnosis, don't be shy about repeating an X-ray, or even getting an MRI (required if there are clear symptoms).

Klinefelter:
What is considered a low upper to lower segment ratio in number:
1:1.3? 1:1.7? What is the cut off?

As many things in life, “it depends”. There are cut offs for birth vs 5 years vs 10 years vs post-pubertal state. Remember that the ratio needs to be **LOW** (need to have LONG legs) and thus you might be given a ratio in <1.0 range in order to diagnose the Klinefelter syndrome.

HEMATOLOGY & ONCOLOGY

What is the TIBC in Iron Deficiency Anemia vs Anemia of chronic diseases?

For iron deficiency anemia, the TIBC is high, with low iron saturation, low total iron value and low ferritin. With anemia of chronic disease, the TIBC can be high or close to normal, but will still have mildly low iron saturation and low total iron. The ferritin will be low as well, but it can be falsely elevated if there is an inflammatory condition occurring.

ITP: Page 284-285 states that IVIG should be used over steroids as first-line treatment of ITP with evidence of bleeding. However, MedStudy and the updated Hematology guidelines recommend steroids first.

In the Core Study Guide, we list IVIG and steroids as possible first-line treatments of ITP. Yes, you are correct that more updated guidelines say that steroids are okay, but you need to make sure that the CBC and all the cell lines are normal. If the patient has early leukemia and receives steroids, they are now elevated to a high risk protocol. So give steroids first ONLY if you are absolutely certain that the patient has ITP (where ONLY the platelets are low). If any other cell lines are affected, you should start with IVIG. Make sure to read the question stem carefully.

Ewing's sarcoma: p. 271 Typical location of Ewing’s sarcoma--does it typically present in epiphysis and diaphysis but typically not metaphysis?

Yes, these are the typical locations of presentation. However, in real life this isn't always the case.

IDA question: p. 280 Do I have it correct that the retic is low in iron deficiency anemia b/c there is not enough building blocks to make RBCs? So the BM cannot respond appropriately to make new RBCs? The fact that the retic is low is ABNORMAL b/c one would expect the number to be higher but the body is not able to respond to anemia appropriately?

Yes, you’re right! In iron deficiency anemia, the retic count will be low as the body doesn't have enough iron to make more RBCs. If we are seeing anemia due to hemolysis (breakdown), the retic count will be high as the body is trying to compensate for low hemoglobin.

Polycythemia question: p. 275 Why does thrombocytopenia occur in polycythemia?

Thrombocytopenia may or may NOT occur with polycythemia. If it's present, think of the mechanism (which we will not go into here) in this mnemonic format: There are too many RBC-producing cells in the marrow and it's leaving no room for the platelet-pro cells.

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pg 276 of 2022 book - How does G6PD deficiency cause oxidative injury?

The reductive power of NADPH, which relies on G6PD enzyme, is protective. So if you are deficient in the G6PD enzyme, you aren't able to reduce the oxidative injury to the cells. However, this question is beyond the scope of the boards. Just focus on how to diagnose/treat this condition and provide education to the families.

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pg 281 of 2022 book - Why is there basophilic stippling in beta thalassemia major? How is it different from the basophilic stippling seen in lead toxicity?

This is too detailed for the boards. We'd like to suggest that you avoid going to this level of depth.

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pg 288 of 2022 book - How does DDAVP help Von Willebrand Disease? Why is vWD associated with menstrual issues in some clinical scenarios?

Thanks for asking! DDAVP temporarily increases vWB antigen and Factor 8. Since this is a bleeding disorder related to clotting, heavier menstrual cycles can occur and those are often the times when this is diagnosed.

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pg 283 of 2022 book - What is the difference between Fanconi Anemia vs. Diamond-Blackfan Anemia? What is aplastic anemia?

Fanconi anemia is an **inherited** formed of aplastic anemia affecting all cell lines AND other skin and skeletal manifestations. So look at the cell lines, the family history and/or possibly some additional genetic defects.

Aplastic anemia is basically bone marrow failure, which causes lack of production of all cell lines. This is not necessarily an inherited disorder, so it is less likely to be related to any family history and/or other genetic disorders.

Diamond-Blackfan anemia ONLY AFFECTS RED BLOOD CELLS with other extraneous manifestations. Usually this is diagnosed at a much younger age because patients are not making RBCs.

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pg 282 of 2022 book - What is Pernicious Anemia and how does that cause vitamin B12 deficiency?

Vitamin B12 deficiency is usually due to an issue with absorption (either missing parietal cells or intrinsic factor or surgery). Pernicious anemia specifically refers to the cause being a parietal cells in the gut, leading to lack of production of intrinsic factor. You need intrinsic factor to absorb vitamin B12. Intrinsic factor deficiency leads to vitamin B12 deficiency, thereby causing anemia.

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pg 273 in 2022 book - Why does Langerhans Cell Histiocytosis (LCH) have papular/petechial rash in the natural body folds and why is LCH associated with Diabetes Insipidus?

We're not sure why those specific areas are affected (possibly more histiocytes in those areas). Regarding DI, there's an infiltration of the histiocytes in the pituitary gland and that causes dysfunction.

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pg 272 and 273 PBR 2022 edition--These statements are made:

Brain tumor is the most common solid tumors found in pediatrics.
Neuroblastoma is the most common extracranial solid tumor in childhood...commonly presents in the abdomen...
Wilms tumor is said to be the most common abdominal malignancy in kids.

Can you explain how these tumors are related and what exactly what is being said about them?

They are all solid tumors. The most common one is in the brain, the most common one outside of the cranium is neuroblastoma and the most common one in the abdomen is Wilms tumor. In terms of your differential diagnosis, look at the entire vignette. If there are only head symptoms and you're thinking cancer, think of a brain tumor. If the patient is generally healthy, but now has an abdominal mass, think Wilms tumor. If the child is sicker (vomiting, not eating), has an abdominal mass and you're thinking cancer, think neuroblastoma because these kids are sicker.

Clarification required for page 270. If a patient has nontender lymph nodes and B symptoms, is the next step an excisional node biopsy or a PPD?

You need to look at the whole picture (history, physical exam and testing results) to determine if this seems like a neoplastic or infectious process. In general, go for the less invasive testing first.

