University of Cincinnati Medical Center - Department of Emergency Medicine

Annals of B Pod

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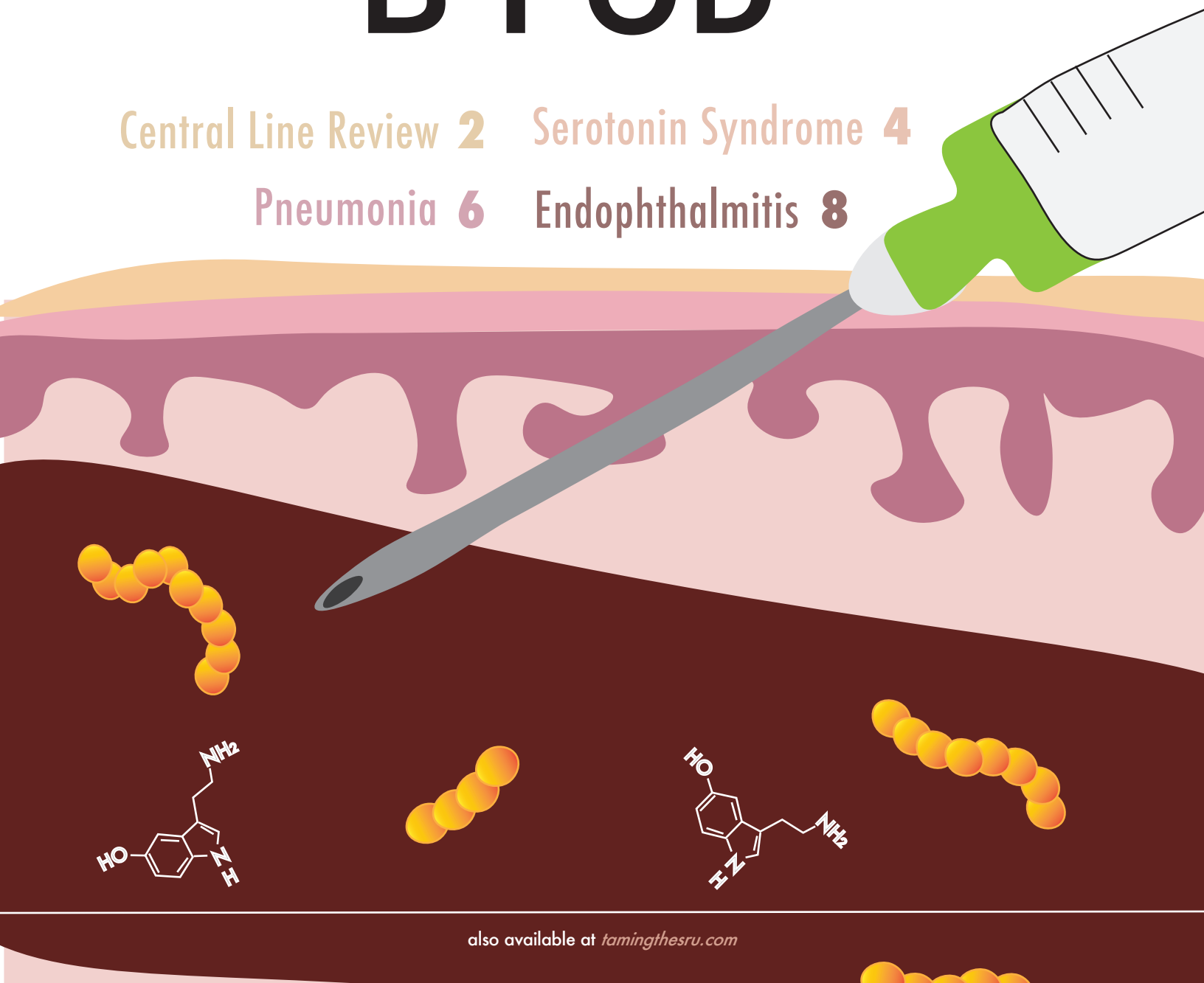
Accessing B Pod



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UC Emergency Medicine
Annals of B Pod
Spring 2017

The topics covered in this Spring's installment of Annals of B Pod highlight the variety of medical knowledge and procedural skills used daily in the Emergency Department. In B Pod, there may be a patient with an intentional ingestion brought in by EMS next door to a patient with a cough who is discovered to have pneumonia, while on the other side of the pod there is a patient complaining of eye pain. All of these patients represent common chief complaints with broad differentials that interns learn how to work up, manage, and treat. Spring also marks a transitional time, when interns start stepping up to into the junior resident role. With this transition, interns broaden their procedural skill set by learning how to place central lines, perform intubations, and insert chest tubes. As the year progresses, interns see more pathology, learn about new disease processes, and acquire procedural skills.

Central Venous Catheters

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From a MAC introducer to a single-lumen trauma catheter, there are countless varieties of central venous catheters available to the Emergency Physician. Prior to insertion of a central line, providers should carefully consider the indication for the procedure. Whether it is administration of medications that cannot be given peripherally or rapid fluid resuscitation, the indication for this procedure can help dictate the type of line that should be placed.

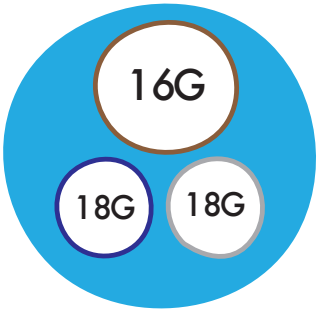
One important factor that must be considered when selecting an appropriate line is the size of the lumen(s). Traditionally, catheters are sized based on their outer luminal diameter. By convention, multi-lumen and very large catheters are measured with the French system, whereas single lumen catheters are generally measured by gauge. The French unit is the outer diameter of the catheter in millimeters multiplied by three. For example, a one millimeter catheter would be a 3 French. Consequently, increasing the French corresponds to an increase in the size of the catheter. In contrary, the gauge of a catheter is inversely proportional to the catheter size, making a 16 gauge larger than a 24 gauge for example. The exact difference between each catheter gauge was derived from wire manufacturing in the 19th century with no true mathematical formula to predict the difference in each gauge.

While sizing is based on the outer diameter of the catheter, the thickness of each catheter can differ, making the inner diameter variable. This leads to difficulty in predicting reliable flow rates for each line. Nonetheless, there are a few basic principles that can help guide providers. First, flow is faster through larger diameter catheters. Second, shorter catheter lengths correspond to higher flow rates. Finally, flow through a catheter is quickest if the lumen is parallel to the direction of flow (in contrast, for example, to the 90-degree angle seen in the introducer, Figure 1D).

Ultimately, selection of the appropriate line varies based upon the clinical presentation of the patient and the indication for the procedure. An understanding of the available options in the Emergency Department will assist providers in selecting the optimal catheter for the desired function.

Triple Lumen CVC

Available in the PAR units along the supply wall in the SRU, this is a triple lumen catheter (TLC) (Figure 1A). The central port projects to the distal end of the catheter and is a 16-gauge lumen. It also has two smaller 18-gauge lumens which each project as side ports just proximal to the end of the eight-inch-long catheter. Ideal for patients requiring multiple medications, this is the most commonly placed catheter in the Emergency Department. It is the smallest external diameter (7 French), anecdotally making it the least painful to insert. This particular catheter has an external antimicrobial layer of chlorhexidine acetate and silver sulfadiazine in addition to internal chlorhexidine, which has shown antimicrobial activity against several line-related infectious organisms. Because of this however, its placement is relatively contraindicated in patients with sulfa allergies as it can result in an adverse reaction to the sulfadiazine in these compounds.



