University of Cincinnati Medical Center - Department of Emergency Medicine

# Annals of B Pod

Since 1970 - Leadership - Excellence - Opportunity

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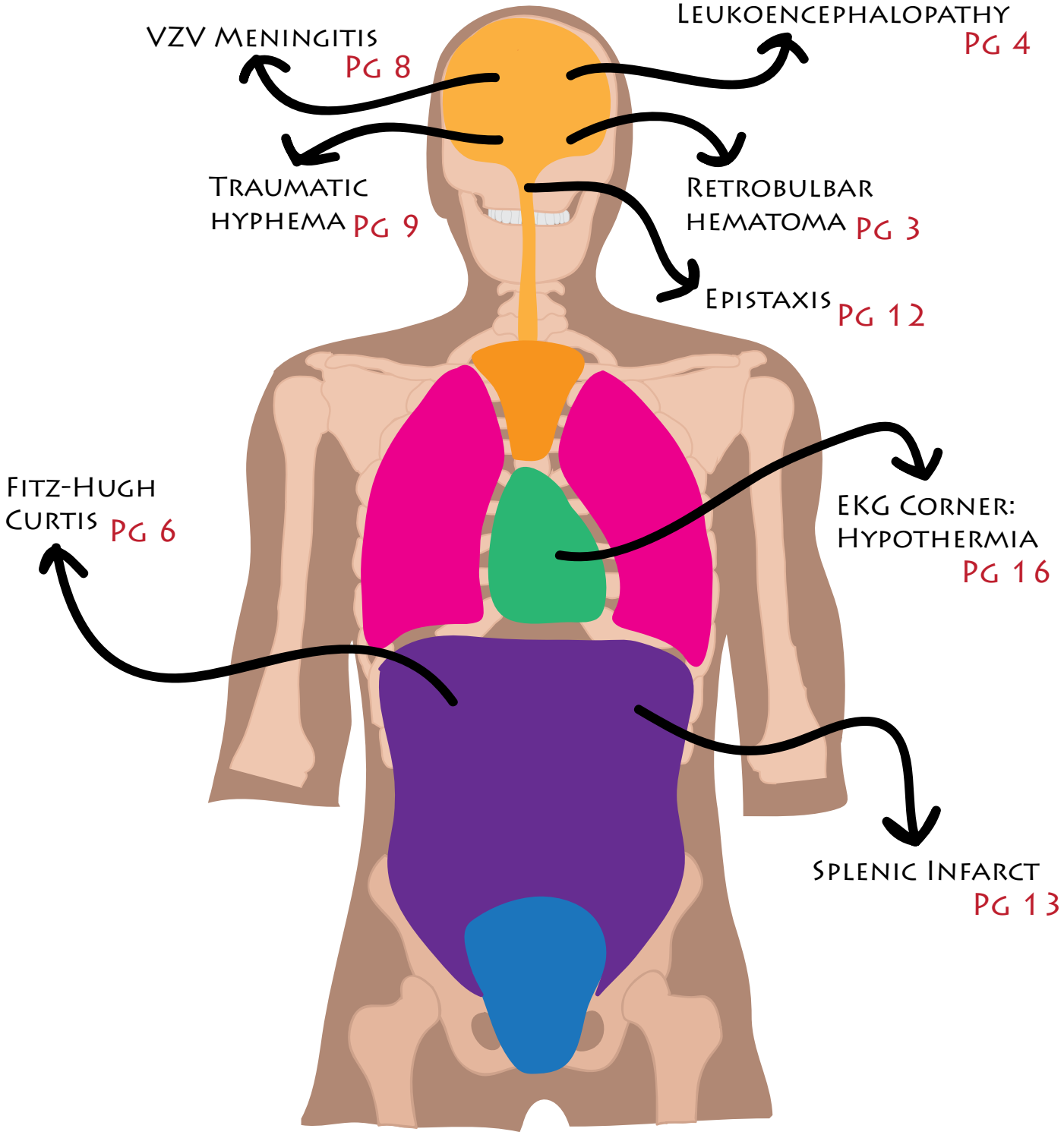


also available at *tamingthesru.com*

EMERGENCY MEDICINE
ANNALS OF B POD
WINTER 2015



## #thewholepatient

Patients typically present to the Emergency Department with one complaint. The chart says "Headache" or "Abdominal Pain" but B-pod implores us to look closer. B-pod teaches us that it is our job to sit down, take a few extra minutes, and look at the entire patient in front of us.

This issue, we look at #thewholepatient. We look at a patient who presents with weakness and leaves with an ultimately life-ending diagnosis. We also look at a patient who presents after falling and ultimately loses her vision. While we may boil down these cases to the literature and science behind the pathology, lets not forget that the reason we show up to every B-pod shift and beyond is to care for #thewholepatient.



# Now you know...

## Quality Matters

Daniel Axelson, MD
University of Cincinnati R3

You've undoubtedly (…hopefully?) mastered your ABCs at this stage, but do you know your PQRS? You've heard of EBM, but are you brushed-up on VBM?

Well, as of 2017 you better be.

Because in 2017 your value as a physician, and thus your Medicare physician reimbursement, can be penalized or rewarded based on quality and cost. Let's explore.

PQRS stands for Physician Quality Reporting System. It is a quality data reporting system to CMS, the Centers for Medicare and Medicaid services, i.e. the government payer for healthcare services, i.e. the source your salary. It's a way the government tracks how efficacious and cost-effective you are as a provider. Since 2007, this program has been voluntary, and physician-groups have been given financial incentives for reporting this cost and outcome data to the PQRS. But as of 2015 such reporting is mandatory, enforced by penalties in physician reimbursement from CMS for non-compliance. Such penalties can take effect starting in 2017. The penalties apply to every physician and every physician group, regardless of size or specialty. Additionally, CMS will begin publicly reporting PQRS data in 2016 on all physicians nationally. Scrutiny grows ever finer. Yes, you're quite literally being watched.

VBM stands for Value Based Modifier. It is a score tied to quality and cost measures on the part of each individual physician. The score is composed of a Quality Composite Score, which is calculated from things such as patient safety, efficiency, and effective clinical care, and a Cost Composite Score, which is calculated from things such as total costs and disease specific costs. (Figure 1) Physicians will now be classified as at, below, or above average in quality and cost measures, and this will affect their reimbursements. (Figure 2) The Affordable Care Act requires that CMS apply this VBM to every physician starting in 2017.

