Pediatrics Board Review

7th EDITION
Your EFFICIENCY BLUEPRINT To
Passing The Pediatric Boards

2017

EASY & EFFICIENT
LEARNING SO THAT YOU
CAN ENJOY LIFE & HAVE MORE FUN!

www.PediatricsBoardReview.com

• 100% Money Back Pass Guarantee •

Written by
Ashish Goyal, MD

PEDIATRICS BOARD REVIEW

Your Certification SYSTEM for

Passing the Pediatric Boards

• 100% Money Back Pass Guarantee •

• MASSIVE Online Community •

• Board-Focused, Manageable Content •

• Powerful Mnemonics •

EFFICIENT LEARNING So You Can

Enjoy Life & Have More Fun!

Written By Ashish Goyal, MD

Edited By Dr. Michael Blyth (A PBR Alum)

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INTRODUCTION TO THE PBR EXPERIENCE! (Please Read This!!!)

HI! My name is Ashish Goyal. I've been fortunate enough help thousands of pediatricians with their board review experience through the "PBR." I'm a double-boarded physician living on the most isolated landmass in the world, yet some of my greatest success stories come for pediatricians across the United States.

My favorite stories are those from pediatricians who had previously **failed 4–6 times before they found the PBR, but then passed** by using the PBR *Certification System*. Those wonderful success stories clearly show that the PBR system is **perfect for first-time AND repeat test takers**. While there are PBR digital, audio and video resources available to streamline and cement the core material, **the Core Study Guide and the Q&A book are at the center of the PBR system and they are essential** towards helping you pass your exam.

PBR is great for residents looking to boost their In-Training Exams (ITE), **for new pediatricians** taking their American Board of Pediatrics (ABP) initial certification exam for the first time, **for pediatricians who have failed** the initial certification exam, and **for busy pediatricians studying for their ABP Maintenance of Certification (MOC) exam**.

PBR is much more than a collection of study resources. It's a group experience and a **system** that provides you with **ALL** of the CONTENT, test-taking TECHNIQUE, GUIDANCE, and COMMUNITY SUPPORT that you need to pass your exam. You truly do NOT need any other board review book to pass your exam.

The national first-time pass rate is usually in the 75%**–**85% range for the (ABP) initial certification exam. By analyzing surveys, PBR's Money Back First-Time Pass Guarantee requests, and emails, we estimate **that PBR's first-time pass rate for the initial certification exams is at least 97%**!

For the ABP MOC recertification exam, the pass rate with PBR has been 100% for practicing general pediatricians (2011 – 2014, 2016), and very similar for pediatric subspecialists. In 2015, only ONE pediatrician failed on his first attempt at the MOC, and he admitted that he barely looked at the PBR resources.

WHY DOES THE *PBR CERTIFICATION SYSTEM* WORK?

EFFICIENCY THROUGH SYSTEMS AND INNOVATION

Most board review books and courses simply hand you a book and say, "good luck." That's how I studied for the USMLE exams, the pediatric board exam (twice) and the Internal Medicine board exam. **I was completely isolated!** After purchasing thousands of dollars of board materials, I was left to go through the books and video courses with no real guidance, no feedback from my peers, and absolutely no advice from the authors (besides a one-page preface).

Because of how excruciatingly painful that was, **I've create a community of pediatricians for you to study with and a blueprint** of what to study, how to study it and how to do so **EFFICIENTLY**!

In fact, **ALL of the PBR resources are created with *your time* in mind**.

* Will the resource be **easy to use**?
* Will it provide **more value** than existing resources AND provide that value in a **more streamlined** fashion?
* Can we make the resource **digital for easy access via smart phones and tablets**?
* Will the resource **reinforce the core concepts** laid out in the PBR and in the Q&A book **instead of overwhelming** with new concepts?
* Can we make the resource **portable** (e.g., audio or video?) so that it can be used at times when a physician, or a mom, or a dad, or a gym-enthusiast, would not normally be able to study?

PBR is a system unlike anything you have ever experience before in your medical career. The Core Study Guide is written in easy-to-understand language and provides you with hundreds of time-saving memory aids. The online systems allow for one-click access to hundreds of high-yield images across the web. The Q&A book has some of the highest yield and most board-relevant questions available.

You also have a ready-made study group of hundreds of pediatricians. It's called the PBR Facebook CREW, and it will help you **EFFICIENTLY blow past trouble spots in your studying**. Plus, if you see an error in the book, or if you would like to submit an official request for content clarification, you can simply submit the info to me through **PBR's error submission portal** (http://www.pediatricsboardreview.com/**error**). **Your submissions will likely be used to create a PDF response** that is made available to ALL PBR members in order to enhance the PBR experience for the entire PBR community.

All of these efficiency-focused systems **SAVE YOU OVER 100 HOURS OF TIME** and give you **flexibility in your life to enjoy your family, your friends, or to reinvest that time** into repetition of the PBR material.

A critical component of ANY individualized board review plan is to go through the study material MULTIPLE times. **PBR is concise, makes the learning manageable,** and will allow you to **feel confident on your test day** because of well prepared you are for your exam.

WHAT ARE THE 7+ RESOURCES THAT YOU HAVE ACCESS TO?

The **PBR Ultimate Bundle Pack** and the **ALL ACCESS PASS** packages have become our two most popular memberships. If you have one of these memberships, **please make sure you take advantage of all of these resources!**

1. **PBR'S COMMUNITY!** This includes the **MEMBERS-ONLY FACEBOOK CREW**, Ashish Goyal, "Team PBR" and PBR's summertime webinar content experts. JOIN THE CREW! Do not study in isolation! You have a community of pediatricians to support you. MANY members say this is one of **the most valuable components of the PBR system**. Studying for a board exam can be GRUELING, but having others to lean on for clarification, advice or just some moral support can make all the difference in your studying experience.

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10 hrs · Worcester, MA, United States

Made it!!! Thank you Ashish, PBR staff and Facebook crew for your help and support with the Boards!!!

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Carolyne

20 hrs

Passed! Thank you Ashish and PBR crew!

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Henna Qureshi

3 hrs •

God is so GREAT!!! After 4 failures I HAVE PASSED. **Ashish Goyal** i used your material for the first time this year and it was God sent. Thank you so much for your test taking strategies, your videos, your mp3s and for restoring the faith that after failure there can be success. Im here for any referrals you need or if any one who has failed wants help please feel free to msg me. THANK YOU THANK YOU THANK YOU

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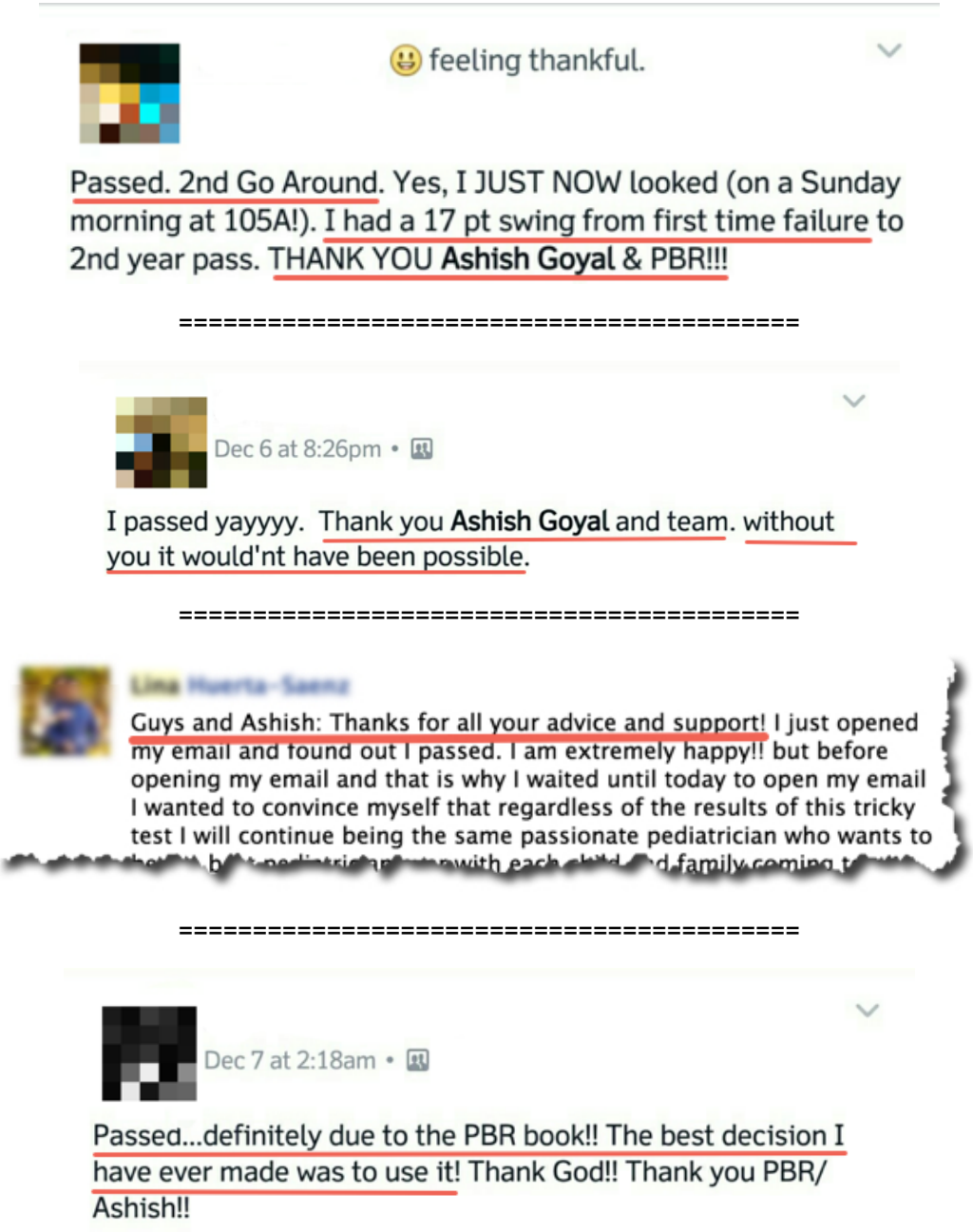

Visit the following link to read more: http://www.pediatricsboardreview.com/**facebook**

2. **HARDCOPY PBR CORE STUDY GUIDE**: YOU WILL LEARN TO LOVE YOUR "PBR!" It is at the center of your success blueprint. Carry it everywhere, highlight it, draw pictures, create mnemonics and add notes to help you cement the 2000 MUST-KNOW topics in this book. After your exam, I promise you that you will MISS IT!
3. **HARDCOPY PBR Q&A BOOK**: KNOW this book! It is NOT a random collection of questions. The material should be considered CORE material for you to study over and over again. Carry it around and mark it up! Make sure you review this book as many times as you review the Core Study Guide.
4. **ONLINE VERSIONS OF THE PBR CORE STUDY GUIDE**: All 2000 topics are available in a scrolling PDF style format and in a topic-by-topic, **searchable** format. Keep this open and use the **one-click**

image links while you study or after each two-hour block of studying. It's **iPhone/smart phone compatible, iPad/tablet compatible and desktop compatible**.

5. **ONLINE VERSION OF THE PBR Q&A BOOK**: Have a few minutes while at work? Open the scrolling PDF version of the Q&A book and go through one or two questions.

6. **PBR WEBSITE**: The website has a TREMENDOUS amount of valuable content. Each article was written to help address a need expressed by pediatricians. Read as many of the articles as you can! There is also a TOOLS section where you can find links to **discounted pediatric board review question banks**.

7. **PBR's TEST-TAKING STRATEGIES & COACHING COURSES**: Physicians are not taught HOW to take tests. **GOOD pediatricians with sound clinical reasoning WRONGLY believe that** a board exam is a measure of one's knowledge base, and thus a measure of one's abilities as a clinician. That is completely false.

   Exams require mastery of the English language, mastery of pacing, mastery of your emotional state during an exam, and an understanding of the **deceptive tactics** employed by question-writers to create **seemingly possible yet blatantly WRONG answer choices**.

   The **PBR TEST-TAKING STRATEGIES & COACHING COURSE** (a paid resource for PBR members - http://www.pediatricsboardreview.com/**strategies**) offers **insights into this "board game"** so that you stop viewing question as miniature patients, and start viewing them as miniature riddles. Riddles with concrete rules and strategies to help you reach the correct answer quickly (**even if you do not have the clinical knowledge to answer it!**). Understanding the rules of the game will completely change your outlook on how to prepare for the exam and how to use board review questions for PRACTICE instead of content.

   I HIGHLY recommend the PBR Test-Taking Strategies & Coaching Course for anyone who is "at risk." This includes you if:

   - You have failed this exam at least once
   - You typically score below the national average on your board exam scores
   - You have failed ANY USMLE Step exam
   - You were classified as "at risk" during residency based on your in-training exam scores
   - You are more than 1 year out of residency

   The course helps you understand the **techniques and skills** associated with answering board-style questions correctly. We've **helped pediatricians finally pass the boards after failing SIX times**, so helping you should be easy.

   To get just a taste of how you can increase your board scores immediately, and to learn a few of the rules to the "board game," click here and read a PBR article I wrote titled, "***3 Strategies To Skyrocket Your Score!***" - http://www.pediatricsboardreview.com/**techniques**

   Also, visit http://www.pediatricsboardreview.com/**strategies** and watch a FREE test-taking strategies session right now.

TEST-TAKING STRATEGY COURSE TESTIMONIALS

(FROM MEMBERS OF OUR ONLINE COURSE AND/OR OUR LIVE COURSE)

Ashish, I did it. I can't thank you enough for creating an amazing system to keep me on on track with my studying. And the $2000 for the live weekend test taking course was well worth it. Doing the technique during the test kept me focused and allowed me to eliminate wrong answers. Thank you for all the great advice, sticking to the material, memorize, memorize, memorize then practice practice practice. After 4 failed attempts it was exhilirating to finally read the words, "we are PLEASED to announce you PASSED!" I will definitely recommend your program.

God Bless

- Dr. Yessenia Castro-Caballero, Board Certified Pediatrician

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*I found myself stuck many times, failing to pick the best answer even though **the correct answer was always between my best 2 options**. Everything was more clear when Ashish recommended to always pick the answer that addresses the "most important clinical issue" of the question. **I started to use this technique this past week, and my test scores have improved remarkably**. Thanks so much!! I am ready for the next webinar!!*

- Dr. HL, Now A Board Certified Pediatrician

Appreciated that Ashish was able to break down the thought process and convey it to me... I was beginning to feel like I was "all over the place" when approaching questions. The techniques were articulated in a way which "clicked" with me.

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*Definitely helped to get a better understanding of the "board game" that Ashish mentions. **I'm sure I've fallen prey to those** traps **in the past**.*

Also, knowing the types of questions and the algorithm to figuring out how to spend my time answering the questions-- never would have thought about the Hybrid approach to just reading the last line of the vignette for "this/these" questions.

Really didn't know that I shouldn't be spending time reading through the whole vignette... or doing the "top to bottom" approach!

Overall it was great and I really appreciate you taking the time and effort putting this together and making sure that we can succeed our first time around.

*Helped immensely with reading/understanding the "English" of the questions - **I actually would've gotten one example question wrong in the past had I not used the AaCNI mnemonic***

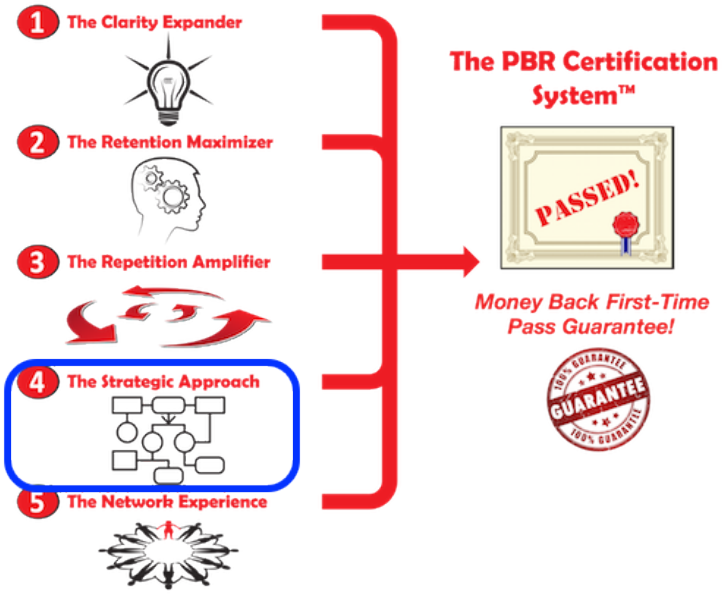
***I had very little time to prepare for the boards...** The core study guide helped me focus on topics that were high yield on the exam. In addition, **the strategies taught by Ashish were very helpful and is what I believe helped me PASS**. I would highly recommend the PBR for anyone needed to review in a short period of time. **It is worth every penny**!*

- Dr. Darlene Melk, Board Certified Pediatrician

The time that you spend learning <u>how</u> to use test-taking strategies to increase your scores will be the HIGHEST yield time of your board prep. The time investment is about one day, but the skills you learn will be used on EVERY single question that you come across. What other pediatric knowledge-based content or chapter can guarantee you the same benefit? NOTHING!

Signup For Your FREE Strategy Session Now

http://www.pediatricsboardreview.com



FULL ONLINE Test-Taking Strategies Course

http://www.pediatricsboardreview.com/strategy

LIVE Test-Taking Strategies Course

http://www.pediatricsboardreview.com/live-tts

DID YOU KNOW THAT I FAILED THE BOARDS?

I took the ABP initial certification exam the year that I graduated from residency. **I used multiple study guides to prepare**. Because there was so much information in front of me (print and video), **I only got through everything once**.

I felt okay going into the exam. I thought, "I've been through the MCAT, three USMLE exams and an Internal Medicine board exam. I did fine in residency and I studied really hard for two months. I'm *sure* I'll be fine."

Coming out of that exam room on test-day, I felt nauseous. I realized that I might have just failed my first medical board exam, ever! **I was upset with myself for getting so scattered with all of those different study materials**, but I was also annoyed because **I still couldn't think of a single resource that I could use as a primary study guide the next time around**.

I went home and made notes about **how I would study differently** if I had failed. What topics would I concentrate on? What topics just don't seem to be "testable"? What information is a waste of time to study?

