FIRST QUARTER, 2024

Volume 8, Issue 1

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A Message from CUCM President Chris Chao, MD

Dear CUCM members,

Are you ready to Recharge in Las Vegas? I am. I hope you are finalizing your plans to join us at the Urgent Care Convention because our members' passion and commitment to Urgent Care makes it special. It is something I look forward to every year. I'm also looking forward to seeing you all at the annual CUCM member's meeting on Monday, April 15, at 7:30 a.m. Breakfast will be provided.

If you have not done so, please vote in the CUCM Board of Directors election. We have some outstanding candidates running for CUCM Board of Directors positions for the 2024-2027 cycle, so make your vote count!

Please join me in congratulating **Jasmeet Bhogal, MD, MBA, FCUCM**, the recipient of the 2024 Sean McNeely Advancing the Specialty Award and **JD Zipkin, MD,** the recipient of the 2024 Joe Toscano Inspiring Excellence Award. Dr. Bhogal and Dr. Zipkin will receive their awards at the Urgent Care Foundation Celebration on Monday, April 15, during the Convention.

Urgent Care: What is the future?

The COVID-19 pandemic has permanently altered the landscape of healthcare in the U.S. During the pandemic, many clinics reduced services except for Urgent Care clinics. Urgent Care rose to the challenge and treated patients who otherwise could not easily access care. Now that the pandemic is over, patients continue to utilize Urgent Care for their healthcare needs.

Due to gaps in primary care and the high costs of emergency medicine, the demand for Urgent Care will continue to grow. The challenges we face include managing patient expectations and redefining our scope of care but also ensuring there isn't scope degradation and supporting individual clinicians to prevent burnout.

As of 2024, we have 27,000 Urgent Care clinicians staffing 14,500 Urgent Care clinics in the U.S. Why are we not recognized as a specialty by our peers? After all, Urgent Care isn't primary care. Urgent Care isn't emergency medicine. Urgent Care is Urgent Care. This is why "Advancing the Specialty" is an important initiative. Part of our completed work included developing a scope and competencies list for Urgent Care clinics and partnering with others to create a skills assessment test. But a lot of work is still needed.

It is my hope that in the future, we will be recognized as a specialty. We are working hard to develop relationships with the AMA, AAPA and AANP to develop tools that clinicians need to succeed in this profession. Ultimately, this can only improve public perception and patient outcomes, which hopefully translates to better reimbursement by payers and higher salaries for clinicians. This is a huge task that will require the coordinated effort of our members, and we need your help. Our collective voice can make this happen.

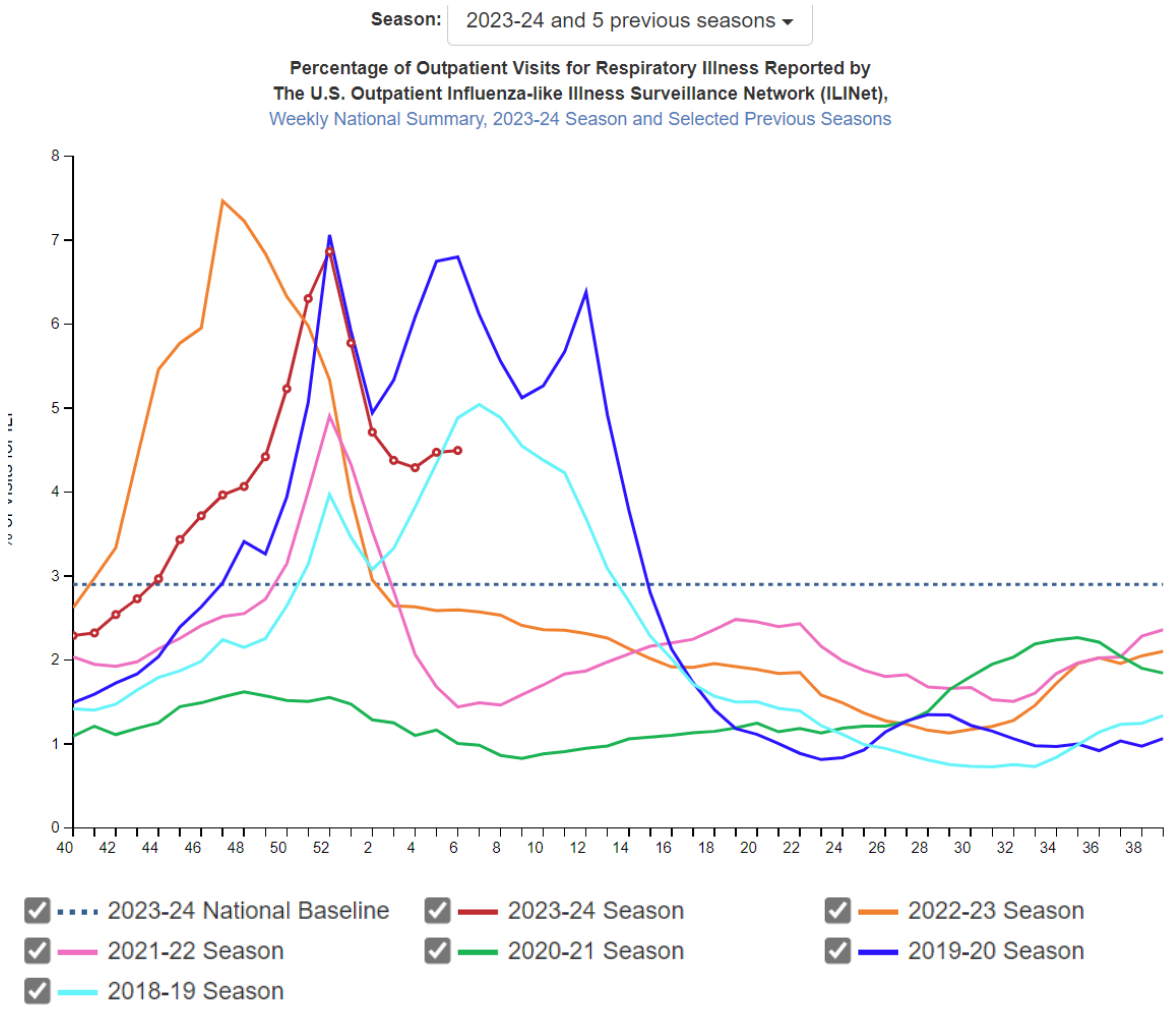
Finally, this will be my last President's letter as my term ends this year. It has been an honor and privilege to serve as President of the College. I look forward to introducing the next President at the member's meeting in Las Vegas.

Sincerely,

Christopher Chao, MD
President College of Urgent Care Medicine

From the Editors-in-Chief

This winter season has been the longest peak season for respiratory infections since the pandemic. We have seen COVID-19, influenza, RSV, and other viruses impacting our communities for many weeks. Does it feel like this respiratory season is never-ending?



Source: CDC

We have been through over 13 weeks of influenza-like illness. Last year, it lasted 11 weeks, and the year before, it was seven weeks. These numbers show a more stable season that matches pre-pandemic years - when the seasons will last for 15-18 weeks.

Nationally, COVID-19 infections are the leading cause of respiratory deaths, but we will have to wait until the CDC reports influenza-related death data, which happens post hoc, to account for underreporting. Hang in there. The season is almost over!

On another topic, we can't wait to see you at the Urgent Care Convention April 13-17 at Caesars Forum in Las Vegas. Last year, the convention was highly successful, and we have raised the bar this year! Come learn, network, and share experiences with your peers. Register here.

The Experience - What's New?

- Specialized journeys for the different roles in Urgent Care
- More unscheduled time to network
- More panel and facilitated discussions
- Wellness sessions
- An immersive garden party - Foundation Celebration

The College of Urgent Care Medicine will have four board positions open, and there are seven excellent candidates: Lindsey Fish MD; Kyla Howrish NP, FCUCM; Joe Toscano MD, FCUCM; Roger Hicks MD, FCUCM; Josh Russell MD, FCUCM; Brad Laymon PA-C, CPC, CEMC, and Patrick Dolan MD, FCUCM. You should have received an email to vote and a link to learn more about these outstanding candidates. The election will close on April 16 during the Urgent Care Convention.
See you in Las Vegas!

Tracey Q. Davidoff, MD, FCUCM

Cesar Mora Jaramillo, MD, FAAFP, FCUCM

Editorials and Opinions: Urgent Care's Future with POCUS — Better Care, Better Business

By Tatiana Havryliuk, MD

At 6 p.m. on a busy Monday, 47-year-old Mr. Jones, whom you saw just three days prior, returns with persistent pain, redness, and swelling in his left axilla. Despite taking cephalexin, his condition has not improved. There is a 4 cm area of erythema, induration, and tenderness, but no fluctuance is evident. You contemplate whether incision and drainage for a suspected abscess is warranted. With some uncertainty, you proceed, but no pus is found. This scenario is not unique. In cases of soft tissue infections without a clear abscess, emergency physicians incorrectly predict the presence of a fluid collection 56% of the time [1]. Essentially, our physical examination is akin to flipping a coin.

Now, consider the possibility of placing an ultrasound transducer on the affected area to instantly determine if a fluid collection is present, as well as its depth and its size. For Mr. Jones, this tool would have confirmed the absence of an abscess, thereby averting a needless and painful procedure and conserving valuable time.

Point-of-care ultrasound (POCUS) has been integral in enhancing diagnostic accuracy, increasing procedural safety, and expediting care in emergency departments for more than two decades. It is increasingly becoming a standard in primary care, and the Urgent Care community should welcome this technology.

Why is now the time to adopt POCUS?

- **Affordability**: POCUS devices are now more budget friendly. Handheld units range from $2,000 to $10,000, a significant reduction from the traditional cart-based systems priced at $20,000 and above.
- **Supported by Evidence**: There is abundant evidence showing how POCUS improves and expedites care. It's backed by multiple medical organizations and is within the scope of practice of Urgent Care providers.
- **Value-Based Care**: POCUS aligns with value-based care models, emphasizing overall cost savings. It aids in reducing unnecessary emergency department visits, more costly imaging, and repeat healthcare facility visits due to initial misdiagnosis or improper treatment.
- **Technologist Shortage Mitigation**: With a shortage of ultrasound and X-ray technologists, POCUS provides a cost-effective alternative.
- **Revenue and Patient Volume**: In a post-pandemic era, when patient volumes and revenue have decreased, introducing a new service like POCUS could attract new patients, broaden care for those with complex conditions, and generate additional revenue.

Which Common Urgent Care Presentations Could Significantly Benefit from POCUS?

- **Soft Tissue Infections**: POCUS excels in the evaluation of soft tissue infections, including detecting peritonsillar abscesses through intraoral and transcutaneous methods. [1-3]
- **Respiratory Complaints**: Lung POCUS can assess for pneumonia, pneumothorax, pulmonary edema, and pleural effusions with more sensitivity than chest X-ray. Plus, it avoids radiation exposure, which is vital for children and young adults. [4-6]
- **Flank Pain, Urinary Retention, or Hematuria**: Renal and bladder POCUS permits the evaluation of bladder volume and presence of hydronephrosis. A POCUS-first approach has been shown to reduce the need for CT scans in 60% of emergency department cases presenting with potential renal colic. [7]
- **Upper Abdominal Pain**: Biliary POCUS is effective for ruling out conditions such as cholecystitis or biliary colic.

- **Lower Extremity Pain or Swelling**: POCUS can be employed to exclude proximal leg deep venous thrombosis (DVT) using a compression technique, preventing unnecessary emergency department referrals. [8,9]
- **Musculoskeletal Pain or Injuries**: Evaluation of tendonitis, tendon ruptures, joint effusions, hematomas, and even fractures and dislocations can be accomplished with POCUS.
- **Ocular Complaints**: POCUS is highly accurate in identifying vitreous hemorrhage, retinal detachment, foreign bodies, and lens dislocation.
- **Procedural Guidance**: POCUS enhances the safety and efficiency of procedures such as difficult IV access, arthrocentesis, and shoulder dislocation reduction.

Additional Advantages of POCUS for Urgent Care Clinicians

Beyond enhancing patient care, creating extra income, and attracting more clients, POCUS offers numerous benefits including:

- Facilitating patient education about their health conditions.
- Ensuring patient adherence to imaging.
- Elevating the overall patient experience.
- Enabling clinicians to make more educated decisions.
- Equipping clinicians with a valuable and marketable skill.

What Is the Return on Investment?

Adopting a POCUS program necessitates an initial investment in ultrasound equipment, an image storage solution, and a training program for medical staff. After achieving proficiency (within 6 months to a year), POCUS procedures can be billed similarly to other medical services, utilizing specific CPT codes for "limited ultrasound." [10] Ensure that your insurance contracts cover POCUS. Medicare reimbursement rates for POCUS range from $56 for lung ultrasounds to $117 for DVT ultrasounds. The potential for significant additional income exists depending on patient volume, types of cases, imaging capabilities, and payor mix.

For instance, based on 2024 nationwide Medicare rates, performing approximately 45 POCUS exams monthly (1.5/day) could yield an additional billable amount of $37,000 annually. Consider using a POCUS ROI Calculator to get a more precise approximation of POCUS revenue for your practice. [11]

For Urgent Cares with Flat-Rate Contracts

In situations with flat-rate contracts, POCUS services are integrated into the patient visit charge. Nonetheless, POCUS can still boost revenue by building patient loyalty and attracting new patients. [12]

So, What's the Bottom Line?

POCUS has proven to enhance and expedite the delivery of care in emergency departments and primary care settings. The devices are now affordable, and training is more accessible. It is well within the scope of practice of Urgent Care providers. It can help manage common Urgent Care presentations, such as respiratory and soft tissue infections, with more precision and better patient outcomes. With appropriate training and workflow, it's time for POCUS to be integrated into Urgent Care.

About the author:

Dr. Havryliuk is an emergency physician with over 15 years of clinical POCUS experience, past Emergency Ultrasound Director at Brooklyn Hospital in NY. She is on the mission to empower clinicians with POCUS to take better and more efficient care of their patients.

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References:

1. Tayal VS, Hasan N, Norton HJ, Tomaszewski CA. The effect of soft-tissue ultrasound on the management of cellulitis in the emergency department. Acad Emerg Med. 2006;13(4):384-388. doi:10.1197/j.aem.2005.11.074
2. Costantino TG, Satz WA, Dehnkamp W, Goett H. Randomized trial comparing intraoral ultrasound to landmark-based needle aspiration in patients with suspected peritonsillar abscess. Acad Emerg Med. 2012;19(6):626-631. doi:10.1111/j.1553-2712.2012.01380
3. Rehrer M, Mantuani D, Nagdev A. Identification of peritonsillar abscess by transcutaneous cervical ultrasound. *Am J Emerg Med*. 2013;31(1):267.e1-267.e2673. doi:10.1016/j.ajem.2012.04.021
4. Ticinesi A, Lauretani F, Nouvenne A, Mori G, Chiussi G, Maggio M, Meschi T. Lung ultrasound and chest x-ray for detecting pneumonia in an acute geriatric ward. Medicine (Baltimore). 2016 Jul;95(27):e4153. doi: 10.1097/MD.0000000000004153. PMID: 27399134; PMCID: PMC5058863.
5. Pirozzi C, Numis FG, Pagano A, Melillo P, Copetti R, Schiraldi F. Immediate versus delayed integrated point-of-care-ultrasonography to manage acute dyspnea in the emergency department. *Crit Ultrasound J*. 2014;6(1):5. Published 2014 Apr 27. doi:10.1186/2036-7902-6-5
6. Baid H, Vempalli N, Kumar S, et al. Point of care ultrasound as initial diagnostic tool in acute dyspnea patients in the emergency department of a tertiary care center: diagnostic accuracy study. *Int J Emerg Med*. 2022;15(1):27. Published 2022 Jun 13. doi:10.1186/s12245-022-00430-8
7. Adhikari S, Amini R, Stolz L, et al. Implementation of a novel point-of-care ultrasound billing and reimbursement program: fiscal impact. Am J Emerg Med. 2014;32(6):592-595. doi:10.1016/j.ajem.2014.02.051
8. Mumoli N, Vitale J, Giorgi-Pierfranceschi M, et al. General Practitioner-Performed Compression Ultrasonography for Diagnosis of Deep Vein Thrombosis of the Leg: A Multicenter, Prospective Cohort Study. *Ann Fam Med*. 2017;15(6):535-539. doi:10.1370/afm.2109
9. Hannula O, Vanninen R, Rautiainen S, Mattila K, Hyppölä H. Teaching limited compression ultrasound to general practitioners reduces referrals of suspected DVT to a hospital: a retrospective cross-sectional study. *Ultrasound J*. 2021;13(1):1. Published 2021 Feb 2. doi:10.1186/s13089-021-00204-y
10. "2024 Medicare Fees for Diagnostic and Procedural POCUS for Urgent Care Centers." Accessed: Feb. 1, 2024. [Online] Available: https://www.hellosono.com/downloadables-1/urgent-care-2024-fees-pocus-exams
11. "Are you leaving revenue on the table?" POCUS ROI Calculator. Accessed: Feb. 1, 2024. [Online] Available: https://www.hellosono.com/pocus-roi-calculator
12. Andersen CA, Brodersen J, Rudbæk TR, Jensen MB. Patients' experiences of the use of point-of-care ultrasound in general practice - a cross-sectional study. BMC Fam Pract. 2021;22(1):116. Published 2021 Jun 18. doi:10.1186/s12875-021-01459

Case Study: Unexplained Bruising

John George, PA-C

Key Words: bruising, thrombocytopenia, ITP, petechiae, purpura, spontaneous bleeding

Introduction

Patients commonly present to Urgent Care with concerns of unexpected bleeding, bruising, and rashes that may be petechiae or purpura. Laboratory testing should be performed if suspicion is high for thrombocytopenia or other bleeding disorders, and if abnormal or if bleeding is severe, patients should be referred to the emergency department.

History

A 36-year-old female presents to Urgent Care with a complaint of bruising on her body for the past 3 days. She also complained of fatigue and myalgia. She stated she had some nasal congestion a week ago that had since resolved. She states she scratched herself in the chest area 3 days ago and subsequently noticed a big bruise in the area. This resolved, but now she has scattered bruising to her arms and legs. The patient notes a past medical history of anxiety and depression, an episode of optic neuritis of unknown etiology about 10 years ago. She also notes she is 6 months postpartum and presently breastfeeding. According to the patient, there were no complications in the last pregnancy. She has had three prior miscarriages prior to 12 weeks gestation. She denies OCP use, any new medications, current pregnancy, personal or family history of thrombocytopenia or autoimmune disease, mucosal, vaginal, rectal, or urinary bleeding, recent trauma, chest pain, shortness of breath, abdominal pain, UTI symptoms, fever, chills, cough, sore throat, any new exposures, insect bites, recent travel, hiking or camping, blurry vision, headaches, dizziness, nausea, vomiting, numbness, or tingling. She voiced no other complaints. She has never had COVID-19 as far as she is aware; her last COVID-19 vaccine was >1 year prior. She had an influenza vaccine approximately 3 months ago. She has been taking sertraline for the last 6 months. She is allergic to sulfa and moxifloxacin.

Clinical Findings

T: 98.7 *F HR: 84 BP:124/78 RR:16 Spo2: 98%
Physical exam: A basic exam was grossly normal. Examination of the skin revealed a few scattered ecchymoses and petechial rashes on the bilateral arms and legs. The face and trunk were spared.