INFECTIOUS DISEASES

VZV ppx - p. 320 for Varicella zoster and ppx, is the reason that no ppx is needed if sx in mom began 6 days PRIOR to delivery b/c it is too late for ppx or b/c the likelihood of transmission is low?

No prophylaxis is needed because the likelihood of transmission is low. By then, the lesions start to crust over and the likelihood of transmission of the varicella virus to the baby is significantly lower. Below is our update for the Core Study Guide to clarify this point.

(DOUBLE TAKE) VARICELLA ZOSTER VIRUS (CHICKEN POX)

The varicella zoster virus causes CHICKEN POX. A chicken pox lesion may be described as a "dew drop on a petal" during the vesicle phase. Lesions are said to come in "crops" at different times, and will therefore appear in different stages on the body (some vesicles, some crusted lesions). The rash goes to the **TRUNK and then to the FACE and EXTREMITIES**. It lasts for 7–10 days and leaves minimal scars.

PEARL: VZIG (VZV immunoglobulin) is given for prophylaxis to newborns if the mom developed symptoms within FIVE days prior to delivery and TWO days after delivery. If symptoms started six days prior to delivery, NO PROPHYLAXIS IS NEEDED because by this time the likelihood of transmission is low. Congenital varicella syndrome can result in low birth weight as well as CNS, eye and skin abnormalities.

PEARL: Any immunocompromised patient should avoid contact with patients who have a case of the chicken pox.

NOTE: It's doubtful you will need to know about the smallpox virus (Variola). In case you do, just know that the lesions all appear at the SAME TIME, so all lesions will look similar. Other facts include –> limited to face/extremities, lasts up to 3–4 weeks and leaves lots of scarring. (Why couldn't it be limited to the trunk and leave scars over there instead?)

(DOUBLE TAKE) MNEMONIC: Imagine a patient who is bored because he's stuck in a NEGATIVE PRESSURE ISOLATION room, and the only channel he gets to watch is **MTV**. Negative pressure isolation is required for **M**easles, **M**ycobacterium **T**uberculosis and **V**aricella. For VZV, droplet precautions are sufficient if only one dermatome is involved. As mentioned in the Aspergillus section, that, too, requires negative pressure isolation.

Erythromycin vs azithromycin question - p. 290 Under erythromycin section it says to use azithromycin b/c of the risk of erythromycin and pyloric stenosis, but on p. 300 it says azithro also increases the risk. Which is correct?

BOTH are associated with pyloric stenosis, but the risk is higher with erythromycin. If the baby is less than 6 weeks old, use azithromycin instead. Below is our update for the next edition for the Core Study Guide.

PYLORIC STENOSIS

Pyloric stenosis results from a gastric outlet obstruction due to a thickening or elongation of the pylorus. Look for **NON**-bilious, projectile emesis in a HUNGRY child. Labs may reveal a hypochloremic **hypOkalemic** metabolic alkalosis and possibly an elevated indirect bilirubin. An upper GI series may show the "string sign" or "railroad track "or "double track" sign. The railroad track sign is due to two lines of contrast created by thick muscle, with a connection due to contrast in rugae. Diagnosis is made by ultrasound showing a pylorus that is **> 14 mm long** or **> 4 mm thick**.

* **SIDE NOTE**: Alkalosis is initially from vomiting out HCl. As the patient becomes dehydrated, there is a superimposed contraction alkalosis. Additionally, hypokalemia results in renal wasting of H+ and K+ ions. This results in even more alkalosis and potassium losses.

* **PEARLS:** If you see a normal potassium level in a patient with pyloric stenosis, know that the total body potassium is still low. If the serum pH is normal or acidotic, it is NOT pyloric stenosis. This occurs in boys > girls.

* **PEARLS:** Erythromycin and azithromycin are BOTH associated with an increased risk of developing pyloric stenosis, but the risk is HIGHER with erythromycin (especially during the first two weeks of life). Give azithromycin when the baby is less than 6 weeks old**.**

* **IMAGE**: (Railroad Track) www.pbrlinks.com/PYLORIC1

* **IMAGE**: (String Sign) www.pbrlinks.com/PYLORIC2

* **MNEMONIC**: **4**yloric stenosis, 1**4** mm, and **4** mm. Remembering the diagnostic criteria can be tough. Use "**4**yloric stenosis" to help you.

Rabies

Why does the management for Rabies change for domestic pets vs wild animals?

Does the onset of symptoms in pets tend to precede the onset of symptoms in humans, considering the very high mortality, I just thought would be safer to give the vaccines + HRIG. Thanks.

Unlike wild animals, domestic pets are usually vaccinated, so domestic pets are at low risk for being infected with rabies.

The incubation period is much shorter in animals. For animals, it can be just days, whereas in humans it can be months or even years. So, it's okay to monitor the animals for 10 days to see if there are any behavior changes.

Neonatal sepsis - On p. 316 in PBR it lists that the most common causes of neonatal sepsis are E. coli and GBS with E. coli being the most common. Where does Listeria fit in?

Listeria is the third most common cause of neonatal sepsis. The most common cause is E. coli, followed by GBS. Bacteria causing early-onset sepsis (from 0-7 days of life) is usually acquired perinatally. From

8-28 days of life, the infection is more likely to be community acquired. Keep in mind that GBS sepsis can still occur after antibiotic prophylaxis due to colonization. Early-onset GBS sepsis causes bacteremia, whereas late-onset GBS sepsis usually results in more localized infections, such as meningitis or osteomyelitis.

Is Rifampin prophylaxis different for H.Inf vs N.meningitidis? Please clarify

Yes, they are different. Rifampin prophylaxis is given over 2 days for N. meningitidis and over 4 days for H. flu. However, this is beyond the scope of the boards. Please don't memorize doses, frequencies or durations of therapy.

What are the HIV testing intervals in infants born to a mom with HIV?

These babies are tested at birth, age 14-21 days, age 1-2 months and lastly at age 4-6 months. If all testing is negative, no need for further testing. Below is our latest update for the Core Study Guide on this topic.