When the results came, I estimated that I **failed by seven to nine questions. I made key strategy changes** based on my previous experience. I studied for hundreds of hours while still working a full-time job. I **focused on efficiency, solid mnemonics for memorization and I stopped trying to learn "all of pediatrics."**

You never feel "great" coming out of a board exam, but the following year I felt like I had a fighting chance. **My score increased by 160 points, and I estimated a pass by about 37–39 questions!** Pretty soon, I even received a letter from the ABP. **The American Board of Pediatrics asked ME to write questions for the boards!!!**

I was really just happy to pass. Failing **the first time had cost me extra time, money and energy** that I would have preferred to spend with my loved ones.

Prior to creating the Pediatrics Board Review experience, I was ashamed that I had failed. Now, **I've taken a horrible experience and I've created something that is helping residents and pediatrician across the country**. I've also realized that **failing the boards did not mean that I was a bad pediatrician**. Nor did passing by such a wide margin mean that I am a great pediatrician.

I'M JUST AN AVERAGE PERSON WHO DID EXTREMELY WELL ON THE EXAM… AND THEN TOOK MY NOTES AND SYSTEMS AND TURNED THEM INTO THE PBR. No matter who you are, I know that you can pass your exam, too. That's why the PBR materials come with a **100% money-back first-time pass guarantee**.

It's the most EFFICIENT and well-integrated *Certification SYSTEM* to help you PASS the pediatric boards. So rest assured that by joining the PBR family, you're already on the right track to success.

JUST FOLLOW THE EFFICIENCY BLUEPRINT!

THE PBR EFFICIENCY BLUEPRINT

The pediatric initial certification exam has **one of the highest failure rates of any medical board exam**. I URGE you to follow just a few of my simple but CRITICAL recommendations as you go through your board review experience. ***ESPECIALLY #1***!

1. **PLEASE STICK TO ONE PRIMARY STUDY GUIDE - the PBR!** Spreading yourself too thin by reviewing multiple resources is the ***BIGGEST MISTAKE*** you can make. **I've gone through thousands of emails, interviews and surveys**. It's clear that this one, single recommendation that will increase your chances of board success more than anything else I can say.

   This is a **key similarity amongst pediatricians who failed** the boards, but then went on to pass using the PBR system. So please **do not spend your time going through other books, DVDs or expensive live board review courses**. **Go through the PBR books** (Core Study Guide + Q&A Book) **and the PBR companion products** (videos, MP3s, digital picture atlas, webinars) exclusively.

2. Approach your PBR material by first simply SEEING all of the PBR content in the Core Study Guide **and** Q&A Book. Spend about 60–90 seconds per page to simply SEE everything that you will need to learn so that you have an idea about the type of knowledge you'll need to acquire in order to pass this exam. **This should take you a full day**. **DO NOT spend time writing notes of any kind during this process.** Do NOT treat the Q&A Book like other questions. This is CORE content.

   During your first official read through, leave no stone unturned. Crosscheck anything that confuses you. Create mnemonics, notes and drawings in the margins so that you understand EVERYTHING. Make sure that you will NEVER have to go outside of the PBR for additional knowledge or clarifications again. If you get stuck on a concept, reach **out to your peers on the PBR Facebook CREW** (http://www.pediatricsboardreview.com/**facebook**)! If you think you've found an error, notify us through our special error submission link (http://www.pediatricsboardreview.com/**error**). **This will help you maintain your PACE and promote EFFICIENCY**! When crosschecking, ONLY go outside of PBR for possible errors or confusion. That's it! **Do NOT go down the black hole of GOOGLE**!

   Your second time should be MUCH faster. Do NOT let your curiosity of non-PBR topics distract you. As you break up your studying time with questions, you WILL want to look up new topics and crosscheck facts between the PBR and PREP®. DO NOT DO IT! It's a guaranteed waste of precious time that could be spent on PBR, the HIGHEST YIELD resource that you will have at your disposal to pass the board exam.

   Your third, fourth and fifth times through the PBR content should strictly focus on adding more information into your long-term memory through **repetition**, through the use of mnemonics, and through the use of **MULTI-MODALITY studying**. Use audio, video, webinars, study buddy sessions, flash cards, etc. Just use *something* to mix things up because it's been **proven to increase learning**!

   Again, you must resist that urge to look up extraneous information and you must **focus on QUALITY study time**. Ensure that your reading is focused on LEARNING and REMEMBERING the concepts. Do not simply read for the sake of reading, and do not study when you're exhausted or irritable.

   Your primary goal is to pass the exam. As long as you KNOW everything from the Core Study Guide + Q&A Book, **you will have enough information in your brain to easily pass**. **However, if you try to learn "all of pediatrics"** you will get overwhelmed and probably **fail the exam**. Map out at least **300 hours of studying** for the initial certification exam (I studied 400+ hours.)

3. **Use PBR's Q&A book as more CORE material. Also use it to get familiar with very high-yield topics and questions**. The format is short and to the point without too much extra information. The questions will help you understand what types of key findings you need to identify on your practice questions and on your exam. Please remember that **the Q&A book is considered CORE CONTENT**. You need to KNOW IT COLD! Do NOT treat the PBR questions like PREP® questions.

4. **Go through at least 1000 practice questions.** Don't go through them all at once (much more on this in the schedule outlines below). As you go through the questions, **work on your timing**. If you can average about 1 minute and 15 seconds per question, you will be fine for the boards. Do not try to understand why every single incorrect answer is wrong. **Just focus on the correct answer, and if your answer is wrong, figure out WHY it's wrong.** Skip explanations about all of the other answer choices.

   When evaluating WHY you answered a question wrong, figure out if it was because of a **CONTENT problem** or if it was due to a **TECHNIQUE problem**. If you're not sure, then it's a TECHNIQUE problem and you must get help – http://www.pediatricsboardreview.com/**strategies**.

   Did you answer a question incorrectly because of a CONTENT issue? Meaning, you had a knowledge deficiency? If so, was the content in the PBR? If the answer is "yes" then you MUST know that information. If the answer is "no" then do NOT worry about it! Do NOT start looking at Nelson's, Harriet Lane, Google, etc. **It's a black hole that you must avoid** because it will only overwhelm you, and it will keep you from the two main goals of **knowing the PBR CONTENT COLD** and **PRACTICING tons of questions** to master your test-taking technique!

   Remember, the AAP writes PREP®, the ABP writes the boards. Going through **three to four years of PREP®** is great, but keep in mind that the resource is great for **CME**. Any single year of PREP® questions is ***not*** designed to be a stand-alone study guide for the ABP. The questions are EXCELLENT for practicing and mastering your test-taking technique, but your highest-yield information will come from the PBR study guides and systems. If you need MORE questions, you can get discounted practice questions by visiting http://www.pediatricsboardreview.com/tools.

   Did you answer a question incorrectly because of a TECHNIQUE issue? Did you add extra information and assumptions to the question or the answers that led you to the wrong answer? Did you spend too much time on a question even though it was clear that you didn't have the knowledge to answer it? Did the question-writer trick you with a distractor? Did the question writer trick you with an English question instead of a clinical question? Did you get anxious or nervous under a timed mock exam? Are you still confused about why the answer you chose is wrong?

   Make notes about the kinds of issues you're having and try to figure out solution and strategies to avoid similar pitfalls in the future. If you notice that TECHNIQUES-BASED PROBLEMS creeping in over and over again, you need to **seek out help through the PBR Test-Taking Strategies & Coaching course** – http://www.pediatricsboardreview.com/**strategies**.

5. **EXTREMELY Important Test Day Tips**: PLAN to be successful. You will find two links below. The first breaks down the number of questions, time per block, etc. The second is **a list of excellent PBR articles**.

   http://www.pediatricsboardreview.com/**examday**
   http://www.pediatricsboardreview.com/category/**test-day-tips**

STUDY SCHEDULE: Resident? First-Time? Failed? MOC? – I GOT YOU!

I have a TON of guidance on how you can schedule your study time. Since PBR is of benefit to pediatricians at all different levels, I've tailored my recommendations accordingly below.

EVERYONE MUST recognize the **difference between clinical practice and what the ABP would want you to do on the exam**. The exam is filled with answer choices that sound like they would be great options in practice, but unless you know what "the book" says, you will have to simply roll the dice.

For anyone taking the **Initial Certification exam**, recognize that the pass rates are DRAMATICALLY LOWER than the USMLE Step Exams. In the 2008–2009 timeframe the **pass rate for the USMLE exams was in the 90s while the pass rate for the ABP initial certification exam was in the 70s**! **Our members' pass rate for first-time test takers of the ABP exams is estimated to be > 95%! So stay focused on your PBR!**

For anyone taking the pediatric **Maintenance of Certification (MOC) exam, you're in luck**! The national pass rate is in the mid-90s for first-time test takers, but **the PBR has had multiple years of pass rates that have been 100% for practicing general pediatricians**!

* **ARE YOU A RESIDENT?** Simply familiarizing yourself with everything in the PBR content before you graduate will dramatically increase your chances of passing the boards.

While on subspecialty rotations, READ and KNOW the associated PBR chapter. While on general inpatient or outpatient rotations, focus on the rest of the book, and take just 15 minutes per day to read the QUICK and high-yield topics about your pateints. Pace yourself so that you can get through the material at least once per year. That's it! If you do that, your in-training scores will skyrocket and you will DESTROY the boards.

* **ARE YOU TAKING THE INITIAL EXAM FOR THE FIRST TIME**? If you have **never taken** the pediatric boards before and you have **never come close to failing** a medical board exam (average or above average board scores), visit the following PBR article for a detailed study schedule:

http://www.pediatricsboardreview.com/**schedule**

* **HAVE YOU EVER FAILED A MEDICAL BOARD EXAM (OR COME CLOSE)?** If you were categorized as being "at risk" of failing based on your in-training exam scores, or if you have ever **failed** ANY medical board exam, or if you scored **below the national average on your USMLE exams**, visit the following PBR article for detailed instructions on how you can avoid failing your next attempt at the pediatric boards:

http://www.pediatricsboardreview.com/**schedule-failed**

* **ARE YOU STUDYING FOR THE MOC?** If you are taking the pediatric recertification exam then your goal should be to get through the PBR materials at least twice and to do at least 550 practice questions. For a video on how to **get 200 FREE ABP questions** scroll to the bottom of this article (for board-certified pediatricians only after logging into the ABP website):

http://www.pediatricsboardreview.com/**abp**

PBR MEMORY AIDS - USING MNEMONICS AND PEGS

MNEMONICS: Mnemonics are memory aids that assist in helping you recall something. They are used throughout this study guide to help you study in a more focused and **EFFICIENT** manner. Not all of them will work for you, but many will. At the time of the exam you WILL use many of the mnemonics in this book to help you answer questions. If you're lucky, you might even get a smile on your face as you think about me acting like a bit of a fool in some of the videos from the **PBR Online Video Course** (http://www.pediatricsboardreview.com/**videos**).

PEGS: Memory "pegs" are typically used to help you remember a list of items. By having 20 pre-memorized pegs that represent the numbers 1–20, you can easily "peg" items to those numbers. For example, in the PEG system outlined in this guide, a CAT symbolizes the number 9 (since cats are said to have "nine lives").

So, if you are trying to memorize a grocery list of 10 items and one of those items is a gallon of milk, then the 9th item could be tied to an image, or a story, about a cat. It could be as simple as visualizing a funky looking BLACK CAT that has white legs drinking from an orange bowl of MILK. The white legs and orange bowl are simply thrown in to add color and imagination. Other strategies would include the use of disproportional size, the use of action, or the use of sound. The crazier the image, or story, the better!

Please note that some of the pegs in this guide will be used in the high-yield mnemonics in this book. Please look through them a few times to see if you can get the hang of it. If you can, then you might even be able to start creating some of your OWN fun and interesting mnemonics. If you cannot, it's okay. Move on since there are only a handful of mnemonics that use one of the pegs listed here. Plus, if I *do* use a peg, I usually try to remind you of the peg association in the book.

Do you have ideas on how to make the pegs or mnemonics in this book more useful?

Please consider sharing your thoughts in the private, members' only community called the **PBR Facebook CREW!** You can also submit them directly to us for consideration through our errors and clarifications portal:

http://www.pediatricsboardreview.com/**ERROR**

TWENTY PEGS

#	USE THIS PEG	DESCRIPTIONS AND EXPLANATIONS OF PEGS
1	TREE TRUNK	Imagine the number 1 looking like a huge, brown tree trunk with limbs full of green foliage sitting at the top of a lush, green hilltop.
2	LIGHT SWITCH	A light switch has 2 positions (ON & OFF). Use a switch OR a bulb for "2".
3	STOOL	Imagine a dark, cherry wood stool with 3 legs.
4	CAR	Cars have FOUR doors and FOUR wheels.
5	GLOVE or HAND	A glove has 5 fingers. Consider making Michael Jackson's shiny glove your peg for the number FIVE.
6	GUN	Another name for a gun is a 6-shooter (since guns used to only hold 6 bullets). GUNS also kill people and put them "6 feet under" the ground.
7	DICE or CARDS	Lucky number 7! Think Vegas, think craps, think gambling with dice or cards!
8	ICE SKATE	Ice skaters are known for performing a move called the figure 8. Eight also rhymes with skate.
9	CAT	Ever heard of the phrase, "Cats have nine lives"?
10	BOWLING BALL or BOWLING PINS	The goal of bowling is to knock down 10 pins.
11	AMERICAN FOOTBALL or GOAL POST	In American football, a field goal occurs when a football is kicked through two, white, vertical uprights (the goal post). A goal post looks like the number 11.
12	EGGS	Eggs usually come in a carton that contains a dozen (12) eggs.
13	HOCKEY MASK	Unlucky number 13 and the unlucky day/movie *Friday the 13th*. The main character in the movie *Friday the 13th* is Jason, a hockey-mask-wearing killer.
14	ROSE or CHOCOLATE HEART	February 14th is Valentine's Day! So think of a long-stemmed, red ROSE or perhaps a big CHOCOLATE HEART.
15	PAYCHECK	You get to give the IRS a huge chunk of your PAYCHECK every single year on TAX-DAY! APRIL 15th. Welcome to healthcare. ☺
16	DRIVER'S LICENSE	Age at which you get a driver's license. Other pegs to consider include CANDLES, CANDY, or a BIRTHDAY CAKE for "Sweet SIXTEEN."
17	MAGAZINE	There is a teen magazine called "SEVENTEEN."
18	VOTING BOOTH	Age when you become a legal adult in the U.S. and are allowed to VOTE.
19	KNIGHTING	Imagine a "KNIGHTING" ceremony (sounds like 19) or a KNIGHT.
20	CIGARETTES	A pack of CIGARETTES has 20 cigarettes in it.

There are TONS of mnemonics throughout PBR. Many will seem brilliant. Others may not work for you at all. If that happens, please CREATE YOUR OWN. It's initially intimidating but gets much easier with time.

Click here to read PBR's article on mnemonics: **http://pbrlinks.com/MNEMONICS**

GETTING THE MOST OUT OF THE PBR FORMAT

* **GRAY HIGHLIGHTING OR YELLOW HIGHLIGHTING**: In the PBR hardcopy resources, gray highlighting is used over a word, phrase or chapter title to feature content that you **MUST KNOW**! These are very high-yield topics and are likely to be seen on the exam as an answer choice. PBR's **online** books may have this content in **red text** or with yellow highlighting.

* **DOUBLE TAKE**: You will LOVE THIS! A "DOUBLE TAKE" alert accompanies topics that are in the book multiple times. Medicine ties together. Ordinarily, that results in flipping back and forth between chapters. Double Take is a PBR-specific system used to **increase efficiency** by reducing the flipping back and forth between related (or similar) topics. Most of these topics tend to be very high-yield.

*** NAME ALERTS**: 

Many disease names sound very similar (e.g., Condyloma Lata versus Condyloma Acuminata, or Shwachman-Diamond Syndrome versus Diamond-Blackfan Anemia). NAME ALERTS serve as reminders to look for these subtle differences.

* **ABBREVIATIONS**: Some disorders are discussed using their abbreviations while others are discussed with their proper names. When searching for a topic online you should do a search for both. If you encounter an unfamiliar acronym, try this tool: http://www.**AcronymFinder**.com

* **MNEMONICS**: If you're much smarter than me, you don't need these. If you have an average memory, like me, you MUST learn to take advantage of memory aids. They can dramatically **increase your efficiency** as you journey to retain thousands of bits of information. The PBR mnemonics may or may not work for you, but many of them **should** serve as excellent examples of the various **types** of memory aids you can begin to create. **As a tip, always use as much action, color, exaggeration and "crazy" as possible**.

* **PEARLS**: These are bits of information that help tie key concepts together for you. Members LOVE THEM! Here's a PEARL for you. ☺ There are only a finite number of ways that the ABP can test you on a disease process. Some PEARLS will show you how information could be presented on the exam.

PBR ERRORS

Are there errors in the PBR? Of course there are! But I also update the PBR every year with new recommendations and guidelines. I'm able to do this because of YOUR support. If you notice ANY error in the PBR materials (e.g., incorrect spelling, grammar, incomplete sentence, contradictory information, etc.), **PLEASE visit the following link to submit the error**:

http://www.pediatricsboardreview.com/**ERROR**

Please DO NOT email individual errors or clarification requests to me. It's WAY too overwhelming. If you have MULTIPLE possible errors, send us a Word document. I LOVE the members who do that!!

Also, because it's impossible for me to respond to every submission individually, I frequently release **PBR CONTENT & CLARIFICATION GUIDES** to active PBR members (FREE). **Please note that THIS IS NOT A GUARANTEED SERVICE, but it is something I have done every single year**. Your submissions drive this process and allow me to providing you with updated pediatric knowledge year after year.