Assessment

The initial impression was that of a previously healthy postpartum female with possible inappropriate bruising, rash, and fatigue after a suspected viral upper respiratory infection. Although this could be nothing more than normal trauma, there was concern for more serious illness such as an acquired bleeding disorder.

The in-house pregnancy test was negative, and a urinalysis was unremarkable.

A STAT complete blood count (CBC) and comprehensive metabolic profile (CMP) were sent to the laboratory. The platelet count was 5,000/microL [Normal: 150,000-450,000/microliter]. The remainder of the CBC and CMP were unremarkable.

Therapeutic intervention

The patient was called about the abnormal platelet count and was advised to proceed immediately to the emergency department for further evaluation and treatment.

Follow-up

At the hospital, a respiratory virus panel was positive for entero/rhinovirus, and negative for COVID-19. HIV, RPR, Hepatitis panel, ANA, and Parvovirus Antibody were all negative.

Hematology was consulted, and the patient was started on 40 mg IV dexamethasone daily for 3 days and pantoprazole 40 mg. She was admitted to the hospital. Breastfeeding was discontinued. The patient was hospitalized for three days and discharged on oral prednisone and was advised outpatient follow-up with hematology. No platelet transfusion was required. Upon discharge, her platelet count had increased to 19,000/microL.

Discussion

Idiopathic thrombocytopenia purpura (ITP) is an acquired thrombocytopenia, which is caused by the destruction of platelets in the reticuloendothelial system due to platelet autoantibodies. This results in destruction as well as underproduction of platelets. (Song) It is one of the more common causes of thrombocytopenia in otherwise asymptomatic adults. The lack of a sensitive or specific diagnostic test for ITP and the large number of other potential causes of thrombocytopenia (e.g. drug-induced thrombocytopenia, hereditary thrombocytopenia) also contribute to the challenges in diagnosing ITP.

Primary ITP is an acquired immune thrombocytopenia due to autoimmune mechanisms, which leads to platelet destruction and platelet underproduction. Primary ITP is not triggered by an associated condition.

Secondary ITP however is precipitated by another condition. The list is long and comprises immunologic, hematologic, and infectious conditions. (See Figure 1) Drug-induced immune thrombocytopenia (DITP) is thrombocytopenia due to drug-dependent platelet antibodies that cause platelet destruction. This syndrome should be distinguished from drug-induced bone marrow suppression, a non- immune phenomenon. See Figure 2 for a list of common offending drugs.

The interval between initiation of a new drug taken daily and drug induced ITP (DITP) is usually less than two weeks. A drug taken daily for several months or longer is rarely associated with DITP. Drugs taken only intermittently may cause DITP even if it has been many years since the initial exposure.

COVID-19 vaccines were initially suspected of causing ITP and exacerbating existing ITP, however, there is no current evidence to support this. (Pishko) ITP rates after a COVID-19 vaccination are no higher than in the general population. Thrombocytopenia may be mistaken for ITP when due to chronic liver disease, hypersplenism, bone marrow suppression, transient drops in platelet counts from infection, or hereditary thrombocytopenia.

ITP may be seen in otherwise healthy pregnant women on their initial pregnancy lab testing. However, pregnancy is also associated with other causes of thrombocytopenia including gestational thrombocytopenia (a physiologic condition) and pregnancy-associated microangiopathic syndromes.

ITP in children, especially in children younger than age 10, is a clinically distinct condition compared to that in adults, with a higher likelihood of spontaneous remission, a lower incidence of underlying diseases and comorbidities, and often a lower risk of bleeding. During adolescence, ITP may be similar to typical childhood ITP or to ITP in adults.

ITP is further categorized into the time elapsed since diagnosis. ITP is newly diagnosed as ITP in the first 3

months and persistent if lasting three months to 12 months and chronic if more than 12 months.

The pathogenesis of ITP is likely multifactorial. Reduced platelet lifespan due to clearance is the predominant cause of thrombocytopenia. The principal mechanism is thought to involve specific autoantibodies (typically, IgG) most often directed against platelet membrane glycoproteins such as GPIIb/IIIa. (Cines) The primary site of platelet clearance for most patients is the spleen, which removes opsonized (antibody coated) cells including platelets. Other mechanisms that may play a role include autoreactive cytotoxic T cells and humoral and cellular autoimmunity directed at megakaryocytes, which causes impaired platelet production.

In the US, the prevalence of ITP is approximately 8 per 100,000 in children and 12 per 100,000 in adults. (Terrell) In younger patients, it is felt that ITP is more common in females, however, there is some debate about this. There is a similar incidence in males and females over age 60.

Clinical manifestations

- Bleeding: Bleeding due to thrombocytopenia may ultimately occur in up to two-thirds of patients. When present, bleeding typically occurs in the skin or mucous membranes. Although the onset of symptoms may be abrupt, it is more often insidious.
- Petechiae: Petechiae are flat, red, discrete lesions that do not blanch under pressure; these often occur in dependent areas of the body such as the legs or sacral areas.
- Purpura: Purpura are lesions caused by coalescence of petechiae. They are nonpalpable. When occurring in the mouth, they may form hemorrhagic blisters called wet purpura, which may be a predictor of more severe bleeding.
- Epistaxis: Mild bleeding with nose blowing is common and may not be clinically important. Continuous epistaxis that requires intervention with nasal packing or cauterization may predict a greater risk of serious bleeding.
- Severe or Critical Hemorrhage: Life-threatening bleeding episodes are not common in ITP.
- Fatigue: Many ITP patients complain of fatigue, which often correlates with the degree of thrombocytopenia but can occur with any platelet count.
- Thrombosis — Thrombocytopenia in people with ITP is not necessarily protective against thrombosis. There may be a small increased risk of thrombosis with ITP. This is not well understood. Causes may include inflammation, antiphospholipid antibodies, or a side effect of treatment such as splenectomy, corticosteroids, or thrombopoietin receptor agonists (TPO-RAs). (Swan)

Patients with ITP can be expected to have normal WBC and RBC counts as there is no effect on these cell lines. Coagulation parameters are also typically normal. Abnormal results of WBC, RBC, or coagulation should prompt an urgent evaluation for conditions other than ITP such as leukemia, thrombotic thrombocytopenia purpura (TTP), disseminated intravascular coagulation (DIC), or aplastic anemia.

Diagnosis

ITP is a diagnosis of exclusion that is made in patients with isolated thrombocytopenia with other cell lines remaining normal. Other possible causes of thrombocytopenia should be ruled out, and conditions that may be responsible for secondary ITP should be considered. Many potential causes will be apparent from the history, physical examination, and review of the complete blood count (CBC). Keep in mind that thrombocytopenia caused by a medication or previous illness may require specific questioning or review of the medical record to identify the responsible agent. The history should be focused on recent infections, medications, underlying conditions such as rheumatologic disorders and liver disease, beverages such as tonic water, herbal remedies, and foods.

The physical examination should be focused on signs of bleeding, specifically on the skin and oral mucous membranes, which would suggest the need for more urgent evaluation and therapy; and the presence of lymphadenopathy or hepatosplenomegaly, which could suggest an underlying condition responsible for the thrombocytopenia.

All patients should have a peripheral smear as platelet clumping may give an erroneous low platelet count. HIV and HCV testing should be performed. Liver function testing should be performed. Coagulation studies such as PT and PTT should be done in any patient with clinically significant bleeding. Testing for *H. pylori* and thyroid testing may also be reasonable in evaluation for secondary causes of ITP. Bone marrow biopsies are no longer recommended. (Neunert) Immunologic studies, such as ANA, etc. are also not routinely recommended, except if there is a suspicion of an underlying immunologic disorder. Antiplatelet antibody testing has a low sensitivity and has no correlation with clinical outcomes and is not recommended.

Treatment

For patients with presumed or known ITP who have severe bleeding, urgent hospitalization with early involvement of a hematologist is necessary to confirm the diagnosis, exclude other potential causes of bleeding, and assist with appropriate therapies. Bleeding should be controlled in traditional ways with direct pressure, nasal packing, etc. Individuals with thrombocytopenia with platelet counts > 20,000/microL that are stable without bleeding may be managed as outpatients if there is close hematologic monitoring and a plan for urgent management of bleeding. Patient education and expedited follow up with a hematologist should be arranged. Patients with platelet counts < 20,000/microL, whether bleeding or not, should be evaluated in the hospital due to the increased risk of bleeding. (Neunert). Long term management of ITP is aimed at preventing significant bleeding, not necessarily to normalize the platelet count. Any secondary causes should be eliminated, if possible, such as drugs and infections. Corticosteroids are often used as initial treatment and in cases of minor bleeding, followed by intravenous immune globulin (IVIG). Anti-D immune globulin may be an alternative to IVIG. Platelet transfusions are reserved for severe or refractory bleeding. TPO-RA's and rituximab may also be considered. Definitive care may require splenectomy. Generally, patients who do not improve within 1 year are considered for splenectomy.

Informed Consent

Consent for publication of this case was not obtained as the patient was unable to be reached.

References

Song F, Al-Samkari H, Management of Adult Patients with Immune Thrombocytopenia (ITP): A Review on Current Guidance and Experience from Clinical Practice, Journal of Blood Medicine, 2021 12:, 653-664, DOI: 10.2147/JBM.S259101

Pishko AM, Bussel JB, Cines DB. COVID-19 vaccination and immune thrombocytopenia. Nat Med. 2021 Jul;27(7):1145-1146. doi: 10.1038/s41591-021-01419-1. PMID: 34108715.

Cines DB, Blanchette VS. Immune thrombocytopenic purpura. N Engl J Med. 2002 Mar 28;346(13):995-1008. doi: 10.1056/NEJMra010501. PMID: 11919310.

Terrell DR, Beebe LA, Neas BR, Vesely SK, Segal JB, George JN. Prevalence of primary immune thrombocytopenia in Oklahoma. Am J Hematol. 2012 Sep;87(9):848-52. doi: 10.1002/ajh.23262. Epub 2012 Jun 5. PMID: 22674643; PMCID: PMC3429719.

Neunert C, Terrell DR, Arnold DM, Buchanan G, Cines DB, Cooper N, Cuker A, Despotovic JM, George JN, Grace RF, Kühne T, Kuter DJ, Lim W, McCrae KR, Pruitt B, Shimanek H, Vesely SK. American Society of Hematology 2019 guidelines for immune thrombocytopenia. Blood Adv. 2019 Dec 10;3(23):3829-3866. doi: 10.1182/bloodadvances.2019000966. Erratum in: Blood Adv. 2020 Jan 28;4(2):252. PMID: 31794604; PMCID: PMC69632

Part 1 of a Series - Vital Signs Are Vital: *Even* Respiratory Rate

Joseph Toscano, MD, FCUCM

Though periodically there are calls to add new data points as "the next vital sign," we still need to pay attention to the traditional ones. Among these, respiratory rate (RR) has always seemed to be the least regarded. Other vital signs can be obtained with the push of a button, and we consider the values we see displayed to be highly accurate. Though wearable devices to measure RR are being used by some high-tech sports enthusiasts, in most medical practices, RR remains an old school assessment. We were classically taught to count respirations for 30 seconds and double the value. At least one study[1] found that even this can miss occasional crucial abnormalities. Counting for longer is always going to be more accurate. Of course, counting for 60 seconds will give you the exact number of breaths per minute, and counting for 120 seconds and dividing by 2 may be a better overall evaluation[1], but who has even 30 seconds these days? The other seemingly obvious issue with RR is that we feel like we should get a general sense of the normality or abnormality of it as part of our gestalt of the patient – if patients look to be in respiratory distress, their RRs will be high. If they appear to have agonal respirations, RR will be low. But another study[2] showed that even some subjective assessments may be flawed.

It is indeed the case that discrete RR numbers are essential parts of some asthma scores, the qSOFA criteria, CURB-65 scores and other clinically useful indicators. But how important are actual RR numbers in everyday Urgent Care practice, particularly when patients either look good overall or have no respiratory complaints?

Consider the following case report:

A 28-year-old male presents to Urgent Care with blurry vision for the past day or so. He denies any trauma to the eyes or chemical or other exposure. He reports no prior eye disease. He does not wear contact lenses or eyeglasses. He has no headache and has had no recent illness, though he does note a lot of stress at work. He has had no drainage from the eyes. On physical exam, he is afebrile with a temperature (T) of 98.2, pulse (P) is 68, blood pressure (BP) 118/68, and RR is documented as 20. Pulse oximetry is 100% on room air. Visual acuity is 20/20 bilaterally. Looking back in the record, his visual acuity was 20/15 when measured at a prior visit a few years ago. Eye exam shows PERRL, EOMI, clear conjunctivae and sclerae, and no abnormal fluorescein uptake. Slit-lamp exam shows normal cornea and anterior chamber and iris. Direct funduscopy does not appear abnormal. The optic discs are sharp, vessels appear normal, and there are no hemorrhages or other retinal abnormalities. The clinician recommends routine referral to an optometrist or ophthalmologist. The patient returns the next day with worsening vision but no other symptoms. At intake, vital signs include T 98.0, P 64, BP 116/72, and RR is recorded as 24. Pulse oximetry is 99% on room air. He is in no distress, and the exam is unchanged except that visual acuity is now measured at 20/30 bilaterally. The prior recommendation to see a specialist is encouraged. The following day, the patient presents with further worsening vision, now with headache and lightheadedness as well. He says that he has an eye appointment the following day but does not feel like he can make it that long. He denies, as he had before, other symptoms of fever, dyspnea, or chest pain. Vital signs include T 98.2, P 68, BP 122/78, and RR is recorded as 28 with room air pulse oximetry of 100%. External eye exam is normal but visual acuity is now 20/50 in both eyes. The clinician decides to recount the respirations and though the patient does not seem to be in any distress, the RR is indeed 28 per minute and lungs are clear to auscultation. The patient is referred to the emergency department for evaluation.

Apart from blurry vision, what about this patient's presentation was abnormal from the start? Why would a respiratory rate be elevated even when the patient has no shortness of breath or other respiratory complaint?

As mentioned, with respiratory complaints, our expectation is that RR matches the overall clinical scenario, whether in response to hypoxemia or hypercarbia, or to abnormal airway resistance, alveolar compliance, or respiratory muscular issues. When the patient looks to be breathing abnormally, they usually are. But when they seem to be in no distress, they may still be tachypneic when you count out their RR, and that may need to be addressed.

This patient in the scenario was referred and examined by an emergency physician and an ophthalmologist. He was found to have inflammatory changes of the optic nerves bilaterally, an anion-gap metabolic acidosis and an osmol gap that led to testing, which eventually confirmed methanol poisoning. He made a full recovery.

Take home points:

We should train staff and ourselves to count RR with proper technique. A longer count is always going to be a better assessment. Normal RRs comprise a range which varies by patient age, and these are hard to memorize. EMRs often flag abnormal values for age, but having a chart or other reference is also helpful to be able to tell how far out of range any patient's values are.

Error is inherent in all data collection, and patient condition can change over time but never ignore an abnormal RR recorded in the chart. If the value recorded by someone else does not make sense to you based on the patient's present appearance, recheck the RR yourself. Conversely, if a normal RR has been recorded previously in the visit, but you think that is incorrect, recount and use the most current value to make decisions. Try to clinically reconcile inconsistencies over time. Did the patient's status change?

Someone with a low O2 saturation will usually have an abnormal RR, but O2 sat can also be completely normal in someone with a truly abnormal RR. Pulse ox saturations indicate only part of a patient's respiratory status.

As we learned from our patient here, an elevated RR with clear lungs and no respiratory complaint, sometimes referred to as "quiet tachypnea", can indicate metabolic acidosis. Think about DKA or other metabolic or toxic conditions as a cause. In infants, consider inborn errors of metabolism or congenital heart disease.

References

1. Drummond, GB et al. Current clinical methods of measurement of respiratory rate give imprecise values. *ERJ Open Research* 2020 6: 00023-2020; DOI: 10.1183/23120541.00023-2020 https://openres.ersjournals.com/content/6/3/00023-2020
2. Latten GHP et al. Accuracy and interobserver-agreement of respiratory rate measurements by healthcare professionals, and its effect on the outcomes of clinical prediction/diagnostic rules. *PLOS ONE* Oct 3 2019; https://doi.org/10.1371/journal.pone.0223155

The Silent Spread: Syphilis on the Rise

Cesar Mora Jaramillo, MD, FAAFP, FCUCM

Syphilis is one of the oldest known sexually transmitted infections. It was first recorded in the 1490s and known with different names, “the French disease", "the Neapolitan disease" and "the Polish disease".

Once thought to be under control, syphilis has reemerged as a formidable public health challenge. Recent data shows a notable upswing in cases including newborn infections. This so-called epidemic is occurring in the U.S., with sustained increases in primary and secondary syphilis. Unfortunately, the epidemic is driven by health disparities, particularly among certain populations - sexual and gender minorities and HIV and substance use disorders.

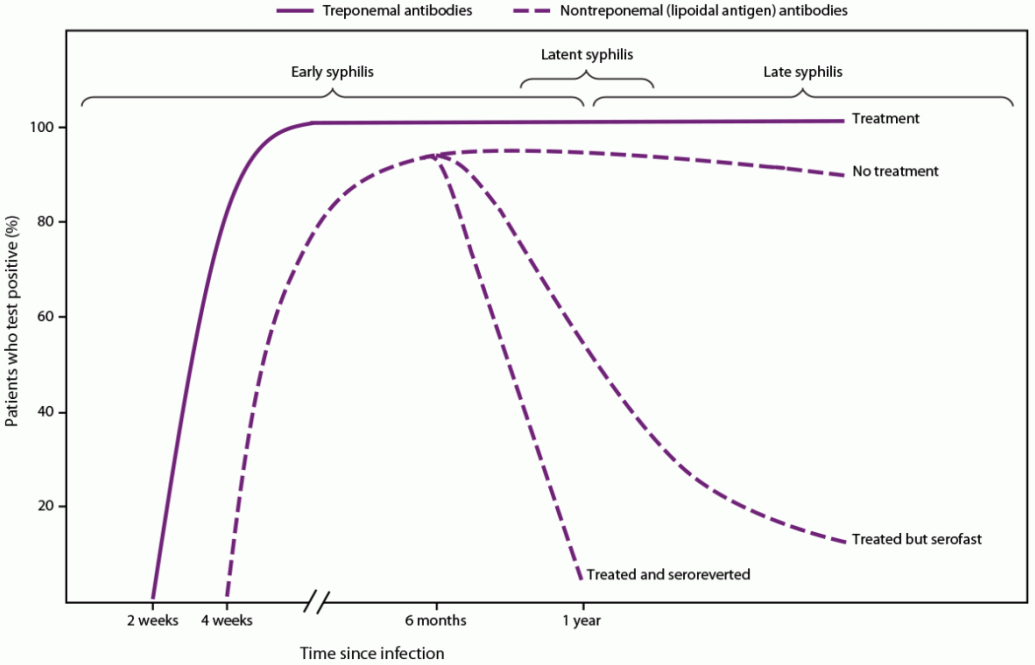
But how can Urgent Care centers (UCCs) assist with the fight against this epidemic? UCCs play a pivotal role in early detection and intervention. Hence, protocols for screening, testing, and treatment within Urgent Care settings are crucial, and centers should emphasize their role in not only managing individual cases but also in stemming the tide of syphilis within our communities. In the context of health disparities, well-established protocols act as a leveling mechanism, ensuring that all individuals, irrespective of their socio-economic background, receive equitable and timely healthcare services. These protocols contribute to consistency in diagnosis, treatment, and follow-up, reducing variations in care that may contribute to disparities. UCCs can implement strategies to decrease disparities in their communities. These strategies should include:

- **Community Outreach and Education** - Tailoring information to specific communities, allowing healthcare providers to address cultural and linguistic barriers that may contribute to disparities
- **Accessible Testing** - Flexible hours, walk-in services, and mobile services particularly in underserved areas
- **Culturally Competent Care** - Healthcare staff receive training to understand and respect diverse cultural backgrounds, thereby fostering trust and increasing patient engagement
- **Collaboration with Community Organizations and Local Health Departments** - Facilitate communication, implement preventative measures, enhance surveillance, outreach, and follow-up care for syphilis cases

On Tuesday, January 30, 2024, the CDC reported syphilis cases had increased 80% in the United States between 2018 and 2022 (from 115,000 to more than 207,000), compounding a decades-long upward trend. The cases increased by 17% between 2021 and 2022 and surged by 32% between 2020 and 2021 to reach the highest number of reported incidences in 70 years. This has led the CDC to warn healthcare entities that this epidemic is showing no signs of slowing, and it has pointed to some "alarming" new trends driving this sudden spike in the disease.