There will be more government regulation over the healthcare you provide. Now you know.

http://www.acep.org/quality/pqrs/. Accessed 11/29/15 at 17:19.

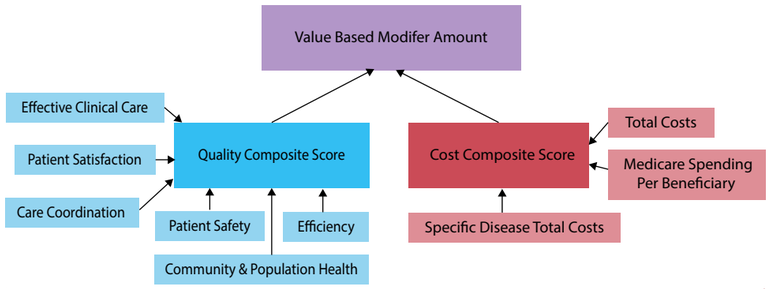


Figure 1. The quality and cost components are based on the categories listed above.

Groups with 2-8 EPs & Solo Practioners

| | Low Quality | Average Quality | High Quality |
|---|---|---|---|
| Low Cost | +0.0% | +1.0x | +2.0x |
| Average Cost | +0.0% | +0.0% | +1.0x |
| High Cost | +0.0% | +0.0% | +0.0% |

Groups with >10 EPs

| | Low Quality | Average Quality | High Quality |
|---|---|---|---|
| Low Cost | +0.0% | +2.0x | +4.0x |
| Average Cost | -2.0% | +0.0% | +2.0x |
| High Cost | -4.0% | -2.0% | +0.0% |

Figure 2. The VBM affects physician reimbursement as noted above





# Ocular Emergency

## A Case of Retrobulbar Hematoma

Courtney McKee, MD
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### History of Present Illness

The patient is a 68 year-old female who presents by EMS after a fall in a parking lot approximately one hour prior to arrival. She fell forward and landed on her face. She believes she simply tripped and fell, but she did lose consciousness and does not know how she ended up on the ground. Per family, she is unsteady on her feet and falls frequently, requiring a cane at baseline. She reports feeling "weak" but no other symptoms preceding her fall. She presents with significant right-sided facial trauma and is unable to open her right eye. She has no complaints of blurry vision in her left eye. She has no headache or other areas of pain or trauma.

### Past Medical History

Hypertension, Hyperlipidemia, Diabetes Mellitus Type II

### Past Surgical History

No surgeries

### Social History

Non-smoker

### Medications

Metformin, sitagliptin, atenolol

### Allergies

No known

### Physical Exam

T 98.7 HR 68 RR 22 BP 220/65 SpO2 95% on RA

Exam reveals a Caucasian female with significant trauma to her right eye, face, lip sitting up in bed in no acute distress but with bloody clothing. HEENT exam reveals a right eye which is swollen shut with significant surrounding ecchymoses and swelling over right maxilla. It is proptotic, with significant chemosis and a fixed mid dilated pupil not reactive to light. She is able to tell light vs. dark but cannot see movement. She is able to move her right eye only very slightly. The rest of her HEENT exam reveals a normal left eye exam, swelling over her right maxilla but no pain on palpation of the face and no Battle's sign. Her neck, cardiovascular, pulmonary, abdominal, and remaining neurological exam are unremarkable.

### Hospital Course

Patient presented one hour after facial trauma with a proptotic right eye, only light/dark differentiation in that eye, as well as loss of extra-ocular movements and an afferent pupillary defect. Together, these findings were classic for retrobulbar hematoma. This was corroborated on her maxillofacial CT (Figure 1). Due to the patient's significant vision deficits on presentation, a right lateral canthotomy with cantholysis was performed urgently at bedside, with successful release of the upper and lower canthal ligaments. After her procedure, the patient had no improvement in her vision, and worsened from light/dark perception to complete vision loss. Her IOPs remained persistently elevated in above 80 mm Hg. Her IOP was acutely managed with timolol drops as well as mannitol, with some improvement in her IOPs. She was admitted to the trauma service for syncope workup as well as management of her ophthalmologic issues as well as multiple orbital and mid-face fractures. The patient ultimately suffered complete vision loss in her right eye.

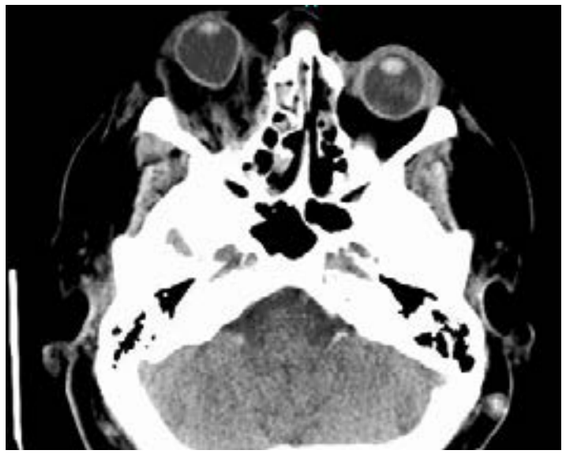

Figure 1: CT maxillofacial: proptosis of the right eye, with teardrop sign (referring to the shape of the eyeball) as well as retro-orbital stranding consistent with blood in the orbital compartment

### Discussion

Retrobulbar hematoma, which can also be conceptualized as "orbital compartment syndrome," is a vision-threatening condition and ophthalmologic emergency. Retrobulbar hemorrhage has been described after trauma as well as after facial surgery, and this presentation may be delayed up to days after injury.[2] This is an uncommon condition, even in the setting of orbital fracture – only 0.45-0.6% of patients with orbital fracture have a coexisting retrobulbar hematoma. However, it is quite morbid, as patients who present with vision loss in the setting of retrobulbar hemorrhage have a 44-52% chance of permanent blindness. The retina may tolerate approximately two hours of ischemia before vision loss is irreversible.[1]

Bleeding into the orbital space increases pressure in a closed cavity. With increased intra-orbital pressure, the globe is displaced anteriorly until it is tethered by the canthal ligaments, resulting in a compressive neuropathy. The optic nerve stretches and suffers ischemia as the globe is displaced anteriorly. Additionally, the pressure in the central retinal artery cannot overcome increased IOP, leading to retinal ischemia. Together, these factors result in the classic proptotic eye with an afferent pupillary defect and progressive vision loss. Additionally, patients may have eye pain, ophthalmoplegia, and findings on fundoscopic exam such as a cherry red macula and nerve head pallor. Intraocular pressures with these secondary findings are typically >40 mm Hg.[3]

Lateral canthotomy with cantholysis is the treatment of choice for decompression of the orbit after retrobulbar hematoma. The only contraindication to lateral canthotomy include suspected globe rupture. See Page 11 for a description of the procedure.