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

Consider HIV in any child with cognitive impairment, FTT, opportunistic or frequent infections, thrush, hepatosplenomegaly, fevers, night sweats, and weight loss. It can present in the first year with HIGH levels of immunoglobulins (which are dysfunctional) and later in life with abnormally LOW immunoglobulins. CD4/helper T cell count should be LOW. The primary mode of transmission for kids is vertical. Pregnant women with HIV should get antiretroviral therapy (ART) with **Zidovudine or Nevirapine** to possibly prevent the vertical transmission. For higher risk babies (born to a mother with no prenatal care, inadequate ART, new HIV infection during pregnancy, detectable viral load at the time of delivery, etc.), get an immediate **DNA PCR** (**not** RNA PCR, which is for a viral load) at birth and at age 2-3 weeks. For lower risk babies, testing can start at age 2-3 weeks. All of babies born to mothers with HIV get 2 additional tests. One at the 1- or 2 month well-child visit and another at the 4- or 6-month well-child visits. If all are negative, the patient is probably negative. If any are positive, **repeat** to confirm.

* CHEMOPROPHYLAXIS: For babies born to mothers who did not get **Zidovudine or Nevirapine** for prevention of vertical transmission, start Zidovudine (AZT) within 72 hours of birth (preferably within 12 hours) and continue for 6 weeks. If four days have passed, it's too late and no prophylaxis is given.

* HIV TESTING: Maternal **antibodies** can persist for up to 18 months. Therefore, antibody tests are **screening** tests in infants and young children. The enzyme immunoassay (EIA) is an antibody test. EIA, or the "rapid antibody test," should be done in children of mothers with HIV at 12-18 months of age to ensure that the maternal antibodies have cleared. If the result is positive, then a confirmatory test which looks for HIV genetic material should be done. A Western Blot, immunofluorescence assay (IFA), nucleic amplification testing (NAT) or polymerase chain reaction (PCR) are all possible answers for this age range on the ABP exam. In older children, antibody testing may be used for diagnosing HIV.

* BACTRIM PROPHYLAXIS: Once a child is diagnosed with HIV, start PCP (AKA PJP) prophylaxis regardless of what the CD4 count is.

* NEEDLE STICKS: If you get stuck with a needle from an HIV patient, you get a 2-drug or 3-drug regimen for prophylaxis. A 3-drug regimen is generally preferred: tenofovir/emtricitabine PLUS dolutegravir OR raltegravir.

* VACCINATIONS: MAY GIVE MMR, VZV, and FLU vaccines as long as there is only **mild** immunosuppression.

* DRUGS: It's doubtful the certification exam would ask you about a drug regimen, but the boards might ask about HIV medication side effects. They include pancreatitis, Stevens-Johnson syndrome, liver toxicity, pneumonitis, CNS effects (neuropathy), neutropenia, and renal problems (insufficiency and stones).

  - **PEARL**: The "d" drugs (ddI & ddC) cause pancreatitis.
  - **MNEMONIC**: Just imagine flipping the "d" upright to make a "p" for pancreatitis!

PBR p294 -"ALWAYS get a culture" - Even when rapid strep is positive?

If the rapid strep test is positive, then you do not have to do throat culture. However, the throat culture should be done if the rapid strep test is negative since the rapid strep test is not sensitive enough and does not rule out GAS infection. Thank you for this question. Here is the updated version to help clarify.

STREPTOCOCCAL PHARYNGITIS (AKA STREP PHARYNGITIS or STREP THROAT)

To identify Streptococcal pharyngitis, look for fever, lymphadenopathy, sore throat, erythematous, or exudative tonsils and the ABSENCE of coughing, sneezing, and rhinorrhea (those are viral symptoms). The pharyngitis is due to Group A Streptococcus and usually resolves in 2–5 days. Diagnose by CULTURE. If a rapid Strep is positive, TREAT. If negative, send for a CULTURE. The only reason Strep throat is such a big deal is because it can lead to **RHEUMATIC FEVER, which is PREVENTABLE**! Treat with **PENICILLIN** or **AMOXICILLIN**. If allergic, use erythromycin or clindamycin.

PEARLS: Antibiotics are NOT given to shorten the course of the illness. They are given to prevent RHEUMATIC FEVER! **Even given 9 days after the onset of symptoms**, RHEUMATIC FEVER can be **prevented**! ALWAYS get a culture if the rapid Strep test is negative. Consider treating if you have a strong clinical case or suspect poor clinical follow up. Rheumatic fever is **NOT** due to GAS skin infections!

PBR p305 - "If mom is asymptomatic AND has a negative chest X-ray, check a PPD in the baby every 3 months. How many times? for how long?

If the mom has latent Tb, you do not need to do any work up for the baby since the mom is not contagious. However, if the mom has active Tb, then you have to do a work up for congenital Tb, which included gastric aspirates and LP. If the work up is negative, you start the baby on isoniazid due to Tb exposure from the mom. Check the PPD for the baby at age 3-4 months. If the PPD is negative, then you continue isoniazid until age 9 months. If the PPD is positive, then you have to repeat the work up for congenital Tb.

PBR p 308-309 - EIA, or the "rapid antibody test", should be done in children of mothers with HIV at 12-18 months to ensure that the maternal antibodies have cleared. If the result is positive, then a confirmatory test.....should be done" Confirmatory test is Western Blot, IFA is also confirmatory, NAT and PCR are also confirmatory?

Use the HIV RNA or DNA NAT test to confirm HIV.

PBR p 326 - Regarding FPIE -"SYMPTOMS include diarrhea +/- emesis and possible BLOODY stool while on formula, but not on clears or not-whole-protein-containing formulas.
What do you mean by "clears", pedialyte

Clears include Pedialyte or any other fluid/food you can see through.

VACCINES, IMMUNIZATIONS AND CONTRAINDICATIONS

ID-vaccines/measles exposures

Can you clarify these scenarios based on table in pg 337 (clarify the table)

If 13 month old got MMR at 12 months and was exposed to measles 2 days ago, they should receive MMR and they are done with the series or need additional routine dose at 4 years of age?

Or no vaccination now since they already received 1st dose of MMR at least 28 days ago?

If patient is 13 months, it has been 4-6 days after exposure and already received MMR #1, they receive no prophylaxis?