If untreated, syphilis can seriously damage the heart and brain and can cause blindness, deafness, and paralysis. When transmitted during pregnancy, it can cause miscarriage, lifelong medical issues, and infant death.

Serologic response to infection with Treponema pallidum, the causative agent of syphilis: source CDC

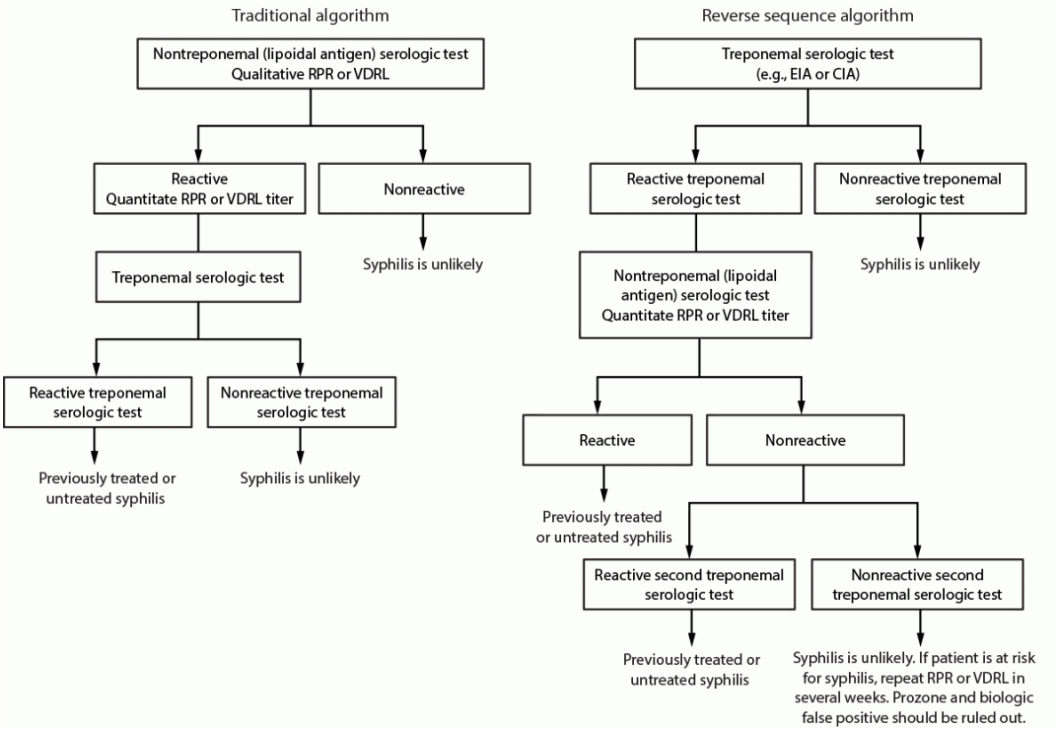


CDC is recommending action to stop the increase of newborn syphilis cases. New CDC data reveal that more than 3,700 babies were born with syphilis in 2022, which was more than 10 times the number in 2012. The increase in newborn syphilis follows rising syphilis cases among women of reproductive age combined with social and economic factors that create barriers to high-quality prenatal care and ongoing declines in the prevention resources. According to the CDC, almost 9 in 10 cases of newborn syphilis in 2022 might have been prevented with timely testing and treatment during pregnancy. They point out that more than half were among people who tested positive for syphilis during pregnancy but did not receive adequate or timely treatment. Nearly 40% were among mothers who were not in prenatal care.

On February 8^{t}, 2024, the CDC reported new recommendations for tests that support a diagnosis of syphilis including serologic testing and methods for the identification of the causative agent *Treponema pallidum*.

These tests can be divided into nontreponemal and treponemal tests (depending on whether they detect antibodies that react to lipoidal antigens shared by both host and *T. pallidum* or antibodies specific to *T. pallidum* respectively. Both types of tests must be used in conjunction to help distinguish between an untreated infection or a past infection that has been successfully treated.

Newer serologic tests allow for laboratory automation but must be used in an algorithm, which also can involve older manual serologic tests. Direct detection of *T. pallidum* continues to evolve from microscopic examination of material from lesions for visualization of the organism. Limited point-of-care tests for syphilis are available in the U.S.; increased availability of point-of-care tests that are sensitive and specific could facilitate expansion of screening programs and reduce the time from test result to treatment.
Here are some of the recommendations from CDC from February 8^{t}, 2024:



Algorithms that can be applied to screening for syphilis with serologic tests — CDC laboratory recommendations for syphilis testing in the United States, 2024.

Recommendation for syphilis serologic testing algorithm. Serologic tests that measure antibodies to nontreponemal (lipoidal) and treponemal antigens related to syphilitic infections should be used when the primary test is reactive to aid in syphilis diagnosis. Sole reliance on one reactive serologic test result can misclassify a patient's syphilis status. Both the traditional syphilis screening algorithm (initial screening with nontreponemal assays) and the reverse syphilis screening algorithm (initial screening with treponemal immunoassays) are acceptable. The preferred algorithm should be based on laboratory resources, including staff, space and costs, test volume, and patient populations served.

Comment and evidence summary. Antibodies detected by nontreponemal and treponemal antigen tests vary by the stage of syphilis, treatment status, and past infection that was treated. Results from both types of serologic tests are required to help diagnose the stage of syphilis. Both traditional and reverse syphilis testing algorithms are used in the U.S. and have about 99% concurrence between the two approaches. The cost-effectiveness of the two algorithms might vary by laboratory setting and need to be considered by individual laboratories.

Recommendation for the direct detection of *T. pallidum* by dark field microscopy. Dark Field microscopy should be maintained if already in use or established in STD clinics where a POC test for primary or secondary syphilis diagnosis would be beneficial for timely patient treatment.

Comment and evidence summary. The sensitivity of darkfield microscopy in detecting *T. pallidum* from primary lesions ranges from 94% to 100% and 81% to 100% from secondary lesions when compared with NAATs (*141*,*187*–*191*). Darkfield microscopy can be more sensitive than serologic tests at the primary stage and offers the advantage of timely detection and rapid treatment of primary syphilis (*186*). The procedure is classified as moderately complex by CLIA, and the settings implementing the darkfield microscopy will require CLIA certification for such a test.

Recommendation for direct detection of *T. pallidum* by immunohistochemistry and silver staining. IHC is preferred over silver staining for FFPE tissue sections regardless of anatomic site.

Comment and evidence summary. The sensitivity of IHC ranged from 64% to 94% whereas silver stain had a sensitivity of 0%–41%.

Recommendation for serologic syphilis testing. Nontreponemal tests (e.g., RPR or VDRL) are not interchangeable when used to determine antibody titers; testing on follow-up samples must be performed with the same type of test. The TPPA test is the preferred manual treponemal test.

Comment and evidence summary. Sensitivity and specificity estimates of RPR and VDRL were similar but not exact in head-to-head studies and studies that used similar reference standards. When assessing changes in antibody titers using nontreponemal tests, it is critical that the same test be used because titers are used by clinicians to classify the infection status of a patient and follow treatment response. A recent study with 959 patients estimated the sensitivity of FTA-ABS and TPPA to be 78.2% and 94.5%, respectively, when testing specimens from patients with primary syphilis (*115*). Two studies that tested specimens from patients with secondary syphilis reported a sensitivity of 92.8%–95.0% compared with 100% for TPPA. Many automated treponemal immunoassays are similar in sensitivity, and certain ones are slightly less specific when compared with the manual TPPA, except for the Trep-Sure test which has inferior specificity. Among the other immunoassays, data are insufficient to recommend one assay based on test performance.

Only the Syphilis Health Check (Diagnostics Direct) and Dual Path Platform (DPP) HIV-Syphilis Assay (Chembio Diagnostics) are FDA cleared and CLIA waived for the detection of *T. pallidum* antibodies. Physician office laboratories and public health field-based screening programs that offer CLIA-waived tests are required to have and maintain a CLIA certificate of waiver that requires these tests to be quality assured and operated by trained personnel according to manufacturer instructions.

POC Tests

Despite years of study internationally, non-laboratory-based POC tests for syphilis are in their infancy in the United States, with only two FDA-cleared and CLIA-waived tests. Additional POC tests and data are needed to increase understanding of their performance in clinical and outreach settings. Additional areas needed for research include well-designed prospective studies on POC test performance in the context of screening algorithms, special patient populations, linkage to treatment and care, and cost-benefits so that recommendations can be made regarding performance and use in the U.S. Also needed are studies comparing POC tests with FDA-cleared, laboratory-based treponemal serologic tests, followed by programmatic recommendations for implementation to guide their appropriate use in syphilis testing algorithms.

Direct Detection Tests

Direct detection of *T. pallidum* has been based on microscopy but is being modernized with molecular methods for detection. No FDA-cleared molecular tests are marketed in the U.S., although certain laboratories offer such testing using in-house, laboratory-developed and validated tests. Molecular tests that are FDA cleared for *T. pallidum* would facilitate their uptake in laboratories. However, additional research is needed in determining optimal specimen types, including genital and extragenital specimens stratified by stage of syphilis, specimen transport and storage, and specimen adequacy; identifying molecular markers that could be used to monitor for the emergence of antimicrobial resistance and strain typing to better aid epidemiological investigations; evaluating the sensitivity of NAATs on whole blood or its components (serum and plasma); and assessing the cross-reactivity of NAATs with commensal *Treponema* spp.

Conclusion

Combating health disparities among syphilis cases in Urgent Care centers requires a multifaceted and inclusive approach. Protocols are not mere guidelines; they are powerful tools for dismantling health disparities. The implementation of tailored protocols, coupled with community engagement and cultural sensitivity, stands as a crucial step in addressing the unique challenges faced by diverse populations. As we strengthen our protocols, we move closer to fostering healthier communities.

It is imperative for Urgent Care centers to be cognizant of the interconnectedness of social determinants, economic factors, and cultural considerations that contribute to disparities in healthcare access and outcomes. Thus, we should incorporate initiatives to address the barriers to care services, proactively bridge gaps in care, and ensure that individuals from all backgrounds have equal access to timely and effective syphilis screening, testing, and treatment.

As we advance towards a future with reduced health disparities, the integration of these strategies into acute care protocols becomes an indispensable part of a resilient and responsive healthcare system. Moreover, community focused strategies and collectively working towards this envisioned future, Urgent Care centers can become transformative agents in the pursuit of health equity and shape a healthcare landscape that is truly accessible, equitable, and responsive to the diverse needs of all individuals and communities.

Ultimately, the commitment to inclusivity in Urgent Care not only improves outcomes for individuals affected by syphilis but contributes to a broader vision of a more just and equitable healthcare system.

References

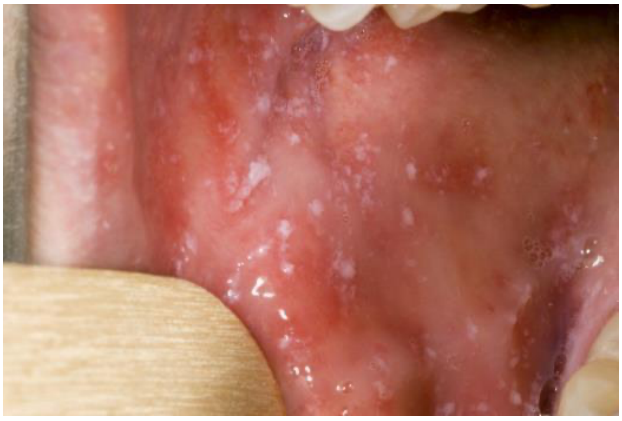
Papp JR, Park IU, Fakile Y, Pereira L, Pillay A, Bolan GA. CDC Laboratory Recommendations for Syphilis Testing, United States, 2024. MMWR Recomm Rep 2024;73(No. RR-1):1–32. DOI: http://dx.doi.org/10.15585/mmwr.rr7301a1.

2022 U.S. syphilis cases reach highest numbers since the 1950s. Centers for Disease Control and Prevention. January 30, 2024. Accessed February 11, 2024. https://www.cdc.gov/nchhstp/newsroom/2024/STI-Surveillance-Report-2022.html.

U.S. syphilis cases in newborns continue to increase: A 10-times increase over a decade. Centers for Disease Control and Prevention. November 7, 2023. Accessed February 11, 2024. https://www.cdc.gov/nchhstp/newsroom/2023/syphilis-cases-in-newborns.html.

Koplik Spots: Early Detection of Measles is Key

Chris Chao, MD



The incubation period of measles is 10-12 days. A patient is not contagious during this time. Once an infected individual develops symptoms (including fever), they are highly contagious with a transmission rate of 90%.

Early symptoms include fever, cough, coryza and conjunctivitis. Unfortunately, early symptoms cannot easily be differentiated from other common viral illnesses.

Source: https://www.nhs.uk/conditions/measles/symptoms/

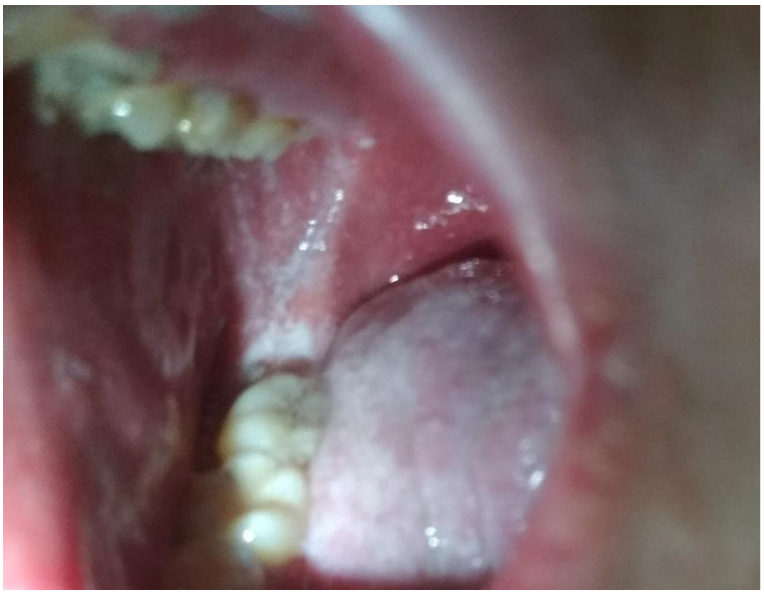
KOPLIK SPOTS ARE THE EARLY SIGNS OF MEASLES

Koplik spots occur 1-4 days before the exanthem and occur in up to 70% of patients with measles. They are typically not painful, which may differentiate Koplik spots from aphthous ulcers or herpangina. Koplik spots initially present as bluish white “grains of salt on a red background” opposite the molars on the buccal mucosa. Spots may extend to the entire buccal mucosa before disappearing as the rash begins.

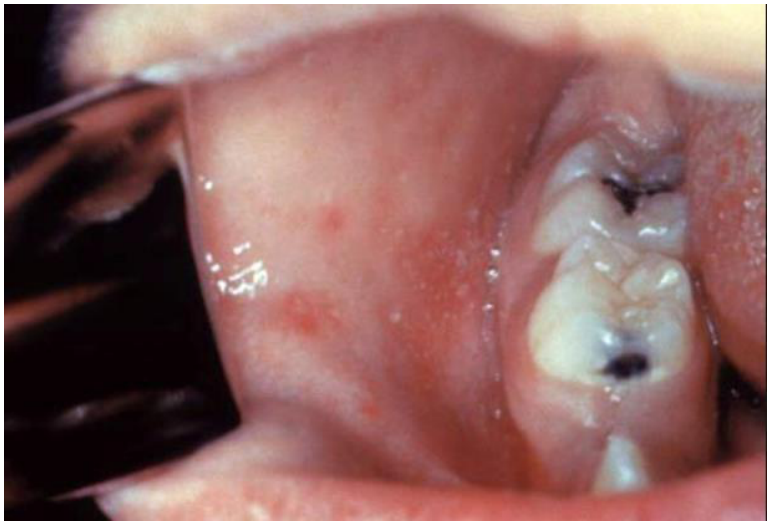
A patient with measles is contagious during the prodrome period up to 5 days after the rash onset. By the time the characteristic rash develops, the patient has been contagious for several days. A patient is contagious when Koplik spots develop.

Since Koplik spots occur before the characteristic rash of measles develops, prompt recognition of Koplik spots is critically important to limit the spread.

To identify Koplik spots, you must look for it, and this includes doing a comprehensive oral exam on a potentially non-cooperative child.



https://commons.wikimedia.org/wiki/File:Koplik_spots,_measles_2019.jpg



https://commons.wikimedia.org/wiki/File:Koplik_spots,_measles_2019.jpg

References:

Zenner, D, Nacul L, “Predictive power of Koplik’s spots for the diagnosis of measles” J Infecti Dev Ctries 2012 Mar 12;6(3):271-5
CDC: measles https://www.cdc.gov/measles/index.html

Oral and Maxillofacial Section—Infection and Inflammation of Intraoral Implants

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Oral & Maxillofacial Surgeon ("OMFS")

The prevalence of dental implants has reached a record high in recent decades. Approximately 6% of the U.S. population had at least one endosseous oral implant by 2015, which represents an 8-fold increase from 1999 that continues to rise.[1] Some projections estimate that up to a quarter of American adults will undergo implant placement in the next decade. Consequently, non-dental healthcare providers are increasingly encountering inflammatory emergencies arising from dental implant failure.

Figure 1. Typical Dental Implant

A typical dental implant complex consists of the crown, the abutment, as well as the implant screw (Figure 1). The crown is the white, esthetic portion that replicates the visible portion of a native tooth (#1). The implant is the titanium- or zirconia-based screw that is inserted into the underlying jawbone (#3). The abutment serves as the interphase between the two (#2). Although any components could contribute to failure, most complications occur at the level of the implant screw itself because it directly integrates with the living tissues.