Continued on page 11

#bpodcase

# Weakness in a HIV patient: Progressive Multifocal Leukoencephalopathy

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## History of Present Illness

The patient is a 30-year-old male who presented to the emergency department with a chief complaint of left-sided weakness. The patient said that over the last month he has had progressively worsening weakness of his left arm and left leg. He first noticed this about a month ago, when his thumb began slipping off of the TV remote. Over the course of the month, he had developed the inability to raise his left arm off of the bed without the assistance of his right arm, and was having difficulty taking care of himself. When his family was in the room he said he had been taking his HIV medications. However, notes from Infectious Disease indicated he had been off of his medications for a few years. He denied fevers, chest pain, or cough but did say he could not talk for more than a couple seconds at a time because he would get short of breath.

## Past Medical History

HIV, ADHD

## Past Surgical History

No surgeries

## Social History

Lives with parents, sexually active with men

## Medications

None

## Allergies

No known

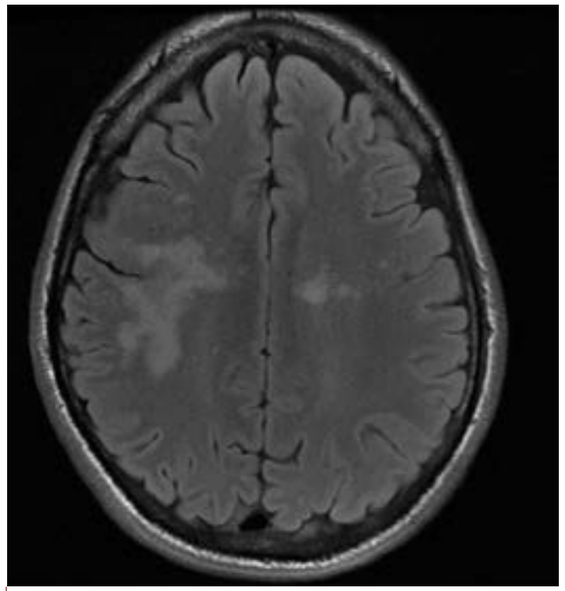

Figure 1: Initial Brain MRI findings suggestive of progressive multifocal leukoencephalopathy in the right frontoparietal white matter and right thalamus with possible other foci of disease in the left corona radiata.

## Physical Exam

T 36.4 HR 79 RR 16 BP 123/73 SpO2 98% on RA

Exam revealed a comfortable appearing male, sitting up in bed, in no acute distress. HEENT, cardiovascular, abdominal, neck and pulmonary exams were unremarkable. His neurological exam revealed an alert and oriented x4 male with an obvious left facial droop but normal facial sensation. He seemed to slur his words slightly but had no signs of aphasia and swallowed without difficulty. He had 5/5 strength in his RUE, 3/5 strength in his LUE, 5/5 strength in his RLE, 4/5 strength in his LLE. He seemed disinterested in the exam and was not particularly concerned about these deficits.

## Labs & Imaging

Absolute CD4 count: 12

CT: Figure 3 MRI(s): Figures 1& 2

## Hospital Course

The patient was admitted to the hospital for treatment of suspected progressive multifocal leukoencephalopathy (PML). He was restarted on his HIV medications and other infectious sources of neurologic pathology were ruled out, including toxoplasmosis, tuberculosis, and Herpes Simplex Virus. He did have a lumbar puncture that was positive for the John Cunningham (JC) virus, which was expected with his MRI findings consistent with PML. He was discharged to rehab with continued neurologic deficits in his left upper and left lower extremities. Remeron was started prior to discharge along with HAART medications and sulfamethoxazole/trimethoprim prophylaxis. Approximately 1 month after diagnosis, he was readmitted for failure to thrive and had a repeat brain MRI (Figure 2) significant for widely progressive PML. At this time he was unable to swallow or talk secondary to weakness and his family decided to pursue hospice care. The patient passed away approximately 6 weeks after the diagnosis was made.

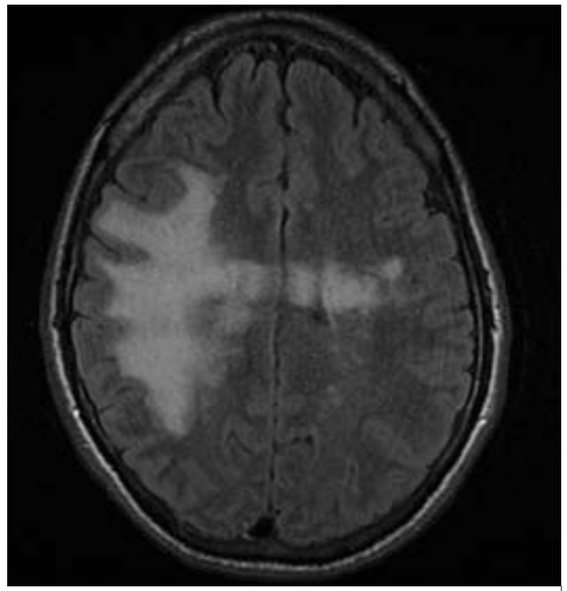

Figure 2: Repeat brain MRI one month later showing extension of the lesions associated with progressive multifocal leukoencephalopathy.