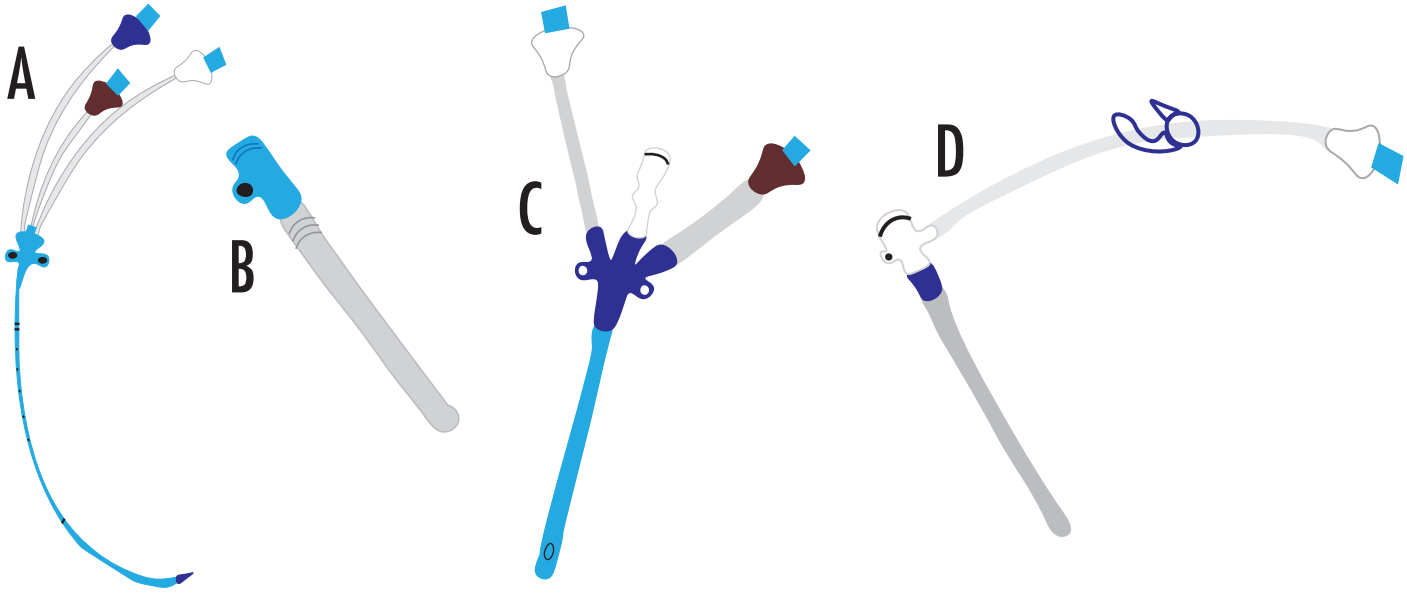


Figure 1. Representative images of various types of central venous catheters. A. TLC B. Trauma Catheter C. MAC D. Introducer catheter

Trauma Catheter

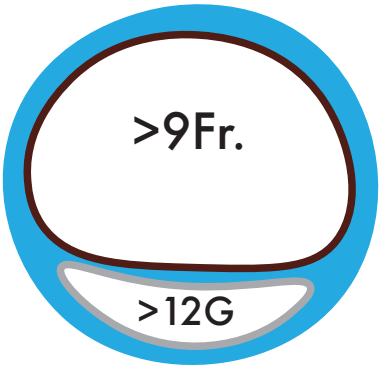
This single-lumen catheter can be found in the shelves at the foot of the beds in SRU 1 and 3 (Figure 1B). This 8.5 French catheter is only 3.5 inches long, making it an ideal lumen for patients requiring rapid administration of large volumes of fluids or blood, although this may be replaced by the MAC described below.

Single Lumen Infusion Catheter

Known as a SLIC catheter, this is a single 7 French lumen which has been designed to be inserted in an introducer sheath. It allows for the addition of an extra infusion site in patients with an introducer in place. Traditionally, these catheters also have the capability to monitor central venous pressure as well. They can be found in the OR or in the cath lab.

Multi-Lumen Access Catheter

Also known as the MAC catheter, this double-lumen catheter has an introducer sheath in addition to a 9 French and 12-gauge line (Figure 1C). The 9 French lumen allows for rapid infusion and aggressive volume resuscitation while the additional 12-gauge side port allows for co-administration of other medications. It is an ideal line for unstable cardiac patients, as another catheter—a Swan-Ganz, a transvenous pacer, a SLIC or even a TLC—can be placed through the introducer while maintaining the two other infusion ports. It has the largest external diameter at 14 French, so generous administration of lidocaine prior to placement is often beneficial. The kit can be found at the bottom of the PAR unit in SRU 1. This line also has the chlorhexidine antimicrobial properties of the TLC, so use caution in patients with hypersensitivities to sulfa drugs.



Introducers

An introducer sheath (*e.g.*, a Cordis) is a single-lumen catheter with a hub on the proximal end that is covered by a one-way valve. (Figure 1D) This hub allows for the insertion of various other catheters or invasive monitoring devices such as Swan-Ganz catheters, transvenous pacemakers and central venous pressure monitors. Most introducers also contain an 8 French side port through which large volume infusions can be administered. These can be found in the PAR unit in SRUs 1 and 3.

Indication	Recommended Line
Administration of medications that cannot be given peripherally	TLC
Administration of inotropes	MAC (for potential Swan)
Rapid fluid resuscitation	Trauma Catheter or MAC
Insertion of transvenous pacemaker	Introducer or MAC
Insertion of Swan Ganz catheter	Introducer or MAC
CVP monitoring	TLC or Introducer (+SLIC) or MAC (+SLIC)
Administration of multiple medications	TLC or MAC (+TLC)

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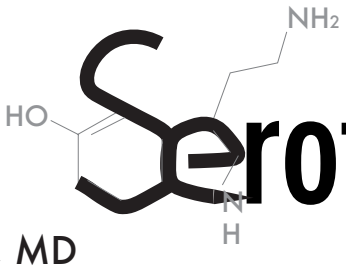

Serotonin Syndrome

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History of Present Illness

The patient is a female in her 20s with a past medical history of depression who presents to the Emergency Department via EMS after an overdose. EMS reports that the patient's friends called 911 after she ingested approximately thirty 150 mg venlafaxine extended release tablets along with alcohol in a suicide attempt approximately four hours prior to presentation.

En route to the hospital, EMS reported one episode of emesis as well as tachycardia in the 150s with otherwise normal vital signs. The patient confirms that she intentionally overdosed in a suicide attempt, but immediately regretted this decision and began to force herself to vomit, regurgitating approximately 10 pills. She states that she has had several stressors including the demands of being a college student as well as a sexual assault one week ago. She currently reports blurred vision as well as anxiety, but denies coingestion of any other substances, chest pain, shortness of breath, or abdominal pain.

Past Medical History

Depression

Medications

venlafaxine XR 150 mg

Vitals

T 38.0 HR 167 BP 106/85 RR 24 SpO2 98% on RA

Physical Exam

The patient is evaluated on the EMS stretcher and appears nontoxic with dried vomitus around her mouth. Cardiovascular exam is significant for marked tachycardia with no murmurs, rubs, or gallops appreciated. Her pulmonary exam shows moderate tachypnea with clear breath sounds bilaterally. Abdomen is soft and non tender. She displays frequent myoclonic jerking movements of the bilateral upper extremities. Her pupils are equally round and reactive and she is alert and oriented to person, place, and time. She is grossly moving all four extremities with 3+ knee and ankle reflexes bilaterally and myoclonus of the bilateral ankles.

Labs & Diagnostics

WBC 27.0 Lactate 20.0
Na 139 K 4.0 Cl 101 CO2 9 BUN 11 Cr 1.49
VBG 7.07 / 38 / 46 / BE -17.6
Acetaminophen/salicylate levels negative

EKG: sinus tachycardia

Hospital Course

As the patient was being transferred from the EMS stretcher to her bed, she exhibited a generalized tonic clonic seizure. She was given 2 mg of lorazepam IM, and the seizure resolved after approximately 75 seconds. She was postictal for approximately 10 minutes before returning to baseline. Labs obtained after she seized revealed an anion gap metabolic acidosis likely secondary to lactic acidosis, as well as significant leukocytosis. Her physical exam was notable for a low grade temperature, tachycardia and myoclonus on exam, consistent with serotonin syndrome. Poison control was contacted, and recommended supportive therapy, as well as cyproheptadine. She was given an initial dose of 12 mg of cyproheptadine orally and was admitted to the MICU.

Approximately six hours after admission, the patient had another generalized tonic clonic seizure that was complicated by aspiration. She was subsequently intubated for hypoxia and airway protection. She went onto develop severe ARDS requiring paralysis and proning. Her course was also complicated by acute renal failure secondary to rhabdomyolysis requiring hemodialysis. On hospital day 14 she was extubated. She spent several additional days in the hospital before being transferred to an inpatient psychiatric facility on hospital day 22.

Discussion

Serotonin syndrome is a condition caused by increased serotonergic activity in the central nervous system. Patients may present with a broad range of symptoms such as anxiety and tremor in mild cases, or altered mental status, cardiovascular compromise, and seizures in severe cases.[1] Mild serotonin syndrome can occur as a side effect of normal medication use for depression or anxiety. Moderate to severe cases, however, are often due to medication interactions or intentional overdose, and can quickly become life threatening, making recognition and prompt treatment of this condition important for the Emergency Physician.

Serotonin is a neurotransmitter found in the central and autonomic nervous system. It is a derivative of tryptophan, and depression has been associated with decreased intra-synaptic levels of serotonin. Selective serotonin reuptake inhibitors (SSRIs) and serotonin norepinephrine reuptake inhibitors (SNRIs) decrease pre-synaptic reuptake of serotonin, increasing the intra-synaptic serotonin concentration.[4] They are primarily used for depression and anxiety disorders. These medications generally have a very

benign side effect profile, with drowsiness, weight gain, and sexual dysfunction being the most common symptoms. However, when taken in large amounts such as in an overdose or when used in combination with other serotonergic medications, especially monoamine oxidase inhibitors (MAOIs) which inhibit the degradation of serotonin, SSRI use can lead to serotonin syndrome.[10]

SSRIs include medications such as citalopram, escitalopram, paroxetine, fluoxetine and sertraline, while SNRIs include the drugs duloxetine and venlafaxine. Other commonly prescribed drugs with significant serotonergic activity include tricyclic antidepressants, trazodone, linezolid, buspirone, dextromethorphan, fentanyl, lithium, tramadol, meperidine, and metoclopramide. Recreational drug use can also lead to significant serotonin toxicity, with the most common culprits being cocaine, amphetamines, and MDMA.