A healthy implant forms a watertight attachment to both the surrounding mucosa (also known as the gingiva) and the underlying bone. If bacterial biofilms of *Fusobacteria*, *Prevotella*, and/or *Streptococci* species form at this interface, the mucosa may inflame and partially detach from the implant, creating a sulcus.[2] An inflamed mucosa would demonstrate erythema, tenderness, hyperplasia, swelling, and potentially bleeding adjacent to the dental implant.[3]

The healthcare provider must first distinguish the extent of the inflammatory disease using a periodontal probe. A periodontal probe is a thin, probe-like ruler (Figure 2). Its tip should be introduced gently through the sulcus to measure the distance from the mucosal surface to the attachment point between the implant and bone (Figure 3). Probing depths of 3mm or less is indicative of peri-implant mucositis (PIM) where the mucosa is inflamed but the underlying bone remains relatively unchanged. [4] Depths 4mm or greater indicates peri-implantitis (PI) where the underlying bone has been destroyed. (Figure 4). Nonmetallic probes are preferred and available at most reputable medical-dental suppliers such as Henry Schein® and Patterson®.

Figure 2. Periodontal Probe

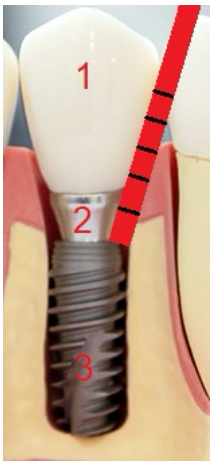

Figure 3. Measuring distance between gum and implant attachment to bone

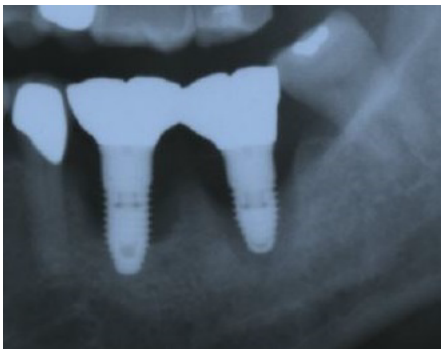

Figure 4. Peri-implantitis. Note the large space between the implant and bone.

Nonmetallic construction is critical as metallic instruments can abrade or damage implant surfaces, creating additional biofilm attachment sites.[5] For both PIM and PI, the provider should use a fine, non-metallic instrument to debride the sulcus. The periodontal probe itself can be used to remove any visible residue. Thicker angiocaths 18g or wider may also be used as they offer flexible tips and can be used to directly irrigate into the debrided areas. Other common and affordable instruments include commercial dental picks such as the Sunstar GUM Soft-Pick® which are equipped with textured surfaces for debris trapping. Irrigation can be conducted using saline or, more preferably, using a topical antibiotic solution such as 0.12% chlorhexidine gluconate. Clinicians may prescribe additional chlorhexidine for at-home use as a mouth rinse until the patient can visit their dental professional.

Peri-implantitis (PI) represents a worsening progression of peri-implant disease but with comparable bacterial populations. Its histology shows increased presences of neutrophils, B-cells, and macrophages when compared to PIM, leading to active destruction of bone.[6] Later stages of PI may be accompanied by frank purulence and implant mobility as well.[7] Treatment should consist of the same debridement process, though a 3% hydrogen peroxide is likely a more effective irrigant. Long-lasting topical antibiotics may be helpful in arresting progression of the bone loss.[8] Minocycline microspheres are commercially available (Arestin™) and sold in injectable syringes. Chlorhexidine is also manufactured in small chips (PerioChip™) and can be placed in any debrided spaces. Although not ideal, clindamycin powder (found within PO clindamycin capsules) can also be placed within the sulcus using small nasal or otic/ear scoops.

Fortunately, outright peri-implant abscesses are quite rare. This is likely because the microbial colonization occurs at or near the surface of the mucosa, which allows for ready drainage of any

purulence. If present, they should be treated like any intraoral abscesses and drained via incision and/or aspiration.

Systemic antibiotics can be used especially in the later stages when purulence becomes evident. Beta-lactams such as amoxicillin, penicillin, and cephalexin – a mainstay for traditional odontogenic infections – should be augmented with clavulanate (in the form of combined amoxicillin-clavulanate 875-125mg formulations) or with metronidazole (usually dosed at 500mg three times daily) to help better address the anaerobes present in PI.[9] Other suitable antibiotics may include azithromycin (500mg qday 1st day, 250mg qday 4 following days) or doxycycline (100mg twice daily) as outlined by the American Heart Association infective endocarditis guidelines.[10] All PI patients must be reminded of good oral hygiene and instructed to visit his or her dental care provider as soon as possible.

It is important to note that systemic metabolic imbalances frequently fuel rapid progression to PI and irreversible bone loss despite adequate treatment of PIM. In particular, poorly managed diabetes mellitus (both type 1 and 2) is associated with increasing rates of implant failures [11] as are osteoporosis and metastatic cancer.[12] Long-lasting topical antibiotics and systemic antibiosis should be considered for these patients even if they did not show evidence of PI. As always, excellent patient interview and history taking is critical.

It is important to note that this article is not meant to direct any treatments or diagnoses. The information presented represents our own protocols and is presented for your academic consideration. All clinical decisions should be made based on the knowledge and experience of appropriately licensed providers.

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References

[1] Elani HW, Starr JR, Da Silva JD, Gallucci GO. Trends in Dental Implant Use in the U.S., 1999–2016, and Projections to 2026. Journal of Dental Research. 2018;97(13):1424–30.
[2] Pokrowiecki R, Mielczarek A, Zaręba T, Tyski S. Oral microbiome and peri-implant diseases: Where are we now? Therapeutics and Clinical Risk Management. 2017;13:1529–42.
[3] Heitz-Mayfield LJA, Salvi GE. Peri-implant mucositis. Journal of Clinical Periodontology. 2018;45(August 2017):S237–45.
[4] Pontoriero R, Tonelli MP, Carnevale G, Mombelli A, Nyman SR, Lang NP. Experimentally induced peri-implant mucositis. A clinical study in humans. Clinical Oral Implants Research. 1994 Dec 1;5(4):254–9.
[5] Prathapachandran J, Suresh N. Management of peri-implantitis. Dental research journal. 2012 Sep;9(5):516–21.
[6] Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. Journal of Clinical Periodontology. 2018;45(June 2016):S246–66.
[7] Stavropoulos A, Bertl K, Pietschmann P, Pandis N, Schiødt M, Klinge B. The effect of antiresorptive drugs on implant therapy: Systematic review and meta-analysis. Clinical oral implants research. 2018 Oct;29 Suppl 1:54–92.
[8] Renvert S, Lessem J, Dahlén G, Lindahl C, Svensson M. Topical minocycline microspheres versus topical chlorhexidine gel as an adjunct to mechanical debridement of incipient peri-implant infections: a randomized clinical trial. Journal of Clinical Periodontology. 2006 May 1;33(5):362–9.
[9] Blanco C, Pico A, Dopico J, Gándara P, Blanco J, Liñares A. Adjunctive benefits of systemic metronidazole on non-surgical treatment of peri-implantitis. A randomized placebo-controlled clinical trial. Journal of Clinical Periodontology. 2022;49(1):15–27.
[10] Wilson WR, Gewitz M, Lockhart PB, Bolger AF, Desimone DC, Kazi DS, et al. Prevention of Viridans Group Streptococcal Infective Endocarditis: A Scientific Statement from the American Heart Association. Circulation. 2021;143(20):E963–78.
[11] Jiang X, Zhu Y, Liu Z, Tian Z, Zhu S. Association between diabetes and dental implant complications: a systematic review and meta-analysis. Acta Odontologica Scandinavica. 2021;79(1):9–18.
[12] Schimmel M, Srinivasan M, McKenna G, Müller F. Effect of advanced age and/or systemic medical conditions on dental implant survival: A systematic review and meta-analysis. Clinical Oral Implants Research. 2018;29(March):311–30.

Coding Corner: Documentation and Coding Pearls for Chronic Illness with Exacerbation and Comorbid Conditions

Brad Laymon PA-C, CPC, CEMC
Section Editor, Coding Corner

Chronic Illnesses with Exacerbation
Consider the following examples:

67-year-old male with complaint of "my chronic back pain has flared up."
He does have a history of chronic back pain and takes Mobic 7.5 mg PRN.
He denies any injury but states it started after raking the back yard for about 45 minutes.
34-year-old female complains of an asthma attack.
She has used her albuterol inhaler with little relief.
She denies any recent URI.

59-year-old male patient who is being seen for a sore throat, but his blood pressure is 168/97.
He does have a history of hypertension.
He takes HCTZ 25 mg daily.

What do these patient complaints have in common?

They all have chronic illnesses which are exacerbated and poorly controlled.

Let's look at the management of these 3 examples:

67-year-old male with complaint of chronic back pain exacerbation
He will start his Mobic 7.5 mg once daily instead of PRN.
He can use OTC Tylenol PRN.

34-year-old female complains of an asthma attack.
1 albuterol nebulizer treatment was given in the center with improvement in her breathing.
She will continue her Albuterol MDI inhaler 2 puffs every 4-6 hours as needed.

59-year-old male patient who is being seen for a sore throat, but his blood pressure is 168/97.
Discussed elevated BP today. He has not been taking as prescribed.
He will take his HCTZ 25 mg once daily.
He will follow up with his PCP for further evaluation/treatment.

What level of service would you code the above examples?
The correct level of service for these (with proper documentation) would be level 4 visits…for all of them!

Coding tips for patients presenting with chronic illnesses which are poorly controlled:

- 1 chronic illness with exacerbation + Rx mgmt. = Level 4
- 1 chronic illness with exacerbation + 3 POC tests = Level 4
- Any chronic illness (diabetes, HTN, asthma, chronic pain, eczema, etc.) which is not at treatment goal, and you prescribe or inform the patient to continue their current prescription medication, will be a level 4 visit.
- You must document the chronic illness in the HPI section and have a treatment plan documented in your MDM section.
- As in the third example above, even if the patient is being seen for an illness/injury which is not related to the chronic illness which is not at goal, with proper documentation, these will be level 4 visits.

COMORBID CONDITIONS

When and Why to Use Comorbid Conditions?

The guidelines state, "*Comorbidities and underlying diseases, in and of themselves, are not considered in selecting a level of E/M services unless they are addressed, and their presence increases the amount and/or complexity of data to be reviewed and analyzed or the risk of complications and/or morbidity or mortality of patient management*."

Examples would include:

- The diabetic patient presenting with a foot wound.
- A COVID-19-positive individual exhibiting multiple chronic conditions.
- A cardiac patient experiencing chest pain and shortness of breath.

Examples would NOT include:

- Notation in the patient's medical record that another professional is managing the problem without additional assessment or care coordination documented does not qualify as being addressed or managed by the clinician.
- Referral without evaluation (by history, examination, or diagnostic study[ies]) or consideration of treatment does not qualify as being addressed or managed by the clinician.

Documentation Key Points

It is not enough to just note the patient has a comorbid condition. The guidelines state the condition must be, "*addressed and their presence increases the amount and/or complexity of data to be reviewed and analyzed or the risk of complications and/or morbidity or mortality of patient management*". A problem is addressed or managed when it is evaluated or treated at the encounter by the provider. For example, "BP is 138/88 today. Patient will continue taking HCTZ 25 mg once daily, log BPs daily, and follow up with his PCP for evaluation." Adding the comorbid condition as a diagnosis is also advised.

Source: American Medical Association. CPT® evaluation and management (E/M) office or other outpatient (99202-99215) and prolonged services (99354, 99355, 99356, 99417) code and guideline changes. Available at: www.ama-assn.org/system/files/2023-e-m-descriptors-guidelines.pdf

A Best Practice from the College of Urgent Care Medicine

Diagnosing and Starting Hypertension Treatment for Otherwise Healthy Adults in the Urgent Care Setting

Date Reviewed	February 28, 2024
Subject	Diagnosis and Initial Treatment of Hypertension in Urgent Care
Patient Population	Otherwise healthy, asymptomatic, nonpregnant adults
Rationale	Hypertension can have major health consequences and may often go undiagnosed and untreated. As a chronic health issue, ongoing management of hypertension (HTN) is usually managed by primary care clinicians, cardiologists, nephrologists and other specialists. Many patients' most frequent access to healthcare, however, is through Urgent Care (UC), and the diagnosis of HTN and lack of treatment may be apparent in this setting. Also, the "silent" nature of HTN as a risk factor, and less ready access to primary or specialty care may impede patient follow-up for this issue subsequent to an UC visit. High-quality guidance exists to enable UC clinicians to diagnose and begin treatment for this disease, thereby reducing morbidity and mortality for the patient.
Introduction	HTN is a major risk factor for myocardial infarction, stroke, and heart and kidney failure. Even small reductions in blood pressure benefit patients. Diagnosis is based on multiple elevated blood pressure (BP) readings over time, which may be apparent in the UC setting. The College of Urgent Care Medicine has previously created a Best Practice Summary for the Management of Asymptomatic Elevated Blood Pressure1, emphasizing confirming elevated BP levels and reasonable time intervals for referrals to primary care. Once HTN is diagnosed in otherwise healthy adults, initiation of treatment is relatively straightforward and does not require

	additional testing beyond vital signs and, at most, basic lab work. Though ongoing follow-up and optimized HTN management may take more time and be better performed in primary care, many patients access healthcare more readily in UC, and the initial treatment for HTN can be within the purview of the acute care clinician. Such treatment could be expected to improve long-term health outcomes for patients. This Best Practice Summary provides evidence- and guideline-based interventions for those UC clinicians and practices desiring to begin such treatment for these patients. The management of elevated BP in patients with significant or multiple comorbidities and with resistant HTN typically requires specialty care and referral. Hypertension is considered resistant when 4 or more medications at optimal doses are required to achieve a goal/target BP, or blood pressure continues to be above goal/target with the patient taking 3 or more medications at optimal doses.
Evidence based guideline with strength of evidence	A multi-specialty conjoint clinical practice guideline2 was published in 2017 summarizing the best evidence for the treatment of HTN. Recommendations were comprehensive and most recommendations were strong, though level or quality of evidence varied based on the recommendation. A practical review3 published in 2022 analyzed data published after the conjoint guideline to provide a more recent high-quality summary of evidence to inform practice. The American Academy of Family Physicians (AAFP) published clinical practice guidelines4 specifically with respect to BP target range, recommending slightly higher-goal blood pressures than the other two references. An earlier Cochrane Review5 supported the AAFP BP target ranges, finding that benefits do not outweigh potential harms with any more aggressive BP target than recommended by AAFP.

Discussion	**The information in this Best Practice Summary applies to asymptomatic adults. Patients who present to Urgent Care with any level of elevated blood pressure and symptoms worrisome for acute end-organ changes, including focal neurological symptoms or signs, shortness of breath, chest discomfort or equivalents, dark urine, etc., should be safely referred and transported to the emergency department in most cases. Also, the evaluation and treatment of elevated blood pressures in children and in pregnant patients should follow different guidance than explained here.** **This Best Practice Guideline should not be construed to create an obligation for UC clinicians to treat patients with hypertension but rather to provide evidence- and guideline-based interventions for those clinicians and practices which desire to do so.** In situations where elevated blood pressures are found in patients without acute-end organ effects, evidence-based recommendations1 do allow for an acute-care specialist referral to primary care with no need for treatment to be started immediately. Patients without end-organ symptoms with elevated BP < 159 systolic and <99 diastolic should be referred within a few weeks. Those with elevated BP 160-180 systolic and 100-110 diastolic should be seen in referral within a few days to a week. "Prompt" referral is recommended for those with average BPs >180 systolic or >110 diastolic if treatment is not initiated in UC. Some patients may have markedly elevated blood pressures (e.g., < 220/120) in UC, either as their main concern or found incidentally. Existing guidelines do not make specific recommendations for patients other than the > 180/>110 threshold, but clinician judgement may always be exercised regarding ED referral, incorporating shared decision-making with patients. If initiating blood pressure medication, the expectation is that blood pressure will be observed to gradually decrease over days to weeks. At no point in any patient's treatment is there a need to rapidly normalize blood pressure in the Urgent Care

	setting. Overly aggressive treatment in this regard can cause more patient harm than benefit. **Basic diagnostic and evaluation recommendations** BP should be measured using appropriate techniques. (**See Table 1**) Home BP readings should utilize a similarly proper technique, and home BP devices (preferred to home auscultation) should be calibrated with BP measurement devices or auscultation in the UC or primary care clinic. Home BP readings may be used to help with the diagnosis of HTN, as well as determining whether "white coat hypertension" or "masked hypertension" (high BP existing out of a medical setting, with normal BPs in the medical setting) exists. Home BP readings can also be used to assess response to BP interventions and medications. Average blood pressure, ideally measured in a variety of settings, is used to make treatment decisions. Based on two or more averaged blood pressure readings and other cardiovascular risk factors, clinicians should recommend follow-up for patients with BP above 120/80. It is recommended that all patients with averaged BP ≥ 140/90 (either systolic or diastolic) begin treatment with medication. According to the cited guidelines2,3,4, those with known vascular disease or events or a 10-year risk of cardiovascular disease (CVD) of ≥ 10% and BP 130-139/80-89 should also be treated. (**See Table 2** for initial treatment details and recommended follow-up). Many online and other calculators exist to help determine 10-year CVD risk (e.g., https://tools.acc.org/ascvd-risk-estimator-plus/#!/calculate/estimate/ or https://www.mdcalc.com/calc/3398/ascvd-atherosclerotic-cardiovascular-disease-2013-risk-calculator-aha-acc). If access to timely subsequent care is limited, initiation of treatment may be considered in the UC setting.

	Several medical issues – renal disease, sleep apnea, drug, and alcohol use – can raise blood pressure and complicate management. Also, though most patients with elevated BP will have primary HTN, a small but important percentage will have secondary HTN, and the underlying cause will need to be sought. Clinicians should consider and screen for these factors (**See Table 3**) as blood pressure medication alone may not be sufficient, and further evaluation and management is beyond the scope of UC. Appropriate referrals should be made in these cases. A basic metabolic panel may be considered in conjunction with prescribing medication with possible renal or electrolyte effects or precautions. Urinalysis and EKG can assess for end-organ changes of HTN and fine-tune medication selection and are useful to obtain as a baseline, but they do not affect initial prescribing. Lipid panels, as well, can help with assessing overall cardiovascular risk but are not essential prior to initial prescribing. More extensive lab-work is needed in cases of resistant or suspected secondary HTN. **<u>HTN treatment recommendations</u>** Certain lifestyle measures (**See Table 4**) should be recommended to all patients. These measures can be considered nonpharmacological treatment for hypertension and have been shown to reduce blood pressure between 4- and 11-mm Hg when consistently followed. BP should still be checked over time because these measures alone may be insufficient to reduce BP to goal levels. Even so, these measures promote good health in general and may lessen the need for higher dose medications. **Goal or target BP** for nonpharmacological and pharmacological treatment according to the conjoint clinical practice guideline2 and cited review3 is < 130/80. The AAFP guidelines4 recommend a standard BP target of < 140/90. This is a strong recommendation based on high-quality evidence. AAFP states that treating to a lower blood pressure target of < 135/85 mm Hg may be considered based on

	patient preferences and values, but this was a weak recommendation and is based on moderate-quality evidence. As a rationale for the different recommended goal BP, AAFP cites evidence that treatment to lower targets does reduce the risk of myocardial infarction but not stroke or mortality, and the rate of side effects may be higher. In patients for whom lifestyle measures alone have not lowered BP adequately (based on the chosen target or goal as shown above) within the follow-up interval, clinicians may begin a first-line medication at the starting dose (**See Table 5**). If, at subsequent follow-up, BP is still not controlled adequately, the dose of the initial medication should be increased toward the maximum before starting another agent. Clinical follow-up is recommended at monthly intervals until a goal/target BP is reached. Patients not achieving adequate BP control at the maximum dose of the initial agent can be started on a second first-line medication at the starting dose. The combination of thiazide diuretics with either ACE-inhibitors or angiotensin receptor blockers is often quite effective. First-line agents are all available in generic forms but may differ in price; patient cost considerations are reasonable to factor into decision-making. Patients should be informed that, different than most medications prescribed for acute care problems, medications for HTN do not cure the condition, but that they constitute ongoing treatment and may need to be taken lifelong. Clinicians should monitor for compliance and for side effects and consider changing agents if undue side effects occur. Clinicians should prescribe sufficient quantities/refills of effective medications to bridge primary or specialty care appointments. Patients who have already documented vascular disease or events (e.g., myocardial infarction, stroke) or other comorbidities (chronic renal disease, congestive heart failure) may be treated to lower BP goals in primary care or specialty settings.