## Discussion

Progressive multifocal leukoencephalopathy (PML) is an infection of the central nervous system by the JC virus that affects patients with HIV.[1] It is a ubiquitous virus that is transmitted via both inhalation and ingestion.[1] The incidence in HIV patients with CD4 counts greater than 200 is much less than those with CD4 counts less than 200, 0.07 vs 0.7 infections/person years.[1] Our patient was more susceptible to the infection because he had a CD4 count of 12 upon arrival. He also had not been on his Highly Active Anti-Retroviral Therapy (HAART) medication for at least a year prior to the diagnosis. Before the implementation of HAART, 3.3% of HIV patients presented with PML, however now the incidence is 1.6% of HIV of patients.[2] Non-adherence to HAART medications also increased the risk of death from PML for our patient; approximately 5% of patients with PML on HAART die within 1 year while 50% not on HAART die within the same time frame.[2] PML contributed to 14% of AIDS related deaths in 2005 and even though HAART has increased survival from 0-30% to 38-62%, its effect on PML is the smallest amongst all AIDS-related illnesses.[1]

The JC virus is a polyomavirus that infects and destroys oligodendrocytes.[1] Patients present with partial neurological deficits that worsen with time as the lesion moves along white matter tracks.[1] This infection pattern leads to the most common presenting complaints including speech difficulties, gait disturbances, and like our patient, limb weaknesses.[2,3] Physical exam often reveals coordination deficits, cognitive deficits, and limb paresthesias that correlate with the most common sites of infection: subcortical white matter, white matter in the cerebellar peduncles, and in the brainstem.[2,3] Patients typically present after the disease has progressed for weeks to months, at which time they finally have enough deficits to notice a change in functionality.[3] Our patient waited to seek treatment until he couldn't use his TV remote anymore but when questioned further, had been developing signs of deficits for approximately a month.

The diagnosis of PML is typically made, as in our patient, in the following sequence: there is clinical suspicion followed by

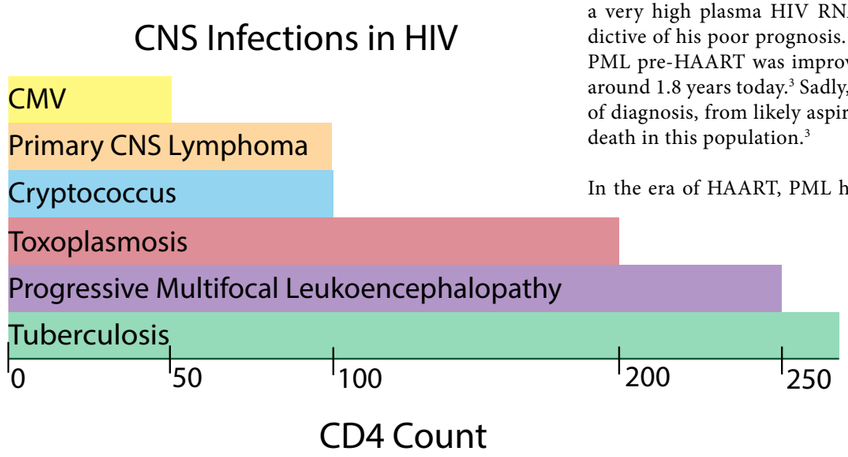


Figure 4: CNS infection risk by CD4 count[6]

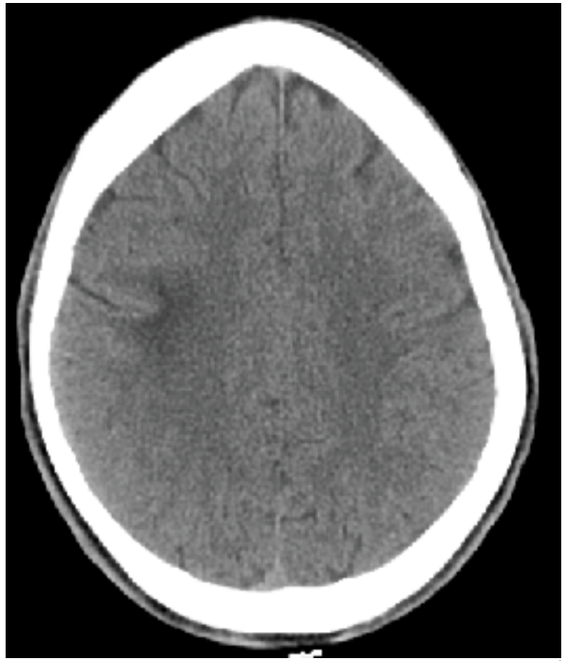

Figure 3: CT Head without conrast: Vasogenic edema in the right frontal lobe, concerning for an infectious or neoplastic lesion.

radiological identification with MRI and then detection of JC virus with CSF PCR.[1] The only treatment for PML is to initiate HAART, which was done promptly upon admission to the hospital. Unfortunately, this only halts progression of the disease in half of patients and ultimately was unable to stop the progression in our patient.[1] HAART cannot reverse the damage already done to the neurons and will leave residual neurologic deficits even those in whom therapy works to stop progression.[1] Poor prognostic factors include lower CD4 counts, higher plasma HIV RNA, higher JC viral loads in the CSF, and any presence of brainstem lesions.[1] Our patient had a CD4 count of 12 and a very high plasma HIV RNA load, both of which were predictive of his poor prognosis. Survival time after a diagnosis of PML pre-HAART was improved from approximately 1 year to around 1.8 years today.[3] Sadly, our patient died within 2 months of diagnosis, from likely aspiration, the most common cause of death in this population.[3]

In the era of HAART, PML has become more uncommon and therefore this particular patient does not present everyday in the ED. However, 60-70% of HIV patients develop CNS complications (Figure 4) and 20% of visits to the ED by HIV+ patients are for neurologic complaints.[4] There is a body of literature attempting to guide the

Continued on page 7

#bpodcase

# Right Upper Quadrant Pain

## Expanding The Differential

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### History of Present Illness

The patient is a 22-year-old female with no past medical history presenting with rib pain. She reports 3 days of pain over the right costal margin. She described it as progressively worsening, moderate to severe, achy in quality, and intermittent. The pain is aggravated by deep inspiration. She has no history of gallstones nor abdominal surgeries. She denies nausea, vomiting, diarrhea, fever, vaginal discharge, dysuria, urinary frequency, chest pain, shortness of breath, and cough. Her last menstrual period ended a few days prior. She is sexually active with one partner and has no history of STIs. She has never been pregnant.

### Past Medical History

Seasonal allergies

### Past Surgical History

No surgeries

### Social History

Non-smoker, sexually active with one partner, occasional condom use

### Medications

Loratadine

### Allergies

No known

### Physical Exam

T 37 HR 120 RR 18 BP 118/67 SpO2 100% on RA

On exam the patient appears uncomfortable and can't sit still on the stretcher. Her HEENT, pulmonary, skin and neurological exam are all unremarkable. Her cardiac exam is remarkable for tachycardia with a regular rhythm. Her abdominal exam demonstrates tenderness to palpation in the right upper quadrant, a negative Murphy's sign, and no rebound or guarding.