Overall, according to the National Poison Center Data System, a total of 2.2 million potentially toxic exposures to serotonergic medications were reported in 2013, resulting in over two thousand fatalities.[7] Less than 5% of these exposures came from antidepressant medications, and the majority of patients had minimal to no symptoms. However, 1% of SSRI overdoses resulted in life-threatening toxicity. As seen in this patient, symptoms can progress quickly.[1] Venlafaxine, the medication involved in this case, is associated with the highest mortality rate of all SSRI/SNRI overdoses.[8]

There is no specific test for serotonin syndrome or toxicity, making this a clinical diagnosis requiring a high level of provider suspicion. A history of serotonergic drug use in combination with signs of sympathetic nervous system activation (e.g., tachycardia, hyperthermia, diaphoresis, mydriasis) and CNS hypersensitivity (most commonly deep tendon hyperreflexia and myoclonus) suggests the diagnosis of serotonin syndrome.[5]

The clinical presentation of serotonin syndrome has significant overlap with other toxidromes, and therefore obtaining a thorough medication history from the patient or family and friends is very helpful in making the diagnosis.[4] However, obtaining a complete medication list may not be possible in the Emergency Department, making the physical exam of the utmost importance. Patients will often present with spontaneous or inducible myoclonus, and may also exhibit tremor, hyperreflexia, ocular clonus or fever. A provider can test for inducible clonus at the bedside by quickly dorsiflexing the ankle and holding the foot in dorsiflexion. If several beats of myoclonic jerking can be felt or observed, this increases the likelihood of serotonin syndrome.

As this condition can often present subtly and progress rapidly, clinical evaluation tools are available to aide in the diagnosis of serotonin syndrome. The most current tool is the Hunter Serotonin Toxicity Criteria (Table 1), which is based on a retrospective analysis of prospective data collected from over 2000 patients who presented with an overdose of a serotonergic drug.[2] This tool was 84% sensitive and 97% specific for the diagnosis of serotonin syndrome. The decision tool requires that the patient has taken a serotonergic medication and exhibits one or several physical findings as described above.

This combination of signs and symptoms allows the clinician to readily differentiate serotonin syndrome from other similar conditions such as neuroleptic malignant syndrome, anticholinergic toxicity, and sympathomimetic toxicity. Patients with neuroleptic malignant syndrome will not exhibit clonus and will instead present with muscle rigidity.[6] Anticholinergic toxicity may appear similar to serotonin syndrome and should remain on the differential of a patient that presents with seizures after an overdose. However, these patients will classically have dry skin as opposed to the diaphoresis seen in serotonin syndrome and sympathomimetic toxicity. Serotonin syndrome and sympathomimetic toxicity can present similarly, but can be differentiated based on the presence of myoclonus.

While laboratory evaluation does not confirm the diagnosis, several abnormalities can be seen in cases of severe serotonin syndrome. These patients may have a severe metabolic acidosis secondary to lactic acid production, as well as leukocytosis and elevated creatinine kinase levels. In severe, life-threatening cases, serious laboratory derangements can develop, including disseminated intravascular coagulation and renal failure secondary to rhabdomyolysis.[5]

Hunter Serotonin Toxicity Criteria

Patient has taken serotonergic agent AND meets one of the following criteria:

- Spontaneous clonus
- Inducible clonus + [agitation or diaphoresis]
- Ocular clonus + [agitation or diaphoresis]
- Tremor + hyperreflexia
- Hypertonia + temperature > 38 C + [ocular clonus or inducible clonus]

Table 1: The Hunter Serotonin Toxicity Criteria. has an 84% sensitivity and 97% specificity for diagnosing serotonin syndrome.

Management of serotonin syndrome is primarily supportive care, with fluid resuscitation and benzodiazepines as the mainstays of treatment. In mild cases, simply withdrawing the offending agent and treating with benzodiazepines for symptom control is adequate. If the patient is severely agitated, chemical sedation with benzodiazepines is the recommended treatment. Choice of benzodiazepine is often determined by provider preference. However, if the patient is actively seizing, a benzodiazepine with rapid onset such as 5-10 mg midazolam or 1-2 mg lorazepam should be chosen initially. Once the seizure has resolved, longer acting benzodiazepines can be given to reduce dosing frequency. Physical restraints should be avoided as this can contribute to rhabdomyolysis. Antipsychotics such as haloperidol should also be avoided, as these inhibit sweating and can worsen hyperthermia.[9]

In severe cases of serotonin syndrome that do not respond to supportive measures, cyproheptadine can be administered.[3] This antidote is primarily an H1 anti-histaminergic medication, but also has both anticholinergic and

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CAP, HAP or VAP?

Making sense of pneumonia acronyms

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History of Present Illness

The patient is a 50 year old male with a history of diet-controlled diabetes who presented to the Emergency Department from work with chest pain, cough and shortness of breath. He describes the chest pain as anterior, non-radiating and throbbing, and states that it started early this morning. The patient states later in the day he noted the development of diaphoresis and shortness of breath, which he described as worse with exertion. He also reported a non-productive cough since waking this morning. He took ibuprofen at home without any relief. He denied fevers, nausea, vomiting, abdominal pain, changes in vision, syncope, and palpitations. He denies tobacco use.

Past Medical History

Diabetes Mellitus
(diet controlled)

Medications

None

Vitals

T 36.1 HR 93 BP 158/89 RR 16 SpO2 96% on RA

Physical Exam

The patient appeared in no acute distress. His lung sounds were clear to auscultation bilaterally with good air entry, and was without wheezes, rales, rhonchi or any signs of respiratory distress. His cardiovascular exam revealed a regular rate and rhythm without murmurs or rubs. He was noted to have tenderness to palpation over his anterior his left chest wall and epigastrium. His abdominal exam was benign, and his skin exam did not reveal any rashes. His neurologic exam revealed no gross deficits.

Labs and Imaging

WBC 12.6 Na+ 135 K+ 3.6 BUN 12 troponin < 0.04 x 3

CXR (Image 1):
New opacification of the right middle lobe with findings suggesting volume expansion, as well as partial obscuring of the elevated right hemidiaphragm

CT Chest:
Elevated right hemidiaphragm with complete collapse of right middle lobe, as well as a patchy infiltrate left lower lobe with surrounding groundglass opacities, consistent with pneumonia

Hospital Course

The patient presented to the ED with chest pain, a new cough, diaphoresis and dyspnea on exertion. The initial work up revealed normal labs with the exception of a mild leukocytosis and normal serial troponins. An EKG was also normal.

However, his initial chest x-ray showed collapse of the right middle lobe of the lung. A CT of the chest was pursued for further characterization of this new x-ray finding, which revealed collapse of the right middle lobe and infiltrate in the right lower lobe, consistent with pneumonia. He was otherwise well-appearing and had no oxygen requirement. His CURB-65 score was 0. This patient was diagnosed with community-acquired pneumonia and was prescribed five days of levofloxacin. He was discharged and did not return to the ED.

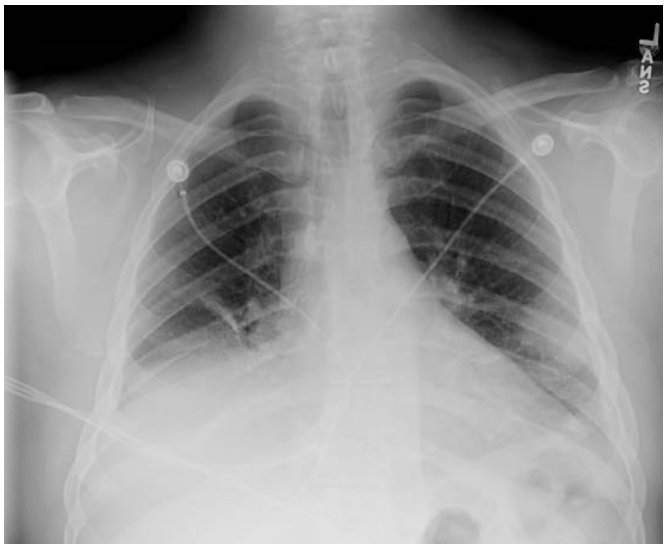

Image 1: Chest x-ray showing opacification of the right middle lobe, with partial obscuring of the right hemidiaphragm.