	For patients 65 years of age and older, the conjoint clinical practice guideline2 and cited review3 recommend a systolic BP goal of < 130 without respect to diastolic blood pressure. The AAFP guideline recommends no specific change in target BP of < 140/90 with aging. At some point, patients started on medication for HTN in UC should have primary care established, even if their BP comes under good control, to begin general health maintenance, cancer screening, etc., which is beyond the scope of UC. **Patient Education** Patient education may include informing patients of the adverse consequences of inadequately controlled HTN; the possibility of side effects and options to change to other medications if these occur; and the potential lifelong need for lifestyle modification and BP medications to reduce morbidity and mortality. Informational handouts or links to these can be curated and/or given to patients, for example: https://www.heart.org/-/media/Healthy-Living-Files/LE8-Fact-Sheets/LE8_How_To_Manage_Blood_Pressure.pdf https://www.heart.org/-/media/Files/Health-Topics/Answers-by-Heart/What-Is-High-Blood-Pressure.pdf There are several, varied elements to lifestyle modification that can be communicated via similar handouts, for example: https://www.heart.org/-/media/Files/Health-Topics/High-Blood-Pressure/What-can-I-do-to-improve-my-blood-pressure.pdf https://www.heart.org/-/media/Healthy-Living-Files/Infographics/Prevention_Infographic.pdf

	Patient education information may be available in many languages and, to have the greatest impact, clinicians should consider the patient's primary language when offering such education.
Summary	HTN is a common and major health issue. Its silent nature makes BP measurement and mindfulness of the numeric values important. There are many effective first-line HTN medications which can be started in UC. Ideally, patients will connect to primary care for ongoing management and health maintenance unless those services are provided by the UC practice.
References	1. Best Practice. Management of Asymptomatic Elevated Blood Pressure https://urgentcareassociation.org/wp-content/uploads/2022/10/ManagementofAsymptomaticElevatedBloodPressure.pdf 2. Whelton PK, et al. 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/ APhA/ASH/ASPC/NMA/PCNA guideline for the prevention, detection, evaluation, and management of high blood pressure in adults: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 2018;71(6):e13-e115. doi:10.1161/HYP.0000000000000065 3. Carey M et al. Treatment of Hypertension A Review. *JAMA*. 2022:328(18):1849-1981. Doi:10.1011/jama.2022.19590 4. Coles S et al. Blood pressure targets in adults with hypertension: a clinical practice guideline from the AAFP. *Am Fam Phys* 2022 Dec;106(6) online. Link 5. Arguedas, JA et al. Blood pressure targets in adults with hypertension. *Cochrane Database of Systematic Reviews* 2020, Issue 12. Art. No.: CD004349. DOI: 10.1002/14651858.CD004349.pub3
Reviewers	This document has been extensively peer reviewed by the members of the Clinical Response Committee.

Attachments (flow charts, graphics, tables, etc.)	Table 1. Accurate Blood Pressure (BP) Measurement Table 2: HTN Treatment and Follow-up based on BP Level Table 3. Considerations for Secondary Hypertension or Complicating Conditions Table 4. Lifestyle Measures to Help Hypertension Table 5: First-line Antihypertensive Medications and Clinical Considerations Figure 1: Summary Algorithm for HTN Diagnosis and Treatment

Table 1: Accurate Blood Pressure (BP) Measurement

Patient positioning and preparation	• Patient should be relaxed, sitting (not recumbent) in a chair with feet on floor and back supported (i.e., not sitting on the exam table) for at least 5 min. • Patient should avoid caffeine, exercise, and smoking for at least 30 min before BP measurement. • Patient should not have the feeling to use the bathroom or be otherwise uncomfortable. • Neither the patient nor staff should talk during BP measurement or in the $\geq$ 5 min rest period beforehand. • BP cuff should be placed directly on the skin with no clothing in between.
Proper Machine, Cuff and Auscultation Technique	• BP measurement devices should be validated and periodically calibrated based on manufacturer recommendations. • Patient's arm should be supported (e.g., resting on a desk). • The middle of the BP cuff should be positioned on the patient's upper arm at the level of the right atrium (the midpoint of the sternum). • The airbladder of a correctly sized BP cuff should encircle 80% of the patient's arm; if a larger or smaller than correctly sized cuff is used, it should be noted. • For auscultated (vs automated) BP measurements, either the stethoscope diaphragm or bell may be used.

Proper determination of BP reading	• At the time of first visit, take the BP in both arms and determine which is higher. This should be noted and be the arm for all subsequent BP measurements. • If repeat BP readings are needed, wait 1-2 minutes between measurements. • Before auscultated BP measurements, inflate the cuff and use the disappearance of the radial pulse to estimate the systolic pressure; then inflate the cuff 20-30 mm Hg above this, letting the cuff delate at a rate of 2 mm Hg per second during auscultation.
Proper BP documentation	• Record both systolic and diastolic measurements. • For auscultation, use the nearest even numbers for recording systolic and diastolic measurements. • Note the time of day that the most recent BP medication had been taken by the patient.
Use the average of multiple readings	Use the average of 2 or more readings on 2 or more occasions to estimate a patient's BP.
Provide BP reading to the patient	Provide BP readings to patients verbally and in writing.

Table 2: Treatment and Follow-up for Averaged Blood Pressure (BP) Thresholds

Average BP Threshold	**Recommended treatment and follow-up**
Normal BP (< 120/80 mm Hg)	Promote healthy lifestyle habits (nonsmoking, proper diet and exercise, weight control) and reassess annually.
Elevated BP (120-129/<80 mm Hg)	Recommend Lifestyle Measures (**See Table 4**) and reassess in 3-6 months.
Stage 1 HTN (130-139/80-89 mm Hg)	If there is no known cardiovascular (CV) disease or prior event (e.g., myocardial infarction, stroke), and 10-year CV disease risk* is < 10%, Lifestyle Measures can be recommended with 3–6-month reassessment.

	If there is known CV disease or prior event, or CV risk is ≥ 10%, Lifestyle Measures should be recommended, and antihypertensive medication started with reassessment in 1 month.
Stage 2 HTN (≥ 140/90 mm Hg)	Lifestyle Measures should be recommended, and antihypertensive medication started with reassessment in 1 month.

* Assess 10-year CV risk using a clinical calculator, such as https://tools.acc.org/ascvd-risk-estimator-plus/#!/calculate/estimate/ or https://www.mdcalc.com/calc/3398/ascvd-atherosclerotic-cardiovascular-disease-2013-risk-calculator-aha-acc

Table 3: Considerations for Secondary Hypertension or Complicating Conditions

Consider secondary HTN	**Complicating issues/conditions**
Hypertension starting < 30 years old	Known renal or renovascular disease
Diastolic HTN starting > 65 years old	Obstructive sleep apnea
Abrupt onset of HTN	Use of nonsteroidal anti-inflammatory drugs
Worsening of previously controlled HTN	Use of corticosteroids
Difficult-to-control HTN	Use of stimulant drugs (cocaine, meth)
Presence of acute end-organ effects	
Unprovoked hypokalemia	

Table 4: Lifestyle Measures to Help Hypertension

Lifestyle Measure	**Goals**
Weight loss (if overweight)	Use diet and exercise to reduce body weight close to ideal body weight.
Reduce sodium intake	Less than 2-2.3 g Na per day; in general, avoid overtly salt food and added salt; eat home-cooked food rather than at restaurant or fast food.
Healthy diet	Consider DASH, Mediterranean, vegan or low carbohydrate diet.

Physical activity	Engage in aerobic activity and/or resistance training 30-60 minutes per session, 5-7 times per week.
Alcohol consumption	Limit alcohol to $\leq$ 2 standard drinks per day for men and $\leq$ 1 standard drinks per day for women.

Twenty Questions (And Answers) About Salter Harris 1 Fracture Management

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UC MAX

1. **What is a physis?**
   - This is a growth plate which separates the epiphysis (end of the bone) from the metaphysis (the area approximating the long bone).
2. **What percentage of pediatric fractures involve the growth plate?**
   - 20-35%
3. **What is a Salter-Harris (SH) 1 injury?**
   - It is a longitudinal fracture through the physis/growth plate which does not involve a fracture through the epiphysis or the metaphysis.
4. **Are SH 2-5 able to be managed in Urgent Care ?**
   - No, these will typically need to be referred.
5. **What percent of SH injuries are type 1?**
   - About 15%
6. **What is the most common SH fracture?**
   - The most common SH fracture is type 2.
7. **What are historical features of an SH injury?**
   - Mechanism may be an impact such as fall from a height or while running or playing, or a twisting motion, often occurring during sports.
   - There will be pain.
   - Sometimes, the inability to bear weight will be present.
8. **What are exam findings of an SH 1 injury?**
   - Ecchymosis
   - Swelling
   - Pain with palpation over physis
   - If severe, possible displacement
9. **Is an SH 1 injury easy to see on an ankle X-ray?**
   - The X-ray may be *normal*, but can also see soft-tissue swelling, an effusion, widening of the physis or an irregular appearance of the physis.
10. **What is the DDx?**
    - Strain (though ankle strains are probably SH 1 fractures)
    - Vascular compromise
    - Infection/septic joint
    - Referred pain
11. **What are the important findings to document?**
    - Appearance of the skin – is there erythema, blue coloration?
    - Neuro/vascular status
    - Temperature – is the foot cold or blue?
    - Deformity
    - Palpate the proximal fibula for a Maisonneuve fracture (spiral fracture of the proximal fibula)
    - Palpate the proximal 5th metatarsal for possible Jones fracture of avulsion fracture

12. **Which other imaging tests could be done with diagnostic uncertainty and concern for more serious injury?**
    - CT or MRI or stress radiographs (uncommon to do in Urgent Care, especially with SH 1 fractures)
13. **How is an SH 1 fracture managed?**
    - Splint
    - Weight bearing is OK as tolerated
14. **When should the patient follow-up?**
    - 7-10 days
15. **What should be done at follow-up?**
    - If there is diagnostic uncertainty, repeat the X-ray and if there are signs of healing fracture, then we have confirmed the DX.
16. **Which SH fractures are most concerning?**
    - SH 3 and above
17. **How do we differentiate SH 1 from SH 5?**
    - They can appear similar but consider the mechanism of injury. This will be much more severe with an SH 5.
18. **How often are there complications of a SH1 fracture?**
- They are likely less than 1% if the time.
19. **Are there SH 1 fractures that are concerning?**
    - The slipped capital femoral epiphysis (SCFE) is technically an SH 1 which can lead to AVN, so it will need an accurate and rapid diagnosis.
    - Also, caution with capitellum due to the fact it could indicate a supracondylar fracture.
20. **How long should the patient be immobilized?**
    - 3-4 weeks

References:

Charlene Jones, Michael Wolf, Martin Herman; Acute and Chronic Growth Plate Injuries. *Pediatr Rev* March 2017; 38 (3): 129–138.
Brown T, Moran M. Pediatric Sports-Related Injuries. *Clin Pediatr (Phila)*. 2019;58(2):199-212.
Salter R, Harris, W. Injuries involving the epiphyseal plate. J Bone Joint Surg Am. 1963;45A:587-622

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Bumped and Bruised Bottoms: Coccyx Injuries

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The holidays may be over, but 'tis still the season for slip and falls on the ice that result in tailbone and coccyx-area injuries. Coccyx injuries are a common chief complaint in Urgent Care during this wintery, icy time of year. Classic coccydynia (aka coccyx pain) is worse when sitting and can worsen when rising from a seated position. At first glance, this seems like a straightforward chief complaint, but it lends itself to opportunities for a more nuanced discussion with patients.

What not to miss

Make sure you do a thorough evaluation of the rest of the pelvis. Just because they slipped and their coccyx region hurts the most doesn't mean they can't have a concomitant fracture in the rest of their pelvis as well. Also, don't let a slip-and-fall be a red herring – a thorough exam here usually means having the patient get undressed so you can visualize the skin. Inspection of the perineum and perianal area is essential to rule out other potential causes such as pilonidal cysts, hemorrhoids, or abscesses. A donut pillow won't fix those!

Not all coccydynia is due to trauma

Minor trauma? Easy. No trauma? This becomes more of a head-scratcher. After a thorough evaluation has you convinced the pain is isolated to the coccyx without a surrounding infectious cause, you might need to broaden your history. Does the patient perform activities that may lead to repetitive microtrauma (think motorcycles or avid Peloton-ers)? The female sex, including recent childbirth, is at higher risk of developing coccyx pain. A higher BMI leads to reduced pelvic rotation while sitting and may predispose these patients to coccydynia. Other less common atraumatic etiologies include avascular necrosis, coccyx bone spurs, sympathetic nerve pain, pelvic floor dysfunction, bursitis, or malignancies.

Not a simple X-ray

Sitting versus standing radiographs should be performed after 10 minutes of standing to make sure that the coccyx is in a neutral position. The sitting radiograph should be performed on a hard stool with the patient having a straight back and thighs horizontal (you might need a footrest), and the seated radiograph performed on a hard stool. When seated, the patient should start with a flat back, position thighs horizontal, and lean back to the point of maximum tenderness before the X-ray is obtained.

Treatment

It's best to advise patients that this is a slow and steady road to recovery – there is no magic healing overnight. While most of our Urgent Cares don't carry donut pillows, I find it helpful to pull up a picture of what one looks like on the website of your friendly, local pharmacy or home medical equipment store, so patients know what to look for. UpToDate does recommend tramadol, but just as we've discussed in other segments, we would recommend NSAIDs and Tylenol as initial mainstays of treatment. Physical

therapy may be helpful as well. Some spicier options have shown some benefit in the literature including trials of using a capsaicin patch at the area of pain.

Treatment

Supplemental oxygen should be initiated for any patient with a blood oxygen saturation <90% or a patient in respiratory distress with a blood oxygen saturation <95%. For critically ill children, a non-rebreather mask should be used. For children with cyanotic heart disease, inquire about baseline oxygen saturation on room air and use the patient's normal range as the goal of oxygen therapy. Although not the sole determinant of final disposition, the IDSA and BTS guidelines recommend admission for oxygen saturation <92% in children with pneumonia.[3,4]

Antipyretics Oxygen

Early administration (preferably in triage) of weight-based dosing of acetaminophen or ibuprofen usually improves general appearance, along with decreasing heart rate and respiratory rate. A young child who has improved clinical status and vital signs after defervescence is reassuring to both parents and the Urgent Care clinician. While the clinician should not base assessment of the patient solely on these grounds, in the fully immunized patient, clinical assessment after antipyretic dosing is a useful adjunct in determining which children should have additional testing performed to assess for serious etiologies of fever. If a child still appears ill, or tachycardia and tachypnea persist after resolution of a fever, more serious causes should be considered.

Intravenous Fluids

Hydration status should be assessed in all patients. Oral hydration is preferred for those who can tolerate it. Aspiration events secondary to respiratory effort are rare. When intravenous fluids are given, care should be taken to avoid overhydration.[121]

Albuterol and Corticosteroids

In the febrile child with respiratory distress, the decision to give a trial of bronchodilators should be based on past medical and family history, in addition to physical examination findings. The absence of the classic end-expiratory wheeze should not deter clinicians from the use of bronchodilators in patients for whom there is a strong suspicion of asthma. The American Academy of Pediatrics guidelines recommend against the use of albuterol and ipratropium in patients with bronchiolitis,[37] but this does not apply to patients with recurrent wheezing and suspected CAP, in whom a reactive component or asthma may be present. If there is no improvement, discontinuation of bronchodilators is appropriate. Systemic corticosteroids should be considered in children with wheezing that is responsive to bronchodilator therapy, even in patients with suspected CAP.[122]

Antibiotics

The IDSA states that "antimicrobial therapy is not routinely required for preschool-aged children with CAP, because viral pathogens are responsible for the great majority of clinical disease."[4] Antibiotic therapy can have numerous side effects including diarrhea and allergic reactions (anaphylaxis, serum sickness, and, rarely, Stevens–Johnson syndrome). Development of antibiotic resistance is also a concern.

A limitation of the guidelines is that specific guidance is not provided for when there should be concern for bacterial etiologies and when antibiotic therapy should be initiated. Based on the available literature and experience, we recommend using a combination of historical factors, physical examination findings, and radiographic findings (when indicated) to determine the need for antibiotics in preschool-aged children with CAP.

Empiric antibiotic therapy for children with suspected CAP is initially guided by the site of care, (ie, inpatient vs outpatient). Children who are well enough to be treated as outpatients are then further classified according to suspected pathogen and age. **Table 2** summarizes the recommended outpatient empiric, antibiotic regimens for children with suspected CAP. For those with uncomplicated CAP of suspected bacterial etiology, high-dose amoxicillin is the first-line antibiotic due to its narrow spectrum and excellent activity against *S pneumoniae*. A multicenter, randomized, blinded, placebo-controlled, noninferiority trial compared low-dose amoxicillin (35-50 mg/kg/day) to high-dose amoxicillin and 3-day-versus-7-day duration of therapy for the treatment of uncomplicated outpatient CAP. The authors of this study concluded that low-dose amoxicillin was noninferior to high-dose amoxicillin for uncomplicated outpatient CAP. The shorter course of amoxicillin was associated with longer cough duration, but no other significant differences were found between the 3-day and 7-day durations for the treatment of uncomplicated outpatient CAP.[123] Cephalosporins are not as effective as amoxicillin for some strains of pneumococcus.[4]

Although rates of resistance vary regionally, most S *pneumoniae* isolates in the United States are resistant to azithromycin, making this a poor choice for first-line therapy in suspected bacterial pneumonia. Azithromycin should only be used as dual therapy when atypical (mycoplasma) pneumonia is suspected. Though azithromycin is the mainstay of treatment for atypical pneumonia, evidence is lacking for its efficacy in treating this infection. High rates of resistance to azithromycin occur among mycoplasma isolates in other parts of the world, especially in Asia.