Approach these situations by first determining whether or not the patient has immunity against measles. if the answer is yes, then there is no need to give immunoglobulin.

To have lifetime immunity, two doses of the MMR vaccine need to administered, with a minimum of 28 days in between the two doses. In this scenario, the 13 month old needs two doses in its lifetime. Give the second MMR dose now, and do not give an additional dose at age 4 years. IVIG is not given in this case because the child had already gotten the first MMR vaccine dose. Keep in mind that the MMR booster does not count if the baby is below age 1 year.

Vaccine question

- Pg 332 vs 333, it seems they are saying different things in terms of when we hold live vaccines after glucocorticoid use. Is it >20 mg/d(or >2 mg/kg/d) and > 14 days or either or(ie >20 mg/d or > 14 days). Seems to be a discrepancy in the book. Thanks.

It has to be BOTH (>14d and >20 mg/d).

Vaccine question

- Could you also clarify the absolute contraindications of holding DTaP vs relative contraindications? Thanks.

Absolute contraindications are when we cannot administer the DTaP vaccine at all because something happened, such as a severe allergic reaction to a vaccine component and encephalopathy not attributable to any other cause within 7-10 days of vaccine administration.

Relative contraindications are simply precautions, where one must weigh the risks vs benefits. These include moderate-severe illness, progressive neurologic disorder (prior to the first DTaP given), uncontrolled seizure disorder, Guillain-Barre syndrome and hypersensitivity (whole leg or whole arm got red).

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pg 335 of 2022 book - If mom's Hepatitis B status is Unknown, i understand that we must give Hep B vaccine within 12 hrs of birth, but why do we wait to figure out mom's status to be HBsAg positive to then give the HBIG immunoglobulin before 7 days of life?

No, we don’t have to wait 7 days. We have a time frame of 7 days where we can still protect the baby by giving the HBIG. After 7 days, it becomes useless. Immunoglobulin can possibly cause volume issues, fevers or other issues. So it is best to determine if the baby really needs it or not by determining the mother’s Hep B status. The earlier that it is given to the baby, the better.

If baby is <2kg when the first Hep B vaccine is administered, then it does not count, and will need to be repeated. Give these babies HBIG and Hep B vaccine within 12 hrs of life.

INBORN ERRORS OF METABOLISM AND METABOLIC DISORDERS

pg 347 of 2022 book - what is "doll-like face" and how does GSD 1 cause this symptom?

In GSD 1 there is a high buildup of lipids in the body resulting in increased fatty deposits. This causes a rounded, or doll-like, face.

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pg 348 of 2022 book - For kids with PKU, why does tyrosine become an essential amino acid? What does it mean to be an essential amino acid? So treatment for PKU would be low phenylalanine but high tyrosine in the diet?

An essential amino acid is one that you must get through your diet. In PKU, tyrosine becomes an essential amino acid because phenylalanine can't be broken down into tyrosine. So, you MUST have oral intake of tyrosine. If a PKU patient is not getting enough tyrosine through their diet, they might need supplementation.

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pg 343 of 2022 book. How and why does L-carnitine help to treat organic acidemias?

These disorders result in a loss of carnitine through the urine. The L-carnitine is used to replete those depleted stores.

Fatty acid oxidation disorders: States that diagnosis requires carnitine and acylcarnitine levels (the levels are to be high? Low??).

This is very complicated and it depends on the disorder and/or its subtype. It can sometimes include ratios to help with specificity. For the boards, you'll likely only need to know that if you suspect a fatty acid oxidation disorder, the next step is to obtain these labs.

Isovaleric Acidemia: Why does isovaleric acidemia NOT present with lactic acidosis? What is the specific pathophysiological reason? (p. 344)

isovaleric acid is a metabolite of leucine, and is not part of the pathway leading to ATP production (pyruvate-lactate and Krebs cycle). Therefore isovaleric acid doesn't create lactate technically speaking (unlike MMA or proprionic acid). HOWEVER, if an infant has a severe exacerbation, with seizures, shock etc., then the baby WILL have a lactic acidosis from being in shock. This last point is unlikely to be tested on the boards though.

Urinary Reducing Substances: what are they, and when are urinary reducing substances testing used?

It's a poor screening test for inborn errors of carbohydrate metabolism. More testing would be needed to rule in/out specific inborn errors of carbohydrate metabolism. Here's the study guide topic for your review:

URINARY REDUCING SUBSTANCES

This is an old test that looks for sugars in the urine to screen for possible inborn errors of carbohydrate metabolism. In many labs, all pediatric urine samples with no glucose found on dipstick will reflexively check for other sugars in the urine with this test. The specificity is poor, newborn screens are better for detecting possible inborn errors, and additional, confirmatory testing is needed if this test is positive.

ACID-BASE DISORDERS AND ABGS

One question regarding acid-base disorders, specifically calculation of the delta-delta gap: when calculating for an overarching anion gap acidosis, should we expect that the delta bicarb be exactly equal to the delta gap, or just approximate (within 1 or 2)? Acid-base calculations are so often considered to be approximate (e.g. sometimes we're told normal pH is "around" 7.4; it's NEVER entirely clear what testers consider to be a normal anion gap, etc). Calculating for this overarching anion gap, in particular, seems to rely on specific values of a "normal anion gap" and a "normal bicarb."

If the delta-delta gap is > 2, then you can diagnose an additional acid-base disorder. If the delta-delta gap is < 2 (for example, the delta anion gap=4 and the delta bicarb=5), then there is no need to diagnose an additional acid-base disorder. On the boards, they will make it more obvious (based on the numbers given) which scenario you are encountering.

Acid-Base - How does a patient clinically present with mixed disorder?

The answer is "it depends." There are so many different causes of acid base disorders that the presentation would be dependent on what is causing the individual disturbances. For example, imagine a diabetic gets a URI which triggers an episode of DKA **and** an asthma exacerbation. You'll have a mixed acid base disturbance with presenting symptoms of both illnesses. In other situations, there may not be any overt symptoms at all (e.g., acidosis from a mild AKI plus an alkalosis from antacids).

FLUIDS & ELECTROLYTES

pg 365 of 2022 book - what is the difference between heat stroke and heat exhaustion?