Discussion

Pneumonia is defined by the 2005 Infectious Diseases Society of America guidelines as "the presence of a new lung infiltrate plus clinical evidence that the infiltrate is of an infectious origin, which includes fever, purulent sputum, leukocytosis, and decline of oxygenation."[1] Affecting millions of Americans each year, the diagnosis carries significant morbidity and mortality, especially if the diagnosis is delayed. When combined with flu, pneumonia is the 7th leading cause of death worldwide and responsible for 1.2 million hospitalizations in the United States annually.[2] As pneumo-

nia represents a significant disease burden, IDSA and the American Thoracic Society (ATS) combined to formulate guidelines for the diagnosis and treatment of pneumonia to improve clinical outcomes. Self et al estimated that 2.2% of all ED visits are for pneumonia, representing 7-8 ED visits per 1000 persons annually.[2] As, such, timely identification of pneumonia and appropriate antibiotic choice is of critical importance for the emergency physician.

Traditionally, the discussion of pneumonia management has been delineated into two discrete patient populations with unique risk factors for specific pathogens, leading to the disease categories of community-acquired pneumonia (CAP) and healthcare-associated pneumonia (HCAP). However, these categories have recently changed, as have the recommendations for management of different patient populations diagnosed with pneumonia.

First suggested as a separate clinical entity in the 2005 Infectious Diseases Society of America and the American Thoracic Society (IDSA-ATS) guidelines, the term HCAP was created to identify patients at risk for multidrug resistant organisms (MDRO) and treat them with appropriate empiric antibiotics. Risk factors for HCAP included: hospitalization greater than 48 hours in the last 90 days; extended-care facility residents; patients undergoing home infusion therapy, home wound care or chronic dialysis within one month; and patients with a family member previously treated for a MDRO.[2]

However, a recent large meta-analysis[4] showed that these defined HCAP criteria did not predict which patients would ultimately be diagnosed with an MDRO. In fact, many of the HCAP patients were ultimately identified to have pathogens classically associated with community-acquired pneumonia, and many of the patients who ended up having MDRO organisms initially presented from the community.

Thus, in a recently-released set of IDSA-ATS guidelines focusing on nosocomial pneumonia, the concept of HCAP was abandoned in favor of the terms hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP).[5] A new set of CAP guidelines are expected to be released this year, and are anticipated to include guidance about patients presenting from the community who are at risk for MDROs.

CURB-65 Score

Confusion (1 point)
BUN >19 mg/dL (1 point)
Respiratory Rate > 30 (1 point)
Systolic Blood Pressure < 90 mmHg or Diastolic Blood Pressure < 60 mmHg (1 point)
Age > 65 (1 point)

Score 0-1: 1.5% mortality risk
Score 2: 9.2% mortality risk
Score 3 or greater: 22% mortality risk

Figure 2: CURB-65 Score for Pneumonia Severity.

Overall, the IDSA recommends that for all patients presenting with concern for pneumonia, empiric antibiotic choice and disease management should be based upon the individual patient's presentation, the severity of their illness, their unique risk factors for drug-resistent pathogens, and the local antibiogram and outcomes.

Community-acquired Pneumonia

Community-acquired pneumonia (CAP) is defined as pneumonia acquired from the community, as opposed to a nosocomial infection. It is a diagnosis made by clinical features such as cough, fever, sputum production, and chest pain, as well as physical exam findings such as rales or bronchial breath sounds.

However, like with the above patient, a new infiltrate on chest x-ray is considered the gold standard for diagnosis.[2] Cross-sectional imaging can also be useful when plain films are equivocal. While most guidelines recommend tailoring antibiotic choice to an identified pathogen, there is no consensus as to how best to obtain a microbiologic specimen, with recommendations ranging from relying on local antibiograms to obtaining sputum cultures, blood cultures and urinary antigens.[6] In general, patients who are being admitted to the hospital for CAP should at the minimum have a sputum culture obtained if feasible, but this is optional for patients managed in the outpatient setting.

Community-acquired pneumonia can be caused by a variety of pathogens, including viral etiologies. Of bacterial causes, *Streptococcus pneumoniae* is the most common, but may also include such other typical organisms like *Haemophilus influenzae*, *Staphylococcus aureus*, or group A strep. Classically "atypical" organisms include *Legionella* species, *Mycobacterium tuberculosis* or *C. pneumoniae*. Viruses include influenza, RSV, parainfluenza and adenoviruses.[6]

While many patients presenting with pneumonia from the community may be appropriate for outpatient management, some may require hospital admission and may even be critically ill. Multiple scoring systems exist to predict CAP severity and aid in disposition planning. CURB-65 and the pneumonia severity index (PSI) are two such tools. The CURB-65 score (Figure 2) uses five prognostic factors–confusion, BUN, elevated respiratory rate, blood pressure and age greater than 65–to predict mortality. In general, CURB-65 scores greater than 2 warrant hospital admission, and those with scores greater than 3 likely need ICU-level care. [7,8]

For patients who can be safely treated in the outpatient setting, the IDSA recommends that these patients receive a first dose of antibiotics in the ED. Treatment should last a minimum of 5 days. In terms of antibiotic choice, the guidelines recommend adhering to local antibiograms for resistance patterns, as well as individual patient risk factors for antibiotic-resistant pathogens (Table 1).[6,9,10]

In general, previously healthy patients without serious comorbidities can be treated with a macrolide such as azithromycin or doxycycline. Patients who have recent use of antibiotics (typically within the last three months) or significant medical comorbidities such as chronic lung disease, end-stage renal disease, heart failure, or diabetes can be treated with a respiratory fluoroquinolone such as levofloxacin or a macrolide with a beta-lactam such as high-dose amoxicillin or amoxicillin-clavulanate.

Continued on page 14

In areas with high

bpodcase

Endophthalmitis

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History of Present Illness

The patient is a female in her mid 30s who presented to the ED as a transfer from an outside hospital for severe left eye pain. The patient works as a welder. She reports that five days ago a metal foreign body struck her left eye. She removed the foreign body by wiping her eye. Since then, she reports worsening pain in the affected eye with associated loss of vision. She reports 10/10 pain and states that she cannot see anything out of the left eye. She is able to move the eye, but has pain with extraocular movements. She endorses fevers, chills, nausea, and headache.

Of note, the patient has a history of a motor vehicle accident in 2013 from which she sustained a left tripod fracture, left orbital blowout fracture with residual CN III palsy, traumatic mydriasis and enophthalmos.

Vitals

T 37.2 HR 118 BP 119/78 RR 21 SpO2 98% on RA

Physical Exam

On exam, this is a well-developed, well-nourished young female in mild distress. She has left-sided periorbital edema extending along the nasal bridge, left cheek, and inferiorly down the left side of the neck. She has left corneal clouding with a visible corneal ulcer that obliterates the visual axis as well as scleral and conjunctival injection and an appreciable hypopyon. Her visual acuity is light perception only in the left eye with a nonreactive pupil and intact extra-ocular movements with the exception of her known CN III palsy. She has no direct or consensual photophobia. Her right eye is within normal limits. She has an otherwise normal HEENT exam with a supple neck with normal range of motion. Her cardiovascular, respiratory, and abdominal exams are within normal limits.

Hospital Course

The patient underwent ophthalmologic evaluation in the Emergency Department due to concern for endophthalmitis. Their exam revealed no visual acuity in the affected eye with a normal intraocular pressure. She was also noted to have a nonreactive pupil without afferent pupillary defect, which was determined to be a stable finding compared to prior ophthalmology notes. She had slightly diminished extraocular movements compared to baseline. Corneal cultures were obtained.

The patient was admitted to the ICU for hourly antimicrobial eye drops and broad-spectrum IV antibiotics. In coordination with the Infectious Disease team, her regimen included vancomycin, bacitracin, tobramycin, voriconazole, and natamycin eye drops along with IV vancomycin and piperacillin-tazobactam. She received broad-spectrum antibiotics for 7 days, which were ultimately discontinued given negative blood cultures. Oral valtrex and fluconazole were added to her regimen as well as pred-forte eye drops. Anterior chamber and vitreous cultures were obtained and the patient received intracameral and intravitreal injections throughout her hospital stay. Cultures ultimately grew *Candida albicans*. Her course was complicated by nonadherence due to pain control issues.

The patient was discharged on hospital day 12 on multiple eye drops, as well as oral antifungals and antivirals. Unfortunately, the patient did not follow up with her outpatient appointments and re-presented to the ED one week after discharge with complete loss of vision in her left eye. At that time, she reported she had been using her eye drops but had not filled her diflucan prescription. She was readmitted, and subsequent ocular cultures ultimately grew gram positive rods and cocci. She underwent enucleation and was discharged on hospital day 5 on oral fluconazole. The patient has not since followed up and her current condition is unknown.