For children who require hospitalization for management of CAP, local resistance patterns and the immunization history must be considered. **Table 3** summarizes the recommended empiric antibiotics that may be started, if available, in the Urgent Care setting prior to transfer for children with suspected CAP. For fully immunized patients with suspected bacterial CAP, ampicillin or penicillin G is recommended unless the patient is in an area with high rates of pneumococcus resistance to penicillin. Ceftriaxone is recommended empirically for unimmunized or under-immunized children, patients in areas with high rates of penicillin resistance, or life-threatening infection, such as empyema or shock.

Table 2. Empiric Outpatient Therapy for Children with Community-Acquired Pneumonia[1]

Empiric Therapy for Presumed Bacterial Pneumonia	Empiric Therapy for Presumed Atypical Pneumonia
First-line: ● Amoxicillin PO 90 mg/kg/day, divided BID or TID, max 4 g/day[a] **Alternatives:** ● Amoxicillin/clavulanate PO (amoxicillin component, 90 mg/kg/day divided BID or TID, max 4 g/day) For patients with a penicillin allergy: ● Cefdinir PO 14 mg/kg, divided BID, max 600 mg/day ● Cefpodoxime PO 10 mg/kg, divided BID, max 400 mg/day For patients with a mild penicillin allergy: ● Clindamycin PO 40 mg/kg/day divided TID[b] ● Levofloxacin PO 16-20 mg/kg/day, divided BID for children aged 6 months-5 years, max 750 mg/dose; 8-10 mg/kg/day once daily for children aged 5-16 years, max 750 mg/dose	**First-line:** ● Azithromycin PO 10 mg/kg on day 1, followed by 5 mg/kg/day once daily on days 2-5, max 500 mg on day 1, followed by 250 mg on days 2-5 **Alternatives:** ● Clarithromycin PO 15 mg/kg/day, divided BID, max 1 g/day for 7-14 days ● Erythromycin PO 40 mg/kg/day, divided 4 times a day, max 2000 mg/day ● For children aged >7 years, doxycycline PO 1 mg/kg BID, max 200 mg/day

Antibiotics are not routinely required for children aged <5 years with pneumonia if there is no suspicion of bacterial co-infection.

[a]For children aged ≥5 years with clinical, laboratory, and radiographic evidence consistent with either pneumococcal or atypical community-acquired pneumonia, a macrolide can be added to a beta-lactam antibiotic for empiric therapy.

[b]Some areas have high rates of clindamycin-resistant *S pneumoniae*. Local antibiograms may be useful to determine the best antibiotic agent.

Abbreviations: BID, 2 times per day; PO, by mouth; TID, 3 times per day.

Table 3. Empiric Antibiotic Therapy for Children Hospitalized with Community-Acquired Pneumonia[1]

	Empiric Therapy for Presumed Bacterial Pneumonia	Empiric Therapy for Presumed Atypical Pneumonia
● Fully immunized with conjugate vaccines for *H influenzae* type B and *S pneumoniae* ● Local penicillin resistance in invasive strains of pneumococcus is minimal (<25%)	**First-line:** ● Ampicillin IV 150-200 mg/kg/day, divided every 6 hours, max 2 g/dose; ● Penicillin G IV 200,000-250,000 units/kg/day, divided every 4-6 hours, max 24 million units/day **Alternatives:** ● Ceftriaxone IV 50-100 mg/kg/day every 12-24 hours, max 2 g/day ● Cefotaxime 150 mg/kg/day, divided every 8 hours, max 2 g/dose ● For suspected CA-MRSA, consider addition of: ● Vancomycin IV 40-60 mg/kg/day, divided every 6-8 hours or dosing to achieve an AUC/MIC ratio of 400 ● Clindamycin PO or IV 40 mg/kg/day, divided every 6-8 hours, max PO 300 mg/dose, max IV 600 mg/dose	**First-line:** ● Azithromycin PO or IV 10 mg/kg on day 1, followed by 5 mg/kg/day once daily on days 2-5, max day 1 500 mg, max days 2-5 250 mg; in addition to beta-lactam if diagnosis is in doubt **Alternatives:** ● Clarithromycin PO 15 mg/kg/day in 2 doses, max 1 g/day ● Erythromycin PO 40 mg/kg/day in 4 doses, max 2 g/day ● Doxycycline PO 2-4 mg/kg/day in 2 doses, max 100 mg/ dose for children aged > 7 years; ● Levofloxacin PO or IV 10 mg/kg/dose, divided every 12-24 hours, max 500 mg/dose, for children who have reached growth maturity or patients who cannot tolerate macrolides
● Not fully immunized for *H influenzae* type B and *S pneumoniae* ● Local penicillin resistance in invasive strains of pneumococcus is significant (>25%) ● Life-threatening infection, including empyema	**First-line:** ● Ceftriaxone IV 50-100 mg/kg/day every 12-24 hours, max 2 g/day ● Cefotaxime 150 mg/kg/day, divided every 8 hours, max 2 g/dose **Alternatives**: ● Levofloxacin PO or IV 10 mg/kg/dose, divided every 12-24 hours, max 500 mg/dose, for children who have reached growth maturity ● For suspected CA-MRSA, consider addition of: ● Vancomycin IV 40-60 mg/kg/day, divided every 6-8 hours or dosing to achieve an AUC/MIC ratio of 400 ● Clindamycin PO or IV 40 mg/kg/day, divided every 6-8 hours, max PO 300 mg/dose, max IV 600 mg/dose	**First-line:** ● Azithromycin PO or IV 10 mg/kg on day 1, followed by 5 mg/kg/day once daily on days 2-5, max day 1 500 mg, max days 2-5 250 mg; in addition to beta-lactam if diagnosis is in doubt **Alternatives:** ● Clarithromycin PO 15 mg/kg/day in 2 doses, max 1 g/day ● Erythromycin PO 40 mg/kg/day in 4 doses, max 2 g/day Doxycycline PO 2-4 mg/kg/day in 2 doses, max 100 mg/dose for children aged > 7 years Levofloxacin PO or IV 10 mg/kg/dose, divided every 12-24 hours, max 500 mg/dose, for children who have reached growth maturity or patients who cannot tolerate macrolides

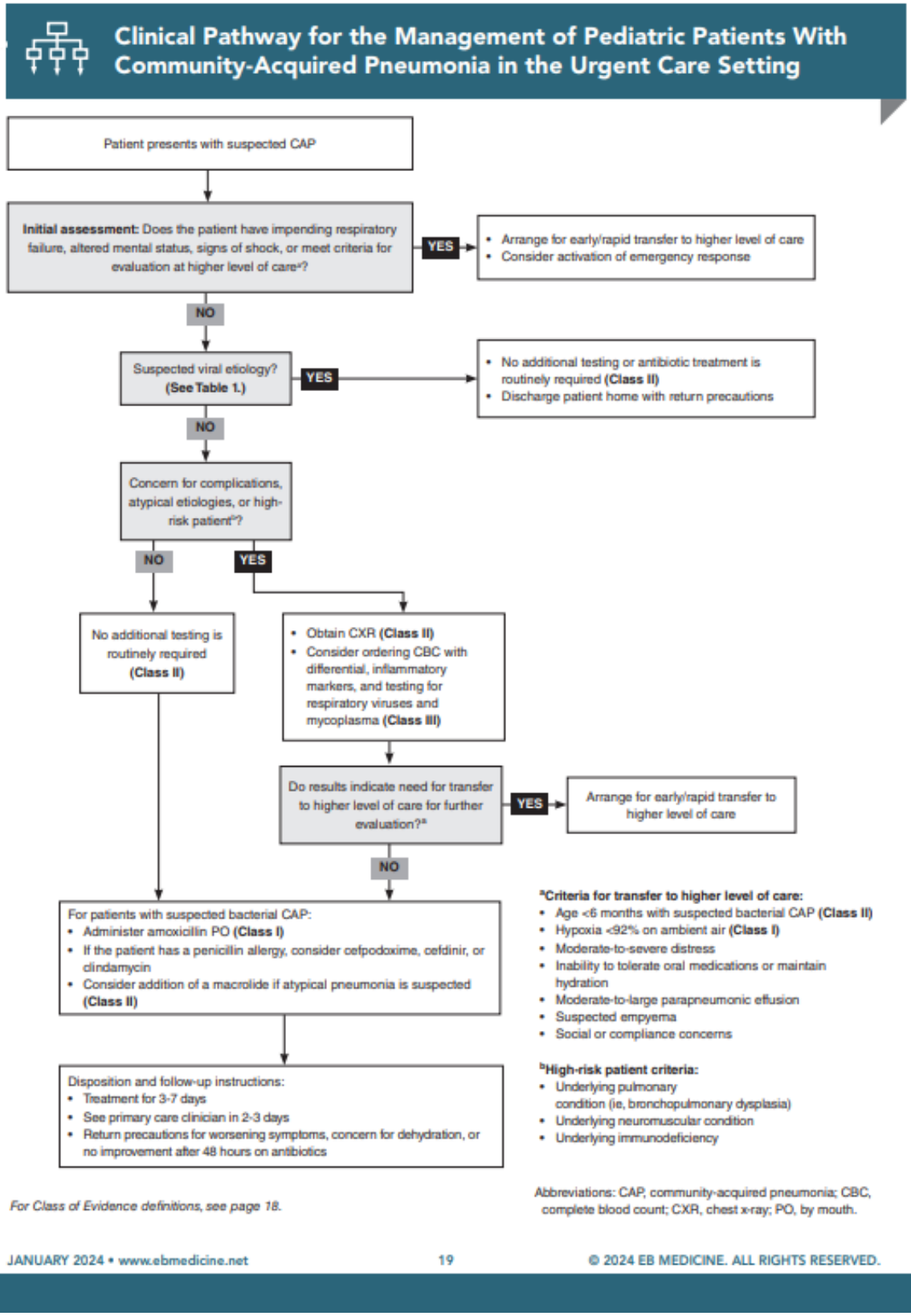
Antibiotics are not routinely required for children aged <5 years with clinical characteristics consistent with viral infection if there is no suspicion of bacterial co-infection.

Treatment should be started in the Urgent Care clinic or emergency department and continued once the patient is admitted.

Abbreviations: AUC/MIC, area under the curve to minimum inhibitory concentration; CA-MRSA, community-acquired methicillin-resistant *S aureus*; IV, intravenous; PO, by mouth.

Management of Parapneumonic Effusion and Empyema

Although small parapneumonic effusions or empyemas (<10 mm or <1/4 thorax opacified) often respond well to antibiotic therapy without additional interventions, most children with these complications will not be managed in the Urgent Care setting. Both the size and degree of respiratory compromise are important factors in determining management. Patients with moderate (>1/4 but <1/2 thorax opacified) or large (>1/2 thorax opacified) effusions are more likely to lead to respiratory impairment and should be transferred to a higher level of care for further evaluation as they are more likely to require drainage.


Clinical Pathway for the Management of Pediatric Patients With Community-Acquired Pneumonia in the Urgent Care Setting
Patient presents with suspected CAP
Initial assessment: Does the patient have impending respiratory failure, altered mental status, signs of shock, or meet criteria for evaluation at higher level of care[a]?
YES
Arrange for early/rapid transfer to higher level of care
Consider activation of emergency response
NO
Suspected viral etiology? (See Table 1.)
YES
No additional testing or antibiotic treatment is routinely required (Class II)
Discharge patient home with return precautions
NO
Concern for complications, atypical etiologies, or high-risk patient[b]?
NO
YES
No additional testing is routinely required (Class II)
Obtain CXR (Class II)
Consider ordering CBC with differential, inflammatory markers, and testing for respiratory viruses and mycoplasma (Class III)
Do results indicate need for transfer to higher level of care for further evaluation?[a]
YES
Arrange for early/rapid transfer to higher level of care
NO
For patients with suspected bacterial CAP:
Administer amoxicillin PO (Class I)
If the patient has a penicillin allergy, consider cefpodoxime, cefdinir, or clindamycin
Consider addition of a macrolide if atypical pneumonia is suspected (Class II)
Disposition and follow-up instructions:
Treatment for 3-7 days
See primary care clinician in 2-3 days
Return precautions for worsening symptoms, concern for dehydration, or no improvement after 48 hours on antibiotics
[a]Criteria for transfer to higher level of care:
Age <6 months with suspected bacterial CAP (Class II)
Hypoxia <92% on ambient air (Class I)
Moderate-to-severe distress
Inability to tolerate oral medications or maintain hydration
Moderate-to-large parapneumonic effusion
Suspected empyema
Social or compliance concerns
[b]High-risk patient criteria:
Underlying pulmonary condition (ie, bronchopulmonary dysplasia)
Underlying neuromuscular condition
Underlying immunodeficiency
For Class of Evidence definitions, see page 18.
Abbreviations: CAP, community-acquired pneumonia; CBC, complete blood count; CXR, chest x-ray; PO, by mouth.
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References

1. Kronman MP, Hersh AL, Feng R, et al. Ambulatory visit rates and antibiotic prescribing for children with pneumonia, 1994-2007. *Pediatrics*. 2011;127(3):411-418. (Population-based surveillance)
3. Harris M, Clark J, Coote N, et al. British Thoracic Society guidelines for the management of community acquired pneumonia in children: update 2011. *Thorax*. 2011;66 Suppl 2:ii1-ii23. (Expert guidelines)
4. Bradley JS, Byington CL, Shah SS, et al. The management of community-acquired pneumonia in infants and children older than 3 months of age: clinical practice guidelines by the Pediatric Infectious Diseases Society and the Infectious Diseases Society of America. *Clin Infect Dis*. 2011;53(7):e25-e76. (Clinical guidelines)
37. Ralston SL, Lieberthal AS, Meissner HC, et al. Clinical practice guideline: the diagnosis, management, and prevention of bronchiolitis. *Pediatrics*. 2014;134(5):e1474-1502. (Clinical practice guideline)
121. Haviv M, Haver E, Lichtstein D, et al. Atrial natriuretic peptide in children with pneumonia. *Pediatr Pulmonol*. 2005;40(4):306-309. (Prospective study; 28 patients)
122. Ambroggio L, Test M, Metlay JP, et al. Adjunct systemic corticosteroid therapy in children with community-acquired pneumonia in the outpatient setting. *J Pediatric Infect Dis Soc*. 2015;4(1):21-27. (Retrospective cohort study; 2244 patients)
123. Bielicki JA, Stohr W, Barratt S, et al. Effect of amoxicillin dose and treatment duration on the need for antibiotic re-treatment in children with community-acquired pneumonia: the CAP-IT randomized clinical trial. *JAMA*. 2021;326(17):1713-1724. (Multicenter, randomized controlled trial; 824 patients)

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The Timing-and-Triggers Approach to the Urgent Care Patient with Acute Dizziness

Urgent Care Evaluation

History and vital signs will usually identify the 50% of patients whose dizziness is caused by some general medical cause. The descriptive word used by the patient (e.g., "lightheadedness" or "vertigo" or "imbalance") for their dizziness is not useful diagnostically and should not, by itself, drive the workup.

The algorithmic approach to the evaluation of the dizzy patient should begin with ATTEST. **(See the Clinical Pathway for the ATTEST Approach to Urgent Care Patients with Acute Dizziness.)** The first 3 letters in the ATTEST mnemonic (Associated symptoms, Timing, and Triggers) refer to historical information: "What happened?", "When?", "Is the dizziness continuous or intermittent?", "Are there associated symptoms?" and "What is the broader context?".

Consider medical causes of the complaint, including:

- Fever, dysuria, and back pain, suggesting infection
- Heavy use of ibuprofen (or other nonsteroidal anti-inflammatory drug) and black stools, suggesting gastrointestinal bleeding
- New antihypertensive or anticonvulsant medication use, suggesting medication side effect
- Moderate-mechanism motor vehicle crash, suggesting cervical artery dissection versus cupulolithiasis versus intracranial bleed
- Abdominal pain, vaginal bleeding, and positive pregnancy test, suggesting an ectopic pregnancy.
- Chest pain and dyspnea, suggesting myocardial ischemia or pulmonary embolus
- Flank and back pain, suggesting aortic vascular complications
- Decreased oral intake, vomiting and/or diarrhea, suggesting dehydration

Each situation suggests a diagnosis or group of diagnoses that would require confirmatory testing. Similarly, the vital signs inform this diagnostic process, i.e., is there fever, tachycardia, hypotension, or hypoxia? Thus, the first diagnostic step in the diagnosis of the patient with acute dizziness is simply to take a history and review the vital signs just as with any other patient. If a general medical diagnosis is likely, I recommend a brief diagnostic "STOP," which takes less than 1 minute to perform.[13,14]

To identify patients who might potentially be mimicking a general medical condition, the 3 components of the "STOP" are: (1) a quick test for worrisome nystagmus (see the "Head Impulse–Nystagmus–Test of Skew (HINTS) Testing—Test 1: Nystagmus Testing" section for a detailed description), (2) arm dysmetria, and (3) truncal ataxia. To test for truncal ataxia, simply have the patient sit up on the exam table without grabbing hold of anything for stabilization. If the "STOP" test is reassuring, then proceed with treatment for the presumed condition. If it is worrisome, consider various vestibular or central conditions.

Acute Vestibular Syndrome

If the history does not suggest a general medical condition (or if the "STOP" is worrisome), then the next question to pose is, "Is the dizziness persistently present and still present at the time of evaluation in the UC?" A "yes" answer identifies patients with the AVS, who have the abrupt or rapid onset of dizziness that has lasted hours to days and is still present at the time of examination, even when the patient is lying still.

Dizziness may decrease when lying still and worsen with head movement, a common occurrence that does not mean that dizziness has a peripheral cause.

Although the strict neuro-otology definition of AVS includes the presence of nystagmus, some patients who otherwise fulfill the AVS definition (such as many with cerebellar stroke) do not. The presence or absence of nystagmus is a key distinction because it affects how one interprets the HIT.[14,1,23,24]

Head Impulse–Nystagmus–Test of Skew (HINTS) Testing
Because, by definition, these patients are acutely symptomatic, one can use physical examination to distinguish between central cause (stroke) and peripheral cause (neuritis), referred to as *head impulse–nystagmus–test of skew* (HINTS) testing. (Note that the HINTS acronym is distinct from the acronym for the head impulse test or HIT.)

An important caveat is that most of the studies that examine the utility of HINTS have been done with neuro-otologists performing the examinations.[51,52] One study conducted by stroke neurologists showed that non–subspecialists can be trained to use the HINTS examination effectively.[53] Another European study of specially-trained emergency physicians (12 hours of special training using Frenzel lenses to interpret the eye findings) also provided evidence of its effective use in the ED.[54,55]

Because HINTS has not been validated in routine practice, 2 additional components must be added to the examination of patients with AVS: a targeted posterior circulation examination and testing of the gait.[14,24] When performing the physical examination, these 5 questions should be asked in the following sequence:

- Is there a central pattern of nystagmus?
- Is skew deviation present?
- Is the HIT worrisome for a central process (i.e., absent corrective saccade)?
- Are there central nervous system findings on the targeted posterior circulation examination?
- Is the patient unable to sit up or walk without assistance?