There is some overlap, but heat stroke is much more concerning than heat exhaustion. In heat exhaustion, there is normal mentation, or possibly mild confusion, that resolves promptly (within 30 min) with treatment and the temperature is less than 104'F. In heat stroke, the temperature is higher than 104'F, there is altered mentation, hypotension and possibly evidence of end organ damage on lab findings (e.g., liver dysfunction, kidney injury, muscle damage, etc.). When in doubt, if there's a high temperature around 104'F with altered mentation, treat as a heat stroke and provide rapid cooling and IVF resuscitation. Below are our Core Study Guide updates on these topics.

HEAT STROKE

Heat stroke is much more serious than heat exhaustion. Heat stroke results in a very HIGH temperature (> 105°F), altered mentation, hypotension and hot/dry skin. There is end organ damage from cytokine and endotoxin release. Give ICE packs to the groin and axilla for rapid cooling and IVF resuscitation.

HEAT EXHAUSTION

In heat exhaustion, the patient is **sweaty**. The skin is NOT DRY. There is normal mentation or mild confusion. Temperature is < 104°F. It's kind of like finding someone at the end of a really long jog (exhausted and sweaty). There can be some overlap with heat stroke, but if there's sweaty skin and a

temperature ≤ 104°F, treat with stopping exercise, removing clothing, moving to shade, and giving cold, electrolyte-rich liquids. Give an IVF bolus if at the hospital.

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pg 368 of 2022 book - How does hyponatremia affect 1) volume status, 2) serum osmolality, 3) urine osmolality, and 4) urine sodium? Would each of these be high or low, and why?

1) The volume status varies. There are many etiologies for hyponatremia, and those etiologies will dictate if the patient has euvolemic, hypervolemic or hypovolemic hyponatremia.

2) All things being equal, higher sodium means a higher osmolality. This can be seen through the equation for serum osmolality:

Serum Osmolality = 1.86(Na) + (glucose/18) + (BUN/2.8) + 9

The equation for serum osms shows that the value is driven mainly by serum sodium.

3) Urine osmolality is also dependent upon the etiology of the hyponatremia.

4) Urine sodium also depends on the etiology. In hyponatremic dehydration, the urine sodium is low since the kidneys are holding on tightly to the sodium. However, in hyponatremia due to diuretics or renal failure, the kidneys are unable to hold onto to the sodium and the urine sodium will be high.

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pg 369 of 2022 book - why is urine sodium high in Syndrome of Inappropriate ADH secretion (SIADH)? Excess ADH means more water retention in the body? What does the word "Inappropriate" refer to? Hyponatremia from retained water?

Yes, in SIADH, there is more water retention in the body and less water released in the urine. More water retention causes hyponatremia. Therefore, the relative amount of sodium in the urine is higher. “Inappropriate” in SIADH means that your pituitary gland is secreting inappropriately high amounts of ADH, especially in the face of euvolemia.

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pg 370 of 2022 book - How and Why does Central Diabetes Insipidus have Low urine osmolality?

Think of Central Diabetes Insipidus as the “opposite” of SIADH because there is a lack of ADH. This lack of ADH means that you are not holding onto as much water as you should. Therefore, you are eliminating large volumes of water, producing very dilute urine (with a low urine osmolality).

How does rapid correction of sodium cause central pontine myelinolysis?

Sodium is added to the system and the sodium concentration rises. The full mechanism is poorly understood, but the rapid shifts cause water to be pulled from brain cells. This process leads to central pontine myelinolysis.

Does ADH cause the kidney to hold onto water first or does it influence sodium in order to hold on to water?

ADH causes the kidney to hold onto the water first.

In diuretic use, if Na is being forced out and there is more Na, and therefore more osmoles, then how is it that urine has low osmolality?

As the sodium excretion into the urine increases, so does the water. The kidneys are not trying to maintain a certain concentration of sodium in the urine, so the amount of water that flows out can result in a lower osmolality than normal urine.

How do I know when I have to give 3% HTS or NS? Can you please elaborate on it.

Use normal saline for fluid resuscitation. Hypertonic saline is used in very specific situations (rapid correction needed to prevent seizures from severe hyponatremia) that you're unlikely to be asked about on the boards. Hypertonic saline is NEVER used for fluid resuscitation. Below is our update for the Core Study Guide to clarify this issue.

DEHYDRATION

If the patient presents with **mild to moderate dehydration**, oral replacement should be tried first if the patient can tolerate small amounts of fluid (preferably with something like Oral Rehydration Solution, which has 2% glucose + 90 mEq NaCl per liter). If admitting into the hospital, give MIVF + FLUID DEFICIT. Give half of the deficit over 8 hours and the rest over the next 16 hours. If the patient is 10% or 15% dehydrated, give boluses of fluid (use **normal saline**) first and then subtract the total amount from the boluses from the 8-hour fluids.

Why can diarrhea cause both hypo and hypernatremia?

There are many types of diarrhea (secretory, osmotic etc.). The etiology of the diarrhea can result in more, or less, loss of water and/or solutes.

GASTROENTERITIS and DIARRHEA: This usually does NOT cause a hyponatremia **until** the patient is only given (or is only tolerating) **hypotonic liquids** (like free water). The patient is volume depleted due to GI losses, so the kidneys hold onto as much sodium as they can. Therefore, the **urine Na+ is LOW** (very low, often < 10). If there happens to be a metabolic alkalosis from vomiting, then the urine sodium could be normal or high, but the chloride level will still be low (low yield).

HYPERNATREMIA

Hypernatremia is much less common than hyponatremia. Correct at no more than 12 mEq/L/day to avoid an intracranial hemorrhage due to fluid shifts resulting in the tearing of bridging blood vessels. The patient can also get pulmonary edema from fluid shifts. The most common causes of hypernatremia include diabetes insipidus (DI), excessive sweating, and increased intake. Hypernatremia can also occur due to diarrhea-related dehydration when more water is lost than sodium. If a patient is noted to have **hyper**natremic dehydration, assume s/he has at least 10% dehydration. Body fluids will be hypertonic with serum osmolarity often in excess of 300 mOsm/kg (300 mmol/kg).

On P. 364 for fluid deficit in L = % dehydration x Wt in Kg --> if 15% dehydration in a 10 kg child --> Can i confirm it means 0.15 x10 kg = 1.5 L?