Discussion

This patient presented with endophthalmitis after a penetrating ocular trauma by a metal foreign body at her work. Endophthalmitis is a purulent inflammation of the intraocular fluids (vitreous and aqueous) due to bacterial or fungal infection. The most common etiologies are exogenous, including post procedural, after penetrating ocular trauma, or from extension of corneal infection, and endogenous via hematogenous spread.[1,2,3] It is an important distinction that while endophthalmitis may result from systemic infection, it is never the cause of bacteremia or fungemia. The most common cause of endophthalmitis is acute post-cataract surgery endophthalmitis; however, it is a rare complication, occurring after 0.1% of all cataract surgeries, one of the most common eye operations performed worldwide.[3]

About 25-31% of all cases of infectious endophthalmitis are post-traumatic, as was seen in this patient. The risk of developing endophthalmitis after an open globe injury is between 0 and 12%, with rates as high as 35% when an intraocular foreign body is present.[2,4] According to the American Trauma registry, the incidence of endophthalmitis did not significantly differ among various types of foreign bodies, although some reviews have reported higher incidence of endophthalmitis with non-metallic intraocular foreign body (IOFB).[5,6]

Risk factors for developing endophthalmitis after ocular trauma include: retained IOFB; delay in wound closure of greater than 24 hours, which increases infection rates by a factor of four; in-

jury in a rural setting; and a ruptured lens capsule. With ruptured lens capsules, organisms have direct access to the vitreous cavity, and the clearance of the organisms is slowed, as the flow of aqueous humor out of the anterior chamber is impeded by the lens.[5] This group tends to have worse visual acuity outcomes than other groups with endophthalmitis.[2]

The most common presenting symptoms are eye pain, photophobia, vision loss, and ocular discharge. Almost all (~95%) patients report visual changes—usually blurry or decreased vision—but almost 25% of patients with endophthalmitis will not have pain on presentation. The presenting complaints and physical exam findings are very similar amongst the different etiologies of endophthalmitis, making an accurate history very important.

Key historic factors include: hammering steel; working with high speed machinery, grinders, weed wackers; and report of ocular trauma, as was the case with this patient.[1] Additionally, a history of ophthalmologic procedures (particularly cataract surgery or trabeculectomy), intravitreal injections, IV drug use, recent systemic infection, or recent travel might suggest non-traumatic etiologies of endophthalmitis.

Physical exam findings include erythema and swelling of the patient's eyelids, conjunctival and scleral injection, chemosis, hypopyon, and uveitis. About 80% of patients present with a red eye. Hypopyon, or a layer of inflammatory cells at the bottom of the anterior chamber, is present in 85% of patients (Figure 1).[2,3,7] Post-traumatic endophthalmitis can present a diagnostic challenge, as the the signs and symptoms of the initial injury can mask those of the infection. Patients with worsening vision, increasing pain, or evidence of vitritis in the setting of recent trauma should be considered to have an infectious component until proven otherwise.

Differential diagnosis includes occult retention of lens cortex or nucleus, hypopyon uveitis (Behcet's or rifabutin), blebitis, keratitis, vitreous hemorrhage, and toxic anterior segment syndrome (TASS). Therefore, in a patient with this presentation, ophthalmologic consultation is indicated.

The diagnosis is largely a clinical one, although it can be a difficult diagnosis to make even after ophthalmologic evaluation. Therefore, aqueous and vitreous cultures are important in elucidating the causative organism and for tailoring antibiotic therapy in refractory cases. Timely PCR analysis is emerging as a means of evaluating aqueous and vitreous fluid, as many cases (up to 38% in one large study) of clinical endophthalmitis are culture-negative. [8,4,9]

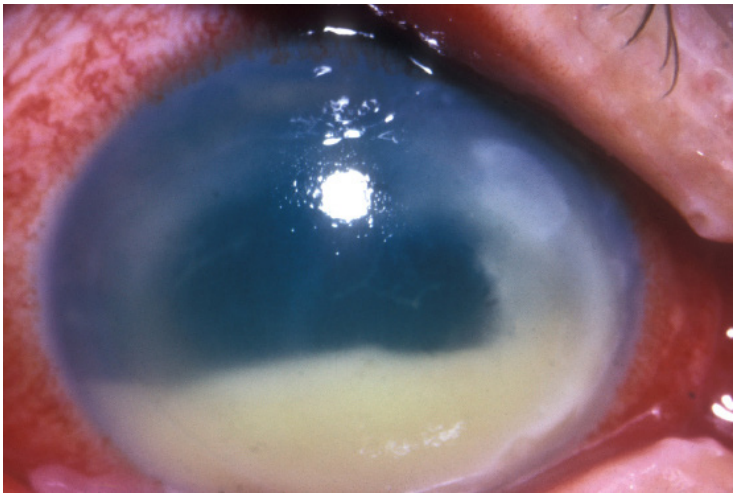

Figure 1: A representative photograph of a patient with endophthalmitis with a hypopyon. From Community Eye Health. www.flickr.com/photos/communityeyehealth/7608314920

Plain films may be helpful in identifying retained IOFB, but may miss more than half of IOFBs. CT may more reliably identify IOFB, but may have limited ability to identify materials such as wood, ceramics, and plastics, which are less radiopaque.

Ultrasonography facilitates assessment of the degree of vitreous opacification, presence of IOFB, status of the posterior hyaloid face, as well as detection of either choroidal or retinal detachment.[5] It is nonspecific, however, and can only demonstrate severity of posterior segment involvement and whether retinal detachment or abscess is present. Ultrasound evaluation is routinely performed when significant media opacification is present and the posterior segment cannot be visualized, which is common in these patients, as up to 79% presented with hazy media.[2,9] Of note, diagnostic quality of ultrasound is operator-dependent and may be compromised by the need to image through a closed lid with minimal pressure in the setting of an open globe.[5]

The most commonly identified organisms in endophthalmitis overall are gram positive agents, with coagulase negative staphylococci being the most common of these. Endogenous cases are frequently caused by *S. aureus*, gram negative bacilli, and candida. Candida is particularly common in IV drug users.[3] The most commonly isolated species in post-traumatic endophthalmitis are gram positive cocci followed by gram negative bacilli. Coagulase negative species, such as *S. epidermidis* and *S. saphrophyticus*, are the most common isolates in culture positive cases, although streptococcus is also common. *Bacillus subtilis* and *bacillus cereus* are other common culprits in post-traumatic endophthalmitis. Fungal endophthalmitis is less common, but often related to organic foreign bodies. Many cases of endophthalmitis are polymicrobial; interestingly, in one study, all of the culture positive polymicrobial cases were coinfected with gram negative bacilli and fungal entities.[3,8]

Endophthalmitis is a vision-threatening emergency, and antimicrobial treatment should be initiated in the Emergency Department. Recommended treatment regimens for post-traumatic endophthalmitis includes topical, subconjunctival, systemic, and intravitreal antimicrobials, in addition to primary closure of any open globe defects or removal of IOFBs, if applicable. As in the case of any endophthalmitis, many patients will need a vitrectomy, and some may require enucleation, as our patient did.

Typical regimens include topical vancomycin and ceftazidime every 1 hour, with addition of topical cycloplegics and steroids. Although there is mixed evidence for systemic antibiotics for treatment of certain types of endophthalmitis, generally they are recommended for post traumatic endophthalmitis, and always recommended for endogenous endophthalmitis. Typical regimens include systemic oral levofloxacin or vancomycin plus ceftazidime, with oral fluconazole or

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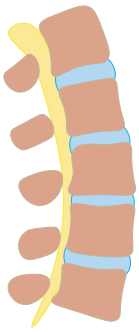
Blood Pressure Management In Acute Spinal Cord Injury



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Although the nomenclature surrouding "spinal cord injury" (SCI) is controversial, all lesions to the spinal cord, conus medullaris and cauda equina should be considered SCI.[1] Damage to the spinal cord may be traumatic (TSCI) or non-traumatic (NTSCI). TSCI can result from a variety of causes, including falls, motor vehicle accidents, occupational and sports-related injuries, and violence. Alternatively, NTSCI is typically secondary to an underlying pathology such as infection, tumor, musculoskeletal problems, or congenital diseases.[1]

Hemodynamic Dysfunction after SCI

Acute spinal cord injury (ASCI) has the potential to be a devastating, life-altering injury. In addition to the residual injury effects on activities of daily living, ASCI can also result in significant autonomic nervous system dysfunction.[2-6] An intact spinal cord is essential for autonomic control of various organs, including the heart and blood vessels. Both the location and completeness of an injury contribute to the severity of cardiovascular effects.