### Lab Work-up

CBC: WBC 15.2, H/H 11.7/35.7, Plt 439
BMP: 137/3.5/102/27/8/0.5/103
LFTs: ALT/AST 6/17, Alk Phos 55, T Bili 0.2, Lipase 16
Urinalysis: negative nitrites, negative leukocyte esterase
Urine pregnancy: negative

### Hospital Course

Our patient presented with colicky right upper quadrant (RUQ) abdominal pain that was initially concerning for gallbladder, kidney, or lung pathology. The workup included a complete blood count, basic metabolic panel, liver functions tests, urinalysis, urine pregnancy test, and chest x ray which were remarkable only for a leukocytosis of 15.2.

Over the course of her Emergency Department stay her pain was difficulty to control and her tachycardia persisted despite IV fluids. She later developed nausea and vomiting. A CT abdomen/pelvis was obtained to evaluate for retrocecal appendicitis. The CT revealed a right-sided high-attenuation tubular structure read as an inflamed distal appendix or prominent periuterine vessel. An additional high-attenuation tubular structure in the left pelvis.

In light of this CT reading, our differential diagnosis was again expanded to include pelvic pathology. A pelvic exam was performed which was concerning for pelvic inflammatory disease (PID). Treatment of PID was started in the ED and she was admitted to gynecology for further management. The patient's pain improved during her hospital stay and she was discharged 2 days later with a diagnosis of PID complicated by Fitz-Hugh-Curtis syndrome (FHC). One day following discharge her endocervical swabs returned positive for chlamydia.

### Pelvic Inflammatory Disease (PID)

**Acute PID**
Causes acute onset of lower abdominal pain and tenderness of the pelvic organs on exam. It is complicated by tubo-ovarian abscess, acute salpingitis, perihepatitis (Fitz-Hugh-Curtis), endometritis, and pelvic peritonitis.

**Subclinical PID**
A subclinical infection of the upper reproductive tract with symptoms that usually do not prompt the patient to seek medical care, but can cause long-term sequele, specifically infertility.

**Chronic PID**
A rare indolent infection associated with tuberculosis or actinomycosis

### Discussion

FHC is a rare extra-pelvic complication of a genital infection that involves the peri-hepatic capsule. While most cases have been described in women in association with PID, in rare cases it has been reported in men.[1] A prospective cohort study in 117 incarcerated adolescents documented a 4% incidence of Fitz-Hugh–Curtis syndrome in those with mild to moderate PID.[2,3] Clinical features include severe RUQ abdominal pain, which may have a pleuritic component and radiation to the right shoulder. Aminotransferases are usually normal or only mildly elevated. If a CT is obtained it may show inflammatory changes in the pelvic and perihepatic regions. CT imaging may also reveal subtle perihepatic enhance-

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# Learn Serve Lead

## AAMC Annual Meeting 2015

Lucia Derks, MD
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I recently attended the Association of American Medical Colleges (AAMC) annual Learn, Serve, Lead meeting in Baltimore as a member of the Organization of Resident Representatives (ORR). Before I delve into the themes of this year's meeting, first let me give a little background information on the AAMC. The AAMC is a not-for-profit association that represents 145 accredited US and 17 accredited Canadian medical schools, approximately 400 major teaching hospitals and health systems, including 51 VA medical centers and close to 90 academic and scientific societies. They are not a regulatory body and do not make specific recommendations. They do, however, make suggestions with the overall goal of improving undergraduate and graduate medical education. Thus, most of those in attendance at the meetings have an interest in academic medicine and range from medical students to undergraduate medical educators to resident medical educators.

Who did I meet? Within the ORR, I met residents across a wide range of specialties, including preventative medicine, surgery, dermatology, ophthalmology, etcetera. This diversity allowed us to compare notes and focus on broad topics that span specialties with the goal of improving medical education globally. The conference also invites the Organization of Student Representatives , Council of Deans, Council of Teaching Hospitals, Customized Assessment Services, Group on Women in Medicine and Science, Council of Faculty and Academic Societies, Group on Student Affairs, Group on Diversity and Inclusion, and many other small committees. These committees allow for a wide range of idea sharing for innovative solutions.

The overarching topic of this conference focused on the underserved and resiliency within medical education. The main speaker was Eugene Robinson, who is a Pulitzer prize winning columnist and editor of the Washington Post. He reflected on the presidency of Barack Obama, the passage of healthcare reform, and more recently the civil unrest in Ferguson and Baltimore. Mr. Robinson set the tone for the conference by underscoring the AAMC's commitment to decrease and hopefully end the racial and socioeconomic gap felt by the most vulnerable in our medical system. Though he is not a medical professional, his words still rang true. The subsequent plenary sessions by the president, CEO, and chair of the AAMC reinforced this commitment. This stringent address of a glaring problem was enlightening, invigorating, and gave me hope for the future of medicine.

> "Mr. Robinson set the tone for the conference by underscoring the AAMC's commitment to decrease and hopefully end the racial and socioeconomic gap felt by the most vulnerable in our medical system."

Regarding resiliency, several smaller (up to 500 attendees) sessions focused on the idea of resiliency across all levels of training and the need to address the problem early. The AAMC brought Frank Warren, the creator of the Post Secret project to help frame this topic for us. Post Secret involves people sending secrets on postcards from all across the world, and he publishes these online and in books. Mr. Warren got the idea as a psychiatric social worker working for the suicide hotline and understood how freeing it can be for people to share their secrets. This is directly applicable to the medical field as medical professionals often do not want to admit inadequacies or failures for fear of shaming or rejection. This underlying message is unconsciously relayed to medical students as they feel pressure to not disclose their inadequacies for fear of being found "not as smart" as their peers. This is thought to foster the development of imposter syndrome and lead to loneliness and depression.

Ultimately, I walked away from the conference with about 10 new ideas for projects, invigorated about the future possibilities for medicine, and excited to have met a group of people so dedicated to this field.