PBR TOPIC CLARIFICATION OR CONFUSION

If you are struggling with a concept, get help from the members only **PBR Facebook CREW!** It's EXTREMELY active (especially starting around June or July of every year). If you find a concept explained poorly and think the PBR needs a revision, feel free to use the error portal to bring it to my attention:

http://www.pediatricsboardreview.com/**ERROR**

PBR IMAGE LINKS

The image links in the PBR lead to PHENOMENAL images throughout the World Wide Web! BUT, these images are located on NON-PBR websites. Some websites go out of business. When this happens, we simply need to replace the image. Typically no more than 3% of the links within PBR are "bad." We have an awesome system that allows us to change the link on our end but we need your help when a link "dies." **Simply submit any "bad link" through the portal below and we'll take care of it!**

http://www.pediatricsboardreview.com/**BADLINK**

PBR & AVSAR – THE NON-PROFIT CONNECTION

WHAT IS AVSAR? I started a non-profit organization, named AVSAR Inc., at the age of 27 to help support existing non-profit organizations that were already doing great work in slum areas.

After medical school I spent one year volunteering in the slums of Mumbai. The need for help was profound and conditions were shocking. Six-year-old children worked as child laborers, using their small, agile fingers to make beautifully detailed handiwork. Others spent their days looking for recyclables in garbage dumps.

I bonded with these children. I then created a non-profit organization under the U.S. IRS, called AVSAR. We recruited volunteers from around the world (college students, dentists, doctors, MBA students) to "help where the help was needed." My personal success stories included the creation of an efficient Western-style clinic for child laborers and the establishment of an adolescent sex-education curriculum.

AVSAR helped thousands of people, but the core volunteer program was shut down in my last year of residency due to lack of funding and my 80-hour workweeks. Even so, the projects and systems created by volunteers live on and **continue** to help thousands more every year.

In order to re-launch AVSAR, we needed funding. Through Pediatrics Board Review (a private company) I donated over $50,000 to AVSAR before ever paying myself a penny.

It's because of my passion for helping people that I created AVSAR, and then the PBR **EXPERIENCE**.

I hope that you're able to use the many resources within the PBR *Certification System* and the PBR community to EFFICIENTLY study and pass your exam.

I very much look forward to being a part of your success. Now let's get started!

PRODUCT REGISTRATION

As mentioned on the PBR site, this guarantee applies to anyone taking an ABP initial or recertification exam for the first time. “Money Back” requests may be made within 90 days of the score release date. The original PBR purchase must have been made at least 45 days prior to the exam. Submission of the product registration form is required for the money back pass guarantee and the form must be submitted within 90 days of your purchase and before you take the exam. For complete details, please visit:

http://www.pediatricsboardreview.com/guarantee

Visit the following link to register all of your product(s):

http://www.pediatricsboardreview.com/register

For hardcopy purchases from PBR, but through Lulu.com, Amazon.com, etc., please contact us through http://www.pediatricsboardreview.com/contact so that you can send us a copy of your receipt.

CHAPTER LIST

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DERMATOLOGY

GENERAL DERMATOLOGY

CONTACT DERMATITIS, A DIAPER RASH

Contact dermatitis is a diaper rash that **spares** the inguinal folds. Treat with more frequent diaper changes and a topical barrier, such as zinc oxide.

(DOUBLE TAKE) CUTANEOUS CANDIDIASIS, A DIAPER DERMATITIS

Cutaneous candidiasis, a diaper dermatitis, can occur secondary to a contact dermatitis or recent antibiotic use. It presents as a beefy red rash with papular satellite lesions. This rash goes **into the inguinal folds**. Use a KOH prep to confirm diagnosis, and treat with a topical antifungal, such as nystatin or clotrimazole.

IMAGE (includes satellite lesions): http://pbrlinks.com/CUTASCAN1

(DOUBLE TAKE) ATOPIC DERMATITIS (ECZEMA)

In babies, atopic dermatitis (eczema) SPARES the diaper folds/flexural surfaces (but not in older kids). It is **PRURITIC** and LICHENIFIED. Food allergies CAN exacerbate eczema. Breastfeeding x 6 months or using hypoallergenic formula may delay the onset of eczema but does not reduce its incidence. The contribution of early food ingestion to the development of atopic dermatitis is controversial. Eggs, fish, milk, peanut, soy, wheat and strawberries are the foods thought to possibly contribute, but delaying their introduction doesn't help. Positive skin and RAST tests for foods are not predictive, either. Treatment options include emollients and topical steroids. Avoid use of steroids in areas where the skin is thin. Use the lowest potency steroids that work. Watch for superinfection if the eczema is not improving with appropriate therapy.

IMAGE: http://pbrlinks.com/ECZEMA1

NUMMULAR ECZEMA

Nummular eczema refers to coin-shaped eczematous lesions usually on the **extensor** surfaces of extremities. Lesions are **uniform**, without any central clearing. Lesions may ooze, crust, or have a scaling pattern. Treat with steroids.

IMAGE: http://pbrlinks.com/NUMMULAR1

MNEMONIC: Imagine that you are standing with your arms in abduction, and you are balancing silver COINS that are **UNIFORM in color** (without central clearing) on the BACK of both of your arms (**extensor surface**).

(DOUBLE TAKE) ECZEMA HERPETICUM

Eczema herpeticum is a potentially life-threatening disseminated herpes (HSV) infection occurring at sites of skin damage, including sites of eczema. Look for HSV Vesicles + Crusted Lesions. Even if a description is not given of a vesicular rash, have a high index of suspicion for a rash "**not improving with steroids and/or antibiotics**." Diagnose with a viral culture for HSV, but do not delay treatment. A Tzanck smear can support the diagnosis. Treat by **STOPPING** topical steroids and/or immunosuppressants and starting **Acyclovir**.

IMAGE: http://pbrlinks.com/ECZEMAHERPE1
IMAGE: http://pbrlinks.com/ECZEMAHERPE2
IMAGE: http://pbrlinks.com/ECZEMAHERPE3

SEBORRHEIC DERMATITIS (AKA CRADLE CAP)

Seborrheic dermatitis (AKA cradle cap), is **a NONpruritic**, inflammatory, flaky rash with white to yellow scales that usually forms in oily areas (e.g., scalp). It is often seen in the first two months of life. After that, it's not very common until adolescence. You may treat with topical antifungal agents or mild steroids. The skin may be left with hypopigmented areas, especially in the folds. If asked to name the hypopigmented areas, choose PITYRIASIS ALBA.

IMAGE: http://pbrlinks.com/SEBORRHEIC1

PSORIASIS

Psoriasis is a **very well-defined,** red, flaky rash covered with **silver**-white patches. It can also be described as thick and scaly (like seborrheic dermatitis). It sometimes results in punctate bleeding when scales are removed (this is called the Auspitz sign). It can occasionally be limited to the diaper area, in which case it goes **into the inguinal folds**.

GUTTATE PSORIASIS

The "guttate" in guttate psoriasis means "drop like" and describes the shape of these discrete psoriatic lesions. This can be preceded by a **Group A Strep** (pyogenes) infection.

IMAGE: http://pbrlinks.com/GUTTATE1

(DOUBLE TAKE) LANGERHANS CELL HISTIOCYTOSIS (LCH) = HISTIOCYTOSIS X

Langerhans Cell Histiocytosis (LCH), AKA Histiocytosis X, is a **PAPULAR** rash that is sometimes associated with petechiae. The rash is located **in the folds** (inguinal folds, supra-pubic folds, perianal area). It can resemble eczema, but the petechiae or PAPULES should guide you towards this diagnosis. LCH is a type of **cancer**. You may be shown a lytic bone lesion (possibly of the skull). Diagnose by skin biopsy. LCH can also be associated with DIABETES INSIPIDUS. Treat by removing the lesion and giving steroids, ± chemotherapy.

PEARLS: Do not confuse this with Wiskott-Aldrich (WiXot**T**-Aldrich, X-linked, low IgM, high IgA, TIE = Thrombocytopenia, small platelets, Infections, and Eczema). Also, if they describe an eczema or seborrheic dermatitis type of rash in a patient with high urine output, LCH is your diagnosis.

IMAGE: http://pbrlinks.com/LANGERHANSCELL1
IMAGE: http://pbrlinks.com/LANGERHANSCELL2
IMAGE: http://pbrlinks.com/LANGERHANSCELL3

RASHES THAT SPARE THE INGUINAL FOLDS

Eczema and Contact Dermatitis should be high on your differential for rashes that spare the inguinal folds.

PRURITIC RASHES

Consider atopic dermatitis/eczema, HSV, scabies, tinea, or Varicella (VZV) in your differential of any pruritic rashes.

KERATOSIS PILARIS

Keratosis pilaris forms due to an overgrowth of the horny skin. It can look similar to eczema and may have a mild erythematous background. No treatment is needed.

IMAGE: http://pbrlinks.com/KERATOSIS1
IMAGE: http://pbrlinks.com/KERATOSIS2
IMAGE: http://pbrlinks.com/KERATOSIS3

LICHEN SCLEROSUS

Lichen sclerosus is a chronic, inflammatory, dry, white, and somewhat scaly rash that is usually found in the genital area. There is no thickening or sclerosis. There are usually no symptoms, although a small percentage of patients have pruritis. Look for a picture of labia with a rash.

IMAGE: http://pbrlinks.com/LICHENSCLEROSUS1

LICHEN STRIATUS

Lichen striatus is a rash that looks like eczema, but is linear or papular and can follow the Lines of Blaschko.

IMAGE: http://pbrlinks.com/LICHENSTRIATUS1
IMAGE: http://pbrlinks.com/LICHENSTRIATUS2
IMAGE: http://pbrlinks.com/LICHENSTRIATUS3
IMAGE: http://pbrlinks.com/LICHENSTRIATUS4

ALLERGIC CONTACT DERMATITIS, A TYPE IV HYPERSENSITIVITY SKIN RASH

Allergic contact dermatitis is a Type IV hypersensitivity skin rash that requires a **prior exposure**, and tends to be pruritic. See if the location of the rash is in an area where a nickel-containing belt buckle, earring, necklace, or other jewelry could have been. A rash may present even after **years** of wearing the irritant. The rash from nickel exposure is more erythematous and can become lichenified. The classic example of Type IV reactions is the rash of **poison ivy**, or other "leaves of 3" (including poison oak and poison sumac). Regarding a contact dermatitis from these plants, it will not spread once the affected area is washed with soap and water. The fluid from within the vesicles **cannot** spread the rash. This reaction is a Type IV Cell Mediated Hypersensitivity Reaction, and is called a **Rhus** reaction (from the old genus name of poison ivy, Rhus radicans). The rash is vesicular and may be in a linear configuration (where the leaves rubbed across the skin).

* **PEARL**: First exposure may take 1 week to develop the rash as helper T cells proliferate and "remember" the agent. After that, the rash may develop within **hours** of exposure. "No wonder I had to go through the 2-step PPD before starting as an attending!"

* **PEARL**: REMINDERS: A PPD and the skin testing of Candida, Mumps, and Tetanus are all Type IV reactions.

* MNEMONICS:

  - "LEAVES OF THREE, LET THEM BE!"
  - Type IV reaction: I + V = the Roman numeral IV = 4, and the 4th letter in the alphabet is **D** = **D**ELAYED. I + V also should you remind you of poison **IV**y.

* **IMAGE:** http://pbrlinks.com/ALLERGICCONTACT1
* **IMAGE:** http://pbrlinks.com/ALLERGICCONTACT2

(DOUBLE TAKE) BIOTIN/BIOTINIDASE DEFICIENCY

Biotin and biotinidase deficiencies may present with a RASH + ALOPECIA + **NEUROLOGIC SIGNS** (ataxia, coma, etc.). Patients may also have lactic acidosis. Treat with biotin.

MNEMONIC: Imagine the TIN MAN from *The Wizard of Oz* walking with an ATAXIC gait as he SCRATCHES his arm. Notice that he has NO HAIR!

PAPULAR URTICARIA

Papular urticaria is a rash due to hypersensitivities to the insect bites of bedbugs, fleas, and mosquitoes that results in edema, erythema, and pruritis. It presents in **RECURRENT CROPS**. It tends to come and go, wax and wane every few weeks or months. Some lesions may be umbilicated. Treat by removing the offending agent (fleas, lice, bedbugs, or outside insects).

PEARL: You may not be given the history of a specific insect or exposure.

MNEMONIC. "CROPular Urticaria." Where do you find insects? In CROPS, of course!

IMAGE: http://pbrlinks.com/PAPULAR1

VITILIGO

Vitiligo results in depigmented macules. Look for a "salt and pepper" type of pattern of re-pigmentation. It is often associated with HALO NEVI.

IMAGE: http://pbrlinks.com/VITILIGO1

(NAME ALERT) ICHTHYOSIS VULGARIS

Ichthyosis vulgaris is a rash that resembles FISH SCALES. It is often seen in atopic dermatitis patients. You may attempt treatment with ammonium lactate or alpha-hydroxyacid-containing agents. The name alert is for lamellar ichthyosis and harlequin ichthyosis.

IMAGE: http://pbrlinks.com/ICHTHYOSIS1

(NAME ALERT) LAMELLAR ICHTHYOSIS (AKA COLLODION BABY)

Lamellar ichthyosis (AKA collodion baby) is noted at the time of birth in newborns. A thin, transparent film is noted on the body. Eyelashes are missing. Eyelids seem everted (ectropion). The name alert is for harlequin ichthyosis and ichthyosis vulgaris.

IMAGE: http://pbrlinks.com/LAMELLAR1
IMAGE: http://pbrlinks.com/LAMELLAR2
IMAGE: http://pbrlinks.com/LAMELLAR3

(NAME ALERT) HARLEQUIN ICHTHYOSIS

Harlequin ichthyosis presents with a newborn that looks much more abnormal than lamellar ichthyosis. The covering is hard ("armor-like") and horny. Movement is restricted. Prognosis is poor comparatively. The name alert is for lamellar ichthyosis and ichthyosis vulgaris.

IMAGE: http://pbrlinks.com/HARLEQUIN1

PYODERMA GANGRENOSUM

The etiology of pyoderma gangrenosum is unknown, but it is known to be associated with other systemic diseases such as Crohn's. Lesions are described as deep, bluish, necrotic, and boggy-looking ulcers.

IMAGE: http://pbrlinks.com/PYODERMA1
IMAGE: http://pbrlinks.com/PYODERMA2

(DOUBLE TAKE) ECTHYMA GANGRENOSUM

Ecthyma gangrenosum is usually a sign of a **PSEUDOMONAS** infection and possibly sepsis in an immunocompromised patient, especially **LEUKEMIA**! Look for a **neutropenic** patient with black, necrotic, ulcerative lesions with surrounding erythema and edema. These lesions are often located in the groin/diaper area.

IMAGE: http://pbrlinks.com/ECTHYMA1

GRANULOMA ANNULARE

Granuloma annulare is a chronic skin condition with an annular (circular) lesion. It may be slightly pruritic. There are **no scales**.

PEARL: This looks kind of like ringworm, but there is **NO SCALING**! Keep this in mind any time you see Tinea as an answer choice.

IMAGE: http://pbrlinks.com/GRANULOMA1

PITTED KERATOLYSIS

Pitted keratolysis is a condition in which there is **pitted** skin in areas of pressure. There will probably be a history of **strong foot odor**.

IMAGE: http://pbrlinks.com/PKERATOLYSIS1

(DOUBLE TAKE) DERMATOMYOSITIS

Dermatomyositis results in a heliotropic, violaceous rash in malar area. Gottron's Papules (erythematous, shiny, pruritic papules over the metacarpals) may be present. Patients will have proximal weakness and possible telangiectasias near the nail folds. Diagnose with a **MUSCLE BIOPSY.** The **CK LEVEL WILL BE HIGH**. These patients can also get calcinosis cutis.

PEARL/REMINDER: Duchenne Muscular Dystrophy also has elevated CK levels.

IMAGE: http://pbrlinks.com/DERMATOMYOSITIS1
IMAGE: http://pbrlinks.com/DERMATOMYOSITIS2
IMAGE: (calcinosis cutis) http://pbrlinks.com/DERMATOMYOSITIS3

STEVENS-JOHNSON SYNDROME (SJS) and TOXIC EPIDERMAL NECROLYSIS (TEN)

The terminology for Stevens-Johnson Syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) varies. Many now view these disorders on a spectrum. SJS and TEN are the same, but TEN is the diagnosis if > 30% of body surface area is involved. Look for bullae or erosions followed by hemorrhagic crusting. There may be severe blistering with the Nikolsky sign when pressure is applied. It is a full thickness rash similar to a burn. Skin lesions may look like a BULLSEYE or TARGET lesion, with the center described as DARK, DUSKY, or VIOLACEOUS. The target CAN be a blister or vesicle. At least two mucous membranes must be involved (most commonly the lips and eyes). If the eyes are involved, this is an ocular emergency!

MEDICATION ASSOCIATIONS: Aromatic seizure medications, penicillins, NSADS, and **sulfa** drugs. The rash usually occurs within 2 months of starting the medication.

ERYTHEMA MULTIFORME

Erythema multiforme is also a confusing topic. It may now also be considered on the SJS/TEN spectrum, especially if mucous membranes are involved. Distinguishing erythema multiforme minor from erythema multiforme major is also confusing, so the terminology is not likely to be tested.

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

IMAGE: http://pbrlinks.com/ERYTHEMULTI1
IMAGE: http://pbrlinks.com/ERYTHEMULTI2
IMAGE: http://pbrlinks.com/ERYTHEMULTI3
IMAGE: http://pbrlinks.com/ERYTHEMULTI4

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

(DOUBLE TAKE) NEONATAL LUPUS

The baby does NOT have lupus. Neonatal lupus occurs in children of mothers with SLE due to fetal exposure to **maternal SLE-related antibodies**. It is rare. Findings may include increased LFTs, petechiae, rash, scaling, thrombocytopenia, **third degree AV heart block with bradycardia,** or hydrops fetalis (fluid accumulation in two or more fetal compartments usually due to heart failure). Diagnose by sending **Anti-Ro** or anti-La antibodies (AKA anti-SS-A or SS-B).

IMAGE: http://pbrlinks.com/NEONATALLUPUS1

RASHES WITH CENTRAL CLEARING (PEARL)

Hives/urticaria, Rheumatic Fever ("jonEs" = E. Marginatum = MARGINs progress to give central clearing), Tinea (raised border/ringworm)

RASHES WITH CENTRAL DARKENING/TARGET LESIONS (PEARL)

SJS/TEN ("target shooting, bull"), Brown recluse spider bite (see Emergency Medicine), Lyme Disease/Borrelia/Erythema Migrans

URTICARIA/HIVES

Urticaria (hives) is a pruritic rash due to an allergic exposure. Pink center with a more erythematous border. Giving histamine blockers (both H1 & H2) may be helpful. Foods are the most likely cause of chronic urticaria.