Yup! That's the deficit, but don't forget about the maintenance fluids needed as well.

NEPHROLOGY

What is usefulness of osmolar gap? When do we use it?

A high osmolar gap can be caused by ingestion of: Methanol, Ethylene glycol, Diuretic, Isopropyl alcohol, Ethanol, and Sorbitol. Do the calculation when you are worried about an ingestion.

Is there a benefit in getting an ultrasound for workup of proteinuria after a 1st am proteinuria is +, and U prot/Cr is > 0.2. The book states go straight to renal biopsy, but wondering if an ultrasound is a bridging step between labs and biopsy.

It is not incorrect to do a renal u/s first to rule out structural anomalies. However, if the urine protein:creatinine ratio is greater than 0.2, then there is renal disease. The patient needs a renal biopsy for a definitive diagnosis. On the boards, choose renal biopsy as the answer. In this case, the renal u/s is a supportive test, not a diagnostic test.

How does a multicystic dysplastic kidney pt present?

This clinical scenario is not often seen in the pediatric world. More than 25 percent of these patients have urinary reflux, so they will get UTIs/pyelonephritis. Start them on antibiotic prophylaxis and then obtain a VCUG to evaluate for reflux and a renal u/s to assess for structural abnormalities. This is a unilateral disease, so one kidney has normal function, and the other's function is abnormal. These patients will have small electrolyte disturbances and an abnormal BUN/creatinine ratio. Once the abnormal kidney is seen on renal u/s, start antibiotic prophylaxis.

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p373 in 2022 book - what is "narrow" female urethra? Is there a measurement? How is that different from Labial Adhesion?

A narrow female urethra means that there is a minor stricture in the urethra. No intervention or workup is needed unless there is a slow voiding stream or difficulty voiding.

Labial adhesions involve the outer aspect of the female anatomy.

For prenatal hydronephrosis, do you do a postnatal US right away or wait a few months to see if it is persistent?

Do the ultrasound right after birth (instead of waiting) to assess what is going on with the hydronephrosis and to see if there is structural pathology or need for further intervention or workup.

Lupus Nephritis: p. 377-Where does lupus nephritis fall into? A glomerulonephritis? Is the mnemonic for MPGN saying lupus pts can get MPGN in addition to a lupus nephritis or is lupus nephritis the same as MPGN?

Lupus nephritis is a type of glomerulonephritis. Lupus patients can also develop MPGN. They are two separate entities.

Nephrotic Syndrome: p. 377 - Why does nephrotic syndrome present with low urine sodium and low FeNa?

In nephrotic syndrome, abnormal sodium and water retention occurs at the kidney level, ultimately causing expansion of interstitial volume and edema. The mechanisms and factors involved remain ill-defined.

IgA Nephropathy: p. 377 In IgA nephropathy, pts typically present with the proteinuria/hematuria DURING a URI correct?

It can occur during or after a URI.

STATISTICS

What is the difference between Cross sectional studies and Case studies?

CROSS-SECTIONAL: Looks at a specific group of people at one specific point in time.
COHORT: Studies a grouping of people over a longer period of time.
CASE: Usually looks at a single case (case study) or a series of cases (case series).

NEUROLOGY

Pseudo-Seizures: How do we differentiate true neonatal seizures from more benign movement disorders like benign sleep myoclonus? Are there other disorders we should be aware of that can mimic neonatal seizures, but are otherwise benign?

The way to differentiate between neonatal seizures and benign movement disorders is to obtain an EEG. There are no other benign disorders in neonates on differential diagnosis.

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pg 388 to 390 of 2022 book - What are the main differences between/among: Guillain-Barre syndrome vs. Cord Compression syndrome vs. Transverse Myelitis vs. Epidural Abscess of spine?

MRI findings and clinical findings are key in answering a question on this topic on the boards. In Guillain-Barre syndrome, rectal tone is maintained and bladder tone (urinary continence) is preserved. There are decreased reflexes and an ascending paralysis. In cord compression syndrome, bladder and rectal tone are lost, and there are increased reflexes. Also, these patients can present with fever, back pain and respiratory compromise. Clinical symptoms of traverse myelitis can mimic that of cord compression syndrome. Patients may or may not have chronic inflammation. On MRI, you will definitely will see inflammation on traverse/cross-section of spinal cord. In an epidural abscess, there will be an encapsulated finding on MRI. Lack of sensation, lack of rectal tone and lack of bladder control.

How is GBS different from Acute Flaccid myelitis? It seems the main differentiating factor is CSF pleocytosis in the latter, and absence of pleocytosis in the former?

These typically present in two different age groups and have different MRI/lab testing findings. GBS often presents in an older kid after a GI infection. These patients will develop paresthesias in the lower extremities, followed by ascending paralysis. The median age for acute flaccid myelitis is age 6 yrs. These patients will have hypotonia and difficulty with speech.

Here are the topics for your review:

GUILLAIN-BARRE SYNDROME (GBS, AKA ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY or AIDP)

Patients suffering from Guillain-Barre syndrome (GBS, AKA acute inflammatory demyelinating polyneuropathy or AIDP) may initially complain of back pain and **fever** and can have a facial palsy and proximal muscle weakness (trouble rising from a chair or shrugging shoulders) prior to lower extremity symptoms. Classically, though, it is an ascending paralysis over several **days to weeks** in which there is ataxia and then an inability to walk. Look for diminished or **absent reflexes in the lower extremities** on exam. **Sensation is preserved** (as is bowel and bladder continence). It can progress to respiratory compromise requiring intubation. Perform a lumbar puncture to look for **albuminocytologic dissociation (increased CSF protein in the absence of increased WBCs)**. FYI, they could say there is an absence of pleocytosis (pleocytosis means an increase in WBCs). For treatment, you can try IVIG or

plasmapheresis. The clinical diagnosis of GBS needs to be confirmed with CSF analysis and nerve conduction studies.