None of these tests is 100% sensitive, so if the answer to *any one of the questions* is "yes," the patient has a central process, likely stroke, and should be promptly transferred for further workup.[14,15,23,24] If the answer to *all 5 questions* is "no," then the patient likely has vestibular neuritis and can be safely discharged with outpatient follow-up. The 5 test elements of HINTS testing, in order, are:

Test 1: Nystagmus Testing
The acronym (HINTS) notwithstanding, do not start with the HIT, but rather with nystagmus. There are several reasons for this. First, nystagmus testing is easy for the patient. Second, if there is no nystagmus, then interpretation of the HIT is problematic, since it has been validated only in patients with nystagmus. Third, if there is no nystagmus, it makes the vestibular neuritis and labyrinthitis very unlikely (in patients presenting in the first 2 to 3 days of their illness). Finally, if there is nystagmus that is of a central type, whatever the results of the remainder of the examination, this is a patient who must be assumed to be having a stroke.

To test for nystagmus, first ask the patient to open his eyes and look forward. Observe whether there is any jerk nystagmus, in which the eyes drift in one direction, then snap quickly back. By convention, it is the rapid phase for which the nystagmus is named. If a patient looks forward and his eyes drift to the left, then snap back to the right, he has a *right-beating horizontal nystagmus*. This is usually very easy to see, especially in the first 2 to 3 days of the patient's onset of symptoms. Next, ask the patient to follow the examiner's finger, going 30° to 40° to the right, then to the left. This is called *gaze-evoked nystagmus*. Also

look for *vertical* or *pure torsional nystagmus*. In patients with the AVS, nystagmus that is vertical, purely torsional, or that changes direction with the direction of gaze is *central*.[13,15]

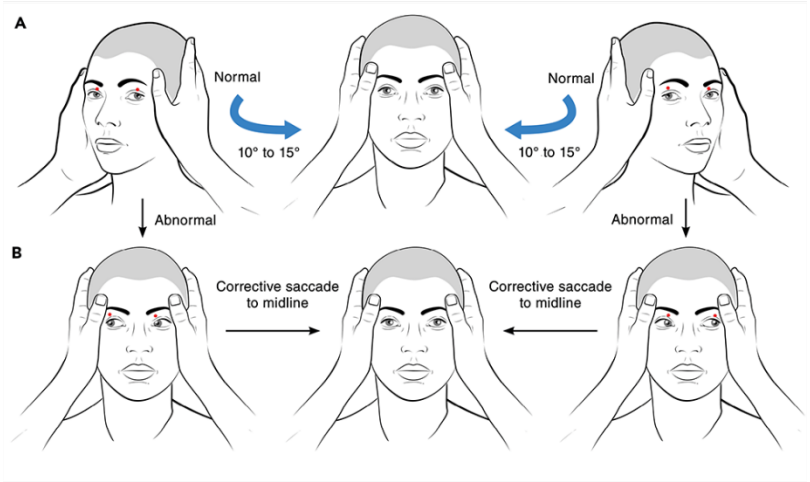
Test 2: Skew Deviation Testing

Next, check for skew deviation using the alternate cover test, which is also very easy for the patient. Stand in front of the patient, instructing him to focus on your nose. Alternately cover one eye and then the other, multiple times, moving from one eye to the next every second or so. The presence of skew deviation—a small vertical correction in the eye when it is uncovered—indicates a brainstem localization of the patient's problem. Although uncommon exceptions exist, from the perspective of a clinician, it is safest to assume that skew deviation is always due to a central problem. It is easiest if one focuses on one or the other eye (it does not matter which) because each will display the vertical correction (one going down while the other goes up).

Test 3: Head Impulse Testing

The third component of the examination is the HIT. **(See Figure 5.)** Again, the patient is instructed to relax his head and neck and to focus on the examiner's nose. The examiner grasps the patient's head on both sides and very rapidly snaps it in one direction or the other over a very small arc (only 10°-15°).[11,14,15,24] Ideally, hold the head 10° to 15° from the midline and then move it very quickly to the midline. The "normal" or "negative" HIT (when the eyes remain focused on the examiner's nose) is worrisome for stroke, whereas the "abnormal" or "positive" HIT (when the eyes move with the head and then snap back in one corrective saccade to the examiner's nose) is reassuring for vestibular neuritis. Therefore, use of the words "normal," "abnormal," "negative," and "positive" to describe the HIT is ambiguous since the "negative" test is worrisome, and the "positive" test is reassuring. It is best to simply state whether a corrective saccade is "absent" or "present."[51,56] Approximately 10% of HITs in which there is a reassuring corrective saccade are false-positives due to strokes,[51] usually of the AICA territory or the labyrinth itself.[24]

Figure 5. Head Impulse Test



To test the left side, grasp and hold the patient's head 10° to 15° to the patient's right of center, then rapidly turn the patient's head to their left, stopping at the midline. To test the right side, grasp and hold the patient's head 10° to 15° to the patient's left of center, then rapidly turn the patient's head to their right, stopping at the midline. In a "negative" or "normal" response (shown in row A), the eyes stay locked on the target. In a "positive" or "abnormal" response (shown in row B), the eyes move with the head, thus going off target, with subsequent corrective saccade to the midline.

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Test 4: Targeted Examination

The fourth component of the AVS examination is to perform a targeted examination to detect any central nervous system findings due to posterior circulation ischemia. In addition to a general motor and sensory examination, this examination targets the cranial nerves, cerebellar function, and visual fields. The latter is not in the posterior fossa, but tests the occipital cortex, which is nourished by the posterior cerebral artery, the terminal branches of the basilar artery. This should not require more than a few minutes, but it must be done systematically. Any (new) abnormality indicates a central finding and would therefore be inconsistent with neuritis. For example, anisocoria and ptosis (Horner syndrome) suggest a lateral medullary infarct. Another detail is that the unilateral facial sensory loss in lateral medullary stroke involves pain and temperature, not light touch, which is the usual modality tested by most non–neurologists. It is important to recognize that acute hearing loss, which is traditionally associated with a peripheral process, can also occur with an acute cerebrovascular event involving either the AICA or labyrinthine artery.[24]

Test 5: Gait Testing

Finally, even if all of the first 4 tests are reassuring, the gait must be tested in patients with AVS. If a patient is unsafe on his feet, he cannot be discharged safely from UC. In addition, the greater the degree of gait abnormality, the more likely it is that the cause of AVS is stroke.[57] In a series of 114 patients with AVS (67% with neuritis, 33% with stroke), most patients with neuritis were able to walk independently, whereas most patients with stroke could not.[57] In fact, two-thirds of the stroke patients could not even stand up independently. Importantly, all of the 10 patients with AICA stroke (whose HIT can be misleading) had severe gait instability.[57]

Spontaneous Episodic Vestibular Syndrome

Patients with the s-EVS report one or more episodes of dizziness of variable duration not triggered by head or body-position changes. Because patients with the s-EVS are, by definition, no longer symptomatic and are not triggerable, physical examination is not useful to distinguish the most common diagnoses, which are vestibular migraine and posterior circulation TIA. Diagnosis relies on history and epidemiologic context.[13] If a patient with vestibular migraine or TIA was still symptomatic at the time of evaluation, he would present and be evaluated as if he had AVS, just as a patient with an anterior circulation TIA who still had symptoms at the time of presentation would be assumed to be having a stroke.

Specific criteria exist for diagnosis of vestibular migraine.[58] There is a strong female predominance for vestibular migraine (5:1).[58] Patients with vestibular migraine have multiple episodes of dizziness, and headaches may occur before, during, or after the dizzy episodes.[59] When headaches do occur, they are usually (but not always) similar to migraines that occur without the dizziness. The duration of the dizziness is variable and, by definition, can last 5 minutes to 72 hours,[60] although rarely the duration is even shorter.[58] Because migraine is a central phenomenon, the associated nystagmus can be of a central type.[61]

Up to half of patients who have posterior circulation TIAs have isolated, transient dizziness.[29] Other symptoms include typical posterior circulation symptoms related to the long tracts that pass through the brainstem, cranial nerve dysfunction, or visual field cuts due to posterior cerebral artery ischemia of the visual cortex. Contrary to conventional wisdom, short-term stroke risk may be higher with posterior circulation TIA than with anterior circulation TIA.[28,62]

Recognizing that none of these elements can be used in a binary, yes/no fashion, factors that suggest vestibular migraine over TIA include younger age, more frequent attacks over a longer period of time, other migraine-related symptoms (such as headache, phonophobia, photophobia), and absence of traditional vascular risk factors.

Patients with Ménière disease (which was relatively uncommon in an ED series of dizzy patients) also present with s-EVS and will usually have ringing or buzzing in the ear and, over time, progressive hearing loss.[63] Treatment is symptomatic, and patients should be referred to an ENT specialist.

Triggered Episodic Vestibular Syndrome

The physical examination is very helpful in patients with a t-EVS and will often establish a specific diagnosis. Although the utility of orthostatic vital signs has been traditionally downplayed, a patient with dizziness when standing up who develops symptoms and orthostatic vital sign changes on standing up is highly likely to have orthostatic hypotension as a cause of the dizziness, and the evaluation is directed at finding the underlying cause.

BPPV should be suspected in patients with very brief episodes of dizziness, generally lasting less than a minute. Brief episodes of dizziness that wake a patient up from sleep are nearly always BPPV.[64-67] One study showed a positive likelihood ratio of 60 for a BPPV diagnosis if dizziness occurred with lying down or turning in bed.[66]

In patients with suspected BPPV, bedside testing can confidently establish the diagnosis. The most commonly affected canal is the posterior canal (pc-BPPV) which is usually tested by the Dix-Hallpike maneuver. If this test is negative on both sides, then the horizontal canal (hc-BPPV) is tested by the supine head roll test. In pc-BPPV, the nystagmus is typically up-beating and torsional, and in hc-BPPV, it is horizontal and direction-changing. This illustrates how the interpretation of nystagmus differs from AVS (where torsional or direction-changing = worrisome diagnosis) from t-EVS (where torsional for pc-BPPV and horizontal direction-changing for hc-BPPV = benign diagnosis).

Occasionally, BPPV patients have no nystagmus.[68-70] Possible causes are a small number of otoliths in the canal, use of vestibular suppressants at the time of diagnosis, or small-amplitude nystagmus that the examiner is not perceiving due to visual fixation by the patient.

Some patients with hc-BPPV will have spontaneous (or more persistent) nystagmus that is normally not seen with BPPV.[71,72] This occurs because, depending on the orientation of the patient's head, otoliths in the horizontal canal may be moving in a patient sitting up and looking forward.

Finally, very rarely, patients with CPPV caused by structural lesions adjacent to the fourth ventricle (usually a tumor, multiple sclerosis plaque, or small brainstem stroke) will exhibit nystagmus or other features that are atypical for BPPV.[34,73] These patients will often have some symptoms (such as headache) that patients with BPPV never have, or they do not respond as expected to a repositioning maneuver. They may have physical findings that localize to the brainstem or cerebellum that patients with BPPV do not have, or they may exhibit nystagmus in the absence of movement or dizziness.

Risk Management Pitfalls for Dizziness in the Urgent Care Setting

1. **"I thought that because the dizziness got worse with head movement, it had to be peripheral."** This is a common misconception. Dizziness at rest in a patient with a cerebellar stroke or tumor often

intensifies with head motion. It is crucial to distinguish dizziness that is triggered by movement (no dizziness at rest, but dizziness develops with movement) versus dizziness that is exacerbated by movement (dizziness is present at rest but worsens with head movement).

2. **"The negative CT ruled out a stroke!"** Brain CT is a great test for hemorrhage but a terrible test for posterior circulation infarction. Hemorrhage is a very uncommon cause of isolated dizziness without other symptoms or signs. One should never rely on a negative head CT to exclude a cerebellar or brainstem infarct.

3. **"I ruled out a posterior circulation TIA because isolated dizziness is never due to ischemia; other brainstem findings will always be present."** This is a misconception that stems from old expert opinion dating back to the mid-1970s. Newer studies make it clear that isolated dizziness is the most common transient symptom that precedes posterior circulation stroke and occurs in approximately 8% of these patients.

4. **"The patient had acute dizziness for 24 hours. The neuroradiologist read the MRI with diffusion-weighted images as negative, so there is no way this is a stroke**." It is important to emphasize that in the first 48 to 72 hours after a posterior circulation ischemic stroke that presents isolated dizziness, diffusion-weighted MRI will miss as many as 1 in 5 patients. There is a reluctance by physicians, in general, to accept that an MRI can be negative in any acute stroke, but the data are clear.

5. **"The patient had a bad headache and said he had some transient double vision, but the Dix-Hallpike test was positive on both sides. I gave him meclizine for his BPPV."** There are some symptoms that never occur with BPPV, including headache and double vision. One can never make a diagnosis of BPPV in a patient with severe headache or diplopia (even if transient). Also, the treatment for BPPV is a canalith-repositioning maneuver such as the Epley maneuver, not meclizine.

6. **"The patient had both hearing loss and dizziness, so it has to be a peripheral problem, right?"** The classic teaching that coexistence of an acute hearing loss plus dizziness is always a peripheral lesion is wrong. A stroke of the AICA territory or of the labyrinthine artery can cause a stroke of the lateral pons or of the vestibular labyrinth and cause both hearing and balance findings.

7. **"The patient was only 32 years old with no vascular risk factors, so there's no way that this is a stroke."** Young patients have strokes! Mechanisms that are more common in young stroke patients include arterial dissection and cardioembolism (especially through a patent foramen ovale), but they also can have a large-vessel disease. Young age is associated with stroke misdiagnosis.

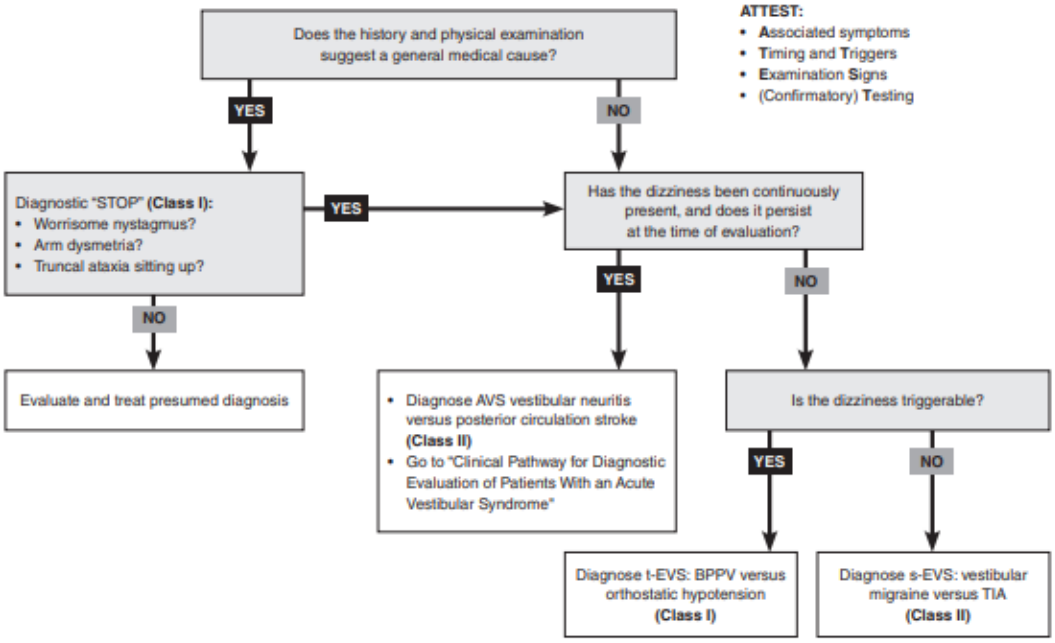
8. **"The patient felt very dizzy on gait examination but had no nystagmus at all, so I ruled out cerebellar stroke."** Only about half of patients with cerebellar strokes have nystagmus, so its absence in no way rules it out. In fact, the absence of nystagmus makes acute vestibular neuritis or labyrinthitis extremely unlikely and probably increases the probability of stroke in a patient with an AVS without nystagmus.

9. **"I know HINTS testing and the HIT was unequivocally positive. I saw a corrective saccade, so the problem must be vestibular neuritis."** No single component of the HINTS testing rules out stroke. Although the HIT is the most sensitive of all the components of HINTS, it still only has a sensitivity of about 85%. It is also important to recognize that this sensitivity was done in studies by neuro-otologists, and the sensitivity in routine emergency medicine and UC practice is not known.

10. **"The HINTS testing was worrisome, but the neurologist said to do an MRI, and if it was negative, to discharge the patient and see him on Monday."** Neurologists are the usual consultant for an acutely dizzy patient, but many neurologists still use the outmoded "symptom-quality" approach to dizziness,

and some are unfamiliar with newer data about the HINTS testing being more sensitive than early MRI. If the physical examination suggests a central cause, it trumps a “negative” MRI.

Clinical Pathway for the ATTEST Approach to Urgent Care Patients With Acute Dizziness[11]



Abbreviations: AVS, acute vestibular syndrome; BPPV, benign paroxysmal positional vertigo; s-EVS, spontaneous episodic vestibular syndrome; t-EVS, triggered episodic vestibular syndrome; TIA, transient ischemic attack.

Class of Evidence Definitions

Each action in the clinical pathways section of *Evidence-Based Urgent Care* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

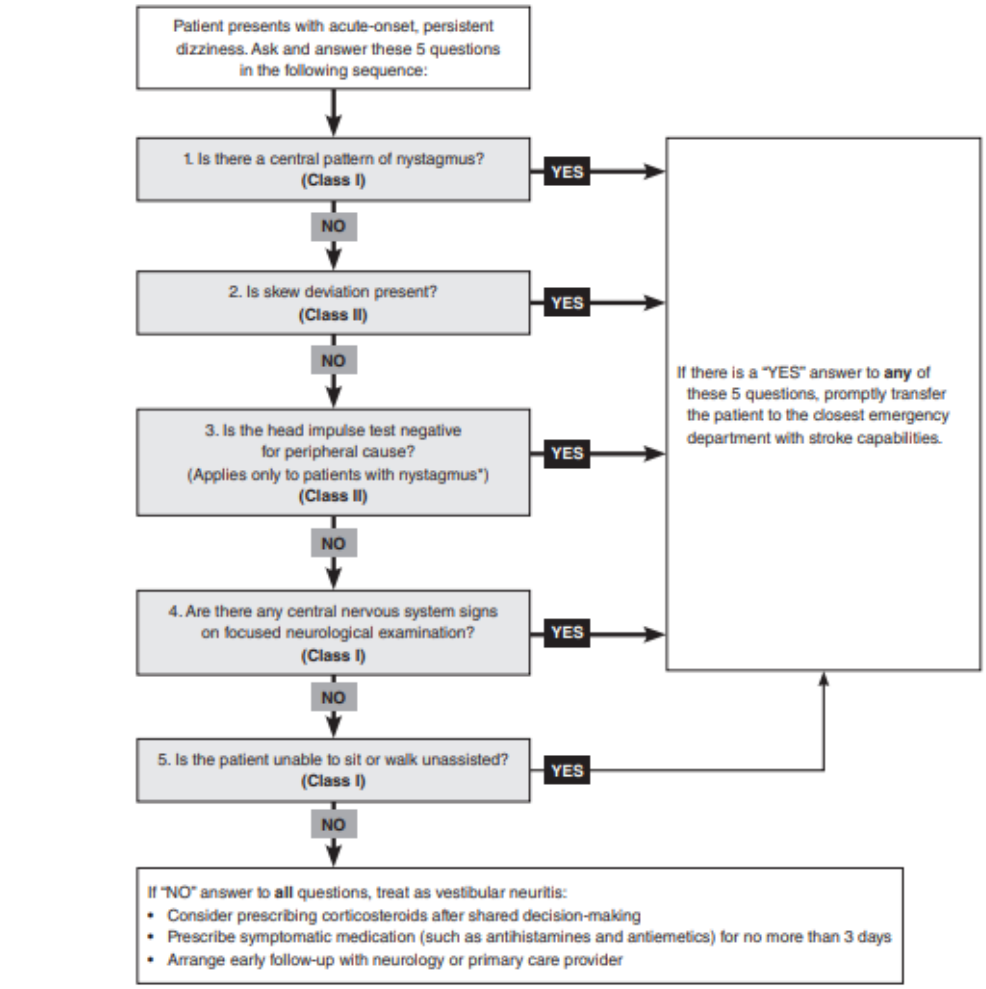
Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Clinical Pathway for Diagnostic Evaluation of Patients With an Acute Vestibular Syndrome[24]



*In patients without nystagmus, the head impulse test may give misleading results; the focused neurological examination and gait assessment become more important in this group. (See "Test 4: Targeted Examination" and "Test 5: Gait Testing" in the "Urgent Care Evaluation—Acute Vestibular Syndrome" section.)