Cervical or high thoracic SCI (above T6) alters patients' ability to control supraspinal sympathetic cardiovascular functions including coronary perfusion, cardiac contractility, and heart rate. In this injury, parasympathetic responses through the vagus nerve are the main controllers of the heart, which leads to hypotension and bradyarrhythmias. This is also known as neurogenic shock.[2-6] Hypotension following ASCI has been associated with worse outcomes.[6] More specifically, reduced spinal cord perfusion resulting from hypotension significantly contributes to secondary ischemic injury and increased morbidity and mortality. In traumatic brain injury, a single episode of hypotension (SBP < 90 mmHg) has been associated with a 150% increase in mortality.[7] This data has been extrapolated to the ASCI population and several researchers have sought to prove that avoidance of hypotension and augmentation of MAP goals to 85-90 mmHg is associated with improved spinal cord perfusion and thus improved clinical outcomes (Table 1).

Vasopressor Considerations

First line therapy for neurogenic shock related to spinal cord injury is always fluid resuscitation. Only once euvolemia has been established should vasopressor therapy be considered. No studies to date have compared outcomes between vasopressor agents in this patient population. Norepinephrine, phenylephrine, and dopamine (Figure 1) are used most frequently for this purpose.

It should be noted that vasopressor utilization does not come without risks. Readdy and colleagues attempted to quantify and describe complications associated with vasopressor use.[17] They conducted a retrospective cohort study which included 34 patients with acute TSCI. Of the 34 patients, 91% received dopamine and 64.7% received phenylephrine to meet a MAP goal of >85 mmHg. Over half of patients required two vasopressors and the majority of patients experienced at least one complication. Most complications were cardiogenic in nature, including atrial fibrillation, tachycardia, ventricular arrhythmias, and bradycardia. In the entire cohort, there was a non-significant trend towards greater complications in the dopamine group. In a subgroup of patients > 55 years of age, all vasopressor complications were significantly higher than their younger counterparts, despite no differences in severity of injury and medical interventions.

Norepinephrine

Improves both peripheral vasoconstriction through alpha and inotropy via beta-1, augmenting both blood pressure and heart rate, making it an ideal agent in this setting.

Phenylephrine

100% alpha agonism, which increases MAP but does not aid and may even worsen instances of bradycardia. This may be less of a concern in high thoracic injuries as bradycardia is less of an issue.

Dopamine

High doses are necessary to obtain alpha agonism (> 10 mcg/kg/min). If a SCI patient has any signs/symptoms of cardiogenic shock, dopamine should be avoided as it has been linked to increased mortality.

Figure 1. Vasopressor therapies in ASCI.

To date, there is weak evidence to suggest that maintaining a mean arterial pressure > 85-90 mmHg directly correlates to improved functional outcomes following acute spinal cord injury. In reviewing the available literature, recommendations can be made to ensure euvolemia and avoid hypotension (SBP < 90 mmHg) immediately following ASCI. It should be noted that the duration of treatment was arbitrarily chosen by individual investigators and should be extrapolated with caution. Careful consideration and monitoring of sustained vasopressor use should be evaluated on a daily basis in patients with ASCI. Future studies are currently underway to clarify ideal MAP goals and length of therapy to guide clinicians in management of ASCI-induced hypotension.

Author (Year)	Type of Study	N	Methods & Intervention	Outcomes & Conclusions
Zach (1976)	Prospective Case Series	117	Prospective assessment; hemodynamic support with Dextran 40 x 7 days and dexamethasone x 10 days	Improved neurological outcome; better outcome for early referrals
Tator (1984)	Retrospective Cohort Control	144	Patients managed per ICU protocol using crystalloid and whole blood or plasma for resuscitation; compared to non-ICU historical cohort	Improved neurological outcome; decreased mortality and LOS with early transfer and ICU care; hard to differentiate if outcomes associated with MAP support or ventilator strategies
Wolf (1991)	Prospective Case Series	52	Bilateral jumped facet injuries received closed reduction within 4 hours, converted to open if unable to reduce; MAP > 85 x 5 days	Improved neurological outcome; only 52% follow-up and challenging to differentiate surgical vs. medical management interventions on outcome
Levi (1991)	Prospective Case Series	103	50 incomplete, 53 complete; invasive monitoring; MAP goal > 85; no control group	Improved neurological outcome; no difference between early vs late surgical intervention
Levi (1993)	Prospective Case Series	50	C-spine injury only; MAP > 90 using dopamine ± dobutamine; no control group	Improved neurological outcome at 6 weeks post-injury
Vale (1997)	Prospective Case Series	77	MAP > 85 with sequence: colloids, blood for HCT > 32, dopamine, norepinephrine; all patients had PA catheters; all treated with steroids; no control group	Improved neurological outcome with volume resuscitation and MAP augmentation, distinct from potential surgery benefit at 1 year follow-up
Cohn (2010)	Retrospective Case Series	17	C-spine injury, complete SCI only; recorded average MAP ranges over a specified time period	Hypotension is detrimental to neurologic recovery; did not assess if higher MAPs correlate to improved outcomes
Hawryluk (2015)	Retrospective Case Series	74	Average MAP and average time spent below certain MAP thresholds recorded; use dopamine, norepinephrine and/or phenylephrine for MAP > 85 x 5 days	Improved neurological outcome with MAP > 85 and those with the least episodes of HOTN but causative relationship cannot be concluded
Martin (2015)	Retrospective Cohort	105	Cervical and thoracic SCI; conducted at a regional SCI center; collected lowest and average MAP for first 72 hrs of hospitalization	More severe SCI was associated with worse ASIA-MS; episodes of HOTN and need for vasopressors not associated with improved ASIA-MS at discharge
Readdy (2016)	Retrospective Cohort	36	Penetrating SCI only (compared to blunt cohort); MAP > 85 x 7 days	Low rates of neurological recovery with penetrating SCI and thus no difference with intervention; high frequency of vasopressor-related complications

Table 1. Review of Primary Literature[7-16]. ICU: intensive care unit; LOS: length of stay; MAP: mean arterial pressure; hrs: hours; HCT: hematocrit; HOTN: hypotension; ASIA-MS: ASIA Motor Score

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caseweponder

A summary of the ongoing department conversation drilling to the bottom of real life cases and the EBM we can use to improve our clinical practice

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Safe discharge in Upper GI Bleed

Identification of patients to safely discharge from the ED is a challenge. Clinical scores like the Glasgow-Blatchford Score have been well-validated to identify a low risk population (Stanley et al 2009) with GBS score 0 who are are safe for discharge. There was discussion about whether increasing the 'safe' score to 1 or 2 was feasable. When evaluated in the literature, clinically significant outcomes are still caught with a sensitivity of 98% (Yaka et al 2015). Group consensus was that the patient's clinical stability is more important than the score. Can the patient ambulate without symptoms? Are they having active hematemesis or melena in the ED? Any of these and they buy themselves a trip upstairs.

The End of the Insulin Drip in DKA?

DKA is a spectrum; does it all need the blunt ICU-binding drip of insulin? Several studies have evaluated the use of short-acting lispro with equal efficacy, incidence of hypoglycemia and time to resolution. This could keep patients out of the ICU depending on your hospital's capability for glucose measurement every two hours. Engaging your medicine colleagues will be key for acceptance here. Also, there is likely a role for observation for mild DKA (anion gap <18) with clear etiology for the DKA, *e.g.* noncompliance.

New Onset Afib Undergoing ED Cardioversion = NOAC?

In patients with known new onset non-valvular atrial fibrillation undergoing cardioversion, what is the role for anticoagulation (AC)? Cardioversion is considered safe within 12 hours of known onset (Nuotio et al. 2014). AC therafter is based off echocardiographic data suggesting that the atria remains stunned after cardioversion and therefore may be prone to clot; however, there are no outcome-based or randomized trials. There is a wide practice pattern variation between practitioners and countries (Rogenstein et al 2012).

In conjunction with cardiology and the patient, current recommendations include the use of age > 65 and CHADSvsasc >1 as cutoffs for initiating AC after cardioversion (Stiell et al 2015).

The Death of Code Bicarb?

While a staple of cardiac arrest since 1970, the data does not support its routine use. It has been been a physiologic concern that the associated hypercarbia of NaHCO3 administration may worsening the already acidotic state of the patient (Velissaris et al 2016). While formal recommendations are lacking, the AHA has de-emphasized its use and NaHCO3 use is best reserved for patients with acidosis despite adequate respiratory compensation.

Role for PCC in GCS 15 ICH?

Previously reserved for the depressed GCS patient, PCCs have shown their efficacy in reducing hematoma expansion (Steiner et al 2016) and concensus is that they should be first line in warfarin reversal for intracranial hemorrhage. Limitations in the literature are the exclusion non-arterial and traumatic etiologies such as subdural hematoma, traumatic subarachnoid hemorrhage and epidural hematoma.

Endophthalmitis
Continued from page 9

voriconazole for confirmed or suspected fungal endophthalmitis. The frequency of eye drop administration often necessitates admission to an intensive care unit depending on local nursing protocols.