## PML

Continued from page 5

Emergency Physician as to the proper radiologic work-up of these neurological complaints in HIV+ patients. One decision guideline recommends obtaining a non-contrast head CT in patients with any of the following presenting complaints or symptoms: new seizure, decreased or altered mental status, headache, or prolonged headache greater than 3 days.[4] In one cohort, all patients with focal CNS lesions presented with one of these findings regardless of whether their CD4 counts were above and below 200.[4] Patients with HIV have a relative risk of 9.1 and 12.7 for ischemic stroke and intracerebral hemorrhage respectively, regardless of their HAART use. It should therefore come as no surprise that vascular lesions are the most common CNS lesion in patients with CD4 counts >200.[4,5] Once a patient's CD4 count drops <200, toxoplasmosis becomes the most common CNS lesion.[4] Other CNS infections to consider include CNS lymphoma, cryptococcal meningitis and TB meningoencephalitis.[6] In general, Emergency Physicians can think of toxoplasmic encephalitis and cryptococcal meningitis as the acute HIV CNS infections, evolving over hours.[6] PML, TB meningoencephalitis, and CNS lymphoma are subacute in onset, and evolve over weeks to months.[6]

1. Cinque, Paola (10/2009). "Progressive multifocal leukoencephalopathy in HIV-1 infection". The Lancet infectious diseases (1473-3099), 9 (10), p. 625.
2. Engsig, Frederik Neess (01/2009). "Incidence, Clinical Presentation, and Outcome of Progressive Multifocal Leukoencephalopathy in HIV-Infected Patients during the Highly Active Antiretroviral Therapy Era: A Nationwide Cohort Study". The Journal of infectious diseases (0022-1899), 199 (1), p. 77.
3. Berenguer, Juan (04/2003). "Clinical Course and Prognostic Factors of Progressive Multifocal Leukoencephalopathy in Patients Treated with Highly Active Antiretroviral Therapy". Clinical infectious diseases (1058-4838), 36 (8), p. 1047.
4. Rothman, R.E. (1999). "A decision guideline for emergency department utilization of noncontrast head computed tomography in HIV-infected patients". Academic emergency medicine (1069-6563), 6 (10), p.1010.
5. Venkat, Arvind (2008). "Care of the HIV-Positive Patient in the Emergency Department in the Era of Highly Active Antiretroviral Therapy". Annals of emergency medicine (0196-0644), 52 (3), p. 274.
6. Tan, Ik Lin (07/2012). "HIV-associated opportunistic infections of the CNS". Lancet neurology (1474-4422),11 (7), p. 605.

#bpodcase

# Connecting the dots.

## VZV Meningitis

Kari Gorder, MD
University of Cincinnati R2

### History of Present Illness

The patient is an otherwise healthy 26-year-old male who presented to the ED with back pain, headache and a rash. He reports that the back pain started at work after lifting some heavy boxes. He describes the pain as located in his lumbar region, worse with flexion, and radiating to his hamstrings and mid-back. Additionally, over the same period of time, he has had a headache, which he describes as worse with lying flat. It is bifrontal and is not accompanied by any nausea, vomiting, neck pain or photophobia. He does not frequently get headaches and denies any trauma. Finally, he reports a 3-day history of a rash on his back. He states it was initially pruritic but is now sharp and painful. He does endorse some chills but denies fevers, IV drug use, recent surgery, immunosuppressive medications or any other concerns.

### Past Medical History

Chicken pox as a child

### Past Surgical History

None

### Social History

None

### Medications

None

### Allergies

No known

### Physical Exam

T98.8 HR 101 RR 18 BP 145/96 O2 Sat 95%

Exam reveals a comfortable appearing male, sitting up in bed, in no acute distress. HEENT, cardiovascular and pulmonary exams are unremarkable. There is some increased pain with neck flexion/extension but no rigidity. Neurologic exam reveals intact strength and sensation. There is a vesicular rash on an erythematous base that extends along the left T5/T6 dermatome.

### Lab Work-up

CBC 7.5 > 14.8/44.0 < 186
BMP 136 / 3.4 / 100 / 28 / 11 / 8 < 113
HIV negative
CSF: total nucleated cells 785 (N 12%, L 75%), glucose 53, protein 186
RBC tube 1: 0 RBC tube 4: 0
CSF VZV PCR: positive

### Hospital Course

This patient presented with back pain, headache and a vesicular rash concerning for herpes zoster. A lumbar puncture was performed, and CSF was concerning for aseptic meningitis, which was confirmed via VZV PCR. While he was initially started on broad-spectrum antibiotics, his regimen was quickly tailored to IV acyclovir when the confirmatory PCR returned. He continued to do well and was discharged on hospital day 4 with no neurologic deficits.

### Discussion

Viral meningitis—also known as aseptic meningitis—describes a clinical presentation consistent with meningitis with a CSF pleocytosis despite negative bacterial cultures. Many different types of viruses, including enteroviruses, herpes simplex virus (HSV), HIV and the West Nile virus, can lead to meningitis. This may progress to or coexist with encephalitis; patients with encephalitis will have altered mental status, motor or sensory defects, or behavioral changes.

The variety of clinical presentations of patients with viral meningitis is broad, and can range from a mild headache and low grade-fever to profound encephalopathy and sepsis. However, patients are typically less systemically ill than those with bacterial etiologies and present with a more indolent course. It is usually not possible to differentiate the specific causal viral agent without CSF studies, although there are some distinguishing clinical features; for example, patients with enteroviral infection often have a flu-like prodrome, and patients with varicella will often have a vesicular rash. Many patients who have HSV meningitis or encephalitis have a known history of HSV-1 or HSV-2 infection; however, up to 50% of HSV CNS infections occur during the primary infection.

The varicella zoster virus (VZV) is the causative agent in two distinct pathologic processes: primary varicella (chicken pox), now rare due to childhood vaccinations, and latent varicella (herpes zoster, shingles, etc.). Herpes zoster, a painful, vesicular eruption along one or more dermatomes, is more common in the elderly or the immunocompromised patient, but can occur in healthy individuals of any age. Other complications of VZV reactivation include post-herpetic neuralgia, myelopathy, vasculopathy, ophthalmologic complications and CNS infections. While initially thought to be a rare cause of meningitis, VZV has been recently identified as the third most frequent etiologic cause of viral meningitis, after enterovirus and herpes simplex virus. Reactivation of the

Continued on page 14

**Lumbar Puncture: Procedural Tips**

By Tim Murphy, MD UCEM R1

1. **Positioning:** Maximize the comfort of the patient
   Maximize the success of the procedure (If sitting, prop up patient's feet on stool to increase interspinus distance)
   Consider anxiolysis
2. **Location:** Iliac crests to identify L4 (77% of physicians were 1 space higher than intended)
3. **Prep and Drape**
4. **Anesthesia:** Make wheal first, then inject a generous amount of lidocaine deeper
5. **Needle Insertion:** Aim needle towards umbilicus to mirror anatomy
6. **Pressure Measurement:** Must be done in lateral decubitus position
   Have patient extend legs to avoid artificially increased pressure
7. **Fluid Collection:** At least 2ml per tube with extra (up to 5ml) in the 4th tube



#quickhit

# Traumatic Hyphema

Isaac Shaw, MD
University of Cincinnati R1

### The Case

The patient is a 45 year old male who presents after sustaining an injury to his right eye with a fishing hook. He states that a three-barbed hook pierced his eye while fishing with his friend. On gross inspection, the hook was noted to have pierced the inferior eyelid causing an obvious right open globe and there was a large hyphema. While he was initially able to count fingers at four feet in his superior visual field, his visual acuity quickly deteriorated to light perception only. Extraocular movements were intact and caused movement of the hook. Ophthalmology was consulted and a CT was obtained. (Image 1). The patient was then taken to the OR for anterior chamber washout, open globe repair, and removal of the fish hook. He was discharged following the surgery with next day follow-up with ophthalmology.