IMAGE: http://pbrlinks.com/URTICARIA1
IMAGE: http://pbrlinks.com/URTICARIA2

SCLERODERMA

Scleroderma patients have thickened skin with an ivory or waxy, appearance. Affects girls more frequently. The limited form is more common than the systemic form in children (located at one site only). Lesions may initially be painful and tender. Skin is often hard and may have a linear appearance. Treat with topical lubricants for limited cases. May have to use steroids or other immunosuppressives in more severe cases.

IMAGE: http://pbrlinks.com/SCLERODERMA1
IMAGE: http://pbrlinks.com/SCLERODERMA2

DERMOID CYSTS (AKA EPIDERMOID CYSTS)

Dermoid cysts (AKA epidermoid 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.

IMAGE: http://pbrlinks.com/EPIDERMOIDCYSTS1
IMAGE: http://pbrlinks.com/EPIDERMOIDCYSTS2
IMAGE: http://pbrlinks.com/EPIDERMOIDCYSTS3

COMEDONAL ACNE

Think of comedonal acne as an OBSTRUCTIVE process that creates white heads and black heads. Treat with a RETINOID keratinolytic agent. You may also prescribe benzoyl peroxide.

PEARL: An answer with topical retinoic acid + benzoyl peroxide twice daily is probably WRONG. Benzoyl peroxide inactivates traditional retinoids (tretinoin), so one should be used at night, and the other in the morning (or at least with some time in between). Newer retinoids, like adapalene and tazarotene, are more stable and may be used at the same time.

INFLAMMATORY ACNE

Inflammatory acne is differentiated from comedonal acne by its RED BASE.

* **Minor cases**: If the acne is localized with small lesions, use a TOPICAL antimicrobial agent, such as Benzoyl peroxide, Clindamycin or Erythromycin. Retinoic acid topicals are also included in most regimens.

* **Severe cases**: If large, nodular, or in multiple areas, use ORAL antibiotics. First line is Tetracycline, Doxycycline, or Erythromycin. Minocycline is a second line agent. These antibiotics provide a bactericidal and an anti-inflammatory effect. You may also try oral contraceptive pills (OCPs) in females for their anti-androgen effects. If all else fails, use ISOTRETINOIN.

ISOTRETINOIN

Isotretinoin is a miracle drug that fights sebum production and bacteria, while also decreasing inflammation and comedonal acne. But it is **TERATOGENIC**, so obtain TWO negative pregnancy tests before starting the medications. Also, patients must use TWO forms of birth control starting one month before starting the medication and until one month after. In addition, they should have monthly pregnancy tests.

PEARL: Acne can begin as early as 8 years of age. If the boards present a 7-year-old child with what looks like acne, CONSIDER ANOTHER DIAGNOSIS! Consider exogenous steroid use, precocious puberty, and TUBEROUS SCLEROSIS.

(DOUBLE TAKE) APHTHOUS ULCERS

Aphthous ulcers are painful lesions found within the oral mucosa (buccal mucosa, lips, and tongue) with a grayish-white base and a rim of erythema. These can occur in isolation or in association with Behcet's or Shwachman-Diamond syndrome.

IMAGE: http://pbrlinks.com/APHTHOUSULCERS1
IMAGE: http://pbrlinks.com/APHTHOUSULCERS2

TEETH ISSUES

TOOTH TIMELINE

Tooth appearance follows a timeline. All anterior teeth are present (eight of them) by about 12 months. Primary teeth are present by about age 2. Some children do not have teeth by 1 year of age, so reassurance is okay. For ABP questions, they will be more focused on **abnormal-looking teeth**.

PEG TEETH

Peg teeth refers to teeth that are smaller than usual. Sometimes they are tapered and look like fangs. This usually affects the lateral incisors and is associated with INCONTINENTIA PIGMENTI and HYPOHIDROTIC ECTODERMAL DYSPLASIA.

IMAGE: http://pbrlinks.com/PEGTEETH1
IMAGE: http://pbrlinks.com/PEGTEETH2

HUTCHINSON TEETH

Hutchinson teeth are found in CONGENITAL SYPHILIS. These children have teeth that are smaller and more widely spaced. They also have notches on the biting surfaces.

IMAGE: http://pbrlinks.com/HUTCHTEETH1
IMAGE: http://pbrlinks.com/HUTCHTEETH2

TETRACYCLINE TEETH STAINING

If tetracycline is used at a young age, teeth can end up having yellow, brown, or blue band-like stains. Avoid tetracycline until patients are at least 8 years of age.

IMAGE: http://pbrlinks.com/TETRATEETH1

FLUOROSIS

Fluorosis is the mottled discoloration of teeth due to excess fluorine use during tooth development (up to age 4).

IMAGE: http://pbrlinks.com/FLUOROSIS1

VASCULAR & PIGMENTED LESIONS

PEARL/MNEMONIC: HEMANGIOMAS are different from VASCULAR MALFORMATIONS (e.g., Port Wine Stains/capillary malformations). VASCULAR MALFORMATIONS tend to have much more associated morbidity. You might say that **VM**s are **V**ery **M**orbid in comparison.

IMAGE: (slideshow on birthmarks) http://pbrlinks.com/VM1

HEMANGIOMAS

Hemangiomas are an abnormal build-up of blood vessels. They eventually self-involute but are dangerous during PROLIFERATION PHASE. They are otherwise benign. They usually look red, but can appear blue if deep (CAVERNOUS HEMANGIOMAS). Proliferation is greatest during the first 6 months, and lesions are largest around 1 year of age. Lesions start to involute around 2 years of age and disappear by 5–10 years of age. If in a benign area, they can be left alone. If in a more sensitive area (near the eyes, ears, nose, throat, or spine), they may require medical treatment with propranolol (first line drug). Second line therapies include systemic steroids, pulsed dye laser therapy and surgery.

COMPLICATIONS: If located in the beard area, look for airway issues. If near the eye, it's okay as long as there is no problem with VISION. Those near the ears, nose, and lips can be troublesome if they ulcerate. If in the lumbosacral area, there is concern for spinal dysraphism (incomplete fusion of a raphe, especially the neural folds/tube). High output congestive heart failure (CHF) can occur due to large, or multiple hemangiomas.

IMAGE: http://pbrlinks.com/HEMANGIOMAS1
IMAGE: http://pbrlinks.com/HEMANGIOMAS2

PHACES SYNDROME

A diagnosis of PHACES syndrome requires a large hemangioma in the face/neck area PLUS one of the following defects:

* **P**osterior fossa malformation (DANDY WALKER)
* **H**emangioma. Often in the distribution of the Facial Nerve. Look for a large **segmental** hemangioma on the **FACE**. Segmental refers to what looks like a nerve distribution (segmented by normal skin in between). This can be associated with STROKES.
* **A**rterial cerebrovascular anomaly
* **C**ardiac anomalies: Especially COARCTATION OF THE AORTA
* **E**ye anomalies: MICROPHTHALMIA, STRABISMUS
* **S**ternal defect

* **IMAGE**: http://pbrlinks.com/PHACES1

(DOUBLE TAKE) KASABACH-MERRITT SYNDROME

In Kasabach-Merritt syndrome, there are large, congenital vascular tumors. They are not true hemangiomas but can cause a severe CONSUMPTIVE COAGULOPATHY (in the form of **thrombocytopenia** and the consumption of coagulation factors) and death. It is most common in infants.

IMAGE: http://pbrlinks.com/KASABACH1
IMAGE: http://pbrlinks.com/KASABACH2

PEARL: Look at the above images closely. Make sure you look closely at images so that you do not get this vascular tumor confused with hemihypertrophy.

MNEMONIC:

- >---< is used by many of us when recording CBC results.

NEVUS SIMPLEX

A nevus simplex is a **S**almon colored lesion often called a **S**tork bite or **S**almon patch. They blanch on pressure and tend to be on the midline or symmetrical (e.g. on both eyelids). These fade with time and are benign. Do not get this term confused with Nevus FLAMMEUS (AKA PORT WINE STAIN).

PEARL: These BLANCH with pressure.

IMAGE: http://pbrlinks.com/NevusSimplex1
IMAGE: http://pbrlinks.com/NevusSimplex2

PORT WINE STAINS (PWS) (AKA NEVUS FLAMMEUS)

Port Wine Stains (PWS), AKA nevus flammeus, are **CAPILLARY** malformations. They tend to be unilateral and segmental, not crossing the midline. They start as pink/flat lesions that become dark red-purple. They then progress to being thick/raised in adulthood. These **P**WSs are **P**resent at birth and are **P**ERMANENT. They are benign if noted in isolation. If noted on the face, they can be associated with glaucoma (increased intraocular pressure that can present as a red eye).

IMAGE: http://pbrlinks.com/PORTWINE1
IMAGE: http://pbrlinks.com/PORTWINE2

PEARL: They grow in proportion to the child and tend to occur in a segmental distribution respecting the midline.

MNEMONIC: Glaucoma is a concern if a PWS is noted in the facial area. Is that why Mikhail Gorbachev wore glasses? Because he has that big FLAME on his head?

STURGE-WEBER SYNDROME (SWS)

The Sturge-Weber Syndrome (SWS) includes the following findings: Port Wine Stain (PWS or NEVUS FLAMMEUS) + **EYE/TRIGEMINAL NERVE DISTRIBUTION** + INTRACRANIAL VASCULAR MALFORMATION (look for with **MRI**) +/- glaucoma +/- Seizures +/- cognitive deficits.

MNEMONICS: "p**WS** = s**WS**"… Ever heard of a basketball player named Chris WEBBER? Think WEBBER = Sports = **ESPN (I know it's a stretch).**

* **E**YE - glaucoma
* SWS
* PWS
* **N**EUROLOGIC issues: Developmental delay, Seizures

CAPILLARY MALFORMATION ASSOCIATIONS

(DOUBLE TAKE) KLIPPEL-TRENAUNAY SYNDROME 

Klippel-Trenaunay syndrome is associated with AV fistulae, causing skeletal or limb OVERGROWTH (hemihypertrophy). Patients with Klippel-Trenaunay have Port Wine Stains and overgrowth of tissue, bones, and soft tissue. Look for unilateral limb overgrowth and CHF.

* **IMAGE**: http://pbrlinks.com/KLIPPELTRENAUNAY1

* **(DOUBLE TAKE) PEARL**: Hemihypertrophy images on the pediatric exam should very quickly clue you in to a few disorders. Highest on your differential should be Beckwith-Wiedemann Syndrome, then Klippel-Trenaunay, then Russell-Silver Syndrome, and then possibly Proteus Syndrome.

* **MNEMONIC**: From now on, say CRIPPLE-**T**. Think of these patients as having a CRIPPLING disorder in which they have one HUGE leg that prevents them from getting around.

* **NAME ALERT:** KLIPPEL-FEIL SYNDROME. This is a completely different disorder. Look for a Torticollis-like photograph (due to fused cervical vertebrae).

IMAGE: http://pbrlinks.com/KLIPPELTRENAUNAY2

(NAME ALERT) KLIPPEL-FEIL SYNDROME

Klippel-Feil Syndrome results in a torticollis-like appearance and results from fused cervical vertebrae. Patients will likely have a short, webbed neck, limited range of motion at the neck, and possibly other anomalies. Etiology is unknown. The "Name Alert" is because this is a completely different disorder from Klippel-Trenaunay Syndrome (limb overgrowth due to AV fistulae).

IMAGE: http://pbrlinks.com/KLIPPELFEIL1 (View images and move on!)

CONGENITAL MELANOCYTIC NEVUS

Congenital melanocytic nevi are commonly referred to as moles. They may present at birth or within the first few months of life. They are generally benign but carry an increased risk of MELANOMA if there are multiple moles (more than three) or if they are > 20 cm. They are associated with spinal dysraphisms and Dandy Walker Syndrome (fossa abnormality).

MCCUNE-ALBRIGHT SYNDROME (AKA POLYOSTOTIC FIBROUS DYSPLASIA)

McCune-Albright syndrome (AKA Polyostotic Fibrous Dysplasia) findings include **IRREGULAR** café-au-lait **MACULES** (either > 3 cm or multiple), **PRECOCIOUS PUBERTY**, BONE PROBLEMS (fractures, cranial deformities), and possibly other endocrine issues (hyperthyroidism). It can cause fractures of long bones and bowing of arms.

IMAGE: http://pbrlinks.com/MCCUNE1

MNEMONIC: Call it **MACULE** Albright Syndrome from now on.

TUBEROUS SCLEROSIS

Tuberous sclerosis is AUTOSOMAL DOMINANT. Look for at least 2 of the following features:

* ASH LEAF SPOTS: These are hypOpigmented lesions, which can be seen with a Woods Lamp. You need at least **3** on the body to help make the diagnosis.

- **IMAGE**: http://pbrlinks.com/TUBERSCLERO1
- **IMAGE**: http://pbrlinks.com/TUBERSCLERO2

* SHAGREEN PATCH (hypERpigmented plaque that can be rough/thick and papular)

- **IMAGE**: http://pbrlinks.com/TUBERSCLERO3
- **IMAGE**: http://pbrlinks.com/TUBERSCLERO4

* ANGIOFIBROMAS (AKA ADENOMA SEBACEUM or SEBACEOUS HYPERPLASIA)

- **PEARL**: Often misdiagnosed as acne. LOOK FOR SPARING OF THE FOREHEAD.
- **IMAGE**: http://pbrlinks.com/TUBERSCLERO5

* PERIVENTRICULAR OR CORTICAL TUBERS: Usually associated with INFANTILE SPASMS or seizures

* CARDIAC RHABDOMYOMAS: Look for a kid with arrhythmias!

* RENAL ANGIOMYOLIPOMA

MANAGEMENT OF TUBEROUS SCLEROSIS: Most of the management has to do with seizures/infantile spasms and cardiac arrhythmias.

- **MNEMONIC**: Imagine a TUBULAR bazooka shooting out WHITE LEAVES. The leaves have DANCING (seizing) tics on them!

- **MNEMONIC**: ASH is typically GRAY/WHITE/HYPOPIGMENTED, whereas a "PATCH of GREEN" is typically DARKER/HYPERPIGMENTED.
- **MNEMONIC**: **ASHES** come from burned WOOD. A Woods lamp is needed to see them.

NEUROFIBROMATOSIS I (NF1)

Neurofibromatosis I (NF1) is an AUTOSOMAL DOMINANT disorder involving the SKIN, BONES, and NERVOUS SYSTEM. Diagnose with at least **2** of the following:

* First-degree relative has the disease

* Neurofibromas

* Lisch Nodules in the iris (they look like mini neurofibromas)

- **IMAGE**: http://pbrlinks.com/NF1

* Optic nerve gliomas. This is the neurologic component.

* 6 **REGULAR** café-au-lait macules. As they get older, the SIZE **DOES** MATTER. If prepubertal, these are > 5 mm, if postpubertal, > 15 mm. Ten years of age is a good cutoff. These macules can be present at birth. Children can have an increase in the **size and number** as they age. Therefore, it is very important that they have regular follow-up, especially if there is a family history of the disorder. As a side note, children can also get pheochromocytomas or renal artery stenosis, so the BP should be monitored regularly.

* Scoliosis or bony abnormalities

* Axillary or inguinal freckling

* **MNEMONIC**: (FOR NF-1) SKIN + "ORTHO" + **N**EURO issues = **S.O.N. This is NF ONE, SON (or daughter!**)!!!

NEUROFIBROMATOSIS 2 (NF2)

(Low-yield topic). Neurofibromatosis 2 (NF2) findings include nonmalignant tumors of the nervous system, especially acoustic nerve tumors (AKA neuromas or schwannomas). These can cause tinnitus or even hearing loss. Patients can also have eye tumors, cataracts, retinal problems, spinal cord tumors, and meningiomas. Look for a family history.

PEARL: Tuberous Sclerosis and Neurofibromatosis are both AUTOSOMAL DOMINANT, BUT they both have a HIGH RATE OF NEW MUTATIONS. Do not exclude these from your differential if they mention that the patient's parents do not have the disorder.

INCONTINENTIA PIGMENTI

Incontinentia pigmenti is a severe X-linked DOMINANT disease that results in DEATH for all MALES. If presented with this as an answer choice, make sure the ABP vignette refers to a FEMALE patient. There are four stages of this disorder: Inflammatory vesicular phase, followed by a verrucous phase, followed by the hyperpigmentation phase noted along the **lines of Blaschko**, and finally a phase in which the hyperpigmentation disappears. This can leave atrophy or hypopigmentation behind.

SYSTEMIC ASSOCIATIONS: **DELAYED DENTITION**, mental retardation, paralysis, **PEG teeth,** and seizures.

IMAGE: http://pbrlinks.com/INCONTINENTIA1
IMAGE: http://pbrlinks.com/INCONTINENTIA2
IMAGE: http://pbrlinks.com/INCONTINENTIA3
IMAGE: http://pbrlinks.com/INCONTINENTIA4

MNEMONIC: As WOMEN age, they tend to have more "INCONTINENTs." Incontinentia = Female patient. Imagine a WOMAN on the ground having a SEIZURE. She becomes INCONTINENT of urine, which streams down her PEG legs and creates black-and-white LINEAR SKIN LESIONS. PEG refers to PEG TEETH.

HYPOHIDROTIC ECTODERMAL DYSPLASIA

Hypohidrotic ectodermal dysplasia is a condition related to INCONTINENTIA PIGMENTI, but this can occur in boys. It is associated with HYPOHIDROSIS, decreased sweating, which can lead to hyperthermia; HYPOTRICHOSIS, sparse hair, so no eyebrows/lashes; DELAYED TOOTH ERUPTION; and DEFORMED/PEG TEETH.

IMAGE: http://pbrlinks.com/HED1
IMAGE: http://pbrlinks.com/HED2

INFECTIOUS SKIN CONDITIONS

(DOUBLE TAKE) ECTHYMA GANGRENOSUM

Ecthyma gangrenosum is usually a sign of a **PSEUDOMONAS** infection and possibly sepsis in an immunocompromised patient, especially **LEUKEMIA**! Look for a **neutropenic** patient with black, necrotic, ulcerative lesions with surrounding erythema and edema. These lesions are often located in the groin/diaper area.