Subconjunctival antibiotics are controversial and may not be necessary. There is also some evidence that administration of subconjunctival antibiotics at the time of open globe repair may decrease postoperative infections. Intravitreal injections, the mainstay of endophthalmitis treatment, include a similar regimen: vancomycin plus ceftazidime or amikacin with optional voriconazole (in suspected fungal endophthalmitis) and/or dexamethasone (contraindicated in fungal endophthalmitis).[5] The duration of treatment for patients with endophthalmitis depends on the etiology, the pathogen and the patient's response to treatment. Although outcomes are patient-dependent, approximately half of patients will recover 20/40 vision. Unfortunately, up to ten percent of patients will have functional vision loss or progress to enucleation.[10]

While endophthalmitis is a relatively uncommon complication of traumatic eye injuries, eye trauma accounts for 3% of all Emergency Department visits. One of the most important aspects of assessing eye trauma is maintaining a high suspicion for open globe injury. Retained foreign bodies are associated with anywhere from 10 to 41% of open globe injuries and of those patients with identified IOFBs, up to 20% had painless presentations.[11] Endophthalmitis occurs after 3-10% of penetrating trauma to the globe, making the distinction between corneal abrasion and globe rupture integral to the initial physical exam in the ED.[2] Patients who present after injury from a metallic object are at higher risk for developing this condition when compared to patients with glass foreign bodies or blunt trauma, and delay to ED presentation is associated with poorer outcomes. Aggressive prophylactic antibiotics for these patients on their initial presentation is important. Choice of antibiotic regimen likely depends on the severity of the globe trauma, and may include oral or systemic antibiotics such as vancomycin and ceftazidime.

In summary, ED management of post-traumatic endophthalmitis includes maintaining a high suspicion in patients with concerning historical features, prompt recognition of open globe injuries, imaging to assess for IOFBs, and emergent ophthalmologic evaluation. Early broad-spectrum antimicrobial prophylaxis should be administered in suspected IOFB or open globe injuries to help prevent post-traumatic endophthalmitis. Finally, close ophthalmology follow-up should be ensured before these patients leave the hospital.

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Serotonin Syndrome
Continued from page 5

anti-serotonergic properties. An initial loading dose of 12 mg is followed by 2 mg every two hours until clinical improvement is demonstrated. Cyproheptadine is only available in oral form.

In severe cases, especially those in which the patient's temperature is greater than 40 degrees or where airway protection is a concern due to seizure activity, endotracheal intubation should be performed. Rapid sequence intubation is safe in these patients. However, ketamine should be avoided as an induction agent due to the serotonergic activity of this drug, and succinylcholine should also be avoided if evidence of rhabdomyolysis is present.[1] Cyproheptadine may be administered via NG or OG tube in patients that require intubation. Ultimately, patients exhibiting signs of moderate to severe serotonin syndrome require close observation in an intensive care unit due to the potential for rapid worsening of symptoms.

Overall, the vast majority of patients who present following a serotonergic drug overdose will have mild to no symptoms, and can often be discharged home after a short period of observation. However, severe serotonin syndrome can be a rapidly progressive disease with significant morbidity and mortality, and Emergency Physicians should have a high clinical suspicion for this disease in overdose patients. Quick screening tests for myoclonus and hyperreflexia can be performed at the bedside, allowing providers to quickly establish a management plan.

Patients can rapidly progress from generalized tremors to full tonic-clonic seizures with the development of serious end-organ damage including acute renal failure. The best treatment strategies for these patients is early and aggressive supportive care with benzodiazepines and fluid resuscitation, and clinicians should have a low threshold for definitive airway management and admission to the ICU.

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rates of macrolide resistant *S. pneumoniae,* the latter regimen is recommended for all patients regardless of comorbidities. This unfortunately encompasses many areas within the United States, so it is important for providers to be familiar with local resistance patterns. The guidelines also recommend prescribing oseltamivir/zanamivir for patients diagnosed with influenza A and whose symptoms have been present for less than 48 hours.[6]

Community Acquired Pneumonia	
Patient Population	**Outpatient Antibiotic Regimen**
Well-appearing patients with no comorbidities and no recent antibiotic use in an area without significant macrolide resistance	a macrolide[1] (*e.g.*, azithromycin 500 mg followed by 250 mg for four days; clarithromycin 500 mg BID) OR doxycycline[2] (100 mg BID)
Well-appearing patients with no comorbidities and no recent antibiotic use in an area with significant macrolide resistance	a beta-lactam (*e.g.*, amoxicillin 1 g TID; amoxicillin-clavulanate XR 2 g BID) plus a macrolide or doxycycline OR a respiratory fluoroquinolone (*e.g.*, levofloxacin 750 mg daily)
Well-appearing patients with significant medical comorbidities[4] or with recent antibiotic use[5]	a beta-lactam plus a macrolide or doxycycline OR a respiratory fluoroquinolone

1. Patients with a history of prolonged QTc interval should not receive macrolides. 2. Doxycycline is contraindicated for pregnant patients. 3. Fluoroquinolones may carry a higher risk for *Clostridium difficile* infection, and carry a black box warning for tendon rupture, although rare. 4. This includes COPD, DM, CHF, ESRD, alcoholism, liver failure, cancer or any other history of immunosuppression. Based on guidelines and recommendations from the ATS and IDSA. 5. Within the last 90 days.

Table1:Asummaryofempiricantibioticchoiceforhospital-acquiredorventilator-associatedpneumoniabasedonpatientriskfactorsandpresenting clinicalsymptoms.Basedonthe2016IDSAandATCGuidelinesforManagementofAdultswithHospital-acquiredandVentilator-associatedPneumonia. Modeled after flowchart from pulmccm.org.

For patients who are more ill and require admission to the hospital, guidelines recommend pursuing a similar antibiotic regimen as above with the addition of specifically-targeted antibiotics based on the patient's risk factors, including coverage for pseudomonas and MRSA as needed. This will often be based on the clinical gestalt of the treating physician.

Although the new IDSA-ATS recommendations for CAP have not yet been released, empiric antibiotic regimens will likely mirror the coverage for HAP and VAP as discussed below. Guidelines also recommend tailoring antibiotic therapy as soon as possible. This can be facilitated in the Emergency Department by sending blood cultures, rapid influenza tests, sputum cultures, tracheal aspirates and urinary antigens.

Unfortunately, the mortality of CAP has not significantly changed despite new antibiotics and guidelines since the advent of penicillin. Both the CDC and IDSA-ATS recommend influenza vaccine for all-comers and the pneumococcal vaccine for adults older than 65 years.[6,11] In the new era of growing pan-resistant organisms, it is likely that infection prevention will have greater effects on morbidity and mortality than infection treatment.

Hospital-associated and Ventilator-acquired Pneumonia

In late 2016, the IDSA released new guidelines for hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP), and did away with the concept of HCAP.[5] Hospital-acquired pneumonia is currently defined as a pneumonia not present at the time of admission that occurs 48 hours or more after entry to a hospital, while VAP is defined as pneumonia occurring 48 hours after endotracheal intubation.[5] Although more germane to inpatient management, these patients may be seen in the Emergency Department as individuals who present from long-term care facilities with ventilator dependence or who were recently discharged from a hospital.

The most recent IDSA-ATS guidelines stress choosing antibiotic coverage using the local antibiogram and blood and sputum cultures. However, if these are not readily known to the treating provider, empiric coverage should be initiated as discussed below and outlined in Figure 3. All patients who present to the Emergency Department with concern for HAP or VAP should be covered for common pathogens such as *staph aureus*, *pseudomonas aeruginosa* and gram-negative bacilli.[5]

Additional antibiotic choices are based on patient risk factors or clinical factors. In both HAP and VAP, the only data-proven risk factor for MDRO is the use of IV antibiotics in the past 90 days. However, other risk factors for MDRO include: septic shock upon presentation; acute respiratory distress syndrome (ARDS); the requirement for acute renal replacement therapy; 5 or more days of hospitalization prior to HAP/VAP development; and treatment in an ICU with known common MRDO isolates.[5]

Patients with these risk factors should be empirically covered for MRSA as well as pseudomonas. Even without these risk factors, patients should be empirically covered for MRSA in hospitals with 10-20% known rates of MRSA. Additionally, patients with significant underlying structural lung disease require additional pseudomonal coverage and MSSA coverage. The new guidelines support antibiotic treatment for at least seven days and to de-escalate as bacterial data becomes available.[5]

In sum, pneumonia is a challenging, multi-faceted disease process that requires early identification and appropriate antibiotic choice. Patients admitted to the hospital should be risk stratified based on individual risk factors and their presenting clinical symptoms. The Emergency Physician must be familiar with the local antibiogram to aid in choosing appropriate outpatient or inpatient medication regimens.