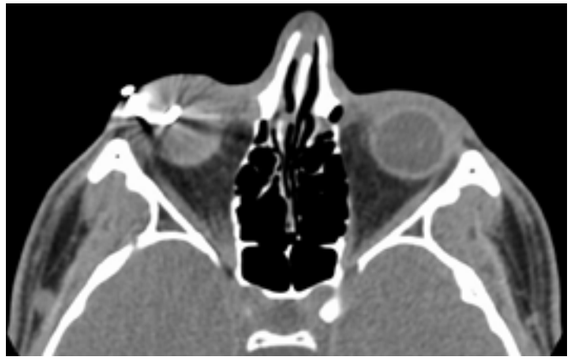

Image 1: CT orbit: Entrance of the hook into the sclera, decreased size of the right globe, and a small hemorrhage anterior to the lens.

### Discussion

A hyphema, defines as a collection of blood in the anterior chamber, is most commonly caused by trauma to the eye.[1] Blunt trauma causes a hyphema by stretching or shearing anterior uveal structures. Penetrating trauma causes hyphema due to the direct vascular injury. Hyphema can also form spontaneously in conditions such as leukemia/lymphoma, ocular neoplasm, coagulopathies, and sickle cell disease.[2]

When evaluating a patient with a hyphema, a slit lamp exam should be performed to rule out a corneal abrasion, evaluate for traumatic iritis, and visualize the extent of bleeding and clot formation. Seidel's test is useful to evaluate for open globe injury. To perform this test ,the clinician anesthetizes the globe with tetracaine and applies fluoroscein just as one would to evaluate for a corneal abrasion. The eye is then examined with a Woods lamp or the blue cobalt slit lamp filter. If perforation or leakage is present, the concentrated dye will be diluted by the aqueous humor from the anterior chamber.

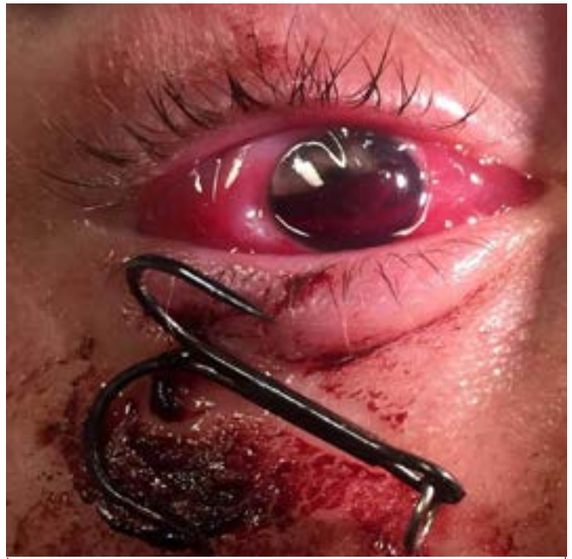

Image 2: Our patient with a fishhook penetrating his eye. Note the presence of a hyphema. Permission granted by patient for use.

Only once an open globe injury is excluded should a clinician obtain intra-ocular pressures. Intra-ocular pressures are necessary because patient's with hyphema are at high risk for secondary glaucoma due to blockage of aqueous humor drainage by clotted blood.[1] Patient's with sickle cell disease are particularly susceptible to this pathology.[3] Management of hyphema focuses on preventing further trauma, preventing rebleeding, and treating complications.[3] An eye shield is typically applied to prevent further trauma.[3] The head of the bed should be raised to at least 30 degrees whenever the patient is supine to prevent secondary glaucoma caused by blood settling posteriorly.[3] A cycloplegic such as atropine can be used to immobilize the pupil to prevent further injury to vessels of the anterior chamber.[3] Topical steroids can be considered to prevent secondary uveitis associated with traumatic hyphema.[3] Given the limited evidence to support these treatments, decisions to use these medications should be made on a case-by-case basis and in consultation with ophthalmology.[3] Antifibrinolytics such as oral aminocaproic acid have been shown to reduce the rate of rebleeding, but do not improve visual acuity.[4] Patients should be instructed not perform any strenuous activity that could lead to rebleeding. Rebleeding typically happens 2-5 days following the injury and is tracked with daily measurements of intraocular pressure.[1]

Indications for hospital admission in traumatic hyphema include involvement of greater than 50% of the anterior chamber, rebleeding, elevated intraocular pressure, suspected child abuse, or poor patient follow-up.[1] Patient compliance is important, as they need next-day follow-up with an ophthalmologist.

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#pharmconsult

# Numbing the Pain:

## A closer look at local anesthetics

Brittany Slocum, PharmD
University of Cincinnati

Local anesthetics are often used for relatively small procedures such as skin surgery, wound repair, abscess drainage, and vascular access to avoid systemic exposure to drug. Although a generally safe method, concerns may arise regarding allergies or toxicity when doses exceed the maximum recommended dose.[1] This overview will address the local anesthetics available for use at University of Cincinnati Medical Center (UCMC), mechanism of action, differences in formulations, dosing, safety, and allergy concerns.