IMAGE: http://pbrlinks.com/ECTHYMA1

STREPTOCOCCAL INFECTIONS OF THE GROIN

Streptococcal infections of the groin or perineum are associated with pain with stooling, pruritis, redness, and possibly a fissure. Unlike zinc deficiency, there is **no desquamation**. If vaginal or vulvovaginitis, look for a history of vaginal discharge. Diagnose by culturing the area. Treat with amoxicillin, penicillin (PCN), or a first generation cephalosporin. Risk factors include abuse and previous instrumentation. Look for a history of recent antibiotics in case the discharge is due to Candida.

(DOUBLE TAKE) CUTANEOUS CANDIDIASIS, A DIAPER DERMATITIS

Cutaneous candidiasis, a diaper dermatitis, can occur secondary to a contact dermatitis or recent antibiotic use. It presents as a beefy red rash with papular satellite lesions. This rash goes **into the inguinal folds**. Use a KOH prep to confirm diagnosis, and treat with a topical antifungal, such as nystatin or clotrimazole.

IMAGE (includes satellite lesions): http://pbrlinks.com/CUTASCAN1

BULLOUS IMPETIGO/STAPH SCALDED SKIN SYNDROME (SSSS)

Bullous impetigo, or Staph Scalded Skin Syndrome (SSSS), is a spectrum of the same disease.

* **IMPETIGO**: Look for honey-colored crusting lesions and bullae. Non-bullous impetigo will look similar but without vesicle/bullae (more oozing/crusting).
    - **IMAGE:** http://pbrlinks.com/SSSS1
    - **IMAGE:** http://pbrlinks.com/SSSS2
    - **IMAGE:** http://pbrlinks.com/SSSS3
* **SSSS**: A very painful and red rash in which large, thin blisters are the result of an exotoxin. There is "**sheet-like"** skin loss/separation. This looks very superficial compared to impetigo. Obtain a BIOPSY to prove that it is SSSS and NOT Stevens-Johnson Syndrome (SJS) or Toxic Epidermal Necrolysis (TEN), both of which have deeper/dermal involvement.

- **IMAGE**: http://pbrlinks.com/SSSS2
- **PEARL**: Lesions are **NOT** in the eyes or mouth but may be **around** the eyes and mouth (as opposed to SJS/TEN, which may be IN the eyes and mouth).

STAPHYLOCOCCUS EPIDERMIDIS

Staphylococcus epidermis is the most likely answer if you are presented with a premature baby that has a skin infection.

CELLULITIS

Cellulitis is defined as a well-demarcated area of erythema, edema, and induration secondary to an infection. It may be associated with bullae. For treatment, start with **Cefazolin** as your first line agent.

TINEA CORPORIS

In tinea corporis, a thin, circular lesion with **THIN SCALES,** a RAISED border, and central clearing is noted. The ring of the "ringworm" looks like a worm.

IMAGE: http://pbrlinks.com/TCORPORIS1
IMAGE: http://pbrlinks.com/TCORPORIS2

TINEA VERSICOLOR

Tinea versicolor results in hypopigmented OR hyperpigmented macules. It's caused by MALASSEZIA FURFUR. Lesions may fluoresce under Woods lamp. Treat with selenium or zinc anti-dandruff shampoo, or with oral fluconazole, ketoconazole, but NOT griseofulvin (use that for T. capitis).

IMAGE: http://pbrlinks.com/TVERSICOLOR1
IMAGE: http://pbrlinks.com/TVERSICOLOR2
IMAGE: http://pbrlinks.com/TVERSICOLOR3

PITYRIASIS ROSEA

Pityriasis rosea presents as oval, parallel lesions with THICK scales. Look for a herald patch (first lesion). It is associated with winter and spring. Lesions are often in a "Christmas tree pattern." Treat with light exposure.

IMAGE: http://pbrlinks.com/PITYRIASIS1

PEARL: Unlike secondary syphilis, there are no lesions on the palms/soles.

MOLLUSCUM CONTAGIOSUM

Molluscum contagiosum results in flesh-colored, pearly papules that are dome-shaped and **umbilicated**. It is caused by the POX virus. NO treatment is needed, but sometimes you may use cryotherapy or topical cantharidin, podophyllotoxin, imiquimod, or potassium hydroxide.

IMAGE: http://pbrlinks.com/MOLLUSCUM1

MNEMONIC:

molluscUMbilicated Papules
O
X

(DOUBLE TAKE) HUMAN PAPILLOMA VIRUS (HPV)

Human papilloma virus (HPV) causes VERRUCA VULGARIS (warts). They can be on the hands, knees, and feet, and in the anogenital region. If genital, the condition is referred to as CONDYLOMA ACUMINATA.

Genital human papilloma virus IS considered to be an STD. In fact, HPV is considered the most prevalent STD of all. Only a small percentage of patients actually carrying HPV get warts. More than 90% of infections are from HPV 6 or HPV 11, which are NOT likely to induce cervical cancer. The risk of cervical cancer **is increased** depending on the subtype (**16 and 18 are most commonly associated with cervical cancer**). Anogenital warts can be due to maternal-fetal transmission and may not present until 3 years after birth! BUT if you note anogenital warts AFTER 3 years of age, think SEXUAL ABUSE. Lesions are NOT tender but easily bleed with minimal trauma. Treat with self-applied topical podofilox or imiquimod. Treatment with cryotherapy or podophyllin is done by a physician.

IMAGE: http://pbrlinks.com/HPV1 (Acuminata)
IMAGE: http://pbrlinks.com/HPV2

MNEMONIC: Don't get confused with molluscum. hp**V** = **VV**arts/Warts = **V**erruca **V**ulgaris = **V**enereal **VV**arts/Warts. "**VV**arts on your hands or knees? It's probably from those darn **V**'s!"

MNEMONIC: The HPV 16 & HPV 18 strains are the two you should remember (associated with the highest risk of cervical cancer): Imagine an adolescent couple. Their birthdays are on the same day, 7/1 (Zodiac of CANCER). The boy is turning 18, and he's excited to finally VOTE. His girlfriend is turning 16, and she's excited because she'll finally get her DRIVER'S LICENSE now that she's celebrating her SWEET SIXTEENTH. As they go to blow out the BIRTHDAY CAKE candles, you notice that she has **VV**arts on her lips! It turns out he also has **VV**arts, but his are **V**enereal (anogenital).

NAME ALERT: 

An "**A**" in **A**cuminata looks like a flipped "**V**," which may help you remember that a diagnosis of Condyloma **A**cuminata represents an hp**V** infection. The "**L**" in Condyloma **L**ata should remind you that you are dealing with syphi**L**is.

CONDYLOMA LATA 

Condyloma lata is found in secondary syphi**L**is = White-gray, coalescing papules. These appear much more FLAT than Condyloma Acuminata.

IMAGE: http://pbrlinks.com/CONDYLOMA1

NAME ALERT: An "**A**" in **A**cuminata looks like a flipped "**V**," which may help you remember that a diagnosis of Condyloma **A**cuminata represents an hp**V** infection. The "**L**" in Condyloma **L**ata should remind you that you are dealing with syphi**L**is.

HERPES SIMPLEX VIRUSES 1 & 2 (HSV 1 & 2)

Herpes simplex viruses 1 and 2 are similar. HSV-2 is usually an STD usually affecting the genitals, while HSV-1 most commonly affects the mouth (gingivostomatitis) but can appear in other sites as well.

Initial infections are often asymptomatic but can be relatively severe with very painful lesions, fever, and lymphadenopathy. Look for multiple painful ulcers or vesicles on the labia or penis (HSV-2) or in and around the mouth (HSV-1). The vesicles are CLUSTERED on an ERYTHEMATOUS BASE. Lesions can also be ULCERATIVE. Diagnose by obtaining a viral culture or HSV PCR. The Tzanck smear is not specific for HSV. Treat with ORAL Acyclovir x 7 days (not topical). Treat babies with **IV** Acyclovir.

HSV becomes latent after the primary infection and can reactivate later. Recurrent infections tend to be less severe and of shorter duration than primary ones. Pain often precedes the appearance of lesions. Patients **DO** shed virus during secondary infections.

IMAGE: http://pbrlinks.com/HSVII1

PEARL: HSV-1 can be associated with a very painful infection called a HERPETIC WHITLOW (typically of a thumb or finger).

IMAGE: http://pbrlinks.com/HSVII2

HERPES SIMPLEX VIRUS ENCEPHALITIS (HSV ENCEPHALITIS)

A question about herpes simplex virus encephalitis (HSV encephalitis) would likely mention fever, seizures, and possibly a CT finding in the **temporal lobe**. Treatment is STAT **IV acyclovir, followed by a lumbar puncture** to obtain fluid for PCR testing. An EEG might show PLEDs (periodic lateralizing epileptiform discharges).

HERPES SIMPLEX VIRUS GINGIVOSTOMATITIS

Herpes simplex virus gingivostomatitis presents with oral and perioral/vermillion border lesions/vesicles. Gingiva is friable and malodorous. There is associated lymphadenopathy. Usually caused by HSV-1.

IMAGE: http://pbrlinks.com/HSVSTOMATITIS1

(DOUBLE TAKE) ECZEMA HERPETICUM

Eczema herpeticum is a potentially life-threatening disseminated herpes (HSV) infection occurring at sites of skin damage, including sites of eczema. Look for HSV Vesicles + Crusted Lesions. Even if a description is not given of a vesicular rash, have a high index of suspicion for a rash "**not improving with steroids and/or antibiotics**." Diagnose with a viral culture for HSV, but do not delay treatment. A Tzanck smear can support the diagnosis. Treat by **STOPPING** topical steroids and/or immunosuppressants and starting **Acyclovir**.

IMAGE: http://pbrlinks.com/ECZEMAHERPE1
IMAGE: http://pbrlinks.com/ECZEMAHERPE2
IMAGE: http://pbrlinks.com/ECZEMAHERPE3

(DOUBLE TAKE) BLUEBERRY MUFFIN SYNDROME

Blueberry muffin syndrome represents extramedullary hematopoiesis. This can be seen in congenital **viral** infections such as **Rubella**, Coxsackie, Cytomegalovirus (CMV), Herpes Simplex Virus (HSV), and Parvovirus. It can also be associated with congenital Toxoplasmosis (a protozoa).

IMAGE: http://pbrlinks.com/BLUEBERRY1
IMAGE: http://pbrlinks.com/BLUEBERRY2

SCABIES

Scabies presents as linear, papular, erythematous, **pruritic**, vesicular, and crusting lesions most often seen in areas with CREASES (wrist, groin, webbing of fingers). You may see burrows. Treat with permethrin overnight from head to toe for the **entire family**. Re-treat if the patient is still having symptoms after 14 days and LIVE MITES are found, because the persisting pruritis can be from residual inflammation. Try topical steroids or antihistamines for that interim.

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

IMAGE: http://pbrlinks.com/SCABIES1

PEDICULOSIS CAPITIS (AKA HEAD LICE)

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

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

IMAGE: http://pbrlinks.com/HEADLICE1

PEDICULOSIS PUBIS (AKA PUBIC LICE or CRABS)

Pediculosis pubis (AKA pubic lice or crabs) is an infection in the groin that results in red, crusted suprapubic macules and possibly bluish-gray dots. There is a STRONG ASSOCIATION with sexual abuse in children.

IMAGE: http://pbrlinks.com/CRABS1

THE "ERYTHEMA" RASHES

ERYTHEMA NODOSUM

For erythema nodosum, look for **PAINFUL**, shiny, red to bluish skin lesions in a patient with a history of a chronic disease or on certain medications. Associations include Crohn's Disease, Ulcerative Colitis, Drugs (oral contraceptives and sulfa drugs), Infections (Yersinia, EBV, Tuberculosis, fungal infections), and Sarcoidosis.

MNEMONIC: For this shiny skin finding, use CUDIS (kind of like CUTIS, which means skin) to help you remember the following associations: **Crohn's**, **U**C, **D**rugs, **I**nfections, and **S**arcoidosis.

IMAGE: http://pbrlinks.com/ERYTHEMA-N1
IMAGE: http://pbrlinks.com/ERYTHEMA-N2
IMAGE: http://pbrlinks.com/ERYTHEMA-N3

(DOUBLE TAKE) ERYTHEMA CHRONICUM MIGRANS

Erythema chronicum migrans (AKA erythema migrans) is caused by BORRELIA BURGDORFERI, the spirochete that causes LYME DISEASE. Look for a large, flat lesion **(> 5 cm) that is annular and has a red border. It is located at the tick bite site in about 75% of patients. The classic description is a "bulls eye" lesion.** The rash shows up 1–2 weeks after the bite. Titers may still be negative during this period. Borrelia is transmitted via the Ixodes deer tick. IF the patient has an acute arthritis, disseminated erythema migrans, a palsy (BELL'S PALSY), or neuropathy, then treat with ORAL medication (**doxycycline if >8 years old, or penicillin or amoxicillin if < 8 years old**). If the patient has **CARDITIS**, neuritis (encephalitis/meningitis), or **RECURRENT** arthritis, treat with INTRAVENOUS medication **(PCN or ceftriaxone)**. Arthritis is usually located at the large joints (especially the **knees**). Diagnosing using labs is often difficult. Obtain **Lyme antibody titers**. If these are positive, confirm with a Western blot. Lyme Disease is often a **clinical diagnosis** (for example, if you see erythema migrans, TREAT).

* **IMAGE**: (BULLSEYE LESION) http://pbrlinks.com/ERYTHEMA-C1

* **IMAGE**: (BELL'S PALSY) http://pbrlinks.com/ERYTHEMA-C2

* **SIDE NOTES**

- BELL'S PALSY: Unilateral facial nerve paralysis (CN VII). It is often idiopathic.
- The Jarisch-Herxheimer reaction results in fever, chills, hypotension, headache, myalgia, and exacerbation of skin lesions during antibiotic treatment of a bacterial disease (typically spirochetes). This is due to large quantities of toxins released into the body. It is classically associated with syphilis but can also occur with Lyme disease. It may only last a few hours.

* **MNEMONICS**:

- From now on, think/say borreLIYME. "Don't ever throw a borreLIYME to MY GRANny!" Or, "Don't ever borre-LIE to MY GRANny." borreLIYME = Borrelia. MY GRANny = Migrans.
- Imagine that BULL'S EYES are made of two bright neon-green LIMES! This should remind of you of the classic description.
- Imagine squeezing LYME into a CAN = **C**arditis, **A**rthritis, and **N**euritis.

(DOUBLE TAKE) ERYTHEMA MARGINATUM

- Erythema marginatum is a transient, erythematous, macular and light colored. It is described as being "SERPENTiginous" (snakelike) and the **MARGIN**s are noted progress as the center clears. It is part of the Jones criteria for Rheumatic Fever.
- **IMAGE**: http://pbrlinks.com/ERYTHEMA1

MNEMONIC: The **E** in **E**rythema is part of the **E** in jon**E**s, and the name **MARGIN**atum should remind you to look for an interesting description of the rash's **MARGIN**s. Erythema **MARGIN**atum.

(DOUBLE TAKE) ERYTHEMA INFECTIOUSUM

Erythema infectiousum IS an INFECTIOUS rash!!! It is caused by Parvovirus B19. It is also called Fifth Disease Look for erythematous facial flushing of the cheeks (sometimes described as "slapped cheeks" appearance). The extremities will have diffuse macular (or morbilliform) erythema (especially on the extensor surfaces) referred to as "lacy" or "reticular." Diagnose with Ig**M** titers. (There is no culture or rapid antigen available.)

PEARLS: The rash occurs AFTER the slapped cheeks rash (often a week later). Patients may also have knee or ankle pain. Parvovirus B19 infection can result in **APLASTIC CRISIS**. Intrauterine exposure can result in **hydrops fetalis**.

MNEMONIC: infectio**5**u**M** = FIFTH disease = "Fiver fingers." Imagine a cheek being SLAPPED with FIVE fingers covered by a white LACY glove with a red M on the back of it (extensor surface). M = Ig**M** titers.

MNEMONIC: ParVoVirus B19: From now on, say/think "par**V**o**V**irus **V**19." **V = Roman numeral 5**!

ERYTHEMA TOXICUM NEONATORUM

See in next section (Newborn Rashes).

ERYTHEMA MULTIFORME

See the Stevens-Johnson syndrome section for more information on erythema multiforme. Look for *target lesions*.

THE NEWBORN RASHES

MILIARIA RUBRA

Look for very superficial vesicles that are easily ruptured in a case of miliaria rubra. This occurs due to obstruction of sweat glands and is also called "prickly heat rash."

IMAGE: http://pbrlinks.com/MILIARIA1

MNEMONIC: Miliaria sounds like malaria, which is usually found in hot countries where you sweat!

MILIA

Milia are small, pearly inclusion cysts that look like little white heads. There's NO associated erythema. If milia are on the nose, they can be very easy to confuse with SEBACEOUS HYPERPLASIA.

IMAGE: http://pbrlinks.com/MILIA1
IMAGE: http://pbrlinks.com/MILIA2

SEBACEOUS HYPERPLASIA

In sebaceous hyperplasia, pinpoint white-yellow papules appear on the nose and central face. There is NO associated erythema. It results due to maternal androgen exposure and is benign.

IMAGE: http://pbrlinks.com/SEBACEOUSHYPERPLASIA1
IMAGE: http://pbrlinks.com/SEBACEOUSHYPERPLASIA2

ERYTHEMA TOXICUM NEONATORUM

Erythema toxicum neonatorum is seen in up to 50% of newborns and consists of **erythematous macules** with raised central lesions (papules **or** vesicles). This is usually seen at birth or by DOL 2. It is a benign rash with an unknown etiology. It usually disappears by DOL 7. Diagnose by noting eosinophils on microscopy.

IMAGE: http://pbrlinks.com/ERYTHEMA-T1

MNEMONIC: Although the name "TOXICum" suggests otherwise, this is a NON-toxic rash resulting in non-toxic looking babies.

MNEMONIC: This is an **E**arly, **E**rythematous, "**E**osinophilled" rash called **E**rythema tox**EEE**cum.

TRANSIENT NEONATAL PUSTULAR MELANOSIS

Transient neonatal pustular melanosis is more common in **African-American kids**. This is a benign rash with NO associated erythema. It starts in utero and is PRESENT AT BIRTH. It resolves within a few days but can leave hyperpigmented macules for a while. Diagnose by examining contents and looking for **PMNs** on Tzanck smear.