PEARLS: Steroids DO NOT help. Pulse oximetry is a poor indicator of neuromuscular respiratory insufficiency. You can, however, try to obtain a negative inspiratory flow (NIF) or a Forced Vital Capacity (FVC) if the child is old enough to participate with the test (at least 5 years of age). Always keep **tick paralysis** in your differential, especially if they mention the summertime, a recent vacation, or the woods! Additionally, if someone presents with GBS a few weeks after a diarrheal illness, they might be referring to C. jejuni infection (a known antecedent to GBS though the mechanism is not understood). Also, when compared to any **CORD COMPRESSION SYNDROME**, GBS maintains rectal tone, bowel/bladder continence, and sensation. It also has **decreased** reflexes. In cord compression syndromes, sensation, tone, and continence are lost, and reflexes are **increased**.

ACUTE FLACCID MYELITIS (AFM)

Acute flaccid myelitis (AFM) is uncommon and causes weakness and decreased reflexes. Cases have been increasing since 2014. Most cases occur in young children, with cases in children ranging from 3 months to 21 years, but with a median age of 6. Symptoms include hypotonia, hyporeflexia, difficulty moving eyes or droopy eyelids, facial droop or weakness, difficulty with speech or swallowing, pain in the arms and legs, and pain in the neck or back. Severe cases can result in respiratory failure and instability of body temperature and blood pressure. The etiology is uncertain but thought to be enterovirus D68. Diagnosis requires an MRI showing a spinal cord lesion primarily in the gray matter and CSF with pleocytosis. Additional body fluids are tested in search of viral etiologies. Treatment is supportive along with rehab including PT and OT.

ORTHOPEDICS & SPORTS MEDICINE

Spondylolisthesis - Can you explain where the abnormality in the 2nd spondylolisthesis image (http://www.pbrlinks.com/spondylolisthesis2) on p. 409 of PBR 2022?

This is an image of L4, L5 and the sacrum. Here, L5 is subluxed anteriorly on top of the sacrum, and L4 is subluxed anteriorly on top of L5. Basically, the whole area has slipped forward.

SPONDYLOLISTHESIS

In spondylolisthesis, a **slipped** vertebral body results in lower back pain. The problem and pain is usually around L5 or S1. Again, pain is **worse with standing**.

IMAGE: www.pbrlinks.com/SPONDYLOLISTHESIS1
IMAGE: www.pbrlinks.com/SPONDYLOLISTHESIS2 (copyright free)
MNEMONICS: Rename it spondylo**SLIP**thesis or spondylo**LISP**thesis.

Ortho - pg 404 of 2022 book - What is a supracondylar fracture? Why does it show a posterior fat pad? How does it cause compartment syndrome?

A supracondylar fracture is a fracture of the distal humerus just above the elbow joint. It usually is caused by a fall with an outstretched hand. Children will present with tenderness just above the elbow.

The posterior fat pad is located in the olecranon fossa. Normally this cannot be visualized on a lateral x-ray. When there is a supracondylar fracture, inflammation pushes the posterior pad out, causing a lucency on the x-ray (which you can now see).

in adolescents, you can also see the posterior fat pad with a radial fracture.

Compartment syndrome occurs when there is pressure on nerves/blood vessels. Typically these kids will have pain out of proportion to the exam. Assess if the hand is neurovascularly intact. If it is not, then the child needs to undergo immediate reduction of the fracture.

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pg 432 of 2022 book - what is the difference between Buckle fracture vs. Bucket fracture?

Remember that buckle fractures are also called torus fractures. In a buckle fracture there is a “wrinkling” of the lateral portion of the bone (where the spongy part of the bone gets compressed during an injury). In other words, no true break of the bone has occurred.

A bucket handle fracture is a little chip fracture in the metaphysis of the bone. This is a growth plate injury, and it is often seen in child abuse cases.

Topic reviews:

TORUS FRACTURE (AKA BUCKLE FRACTURE)

In a torus fracture (AKA buckle fracture), compression of bone via longitudinal force causes a fracture on one side that’s usually associated with a protuberance, but has NO fracture or deformity on the other side. It also does NOT go through the growth plate. It’s not that serious and is common in children due to their soft bones.
IMAGE: www.pbrlinks.com/TORUS1
IMAGE: www.pbrlinks.com/TORUS2
IMAGE: www.pbrlinks.com/TORUS3 (copyright free)

BUCKET HANDLE FRACTURES AND CORNER FRACTURES

Bucket handle fractures and corner fractures are **highly specific for abuse**. They are fractures of the metaphysis caused by sudden pulling, which causes avulsions.
IMAGE: www.pbrlinks.com/CHILDABUSE2 – Bucket handle fracture
IMAGE: www.pbrlinks.com/CHILDABUSE3 – Corner fracture

NAME ALERT/PEARL: 

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

On page 406, under Infantile blount disease, it mentions that we need to differentiate it from genu varum. What are usually the main differentiating features in the question stem?

Genu varum, which is normal, usually improves and resolves by 24 months. Blount's disease is pathological. The bowing of the legs gets worse and does not improve. This is usually delineated in the question stem to differentiate between these diagnoses.

RHEUMATOLOGY

How is HSP different from IgA deficiency?

These are two different entities altogether. **Henoch-Schonlein purpura** is now known as **IgA vasculitis.** Patients present with a rash, abdominal pain, blood in the stool and intussusception. At first, the rash is blanchable, but then becomes purpuric. This is a clinical diagnosis, and usually kids do very well. They will require some monitoring.

JIA vs Lupus question - There seems to be a lot of overlap b/w systemic JIA and lupus. What are the key differences in clinical presentation from your perspective and how might a question writer want you to consider one dx vs the other? Also what would the 'first step' be in diagnosing systemic JIA?

One of the biggest differences between the two illnesses are the rash presentations: SLE rash (malar/butterfly rash) vs. JIA rash (silvery-white patches due to dead skin accumulation). Also, the timeframe of the diseases are different. Remember that symptoms must be present for at least 6 weeks to even consider JIA diagnosis. Kids with JIA are below the age of 16 years, and all other conditions have been ruled out. To diagnose lupus, multiple tests are conducted to rule in/rule out other diseases (blood./urine testing).

There is no real "first step" that I can think of, but ordering an ANA profile will at least start you on the path of doing the workup.

PULMONOLOGY

Pneumonia and CPT: Is chest physiotherapy indicated for pneumonia?