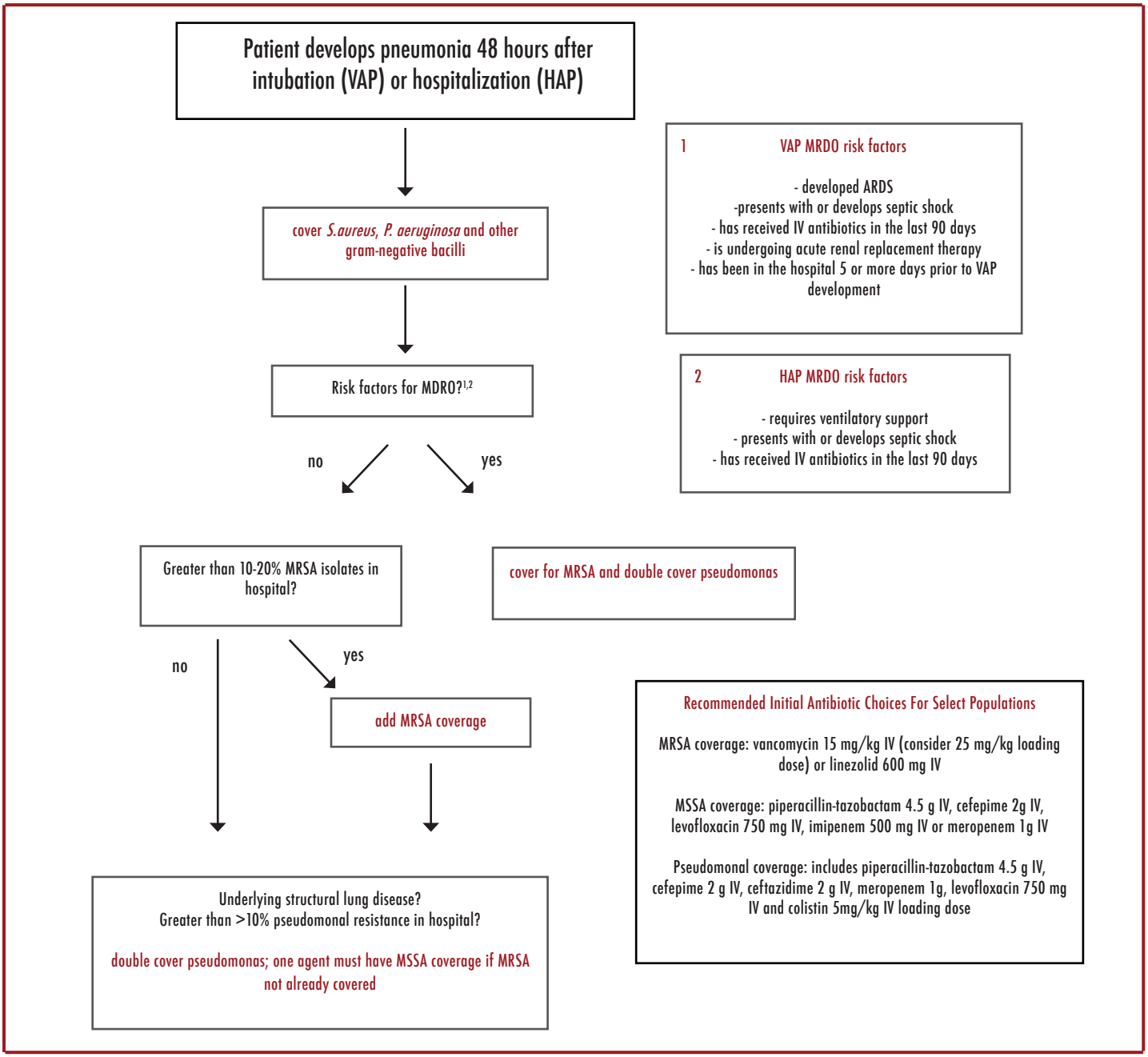

Figure 3: A summary of empiric antibiotic choice for hospital-acquired or ventilator-associated pneumonia based on patient risk factors and presenting clinical symptoms. Based on the 2016 IDSA and ATC Guidelines for Management of Adults with Hospital-acquired and Ventilator-associated Pneumonia. Modeled after flowchart from pulmccm.org. [12]

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Wolff-Parkinson-White EKG focus

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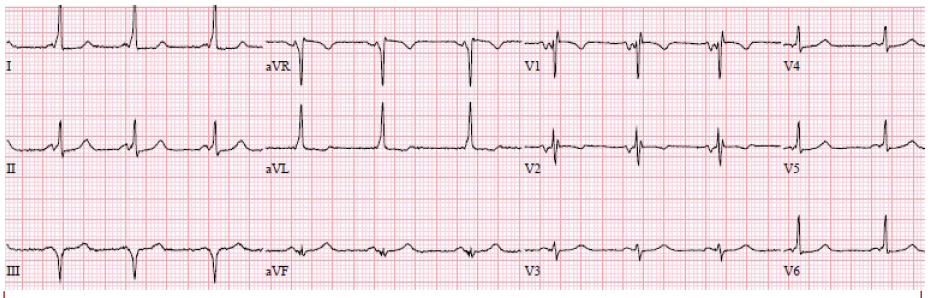


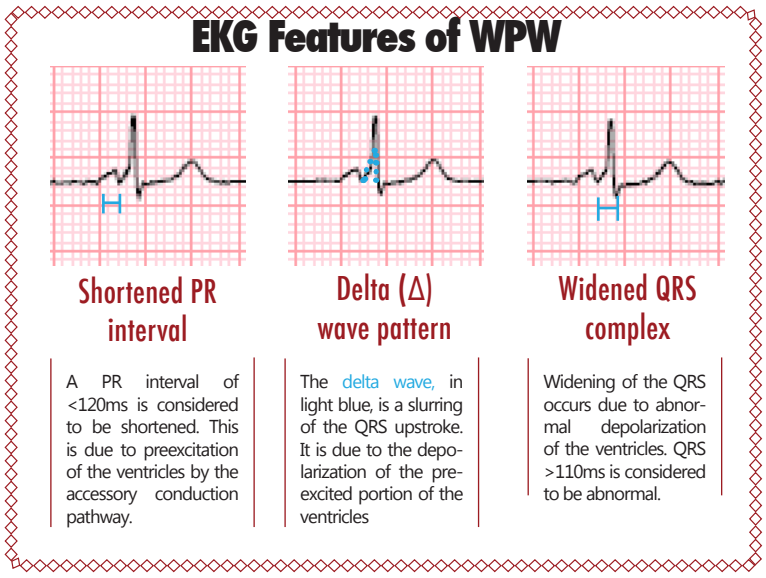
Figure 1. The patient's EKG showing features consistent with Wolff-Parkinson-White (WPW). This EKG shows a shortened PR interval, delta wave pattern, and a widened QRS complex.

History of Present Illness

The patient is a female in her 30s who presents with shortness of breath five days after delivering her second child. She has no significant past medical history and denies any chest pain or palpitations. The patient had an EKG which showed a Wolff-Parkinson-White (WPW) pattern. This was thought to be an incidental finding and because she was asymptomatic from her WPW, she did not receive any additional intervention.

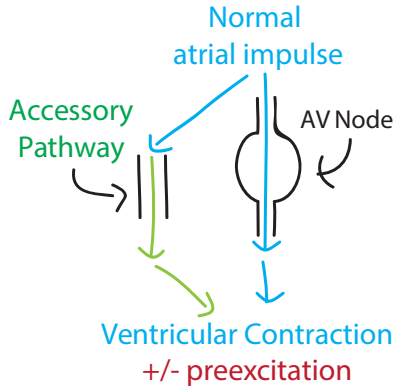
WPW Treatment

Asymptomatic patients, specifically those with no history of AVRT, syncope, chest pain, or palpitations, can follow up with cardiology or electrophysiology as an outpatient. Symptomatic patients should be admitted to the hospital and cardiology should be consulted.



WPW, Preexcitation & Accessory Pathways

Preexcitation occurs when there is depolarization of all or part of the ventricles earlier than expected from the normal AV node/His system, due to the presence of accessory conduction pathways (APs). APs are alternative electrical connections between the atrium and ventricle that can cause reentry tachycardias. These APs are normal in the fetal heart and then degenerate before birth, leaving the "typical" AV node/His system. However, in about 0.1-0.3% of the population, one or more APs persist and are usually capable of antegrade and retrograde conduction. WPW describes a preexcitation EKG pattern that is associated with AP related tachycardias, typically atrioventricular reentry tachycardia (AVRT).



EKG and Case referred by

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Please submit cases in the composition book located in B Pod. Make sure to include the R1/R4 involved in the case.

List of Submitted B Pod Cases

Case	Case Physicians
Serotonin syndrome	Klaszky/Axelson
Pericardial effusion	Jarrell/Kircher
Orbital abscess	Gauger/Scupp
SMA stenosis	Scanlon/Derks
Endophthalmitis	Axelson
Community-acquired pneumonia	Oewns/Axelson
Infected renal cyst	Spigner/Riddle
Diaphgragmatic hernia	Harty/McKean
Abdominal compartment syndrome	Harty/Devries