IMAGE: http://pbrlinks.com/TRANSIENT1
IMAGE: http://pbrlinks.com/TRANSIENT2

MNEMONICS: Transient neonatal PUStular melanosis should remind you of the PMNs on the Tzanck smear in the PUS-like contents of these PUStules. MELANosis should make you think about dark-skinned individuals (AA kids) and the dark macules that can be left behind.

NEONATAL ACNE (AKA NEONATAL CEPHALIC PUSTULOSIS)

Neonatal acne (AKA Neonatal Cephalic Pustulosis) occurs within the **first month** of life and resolves by 4 months of age. Look for inflammatory pustules on the cheeks and forehead without comedones. This is a benign rash that requires no treatment.

IMAGE http://pbrlinks.com/NCP1

MNEMONIC: NEONATal = FIRST MONTH OF LIFE!

INFANTILE ACNE

Infantile acne looks like typical pubertal acne, but it is found in babies. Onset is usually around 2–3 months of age, and it is due to androgenic stimulation. There can be COMEDONES (whiteheads and blackheads). The rash can resolve in a few weeks or it can take up to a year to resolve.

MNEMONIC: INFANTile = Infants. Don't choose this if the baby is 4 weeks old.

IMAGE: http://pbrlinks.com/INFANTILE1

LIVEDO RETICULARIS (AKA CUTIS MARMORATA)

Livedo reticularis (AKA cutis marmorata) presents as a mottled, reticulate patterned rash and may be described as a lacy rash. It is benign and resolves by 1 month.

IMAGE: http://pbrlinks.com/LIVEDO1
IMAGE: http://pbrlinks.com/LIVEDO2

PEARL: If the baby is healthy and without any concerning symptoms, choose this. If not, consider sepsis in your differential.

ALOPECIA & HAIR FINDINGS

ALOPECIA AREATA

In alopecia areata, there are round/well-circumscribed area(s) of alopecia. Alopecia can be on the scalp or in other areas. Hairs at the periphery of the areas are short, **pluckable**, and may resemble an **exclamation point!**

IMAGE: http://pbrlinks.com/ALOPECIA-A1
IMAGE: http://pbrlinks.com/ALOPECIA-A2
IMAGE: http://pbrlinks.com/ALOPECIA-A3

ALOPECIA TOTALIS

Alopecia totalis is the loss of all hair on the HEAD.

IMAGE: http://pbrlinks.com/ALOPECIA-T1

ALOPECIA UNIVERSALIS

Alopecia universalis is the loss of all hair on the entire BODY. There is usually a **SYSTEMIC** etiology such as hypothyroidism, a nutritional deficiency, or even lupus (SLE).

(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**: http://pbrlinks.com/ZINC1
* **IMAGE**: http://pbrlinks.com/ZINC2
* **IMAGE**: http://pbrlinks.com/ZINC3
* **IMAGE**: http://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!

(DOUBLE TAKE) ACRODERMATITIS ENTEROPATHICA

Acrodermatitis enteropathica is an inherited condition (autosomal recessive) in which there is a zinc transport defect. It can result in **alopecia**, diarrhea, failure to thrive (FTT), and the **rash** of zinc deficiency.

IMAGE: http://pbrlinks.com/ACRODERMATITIS1

(DOUBLE TAKE) BIOTIN/BIOTINIDASE DEFICIENCY

Biotin and biotinidase deficiencies may present with a RASH + ALOPECIA + **NEUROLOGIC SIGNS** (ataxia, coma, etc.). Patients may also have lactic acidosis. Treat with biotin.

MNEMONIC: Imagine the TIN MAN from *The Wizard of Oz* walking with an ATAXIC gait as he SCRATCHES his arm. Notice that he has NO HAIR!

TELOGEN EFFLUVIUM

Telogen effluvium is a form of acute hair shedding that occurs diffusely. Instead of patches, you see "thinning" of the hair. The hair that is shed can be recognized by a small bulb of keratin on the root end. It was too young to shed. This is often related to a psychological or medical stressor. Treat with REASSURANCE because the hair will grow back.

IMAGE: http://pbrlinks.com/TELOGEN1
IMAGE: http://pbrlinks.com/TELOGEN2

TINEA CAPITIS (AKA RINGWORM)

Tinea capitis (ringworm) results in broken hair that looks like "**black dot alopecia."** There is often inflammation, and this condition can be associated with a kerion (a raised spongy lesion). Treat with GRISEOFULVIN. You do not need any baseline labs.

IMAGE: http://pbrlinks.com/TINEACAPITIS1
IMAGE: http://pbrlinks.com/TINEACAPITIS2

TRICHOTILLOMANIA

Trichotillomania is a body-focused repetitive behavior in which patients pull out their hair. (This may be on a location other than the scalp.) Look for loss of hair in an irregular pattern (not a nice circle). Also, the irregularly shaped patches will contain incomplete hair loss in which you will see hair of **differing lengths.**

IMAGE: http://pbrlinks.com/TRICHOTILLOMANIA1
IMAGE: http://pbrlinks.com/TRICHOTILLOMANIA2

(DOUBLE TAKE) ESSENTIAL FATTY ACID DEFICIENCIES

Essential fatty acids include **LINOLEIC ACID** and alpha-linolenic acid. Deficiency results in alopecia, a scaly dermatitis, and **thrombocytopenia**. Treat with IV lipids.

MNEMONIC: Imagine a fish whose red SCALES are shaped like HAIRY PLATELETS. As the fish struggles to find food, it becomes SKINNIER and skinnier (malnourished) and the hairy platelets begin to fall off. What's left is a SKINNY (fat-free), BALD, and THROMBOCYTOPENIC fish!

APLASIA CUTIS CONGENITA

In aplasia cutis congenita, there is a congenital absence of the skin in an area. It is usually in a single location (most often the scalp) but can be in multiple areas. After the lesion heals and scars, a BALD SPOT is left behind. Aplasia cutis can be associated with underlying spinal dysraphisms and underlying skull defects.

IMAGE: http://pbrlinks.com/APLASIACUTIS1
IMAGE: http://pbrlinks.com/APLASIACUTIS2

PEARLS: Look for the HAIR COLLAR SIGN. This is a hairless area with a collar of dense hair at the edges. If given a picture of a scalp with the hair collar sign, get an MRI.

IMAGE: http://pbrlinks.com/APLASIACUTIS3

GASTROENTEROLOGY

LIVER DISEASE

CONGENITAL HEPATIC FIBROSIS

Congenital hepatic fibrosis is associated with POLYCYSTIC KIDNEY DISEASE and can lead to varices and portal hypertension.

HEPATOMEGALY

Hepatomegaly is defined as a palpable liver > 1 cm below the costal margin, or a liver that crosses the midline. A palpable liver edge is normal, and is especially notable in a newborn.

GALLBLADDER HYDROPS

Gallbladder hydrops refers to RUQ pain from acute swelling or distension of the gallbladder in the absence of any gallstones. It may be associated with FASTING, HENOCH-SCHONLEIN PURPURA, KAWASAKI SYNDROME, STREP PHARYNGITIS, and TPN.

MNEMONIC: Imagine a Las Vegas "STREPPer" who has breasts that look like giant GREEN WATER BALLOONS. She's riding a KAWASAKI motorcycle through a drive-thru to get a BURGER IN A BAG. She gets on the road; her speedometer only reads FAST or slow. She goes FAST, but when she tries to eat her BURGER IN A BAG, she flies off the bike and gets BRUISES on her BUTT and LEGS.

* **KEY:** STREPPer = Strep pharyngitis, GREEN WATER BALLONS = Gallbladder, KAWASAKI = Kawasaki Syndrome, BURGER IN A BAG = TPN, and the BRUISED BUTT and LEGS = HENOCH-SCHONLEIN PURPURA.

HEPATOBLASTOMA

A hepatoblastoma is a malignant liver neoplasm in infants and children. It usually presents as an abdominal mass by 3 years of age. ALPHA-FETOPROTEIN (AFP) levels are high. The prognosis is poor.

PRIMARY SCLEROSING CHOLANGITIS (PSC)

Primary sclerosing cholangitis (PSC) is a chronic cholestatic liver disease resulting from autoimmune inflammation leading to fibrosis of the intrahepatic and extrahepatic biliary tree. It is diagnosed with CHOLANGIOGRAPHY (AKA ERCP or endoscopic retrograde cholangiopancreatography), which looks for beading and stenosis of the biliary ducts. ULCERATIVE COLITIS and elevated p-ANCA ((Perinuclear Anti-Neutrophil Cytoplasmic Antibodies) levels are both frequently associated with PSC. "PSC often = UC."

PEARL: Since the biliary tree is affected, **look for GGT elevation**. Bilirubin levels are elevated in the advanced stages.

HEPATOBILIARY IMINODIACETIC ACID SCAN (AKA HIDA SCAN or CHOLESCINTIGRAPHY)

A hepatobiliary iminodiacetic acid scan (AKA HIDA scan or cholescintigraphy) utilizes a nuclear medicine tracer that is injected into an IV. The gallbladder should then be visible within one hour post-injection. If the gallbladder is not seen, there is either CHOLECYSTITIS or CYSTIC DUCT OBSTRUCTION.

IMAGE: http://pbrlinks.com/HIDASCAN1

TRANSAMINITIS

Mild transaminitis is common with many viral infections. If transaminases are in the thousands, the diagnosis is likely VIRAL HEPATITIS.

PEARLS: If the ALT is higher than the AST, that is also suggestive of VIRAL HEPATITIS. AST > ALT usually means there is an alcoholic hepatitis. This would only be presented in a teen patient.

ALKALINE PHOSPHATASE

Alkaline phosphatase levels are elevated in biliary and bone disease (e.g., biliary obstruction, bony tumors/metastases, PAGET'S DISEASE).

PEARL: A Gamma-Glutamyl Transpeptidase (GGT) level will guide you to the source of an elevated alkaline phosphatase. GGT is elevated in hepatic disease. It is normal in diseases of the bone.

BILIARY OBSTRUCTION

After a biliary obstruction, AST becomes elevated first, followed by alkaline phosphatase.

CAUSES OF JAUNDICE

Jaundice may be due to hyperbilirubinemia from common neonatal jaundice etiologies (discussed in the Neonatology section), hemolytic jaundice (discussed in the Hematology section), or liver or biliary diseases (discussed in this chapter).

PEARL: When evaluating a patient with jaundice, if a hepatobiliary etiology is suspected, look at the transaminase and alkaline phosphatase levels to help differentiate CHOLESTATIC DISEASE from HEPATOCELLULAR JAUNDICE. In hepatocellular jaundice, there will be an associated transaminitis. In cholestatic jaundice, there will be a marked elevation in alkaline phosphatase.

CHOLESTASIS

Cholestasis may present in the neonatal period. Look for possible acholic (pale or gray) stools, hepatomegaly, and an elevation in the DIRECT (or CONJUGATED) bilirubin. A **HIDA scan** will show hepatic uptake without biliary excretion due to an obstructive process.

BILIARY ATRESIA

In cases of biliary atresia, look for an elevation in the direct bilirubin (AKA conjugated bilirubin) in a neonate. If found, obtain an abdominal ultrasound followed by a HIDA scan. If left untreated, this can result in liver failure. Until the liver fails, conjugation of bilirubin continues. KERNICTERUS only occurs once the liver fails and indirect bilirubin (AKA unconjugated bilirubin) can no longer be conjugated. (Only unconjugated bilirubin can cross the blood-brain barrier.)

CHOLEDOCHAL CYSTS

Choledochal cysts are congenital cystic dilations of the biliary tree. Along with **jaundice**, other symptoms may include an **abdominal mass, RUQ abdominal pain**, nausea, vomiting, and pancreatitis. These patients carry an increased risk of cancer. Treatment is surgical removal of the cyst.

IMAGE: (Various types of choledochal cysts) http://pbrlinks.com/CHOLEDOCHAL1

PROGRESSIVE FAMILIAL INTRAHEPATIC CHOLESTASIS (PFIC)

There are three types of Progressive Familial Intrahepatic Cholestasis (PFIC). Various types of defects can lead to cholestasis with hyperbilirubinemia and eventual cirrhosis. Average age of onset is 3 months, though may be as late as adolescence. The classic triad of intermittent abdominal pain, jaundice and a right upper quadrant abdominal mass is rare. PFIC is more common in Asia. In PFIC type I, bilirubin is formed but it is formed improperly. Look for a **direct hyperbilirubinemia and severe pruritus**. In PFIC I and II, since the biliary tree/piping is normal, the GGT will be normal. In PFIC III, there is a mutation that causes damage to biliary duct epithelium. This results in very high GGT levels.

PEARLS: The initial disorder was described in the Amish community, so keep an eye out for such an association on the exam.

MNEMONIC: "pfic thrEE has the HIGH ggTEE!"

ALAGILLE SYNDROME (AKA ARTERIOHEPATIC DYSPLASIA)

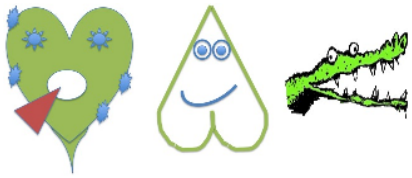
Alagille Syndrome (AKA arteriohepatic dysplasia) is a genetic disorder in which jaundice is noted in the newborn period. Look for a child with LIVER and HEART disease. Here are some associations: **paucity of bile ducts (AKA intrahepatic biliary atresia or hypoplastic biliary ducts)**, **pulmonary stenosis, a triangular face** (underdeveloped mandible → small, pointed chin), Tetralogy of Fallot, hypercholesterolemia with xanthomas, eye abnormalities, and acholic stools.

IMAGE: http://pbrlinks.com/ALAGILLE1

MNEMONIC: (image #1) The GREEN ALLIGATOR has a TRIANGULAR FACE that is green because it is filled with BILE. His head is shaped like a HEART to remind of the PULMONARY STENOSIS. He also has funny-shaped EYES and XANTHOMATOUS lumps all over his face. Also, notice what he's eating. It's a LIVER!

MNEMONIC: (image #2) This cute little guy is named Alagille. He has a TRIANGULAR head and a funny-shaped JAW that is in the shape of a HEART to remind you of PULMONARY STENOSIS.

MNEMONIC: (image #3) That's Al the Green Alligator.



IDIOPATHIC NEONATAL HEPATITIS

Idiopathic neonatal hepatitis is a diagnosis of exclusion! You must do a workup first. If nothing is found except for enlarged hepatocytes on biopsy, it is likely this. This will likely resolve by 8 months of age.

VIRAL HEPATITIS

PEARL: When evaluating a viral hepatitis serologies, always note whether the exam gives you A**g** or A**b**. Knowing how to interpret the presence of antigens and antibodies is key for this section.

MNEMONIC: There are many types of viral hepatitis: A, B, C, D, and E. To remember which ones are transmitted fecal-orally, imagine that A is the beginning of the GI tract, and E is the end. So Hep A and Hep E are transmitted fecal-orally, while the rest (B, C, and D) are transmitted through B-C-D (Blood, Cex/Sex, and Drugs).

HEPATITIS A

The incubation period of hepatitis A is approximately 4–6 weeks. Children may be asymptomatic (symptoms are much worse in adults). Jaundice can relapse for up to one year.

- **PEARLS:** Look for a child with elevated transaminases, recent travel, and what sounds like a viral syndrome. DO NOT OBTAIN AN IgG LEVEL FOR DIAGNOSIS. It is persistent for life. Diagnose with IgM. Also, keep in mind that many viral syndromes can cause a mild transaminitis. In viral hepatitis, look for elevation in the many hundreds to thousands.

HEPATITIS B

Hepatitis B SURFACE ANTIGEN (HBsAg) persistence beyond 6 months means there is a chronic infection. HEPATITIS B "E" **ANTIGEN** (HBe**Ag**) presence means there is high replication resulting in a high viral load and **high infectivity**. The WINDOW PERIOD is the period after the Hep B surface **Ag** presents and before the Hep B surface Abs (anti-HBs) are made. Diagnosis during this time can be difficult **UNLESS anti-HBc** (IgM, anti-core antibody) is obtained. If this test is positive and everything else is negative, then this is the WINDOW PERIOD.

If the IgG antibodies to core and surface antigens are both positive, this represents a PAST INFECTION (not immunization). If only anti-HBs is positive, that represents prior immunization.

- **IMAGE**: http://pbrlinks.com/HEPATITISB1
- **IMAGE:** http://pbrlinks.com/HEPBSEROLOGY
- **PEARL**: Vertical transmission is a very common mode of transmission.
- **MNEMONIC**: HB**e**Ag reflects high "**E**nfectivity"

HEPATITIS C

Hepatitis C is the most common blood-borne infection in the U.S. and the most common etiology of chronic viral hepatitis. Kids with Hep C are mostly asymptomatic. Symptoms show later with liver CA and cirrhosis.

GILBERT'S SYNDROME (AKA GILBERTS SYNDROME)

In Gilbert's Syndrome, there is a glucuronyl transferase deficiency (therefore conjugation of bilirubin is decreased). It's the most common inherited cause of **indirect** hyperbilirubinemia. The **intermittent indirect** hyperbilirubinemia is mild (usually with levels < 3), extremely common, and BENIGN. It is usually autosomal recessive. It is usually noted at times of illness and physiologic stress (dehydration, fasting, and even in vigorous exercise).

MNEMONIC: g**I**l**B**ert's. The "**I**" represents Indirect. The "**B**" represents both **B**ilirubinemia and the fact that this is a **B**enign condition.

CRIGLER-NAJJAR SYNDROME

In Crigler-Najjar Syndrome, there is a glucuronyl transferase deficiency (therefore conjugation of bilirubin is decreased). This results in **indirect** hyperbilirubinemia and is rare.

* TYPE 1: **NO DIRECT BILIRUBIN** (zero) because of a complete lack of glucuronyl transferase. There is severe INDIRECT hyperbilirubinemia and jaundice within the first days of life. Type 1 requires lifelong phototherapy.
* TYPE 2: Glucuronyl transferase function partially exists, so patients do well with the partial conjugation indirect to direct bilirubin. They do not need phototherapy.

MNEMONIC: Najjar sounds like NINJA. Imaging two ninjas born to a man named Craig. The first-born carries 1 sword (Type 1) and is always seen **wearing protective goggles** because he'll need life-long phototherapy to protect himself.