Chest physiotherapy is an adjuvant treatment for pneumonia, but not a required component of therapy.

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pg 422 of 2022 book - What is the difference between Obstructive process vs. Restrictive process? How do they look different on spirometry? If asthma is obstructive, what is categorized as restrictive?

In an obstructive process there is difficulty in pushing air out of the lungs because of something blocking the airway (inflamed airways in asthmatics, foreign body, etc.). A restrictive process prevents the lungs from inflating due to parenchymal disease. These lungs are stiff.

There are some good images **on this page**. Please do an on-page search for “plot out spirometry findings.”

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pg 421 of 2022 book - How and Why does persistent pulmonary hypertension occur?

The full name of this condition is persistent pulmonary hypertension of the newborn. A fetus has high pressure systems in utero because it doesn’t need to use the lungs. Vasculature is clamped down so that the blood can be shunted to the appropriate areas to get oxygenated. Once the baby is born, that pressure in the lungs should come down and allow blood to flow normally into the lungs. PPH occurs for various reasons (infections, abnormal development of the lungs, meconium aspiration, being LGA, etc.).

Asthma: What are the guidelines to decide if the patient has outgrown asthma?

Most sources say that asthma is a lifelong disease. Practically speaking, healthcare professionals may choose to stop prescribing inhalers once a child has not needed any meds in 2-3 years. Spirometry can also be redone to provide reassurance.

Is there a certain weeks of gestation for Palivizumab cut off?

According to the 2021-2022 guidelines, children who qualify for Palivizumab should be age 12 months or younger AND less than 29 weeks gestational age at birth.

Why doesn’t pulmonary sequestration cause the same symptoms(neonatal distress) as CPAM if the pathophys is so similar?

The pathophysiology is not similar because they are different types of tissue. Sequestration refers to lung tissue without a connection to the bronchial tree, whereas a CPAM is a pathologic mass. Both are prone to infection, but a CPAM is a mass that, depending on size, can physically impact the lower airways even at birth. They are both susceptible to recurrent infections later on in life.

PSYCHIATRY & SOME SOCIAL ISSUES

pg 429 of 2022 book - is it possible to have some of the stages/emotions of grief at the same time? How do children process and let go?

In real life, yes this is possible. However, for the boards, they would likely need to give you the description of a specific stage to think about. In the PBR Core Study Guide, there is a good summary of how kids at different ages process and let go of grief. For these types of questions on the boards, do not choose the option “reassurance” or “do nothing.” Instead, choose an answer that provides an intervention like counseling, which means that you are taking this issue seriously.

ETHICS IN PEDIATRICS

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

Think of futility as a concept that has to do with whether something is useless. Will it provide any benefit to the patient? If not, it's futile. So, if there is no data that a treatment will provide any benefit to the patient's disease process, that treatment is futile. Or, if a patient is legally dead, then all treatment is futile.

The idea of "quality of life" is very subjective. If someone is not brain dead and is minimally responsive, some may argue that the treatment of acne will not result in any significant increase in the quality of life. However, the family of the patient may have many reasons why they would disagree.

PATIENT SAFETY AND QUALITY IMPROVEMENT

pg 444 of 2022 book - What is the difference between Near Miss vs. Adverse Event?

A near miss event is a "close call" in which an error could have caused harm, but no harm occurred. For example, an MD orders the wrong dose of an antibiotic, but the pharmacy catches it and corrects it before the patient gets the medication.

In an adverse event, harm does occur to the patient. There are preventable adverse events (wrong medication given) and non-preventable adverse events (patient gets a medication they have never received before and they break out into a rash).

PEDIATRIC LAB VALUES

There were no Pediatric Lab Values clarifications for 2022!

PEDIATRIC VITAL SIGNS

There were no Pediatric Vital Signs clarifications for 2022!

QUESTIONS & ANSWERS BOOK

There were no Questions & Answers Book clarifications for 2022!

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1. Read the PBR "Exam Day" article. It's a MUST read. It will give you a great deal of insight into your exam day. I'll list the link at the end of the document.
2. **Go back to your core PBR study material!** At the end of the day, THAT is what will help you pass the boards.
3. If you're not a PBR member yet, this is a GREAT time to join!
4. If you're feeling pretty good about your pediatric KNOWLEDGE/CONTENT, then work on your TEST-TAKING STRATEGY by going through the CRASH COURSE on Test-Taking Strategies (it has been a HUGE HIT)!

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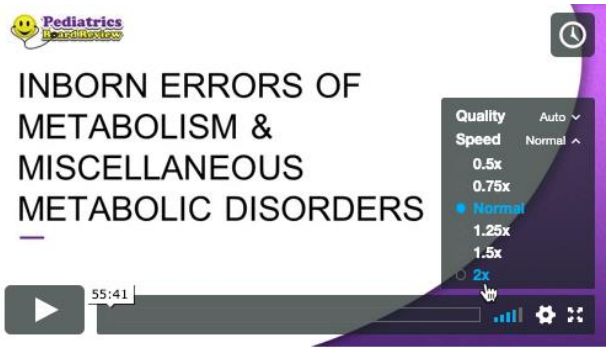

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PBR'S ANNUAL CORRECTIONS AND CLARIFICATIONS GUIDE

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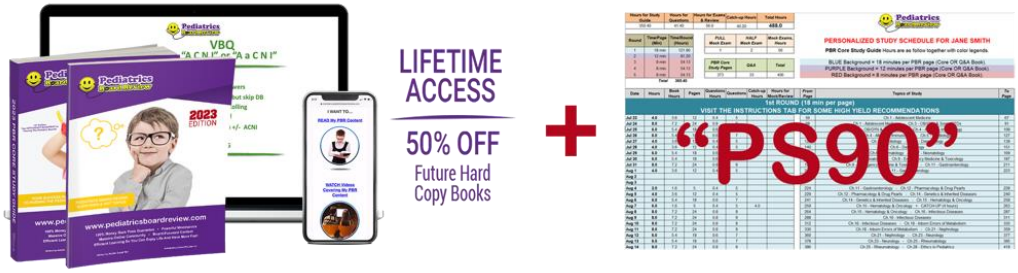

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Sincerely,

Ashish & Team PBR