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- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Excerpted from: Toscano J. The timing-and-triggers approach to the Urgent Care patient with acute dizziness. *Evidence-Based Urgent Care*. 2024 February;3(2):1-27. [Content was adapted from: Edlow JA. The timing-and-triggers approach to the patient with acute dizziness. *Emerg Med Pract*. 2019;21(12):1-24. Used with permission of EB Medicine.] Reprinted with permission of EB Medicine.

References

11. Edlow JA. Managing patients with acute episodic dizziness. *Ann Emerg Med*. 2018;72(5):602-610. (Review article)
13. Newman-Toker DE, Hsieh YH, Camargo CA, Jr., et al. Spectrum of dizziness visits to US emergency departments: cross-sectional analysis from a nationally representative sample. *Mayo Clin Proc*. 2008;83(7):765-775. (Cross-sectional analysis of a large national database)
14. Edlow JA, Gurley KL, Newman-Toker DE. A new diagnostic approach to the adult patient with acute dizziness. *J Emerg Med*. 2018;54(4):469-483. (Review article)
15. Edlow JA, Newman-Toker D. Using the physical examination to diagnose patients with acute dizziness and vertigo. *J Emerg Med*. 2016;50(4):617-628. (Review article)
23. Edlow JA. A new approach to the diagnosis of acute dizziness in adult patients. *Emerg Med Clin North Am*. 2016;34(4):717-742. (Review article)
24. Edlow JA. Diagnosing patients with acute-onset persistent dizziness. *Ann Emerg Med*. 2018;71(5):625-631. (Review article) DOI: 10.1016/j.annemergmed.2017.10.012
28. Gulli G, Marquardt L, Rothwell PM, et al. Stroke risk after posterior circulation stroke/transient ischemic attack and its relationship to site of vertebrobasilar stenosis: pooled data analysis from prospective studies. *Stroke*. 2013;44(3):598-604. (Pooled analysis of prospective studies; 359 patients)
34. Soto-Varela A, Rossi-Izquierdo M, Sanchez-Sellero I, et al. Revised criteria for suspicion of non-benign positional vertigo. *QJM*. 2013;106(4):317-321. (Review and opinion paper)
51. Kattah JC, Talkad AV, Wang DZ, et al. HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging. *Stroke*. 2009;40(11):3504-3510. (Prospective study; 101 high stroke risk AVS patients)
52. Saber Tehrani AS, Kattah JC, Mantokoudis G, et al. Small strokes causing severe vertigo: frequency of false-negative MRIs and nonlacunar mechanisms. *Neurology*. 2014;83(2):169-173. (Ambispective study; 190 high risk AVS patients)
53. Chen L, Lee W, Chambers BR, et al. Diagnostic accuracy of acute vestibular syndrome at the bedside in a stroke unit. *J Neurol*. 2011;258(5):855-861. (Prospective study; 24 AVS patients)
54. Vanni S, Nazerian P, Casati C, et al. Can emergency physicians accurately and reliably assess acute vertigo in the emergency department? *Emerg Med Australas*. 2015;27(2):126-131. (Convenience sample; 94 dizzy ED patients)
55. Vanni S, Pecci R, Edlow JA, et al. Differential diagnosis of vertigo in the emergency department: a prospective validation study of the STANDING algorithm. *Front Neurol*. 2017;8:590. (Prospective validation study; 252 dizzy ED patients)
56. Cnyrim CD, Newman-Toker D, Karch C, et al. Bedside differentiation of vestibular neuritis from central "vestibular pseudoneuritis". *J Neurol Neurosurg Psychiatry*. 2008;79(4):458-460. (Retrospective analysis; 83 AVS patients)
57. Carmona S, Martinez C, Zalazar G, et al. The diagnostic accuracy of truncal ataxia and HINTS as cardinal signs for acute vestibular syndrome. *Front Neurol*. 2016;7:125. (Retrospective cohort; 114 AVS patients)
58. Neuhauser H, Lempert T. Vestibular migraine. *Neurol Clin*. 2009;27(2):379-391. (Review article)
59. Furman JM, Marcus DA, Balaban CD. Vestibular migraine: clinical aspects and pathophysiology. *Lancet Neurol*. 2013;12(7):706-715. (Review article)
60. Dieterich M, Obermann M, Celebisoy N. Vestibular migraine: the most frequent entity of episodic vertigo. *J Neurol*. 2016;263 Suppl 1:S82-89. (Review article)
61. Polensek SH, Tusa RJ. Nystagmus during attacks of vestibular migraine: an aid in diagnosis. *Audiol Neurootol*. 2010;15(4):241-246. (Retrospective study; 26 patients)
62. Flossmann E, Rothwell PM. Prognosis of vertebrobasilar transient ischaemic attack and minor stroke. *Brain*. 2003;126(Pt 9):1940-1954. (Meta-analysis)
63. Sajjadi H, Paparella MM. Meniere's disease. *Lancet*. 2008;372(9636):406-414. (Review article)
64. Bisdorff A. Vestibular symptoms and history taking. *Handb Clin Neurol*. 2016;137:83-90. (Review article)
65. Ichijo H. Onset time of benign paroxysmal positional vertigo. *Acta Otolaryngol*. 2017;137(2):144-148. (Retrospective cohort study; 351 BPPV patients)
66. Lindell E, Finizia C, Johansson M, et al. Asking about dizziness when turning in bed predicts examination findings for benign paroxysmal positional vertigo. *J Vestib Res*. 2018;28(3-4):339-347. (Prospective survey; 149 patients)
67. Luscher M, Theilgaard S, Edholm B. Prevalence and characteristics of diagnostic groups amongst 1034 patients seen in ENT practices for dizziness. *J Laryngol Otol*. 2014;128(2):128-133. (Prospective observational study; 1034 patients)
68. Balatsouras DG, Korres SG. Subjective benign paroxysmal positional vertigo. *Otolaryngol Head Neck Surg*. 2012;146(1):98-103. (Prospective cohort study; 63 patients)
69. Huebner AC, Lytle SR, Doettl SM, et al. Treatment of objective and subjective benign paroxysmal positional vertigo. *J Am Acad Audiol*. 2013;24(7):600-606. (Retrospective cohort study; 63 patients)
70. Tirelli G, D'Orlando E, Giacomarra V, et al. Benign positional vertigo without detectable nystagmus. *Laryngoscope*. 2001;111(6):1053-1056. (Prospective cohort study; 43 patients)
71. De Stefano A, Kulamarva G, Citraro L, et al. Spontaneous nystagmus in benign paroxysmal positional vertigo. *Am J Otolaryngol*. 2010;32(3):185-189. (Retrospective cohort study; 412 patients)
72. Imai T, Takeda N, Sato G, et al. Differential diagnosis of true and pseudo-bilateral benign positional nystagmus. *Acta Otolaryngol*. 2008;128(2):151-158. (Retrospective cohort study; 20 patients)
73. Macdonald NK, Kaski D, Saman Y, et al. Central positional nystagmus: a systematic literature review. *Front Neurol*. 2017;8:141. (Systematic review)

Urgent Updates

Stay Alert for Measles Cases-CDC Clinician Outreach and Community Activity!

Between December 1, 2023, and January 23, 2024, the Centers for Disease Control and Prevention (CDC) was notified of 23 confirmed U.S. cases of measles, including seven direct importations of measles by international travelers and two outbreaks with more than five cases each. Most of these cases were among children and adolescents who had not received a measles-containing vaccine, even if age eligible.
Full Access: CDC

Recommendations for Healthcare Providers

Isolate	Do not allow patients with suspected measles to remain in the waiting room or other common areas of the healthcare facility; isolate patients with suspected measles immediately, ideally in a single-patient airborne infection isolation room (AIIR) if available, or in a private room with a closed door until an AIIR is available.
Precautions	Healthcare providers should be adequately wearing standard and airborne precautions should be followed, including: ◦ Use of a fit tested NIOSH-approved N95 or higher-level respirator. ◦ Use of additional PPE if needed for task (e.g., gloves for blood draws). ◦ Cleaning hands before and after seeing the patient. ◦ Limiting transport or movement of patients
Notify	Immediately notify local or state health departments about any suspected case of measles to ensure rapid testing and investigation.
Test	Follow CDC's testing recommendations and collect either a nasopharyngeal swab or throat swab for reverse transcription polymerase chain reaction (RT-PCR), as well as a blood specimen for serology from all patients with clinical features compatible with measles. RT-PCR is available at CDC, at many state public health laboratories, and through the APHL/CDC Vaccine Preventable Disease Reference Centers.

Manage	In coordination with local or state health departments, provide appropriate measles post-exposure prophylaxis (PEP) to close contacts without evidence of immunity, either MMR or immunoglobulin. The choice of PEP is based on elapsed time from exposure or medical contraindications to vaccination.
Vaccinate	Make sure all your patients are up-to-date on measles vaccine, especially before international travel. People 6 months of age or older who will be traveling internationally should be protected against measles.

CDC Respiratory Virus Guidance

Each year, respiratory viruses are responsible for millions of illnesses and thousands of hospitalizations and deaths in the United States. CDC has updated isolation requirements for patients with COVID-19 for non-healthcare workers. Full Access: CDC

US Respiratory Virus Levels Remain High as Flu Rises in Central States

Respiratory illness levels in the United States remained high but stable last week, with flu activity rising in some regions of the country and indicators declining for both COVID-19 and respiratory syncytial virus (RSV). **Full Access:** CIDRAP

Measles Outbreak Threatens US Status Of 'Eliminating' Virus

The rash of measles outbreaks around the country has sparked concerns that the U.S. risks losing its status as a country where the disease has been eliminated, a distinction held since 2000. The CDC recommends people without immunity to measles should isolate after potential exposure for 21 days. **Full Access:** The Hill

Can AI Tool Improve Dx of Ear Infections?

Out of an original pool of 1561 videos, experts identified acute otitis media in 305 videos (26.5%) and no acute otitis media in 846 videos (73.5%). The tool achieved a sensitivity of 93.8% and specificity of 93.5%, with bulging of the tympanic membrane being the most indicative feature of acute otitis media, present in 100% of diagnosed cases, according to the researchers. **Full Access:** Medscape

A New Diagnostic Tool for Gonorrhea?

In preliminary studies, a novel point-of-care assay for detecting Neisseria gonorrhoeae accurately identified the infection in 96.5% of symptomatic men and 95.5% of symptomatic women. This test may provide an important addition to the toolbox for diagnosing STIs in the resource-limited settings. **Full Access:** NEJM

SMART Initiation Low Among PCPs, But Several Interventions Could Improve Adoption

In this study, presented at the American Academy of Allergy, Asthma & Immunology Annual Meeting, only seven out of 22 children initiated on SMART were initiated by a PCP.

The researcher concluded that single maintenance and reliever therapy was not commonly initiated by primary care providers, although PCPs supported efforts to improve adoption. Full Access: Helio.

Urgent Updates in Pediatrics

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Fatigue Recovery Following Concussion

Fabiano F, Takagi M, Anderson N, et. al. . Br J Sports Med 2024;58:59–65. doi:10.1136/bjsports-2023-106894

This was a single site, prospective, longitudinal study of children and adolescents presenting to the emergency department (ED) of a tertiary pediatric hospital with a diagnosis of concussion. Participants completed assessments in the form of questionnaires over five time points: T0: during the initial ED admission; T1: 2–4 days post injury; T2: 2 weeks; T3: 1 month and T4: 3 months post injury.

The authors found that in the 240 children reviewed, fatigue decreased between the acute post-injury stage to 3 months following concussion. There was a linear decrease in fatigue symptoms over the course of time with most children gaining energy levels up to pre-injury states at the 3-month mark. Child behavior issues were the most significant factor when related to fatigue when incorporated in a regression with other covariates.

The setting of the study may limit its generalizability as most patients with head injuries do not present to the ED. It does highlight the need for us as UC clinicians to consider mental health history and issues when assessing children presenting with head injuries.

Full Access: BMJ

Shorter Courses of Antibiotic Treatment

Vernacchio L, Hatoun J, Patane LB, et al. Improving Short Course Treatment of Pediatric Infections: A Randomized Quality Improvement Trial. Pediatrics. 2024;153(2):e2023063691

Antibiotic stewardship is one of the important areas of prescribing within Urgent Care Centers. This was a site-randomized quality improvement trial to test the effectiveness of education with performance feedback and clinical decision support (CDS) delivered at the point of care or a combination of both in improving prescribing habits. The study was set within an independent practice association of 76 privately-owned pediatric practices with 500 pediatric primary care clinicians (PCC; generally, physicians and pediatric nurse practitioners) affiliated with Boston Children's Hospital. The practices were randomly assigned in blocks of 4 to 1 of 4 intervention groups using a random number generator: education and feedback; CDS; both education and feedback plus CDS (combined group); and control (no intervention). Prescribing of antibiotics for soft tissue skin infection and pneumonia were studied.

The authors found the combination of the 2 techniques was substantially more effective than either approach alone. There was a 23% to 26% absolute improvement in the groups assigned to education with performance feedback alone or to CDS alone, and with 42% absolute improvement in the group assigned to the combination of the 2 intervention strategies.

This study was limited by its setting, which was in a large practice group whose clinicians were accustomed with QI projects. However, there it does highlight the benefits of ongoing education and support for clinicians, to improve prescribing practices.

Full Access: AAP

Trends of RSV Infection Severity in Children Pre- and Post- Covid

Garcia-Maurino C, Brenes-Chacón H, Halabi K, et. al. Trends in Age and Disease Severity in Children Hospitalized with RSV Infection Before and During the COVID-19 Pandemic JAMA Pediatric. 2024 Feb 1;178(2):195-197. doi: 10.1001/jamapediatrics.2023.5431

The authors of this cohort study analyzed hospitalization trends and disease severity in children younger than 5 years with RSV infection at Nationwide Children's Hospital, Ohio, during 8 RSV seasons. These included 6 pre-pandemic years, 1 pandemic year and 1 post-pandemic year.

They found the proportion of hospitalized children younger than 5 years with RSV infection increased significantly after 2020, the first year of the COVID-19 pandemic. Disease severity parameters were increased in infants younger than 12 months during pandemic and post-pandemic seasons vs pre-pandemic years. Analyses stratified by age showed that disease severity gradually increased from pre-pandemic to 2021 and 2022 to 2023 and was significant among the groups aged younger than 6months and 6months to younger than 12 months.

This study highlights the severity of RSV infections in younger children that are presenting, and, therefore, vigilance is needed during consultations in the Urgent Care to ensure appropriate referrals and discharge safety-netting are given.

Full Access: JAMA

Email your clinical questions to the Editors:
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Cause for Applause Q4 2023—The College's Newest Fellows

We would like to welcome the following new fellow of the College of Urgent Care Medicine! Fellows represent the best of us who work every day to provide the highest quality of medicine and advance the specialty of Urgent Care Medicine. The following clinician applied and earned the distinction of Fellow in the College of Urgent Care Medicine since our last announcement in December 2023.

Jackie McDevitt-Capetola, PA-C, FCUCM

Do you want to be recognized? Requirements to become a fellow include actively practicing as a physician, PA, or NP with a solid foundation in Urgent Care and being an active member of CUCM for at least one year. Further requirements can be found here. Those who achieve fellowship status will be entitled to use the initials FCUCM for as long as they remain members of the College.

CONTINUING MEDICAL EDUCATION (CME)

Target Audience

This CME activity is intended for medical professionals who practice medicine in on-demand space, including Urgent Care, retail medicine, and other similar venues. These providers may include physicians, nurse practitioners, and physician assistants.

Designation Statement

The Urgent Care Association (UCA) designates this enduring material activity for a maximum of 3 AMA PRA Category 1 Credit(s)™. Physicians should claim credits only commensurate with the extent of their participation in the activity. Credits may be claimed for one year from the date of release of this issue.

CME Objectives

1. Provide updates on the diagnosis and treatment of clinical conditions commonly managed by on-demand clinicians
2. Alert on-demand providers to potential unusual cases that may present to them
3. Utilize tips and tricks to improve patient care in the on-demand space

Accreditation Statement

This activity has been planned and implemented in accordance with the accreditation requirement and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of the Urgent Care Association and the College of Urgent Care Medicine. UCA is accredited by the ACCME to provide continuing medical education for physicians.

CME Credit Instructions

Once you have read the article, please log into your UCA profile. Once you are logged in go to Learn-> CME->Request CME. Complete the survey with the requested information for Urgent Caring. Your certificate will then be emailed to you within 3-5 business days. Please email learning@urgentcareassociation.org with questions.

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Medical practice and knowledge are constantly evolving and changing. This information is peer-reviewed but should not be your only source. Clinicians should use discretion when applying knowledge to any individual patient.

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TWENTY QUESTIONS (AND ANSWERS) ABOUT SALTER HARRIS 1 FRACTURE MANAGEMENT was contributed by EM RAP and UC MAX

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Bumped and Broken Coccyx Injuries was brought to you by Hippo Education

For more information on coccyx pain and some fun coccyx factoids, listen to Bumped and Broken Bottoms: Coccyx Injuries on January's Urgent Care RAP podcast.

EMPOWERING PHYSICIANS WITH EVIDENCE-BASED CONTENT

Excerpted from: Nedved A. Pediatric Community-acquired pneumonia: diagnosis and management in the Urgent Care setting. *Evidence-Based Urgent Care*. 2024 January;3(1):1-27. Reprinted with permission of EB Medicine.

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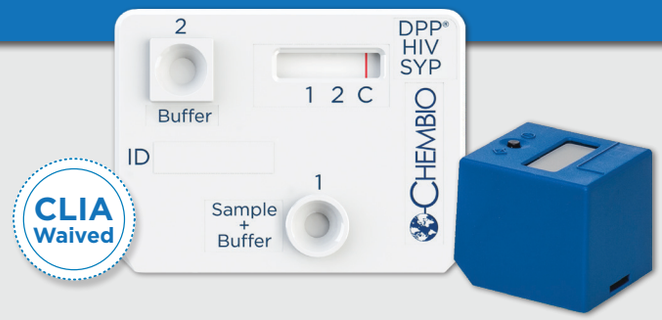

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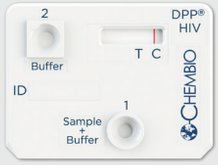

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