MNEMONIC: (image)

DUBIN JOHNSON SYNDROME

In Dubin Johnson Syndrome, there is a mild **DIRECT** hyperbilirubinemia. It is benign.

MNEMONIC: **D**u**B**in Johnson = **D** for **D**irect, **B** for **B**enign.

REYE'S SYNDROME (AKA REYES SYNDROME)

Reyes syndrome is an acute non-inflammatory encephalopathy associated with liver function abnormalities. It is rare. Look for a recent viral URI or varicella infection in the setting of aspirin use. Symptoms will include encephalopathy, possible coma, abnormal LFTs, possibly an elevated PT, and hyperammonemia.

PEARL: This disease only occurs once in a patient's life.

(DOUBLE TAKE) WILSON'S DISEASE

Wilson Disease (AKA Wilson's Disease) is an autosomal recessive disorder resulting in excess copper accumulation, especially within the liver and brain. Accumulation in the liver can lead to **hepatomegaly**, spider nevi, esophageal varices, and a Coombs-negative hemolytic anemia. Accumulation in the brain can lead to **neurologic changes** including tremors, poor school performance, ataxia, abnormal eye movements, and spasms. On eye exam, a Kayser-Fleischer ring may be visible. Copper levels are **low** in the serum but high in the **tissues**. Diagnose by LIVER BIOPSY. Treat with PENICILLAMINE, a copper chelator.

PEARLS: Diagnose by LIVER BIOPSY. Abnormal eye movements and a Fleischer ring may be present, but there is no visual disturbance. Kayser-Fleischer rings are seen in 90% of symptomatic patients, and almost 100% of patients with neurologic manifestations. **Screen** family members with CERULOPLASMIN levels. Ceruloplasmin is made in the liver and is the primary copper-carrying protein. If the level is **LOW**, that suggests Wilson's Disease because excess copper is not being incorporated into ceruloplasmin, and is therefore still in the TISSUES. Therefore, supportive labs may include a low serum ceruloplasmin, high tissue copper levels, high urine copper levels and low serum copper levels.

IMAGE: http://pbrlinks.com/WILSONS1
IMAGE: http://pbrlinks.com/WILSONS2

MNEMONICS: Treat with a "**COPPER PENNY-**cillamine." Also, ever heard of Wilsons, the leather company that **makes** baseball gloves? See the image below to note the strong resemblance in color to a COPPER PENNY. This should help you remember that Wilsons Disease has to do with copper, and that it is treated with "PENNY-cillamine."

CHOLECYSTITIS 

Cholecystitis is an inflamed and thickened gallbladder wall, usually due to gallstone obstruction of the cystic duct. The illness presents with fever and RUQ abdominal pain (Murphy's Sign). A RUQ mass is sometimes palpable. Unlike adults, many children also have JAUNDICE. Fatty meals exacerbate the pain, which may radiate to the right scapula or shoulder. Acalculous cholecystitis occurs in the absence of gallstones and may be associated with hemolysis, TPN sepsis, prolonged fasting, or obesity. Diagnosis is usually made by ultrasound, though a HIDA scan may be needed in some cases to confirm.

PEARL: Instead of abdominal pain, you may be presented with a child complaining of SHOULDER pain (referred).

NAME ALERT: CholeCYSTitis refers to the inflammation of the gallbladder (cyst = bladder, the same way cystitis refers to inflammation of the bladder). CholeLITHiasis simply means there are stones in the gallbladder. CholANGItis is a medical emergency that classically presents with Charcot's triad of fever, RUQ pain, and leukocytosis from an infection in the biliary tract.

CHOLELITHIASIS

Cholelithiasis is a term for gallstones in the gallbladder. The patient may present with similar symptoms as cholecystitis. Patients are more likely to have jaundice and icterus (yellowing of the sclera). Unlike in cholecystitis, hepatosplenomegaly may be present. Cystic fibrosis, TPN use, and a history of ceftriaxone use increase the risk of cholelithiasis.

ICTERUS

Icterus is a yellowing of the sclera.

PEARL: A patient who presents with yellow-orange skin but DOES NOT HAVE ICTERUS likely has had excessive beta carotene ingestion (apricots, carrots, sweet potatoes, pumpkins, etc.).

CAUSES OF ABDOMINAL DISCOMFORT & PAIN

CLASSIC FUNCTIONAL ABDOMINAL PAIN OF CHILDHOOD

Classic functional abdominal pain of childhood is a periumbilical, crampy abdominal pain that does not radiate in an otherwise healthy, prepubertal child. It may be recurrent.

CONSTIPATION

For cases of constipation, use stool softeners, fiber supplements, and osmotic agents/laxatives to "clean them out." Then focus on good bowel habits, appropriate fiber intake and avoidance of withholding through positive reinforcement.

PEARL: Avoid suppositories and enemas in simple constipation, as they may be traumatic.

FECAL OVERFLOW ENCOPRESIS

Patients with fecal overflow encopresis may present with LLQ pain. You may use more aggressive measures, such as enemas and suppositories for initial "clean out."

HELICOBACTER PYLORI

* PEPTIC ULCER DISEASE (AKA H. pylori induced PUD)

If a patient is diagnosed with an ulcer of any type that is found to be positive for *H. pylori*, treatment will require a proton pump inhibitor (PPI) and antibiotics. Possible regimens include:

- PPI + Amoxicillin + Clarithromycin
- PPI + Amoxicillin + Metronidazole (good if the patient can't tolerate clarithromycin)
- PPI + Clarithromycin + Metronidazole (good if the patient is allergic to penicillin)

* NODULAR GASTRITIS: The most common etiology is H. pylori. An EGD with biopsy (samples sent for pathology) is the gold standard for diagnosis. This can also be found in Crohn's disease.

  - **IMAGE:** http://pbrlinks.com/HELICOBACTER1

* CAMPYLOBACTER-LIKE ORGANISM TEST (AKA CLO test or Rapid Urease Test): Just know that this can be used at the time of an EGD to help diagnose. It's faster and cheaper than sending a biopsy specimen to pathology, but it is not as specific as an EGD with biopsy.

* UREASE BREATH TEST: This is a noninvasive means to attempt diagnosis of Helicobacter pylori.

NSAID-INDUCED DYSPEPSIA, ULCERS, AND EROSIVE GASTRITIS

NSAID-induced dyspepsia, ulcers, and erosive gastritis result from the inhibition of PGE and thus a decrease in the protection of the gastric mucosa.

PEARL: Especially consider this diagnosis in any patient with a chronic pain illness, such as Sickle Cell Anemia or Juvenile Rheumatoid Arthritis.

EROSIVE GASTRITIS AKA EROSIVE GASTROPATHY

Erosive gastritis (AKA erosive gastropathy) may be caused by NSAIDS, exercise, *H. pylori*, or the stress of surgery. NSAIDS are the most common cause. "Erosive" refers to mucosal injury. Erosive gastritis and duodenal ulcers may cause black stools!

NON-EROSIVE GASTRITIS

Non-erosive gastritis is associated with Crohn's Disease, eosinophilic esophagitis and H. pylori. "Non-erosive" refers to the lack of mucosal injury (only erythema is noted on EGD).

NON-ULCER DYSPEPSIA

Non-ulcer dyspepsia is a chronic or recurrent epigastric discomfort associated with early satiety, bloating, belching, nausea, and possibly reflux-like symptoms.

ZOLLINGER-ELLISON SYNDROME

Zollinger-Ellison Syndrome results in multiple GI ulcerations due to a gastrin tumor of the pancreas. Diagnose by checking for a high **fasting** gastrin level. If diagnosed, you will need to rule out Multiple Endocrine Neoplasia Type 1 (MEN Type 1), which is associated with tumors of the PPP: Pituitary gland, Parathyroid hormones, and Pancreas.

INFANTILE GASTROESOPHAGEAL REFLUX (GERD)

Infantile gastroesophageal reflux (GERD) may be noted in up to half of all children 4–6 months of age and usually resolves by 12 months of age. There is no need to treat if the child is healthy and doing well otherwise.

(DOUBLE TAKE) IRRITABLE BOWEL SYNDROME (IBS)

Irritable bowel syndrome (IBS) is a crampy abdominal pain associated with diarrhea **or** constipation. Symptoms may alternate. This is a diagnosis of EXCLUSION. Treat with fiber.

PEARLS: There must be some type of poop issue! There's often an emotional component as well. Do not choose this answer unless at least some type of workup has been done already. If no workup has been done, start with noninvasive tests such as a CBC, ESR, anti-TTG, and stool guaiac. Do not choose an invasive test unless other tests are negative and the patient failed a FIBER trial. Non-invasive testing -> Fiber trial -> EGD and/or Colonoscopy.

INFLAMMATORY BOWEL DISEASE (IBD) – CROHN'S AND ULCERATIVE COLITIS

Know the similarities and differences between the different inflammatory bowel diseases (Crohn's Disease and Ulcerative Colitis). Both are associated with HLA B27 and toxic megacolon. Both also have similar treatments.

* **ULCERATIVE COLITIS** (UC) typically presents in a TEEN of an **Ashkenazi (European) Jewish** descent. Look for a history of chronic, crampy lower abdominal pain that may or may not be associated with bloody stool. If there is severe colitis, fever may be present. Lab findings may include hypoalbuminemia and anemia. Diagnosis is by colonoscopy and biopsy. First-line treatment includes 5-ASA (AKA mesalazine or 5-aminosalicylic acid). Second-line therapy options include steroids, metronidazole, azathioprine, cyclosporine, methotrexate, and tacrolimus.

    - **PEARLS:** An acute ulcerative colitis flare can make the colon very fragile and susceptible to PERFORATION. If "barium enema" is an option, cross it out. Also, remember that this disease refers to a COLITIS and is primarily a LARGE BOWEL disease.

* **CROHN'S DISEASE** may present simply as short stature and weight loss prior to the onset of any of the usual GI symptom of diarrhea. GI findings may include transmural ulcers in a "skip lesion" pattern, noncaseating granulomas of the upper GI tract, and perianal fistulas. Other manifestations include hepatic disease, erythema nodosum, pyoderma gangrenosum, and uveitis. Supportive laboratory data may include elevated inflammatory markers (ESR or CRP) and anti-Saccharomyces antibodies. Treatment options include 5-ASA, steroids, metronidazole, and immunomodulators/immunosuppressives.

    - **PEARL**: Unlike UC, Crohn's lesions can occur anywhere from the mouth to the anus. Also, a future conversion to cancer is the RULE for ulcerative colitis patients, but that is not the case for Crohn's patients.
    - **MNEMONIC**: A positive "anti-sa**CROHN**amyces" antibody can help make the diagnosis.

APPENDICITIS

Consider a diagnosis of appendicitis in any child **> 2 years of age** with abdominal pain. Look for a child who is not hungry and has periumbilical abdominal pain that migrates to the right side of the abdomen (RLQ at McBurney's Point). This may be associated with diarrhea. A psoas sign might be noted on exam, and a "sentinel loop" of bowel with absence of air in the RLQ may be noted on an X-ray. Inflammation may be seen on a CT of the abdomen. An ultrasound can also be used, but all you truly need to make the diagnosis is a good history. Treatment is surgery (if the story fits, take the child to surgery). If the appendix has already ruptured, the patient may suddenly be PAIN-FREE. In that case, an acceptable choice may be to give IV antibiotics with plans for a delayed appendectomy (weeks later!).

PEARL: The psoas sign is the finding of **abdominal pain** elicited by passively extending the thigh of a patient lying on his side with knees extended, or asking the patient to actively flex his thigh at the hip.

IMAGE: (video) http://pbrlinks.com/APPENDICITIS1

PANCREATITIS

Pancreatitis is an inflammation of the pancreas resulting in epigastric pain that can be associated with rebound, guarding, nausea, vomiting, or fever. Pain often radiates to the back. Possible etiologies include trauma, cystic fibrosis, hereditary pancreatitis, and pancreatic duct obstruction, but it's most often idiopathic. Diagnose with abdominal ULTRASOUND (extremely specific) or CT of the abdomen. Supportive labs include elevated biomarkers, such as ISOamylase, amylase, and lipase. An ERCP may be obtained if the patient has RECURRENT bouts of pancreatitis.

PEARL: Many GI diseases result in hyperactive bowel sounds. With pancreatitis, bowel sounds can be DECREASED.

INTUSSUSCEPTION

Intussusception is a telescoping of the bowel into an adjacent segment of bowel, often in the ileocecal area (AKA ileocolic area). This can result in **intermittent** episodes of abdominal pain, currant jelly stools, bilious emesis, a palpable mass, and even a septic clinical picture without fever. It usually occurs in children 3 months to 6 years of age. Treatment options include either an air contrast enema with a small amount of barium, or a barium enema.

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

MNEMONIC: **intusSIXception** should help to remind you that this condition is seen in kids up to **SIX** years of age.

NAME ALERT: 

Currant jelly SPUTUM is a buzz word for KLEBSIELLA pneumonia.

(DOUBLE TAKE) GIARDIA

Giardia presents with intermittent watery diarrhea that has been going on for weeks. This may be accompanied by abdominal distension and may eventually cause malabsorption. Diagnose with a STRING TEST + ELISA, and **then** treat with metronidazole.

PEARLS: History may include a camping trip or a child in daycare.

ABDOMINAL PAIN PEARL

Keep in mind other causes of abdominal pain and discomfort, such as pneumonia, testicular torsion, torsion appendix, and inguinal hernias.

CAUSES OF DIARRHEA

CHRONIC NONSPECIFIC DIARRHEA

Chronic nonspecific diarrhea is very common. The patient presents with malodorous stool that is often **intermittently** loose and may contain particles of food. This is usually seen in children with a diet that is low in fat and high in carbohydrates. Treat by increasing fat intake and decreasing carbohydrates.

(DOUBLE TAKE) LACTOSE INTOLERANCE (AKA LACTASE DEFICIENCY)

It is **not** common for kids < 5 years old to have lactose intolerance (AKA lactase deficiency). So for the pediatric boards, if the child is less than 5 years of age, suspect a different diagnosis!

* SYMPTOMS: Diarrhea ± abdominal pain.

* The **HYDROGEN BREATH TEST** can be used to help diagnose a lactase deficiency (as well as bacterial overgrowth). When the patient takes a carbohydrate load; if he or she is unable to digest the carbs because of a lack of lactase, the bacteria will digest the carbs and release hydrogen (which can be measured in the breath).

* TREATMENT: SOY milk. It does not contain lactose. It contains sucrose.

* **PEARLS:** Stool is NOT malodorous and does NOT have food particles. These patients do NOT vomit and do NOT have an associated rash. consider the diagnosis of an ALLERGY if you're presented with a patient with such symptoms.

* **MNEMONICS:** "**LACT**ose comes from the **LACT**ating breasts of women and cows, NOT from soy beans."

BACTERIAL OVERGROWTH

Bacterial overgrowth is often seen in children with short bowel syndrome and will result in malabsorption. As mentioned above, an elevated fasting breath HYDROGEN test can make the diagnosis. Diagnosis is also supported by an elevated D-lactic acid level (not L-lactic acid level).

CELIAC DISEASE (AKA CELIAC SPRUE)

Classic symptoms of celiac disease (AKA celiac sprue) include malodorous stool that is bulky and frothy. The patient may also have a distended abdomen and signs of malabsorption. Patients have a GLUTEN sensitivity. Malabsorption may be evidenced by noting reducing substances in the stool (caused by **patchy villous atrophy** of the GI mucosa leading to lactase deficiency), or noting split fats in the stool (indicates the pancreas is working and there is adequate lipase, but malabsorption is present). Supportive blood tests include the presence of antiendomysial antibodies, anti-Tissue TransGlutaminase (anti-TTG is much more sensitive, similarly specific, and easier for labs to process), and the newer anti-deamidated gliadin peptide (DGP) assays (highest specificity). The gold standard for **diagnosis** is upper endoscopy and duodenal biopsy showing villous atrophy. Treat with a gluten-free diet.

PEARLS: This is often associated with Type 1 Diabetes Mellitus. If presented with a DM patient that is having GI symptoms, or is losing weight, consider CELIAC DISEASE. If there is a high suspicion for celiac disease, you may empirically treat with a gluten-free diet and monitor for improvement rather than performing an invasive procedure (upper endoscopy).

INFECTIOUS DIARRHEAL ILLNESSES

SEE ID SECTION FOR AN EXTENSIVE LIST OF INFECTIOUS DIARRHEAL ILLNESSES

CAUSES OF CONSTIPATION

FUNCTIONAL CONSTIPATION

Look for functional constipation in a child who has, or had, difficulty with toilet training and has had problems with frequent soiling or stool incontinence. The child may have a tendency to withhold stool.

PEARL: Diagnosis of exclusion. Look for a child with a history of soiling himself who has **stool in the rectum** on rectal exam. This can be differentiated from IBS by the **absence** of diarrhea, bloating, etc.

(DOUBLE TAKE) IRRITABLE BOWEL SYNDROME (IBS)

Irritable bowel syndrome (IBS) is a crampy abdominal pain associated with diarrhea **or** constipation. Symptoms may alternate. This is a diagnosis of EXCLUSION. Treat with fiber.

PEARLS: There must be some type of poop issue! There's often an emotional component as well. Do not choose this answer unless at least some type of workup has been done already. If no workup has been done, start with noninvasive tests such as a CBC, ESR, anti-TTG, and stool guaiac. Do not choose an invasive test unless other tests are negative and the patient failed a FIBER trial. Non-invasive testing -> Fiber trial -> EGD and/or Colonoscopy.

CONGENITAL HYPOTHYROIDISM

Look for constipation + delayed anterior fontanelle closure, a hoarse cry, poor growth, or an umbilical hernia to indicate congenital hypothyroidism.

CYSTIC FIBROSIS (CF)

Cystic fibrosis (CF) should be top on your list for any newborn who does not produce stool within 48 hours!

HIRSCHSPRUNG DISEASE

Hirschsprung Disease results in constipation **early** in infancy and tends to present prior to 2 years of age. There are no problems with soiling. It can be associated with poor oral intake, abdominal distension, occasional diarrhea, and bilious emesis. It may present as FTT. Boys are more often affected. Patients have an aganglionic (lack of parasympathetic innervation) segment of bowel that is **narrow or contracted, and can eventually result in megacolon proximal to that segment**. There are strong associations with DOWN SYNDROME and CYSTIC FIBROSIS. Diagnosis is by biopsy.

PEARLS: Look for early history of constipation, delayed passage of meconium, an absence of fecal incontinence, and the ABSENCE of stool in the rectum on rectal exam. Also keep in mind the associations with Trisomy 21 (constipation + syndromic features) and CF patients (constipation + foul-smelling stools). If you are presented with images, remember that the **narrow** segment of the bowel (next to normal or dilated bowel) is the affected/aganglionic segment.

IMAGE: http://pbrlinks.com/HIRSCHSPRUNG1
IMAGE: http://pbrlinks.com/HIRSCHSPRUNG2

MECONIUM ILEUS

Meconium ileus results in abdominal distension and vomiting after birth. It's due to thickened meconium causing an obstruction in the ileum. You may find palpable bowel cords on exam. X-ray may show ground glass or "soap and bubble" stool, calcifications, or air-fluid levels. Contrast enema may show a microcolon (small from the splenic flexure to the anus). There may be dry meconium pellets in the small intestine.

PEARLS: Meconium ileus is often the presenting symptom of **CYSTIC FIBROSIS**.

CAUSES OF VOMITING

GASTROESOPHAGEAL REFLUX DISEASE (GERD)

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

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

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+ ions in an effort to hold on to K+ ions. This results in even more alkalosis.

* **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.

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

* **IMAGE**: (String Sign) http://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.

ANTRAL WEB

An antral web is a membrane in the antrum of the stomach that can cause gastric outlet obstruction. It is usually formed before birth. It can present as polyhydramnios in utero, or non-bilious emesis in an infant less than 6 months of age. Imaging with barium may reveal a filling defect in prepyloric region.

ESOPHAGEAL WEB

An esophageal web can cause reflux-like symptoms, esophageal impaction, and chest pain. It results from the failure of the esophagus to re-canalize in utero. The web then acts as an obstruction to the passage of a food bolus. Liquids, however, pass through more easily. Treatment requires dilation of the esophageal web.

IMAGE: http://pbrlinks.com/ESOPHAGEALWEB1
IMAGE: http://pbrlinks.com/ESOPHAGEALWEB2

PEARL: The "jet phenomenon" refers to the thin area of barium seen when looking at a barium swallow. It starts at the initial point of constriction. When that area is tortuous (http://pbrlinks.com/ESOPHAGEALWEB3), it can resemble a TE fistula. When it's linear it does not (http://pbrlinks.com/ESOPHAGEALWEBPDF – page 1 – see it and move on!).

ACHALASIA

Achalasia is caused by a dysmotility problem, or a lack of relaxation at the lower esophageal sphincter, which results in forceful emesis. There is eventual esophageal dilatation and loss of peristalsis ability. Look for forceful vomiting, difficulty swallowing (dysphagia), and weight loss or FTT.

VOLVULUS

A volvulus can cause bilious emesis, abdominal distension, and possibly even bloody stools (from ischemia and necrosis of bowel). This occurs due to a rotational defect during embryology resulting in poorly-fixed bowel (the rotational defect is called a MALROTATION). This leads to the bowel wrapping around the superior mesenteric artery (SMA) and causing bowel ischemia, and therefore requires emergent surgery. A double bubble may be seen on imaging.

PEARLS: An upper GI series is the preferred and gold-standard study, though a barium enema can be helpful when the UGI is inconclusive. There may be a "corkscrew" appearance of the duodenum. On barium enema, the cecum may be in the wrong place (abnormally high). An abdominal X-ray may be shown with a "double bubble" sign.

IMAGE: http://pbrlinks.com/VOLVULUS1 (corkscrew)
IMAGE: http://pbrlinks.com/VOLVULUS2 (corkscrew)
IMAGE: http://pbrlinks.com/VOLVULUS3

ANNULAR PANCREAS

Look for a history of polyhydramnios and then vomiting in a neonate. The annular pancreas forms a ring around the intestine. This causes poor swallowing in utero, resulting in polyhydramnios, and then vomiting in the neonatal period.

IMAGE: http://pbrlinks.com/ANNULARPANCREAS1

CYCLIC VOMITING

Cyclic vomiting is associated with intermittent episodes of repeated vomiting with periods of complete normalcy. There is likely to be an emotional component to the question, either in the patient or the family, and there may also be a history of migraines or IBS. Treatment may include hydration and/or prophylactic medications similar to those used in migraine patients: Amitriptyline (or similar tricyclic antidepressant/TCA), cyproheptadine, or propranolol.

PEARL: This is a diagnosis of exclusion, so make sure somewhat of a workup has been done before choosing this answer.

RUMINATION

Rumination is when a child chews something over and over again. This occurs in patients with mental retardation and in some children who are emotionally disturbed.

BILIOUS EMESIS IN A NEWBORN

Bilious emesis is a surgical emergency in a newborn! Look for evidence of duodenal atresia or malrotation. In older children, bilious emesis can be less severe/emergent.

* Duodenal Atresia = bilious emesis on **1st DOL**, double bubble on KUB. The patient could have jaundice due to increased enterohepatic circulation. If there is "complete" atresia, there will be NO SECOND BUBBLE—NO AIR BEYOND ATRESIA. Remember, 1st DOL, NOT at 2 months. (If a 2-month-old baby has bilious emesis, consider pyloric stenosis, although that's typically NON-bilious!!!)

PEARL: For enterohepatic circulation, think of it as the following circuit: the LIVER processes "something" (bilirubin, medications, etc.) → that "something" gets excreted into BILE → the bile goes into the BOWEL → and then that "something" can potentially be absorbed AGAIN from the bowel! So if stool isn't moving along, reabsorption of that "something" will be increased.

DOUBLE BUBBLE

A "double bubble" refers to the radiologic sign noted when there is a duodenal obstruction. There will be a large "bubble" and a small "bubble." These represent a dilated stomach and a dilated duodenum, respectively. Associated conditions include **volvulus due to malrotation, duodenal atresia, duodenal webs, and antral webs.**

IMAGE: http://pbrlinks.com/DOUBLEBUBBLE1

VOMITING PEARLS

* One episode of vomiting in an otherwise healthy child probably warrants reassurance.

* Always keep in mind infections (pneumonia, urinary tract infections, gastroenteritis, rotavirus, etc.) as possible causes of emesis.

* Inborn errors of metabolism and Diabetic Ketoacidosis (DKA) are a couple of metabolic causes of emesis.

GI BLEEDING

GI BLEEDING PEARL

STEP 1 is to NG LAVAGE! This is done to evaluate for an upper GI bleed. A brisk upper GI bleed can result in what looks like lower GI bleed because the blood acts like a laxative.

LOWER GI BLEEDING (LGIB)

* **DIFFERENTIAL FOR LOWER GI BLEEDING (LGIB) IN THE NEONATAL PERIOD:**

- Maternal Blood: Perform an Apt test on the blood, which will differentiate fetal hemoglobin from adult (maternal) hemoglobin.
- Malrotation with Volvulus
- Necrotizing Enterocolitis (NEC): Particularly in premature neonates

* **DIFFERENTIAL FOR LOWER GI BLEEDING (LGIB) AT 1–2 YEARS OF AGE**

- Anal Fissure: Usually secondary to constipation; commonly located anteriorly.
- Intussusception
- **MNEMONIC**: intus**SIX**eption) = ages 3 months – **6 years**
- Juvenile Polyp: Painless bleeding. There may be a history of an intermittently seen mass protruding from the rectum.
- **PEARL**: There is no increase in the risk of cancer with juvenile polyps.

* **DIFFERENTIAL FOR LOWER GI BLEEDING (LGIB) AT 2–5 YEARS OF AGE**

- Meckel's Diverticulum: Painless; CAN be melenic; do Technetium-99m study
- Juvenile Polyp: Painless. Not associated with increased cancer risk.

* **DIFFERENTIAL FOR LOWER GI BLEEDING (LGIB) IN SCHOOL-AGED KIDS**

- Meckel's Diverticulum: Painless; CAN be melenic. do Technetium-99m study
- Juvenile Polyp: Painless. Not associated with increased cancer risk.
- Familial Adenomatous Polyposis: 100% chance of future malignancy.
- Ulcerative Colitis
- Crohn's Disease

PAINLESS RECTAL BLEEDING

Painless rectal bleeding can be due to anal fissures, polyps, hemorrhoids, and Meckel's diverticulum.

PEARLS: Polyps, fissures, and hemorrhoids have small-volume bleeding. Hemorrhoids usually do not result in streaks of blood on the stool. Meckel's Diverticulum usually results in large-volume bleeding.

MECKEL'S DIVERTICULUM (AKA MECKELS)

Meckel's diverticulum is a true diverticulum of the small intestine containing all three layers of bowel wall. It is present at birth and can produce LARGE volumes of PAINLESS rectal bleeding. Diagnose with a Meckel's scan.

PEARLS: Bleeding is usually red, but CAN be melenic. Painless rectal bleeding due to Meckel's diverticulum is MUCH more common than bleeding due to polyps.

MNEMONIC: The "Rule of 2s" refers to the fact that 2% of the population have a Meckel's Diverticulum, most of them are located 2 feet from the ileocecal valve, and most are 2 inches in length.

FYI: The Meckel's scan is a technetium-99 scan that looks for ectopic gastric or pancreatic cells in the small bowel. These ectopic cells are present in 50% of Meckel's diverticuli. Technetium scans can also be used to help diagnose a small bowel obstruction and intussusception.

FAMILIAL ADENOMATOUS POLYPOSIS (FAP)

Adenomatous (adenomas) polyps are extremely rare in children less than 10 years of age unless you have familial adenomatous polyposis (FAP), an autosomal dominant disorder. Adenomas are not cancerous, but can be precursor lesions to colon cancer. If a child younger than 10 is noted to have a polyp (possibly after presenting for painless rectal bleeding), resect and send for pathology. If the polyp is adenomatous, obtain GENETIC TESTING for the APC mutation (Adenomatous Polyposis Coli mutation). Children with FAP have a 100% chance of ending up with cancer. For APC+ patients, screen for colonic adenomas every year starting at 10 years of age. In order to prevent cancer, the colon is resected once large adenomas (> 1 cm) are noted, once adenomas are noted to have high-grade dysplasia (or villous histology), or once the patient turns 25.

MISCELLANEOUS GI CONDITIONS & TERMINOLOGY

OMPHALOCELE

An omphalocele refers to a herniation of bowel ± organs through the umbilicus. The herniated material is SEALED in by overlying membranes. Associated Beckwith-Wiedemann Syndrome and various chromosomal defects.

GASTROSCHISIS

Gastroschisis occurs when there is herniation of UNCOVERED bowel NEAR (not through) the umbilicus. The organs remain in the abdomen. Treat by placing a nasogastric tube for decompression and keeping the bowel moist until surgical repair.

NASOGASTRIC TUBE FEEDINGS (NG TUBE FEEDINGS)

Mild diarrhea is common with nasogastric tube feedings (NG tube feedings). Bolus feeds are recommended for children struggling with oral motor coordination in order to "help them learn." For all other patients, give CONTINUOUS NG tube feeds.

ESOPHAGEAL PERFORATION

There is a strong association between esophageal perforation and Marfan syndrome, Ehlers-Danlos, and Epidermolysis bullosa. Ingestion of bases (or strong acids) can also result in esophageal perforation. Weaker acids can cause esophageal strictures.

IMPERFORATE ANUS (AKA ANAL ATRESIA)

Imperforate anus (AKA anal atresia) presents with abdominal distension within the first 48 hours of life and the failure to pass meconium. The anus might be more anteriorly located. Some patients may have a fistula leading to the vaginal or urinary tract.

IMAGE: http://pbrlinks.com/ANALATRESIA1

* **(DOUBLE TAKE) VACTER-L (AKA VACTERL or VATER) SYNDROME**: VACTER-L (AKA VACTERL or VATER) syndrome is an acronym. VACTER**L** is now used instead of VATER because it stands for **V**ertebral anomalies, **A**nal atresia/imperforate anus, **C**ardiac defects (especially VSD), **T**racheoesophageal fistula, **R**adial hypoplasia and **R**enal anomalies, and **L**imb abnormalities. These children have a normal IQ. When associated with hydrocephalus, this can be an X-linked disorder.

PEARL: The patient may present with a single umbilical artery.
MNEMONIC: Imagine Darth VACTER cutting off his own son's ARM (radial hypoplasia and limb abnormalities) and then using the ARM as a light saber to create ANAL ATRESIA and a TE Fistula."

IMAGE: (go to 5:30 in the video) http://pbrlinks.com/VACTERL1
IMAGE: http://pbrlinks.com/VACTERL2

PERSISTENT CLOACA

Persistent cloaca refers to the presence of a single channel consisting of the rectum, vagina, and urinary tract (which did not separate in utero). It requires immediate surgery to prevent severe urinary tract complications. This should be suspected clinically in any female child with an imperforate anus.

RECTAL PROLAPSE

Rectal prolapses are usually due to **CONSTIPATION** or diarrhea (or a polyp), but **screen for cystic fibrosis**. Also caused by "TRICK YOur A$$ **out"** or "WHIP your A$$ **out**!" = Trichuris = Whipworm. Also by Shigella!

TYPHLITIS

If you are presented with a clinical description in an older child with leukopenia or neutropenia that sounds like NEC, choose typhlitis as the answer.

* **(DOUBLE TAKE) NECROTIZING ENTEROCOLITIS**: Necrotizing enterocolitis is often located at the ileocecal (AKA ileocolic) junction. Look for thrombocytopenia, bloody stool, distended, tender abdomen, erythematous abdominal wall, poor feeding, PNEUMATOSIS INTESTINALIS ± air in biliary tree. This is associated with infection and hypoxic injury (apnea, asphyxia, RDS, etc.). Do not feed these kids for **3 weeks**.

- **MNEMONIC**: NECKrotizing enterocolitis usually occurs at the NECK of the colon (ileocecal junction).
- **PEARL**: If you are presented with a similar clinical description in an older child with leukopenia or neutropenia, choose typhlitis as the answer instead.

I really hope that you've enjoyed this free chapter. The links are active to show you how valuable an online learning experience can be. My sincere recommendation is that you purchase a PBR **bundled** product that includes **both** the online AND the hardcopy versions of the PBR materials so that you can mark things up, make notes, but also be EFFICIENT!

Now… how about a handful of free questions?

Scroll to the next page to get a sample of the PBR Questions & Answers.

QUESTIONS

1. A premature baby needs:
    a. More sodium than a full term neonate. Sodium supplementation should be started immediately.
    b. More sodium than a full term neonate. Sodium supplementation can be started after 24 hours.
    c. Less sodium than a full term baby.
    d. The same amount of sodium as a full-term baby.
2. A premie is born at 33 weeks in a taxi. In the ER, the baby is noted to have a temperature of 35 degrees Celsius. The child should be placed:
    a. In a bassinette.
    b. In an incubator at 40 degrees Celsius.
    c. Under a radiant warmer at max temperature.
    d. Under a radiant warmer at preferred skin temperature.
3. An LGA baby is noted to have a firm, freely mobile, erythematous and nodular mass with distinct borders at the upper cheek on DOL 13. This is likely:
    a. Fat necrosis of the newborn.
    b. A lipoma
    c. A sarcoma
    d. Related to child abuse.
4. Which abnormality is common in the recipient of a PRBC transfusion and also in the recipient twin of a twin-to-twin transfusion?
    a. Hyponatremia
    b. Hypokalemia
    c. Hypocalcemia
    d. Hypophosphatemia
5. A child is born by a normal vaginal delivery. About an hour later he is noted to be tachypneic and pale. Labs show that he is anemic. Reticulocyte count is 15%. The RBCs are noted to be normal under microscopy. What is the likely etiology of these finding?
    a. Chronic intrauterine blood loss.
    b. Acute blood loss at birth.
    c. Congenital heart disease.
    d. Congenital syphilis

ANSWERS?

WHERE ARE THE ANSWERS?
Sorry, these are real questions from the PBR Q&A Book. You'll need to get the PBR Ultimate Bundle Pack or the PBR 12-Month ALL ACCESS PASS package by visiting:
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Best,
- Ashish

Hope You've Enjoyed It!

A Few [CRITICAL] Reminders

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This exam is MUCH HARDER than the USMLE exam. Go through the PBR Core Study Guide and the Q&A Book 3–5 times. Go through the material AT LEAST three times if you're recently out of residency, studying for the initial certification boards, and typically score above the national average on board exams and in-training exams. Go through the material AT LEAST FIVE times if you typically score below the national average on medical board exams, if you have ***ever*** *failed a medical board exam or if you come from an "at risk" residency program with less than a 90% first-time pass rate.*

TAKING THE MOC? READ MORE!

Going through the PBR Core Study Guide and Q&A Book 2–3 times should be enough. The pass rate for the PBR has been100% (2011 – 2014, 2016) for practicing general pediatricians, and in the "super high" 90s for subspecialists.

"LOW-ISH" USMLE SCORES? FAILED A BOARD EXAM? WORK ON TECHNIQUE!

Seriously, seriously, SERIOUSLY! This exam can wreek havoc and chaos in you life. The techniques that are taught in PBR'S Test-Taking Strategies & Coaching courses have helped pediatricians finally pass the boards after they had MULTIPLE times. Learn the "board game" by understanding question-answering STRATEGIES. You can see an increase in your practice exam scores IMMEDIATELY. ***PBR has helped pediatricians pass after they had up to SIX failed attempts using other board review resources!*** *So helping YOU should be easy. Don't have regrets. Visit the following link and learn more about our online and in-person Test-Taking Strategies Courses:*

*http://www.pediatricsboardreview.com/**technique***

DON'T FORGET TO DO TONS OF BOARD REVIEW QUESTIONS… FOR PRACTICE!

*Do at least 700 – 1000 practice questions if you're studying for the ABP initial certification boards, and at least 300 if you're taking the ABP MOC recertification exam. The first choice is the AAP PREP ® series of questions, but PBR also has trusted affiliate relationships with other great companies that give you MASSIVE discounts on questions to use for PRACTICE! Just visit http://www.pediatricsboardreview.com/**tools***

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Can't Decide What To Use Next? You Don't Have To!

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- Ashish & Team PBR

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