Local anesthetics prevent transmission of pain by reversibly blocking sodium channels in nerve fibers which disrupts depolarization of the nerve[2]. Local anesthetics are either amides or esters, and contain a lipophilic aromatic ring as well as a terminal amine. The terminal amine allows the molecule to transition between a lipid soluble and water soluble conformation. In order to increase stability of the drug it is stored in an acidic environment, in which the molecule is in its water soluble charged conformation. Upon injection into the physiologic pH of the human body, the molecule loses a hydrogen ion to become uncharged (Figure 1). The uncharged molecule diffuses through the interstitial tissue and transports across the nerve membrane. Once in the nerve membrane, the molecule gains a hydrogen ion to once again become water soluble. This conformation confers the ability to block sodium channels. The low pH of the drug formulation makes injection painful to the patient. Lidocaine may be buffered to decrease pain upon injection by co-administering sodium bicarbonate as a 1:9 mixture made of one part sodium bicarbonate 1 mEq/mL to 9 parts 1% lidocaine[1]. This should be used promptly as the shelf life of the lidocaine will decrease upon buffering. Some local anesthetics are formulated with epinephrine to create local vasoconstriction for the purpose of decreasing systemic absorption of the anesthetic.[1,2] Epinephrine may extend the duration of action in certain anesthetics such as lidocaine (Table 1). Epinephrine formulations should be avoided when the patient has comorbidities including severe hypertension, hyperthyroidism, pheochromocytoma, coronary artery disease, or suspected recent cocaine use[1,2]. There is no maximum dose recommendation; however 40 mcg per 30 minutes is reported in dental literature[3]. For reference, 300 mcg is the intramuscular dose to treat anaphylaxis[2]. A list of UCMC formulary local anesthetics class, dose, and pearls are listed in Table 1.

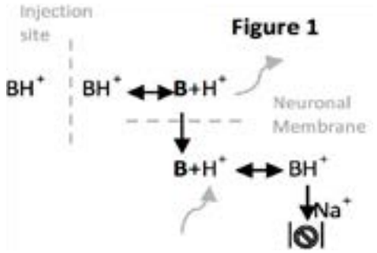


Figure 1: Reactions undergone by local anesthetic medications from injection until the medication binds to and blocks sodium channels in neuronal membranes producing the sought after anesthetic effect.

True allergic reactions to local anesthetics are rare. It is important to carefully question the patient on their allergy as patients often mistake syncope or tachycardia for allergies[2]. If the allergy is not a major reaction such as diffuse hives, throat swelling, or anaphylaxis, a different anesthetic without preservatives or epinephrine may be used as adverse events are hypothesized to be more from the additives then local anesthetics themselves. If the allergy is major, neither amides nor esters should be used as there is a possibility of cross reactivity among the classes. The patient may be referred to an allergist for further investigation and possible future use. Diphenhydramine is an alternative agent that can be considered for local anesthetic; however concern for necrosis limits its use to small skin procedures[4]. Efficacy was measured by pinprick in a trial involving 24 blinded patients randomly given 0.5 ml of placebo, 1% lidocaine, 1% diphenhydramine, and 2% diphenhydramine5. 1% lidocaine and 1% diphenhydramine displayed no difference (p=0.889). A desired 1% solution can be made by drawing up 50 mg/mL diphenhydramine with 4 ml of normal saline. Onset of action has been reported to begin within 5 minutes, and duration lasting 30-40 minutes[6].

| Anesthetic | Class | Onset (min) | Duration (min) | Max dose | Available at UCMC | Clinical Pearls |
|---|---|---|---|---|---|---|
| Lidocaine | Ester | 1-5 | 30-90 | 4-5mg/kg (max total dose 300mg) | 1%, 2%<br>PF 0.5%, 1%, 2%, or 4% | Most commonly used. May be combined with bupivacaine to create a short onset and extended duration (pay close attention to max dose when combining, additive effects). |
| Lidocaine +epinephrine | | 1-5 | 60-180 | 5-7mg/kg (max total dose 500mg) | 1:100,000 as 1% or 2%<br>1:200,000 as 0.5%, 1%, 1.5%, or 2% | |
| Bupivacine | Ester | 5-10 | 3-8H | 2mg/kg (max total dose 175mg) | 0.125%, 0.25%, 0.75%<br>PF 0.25%, 0.5%, 0.75% | Epinephrine does not extend duration, however does decrease risk of toxicity by local vasoconstriction |
| Bupivicane +epinephrine | | 5-10 | 3-8H | 3mg/kg (max total dose 225mg) | 1:200,000 as 0.25%, 0.5%, or 0.75% | |
| Mepivicaine | Ester | 30-120 | 30-120 | 4-5mg/mg (max total dose 300mg) | PF 1% | Similar onset and duration as lidocaine |
| Tetracaine | Amide | 8H | 8H | 1mg/kg | 1% | |
| Chloroprocaine | Amide | 30 | 30 | 10mg/kg (max total dose 800mg) | 2% or 3%<br>PF 2% | |

Table 1: UCMC Local Anesthetics[1,7,8,9]

Toxic complications may occur if dose exceeds the maximum recommended dose or if drug is injected into a major vessel. Patients with renal or hepatic disease should receive a 50% dose reduction to avoid toxicity[7]. Metallic taste, tinnitus, and tingling lips are the first signs of lidocaine toxicity; seizures may then occur, which may be treated with lorazepam. Bupivacaine has the highest risk for toxicity. If ventricular arrhythmias or cardiac arrest occurs, IV fat emulsion 20% can be administered at a dose of 1.5 mg/kg bolus, followed by 0.25 ml/kg/min infusion until hemodynamic recovery is obtained. Reversal of the sodium blockade may be overcome by administering sodium via sodium bicarbonate or hypertonic saline infusions or boluses. Table 2 summarizes the anesthetic adverse effects.

| CNS Effects | CV Effects |
|---|---|
| Metallic taste | Bradycardia |
| Tinnitus | Decreased myocardial contractility |
| Tingling lips | AV block |
| Agitation | Vasodilation |
| Seizures (dose dependant) | Ventricular arrhythmias |

Table 2: Anesthetic Adverse Effects[7]

| Common Anesthetic Concentrations | |
|---|---|
| 2% | 20mg/mL |
| 1% | 10mg/mL |
| 0.75% | 7.5mg/mL |
| 0.5% | 5mg/mL |
| 0.25% | 2.5mg/mL |
| 1.25% | 1.25mg/mL |
| 1:100,000 | 10mcg/mL of epinephrine |
| 1:200,000 | 20mcg/mL of epinephrine |

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## Retrobulbar Hematoma

Continued from page 3

Ideally, decompression is performed as soon as possible after the injury, to mitigate the effects of ischemia on the nervous structures of the orbit. If decompression is unsuccessful in lowering IOP, adjunctive therapies include pharmacologic interventions to decrease intra-ocular pressure by reducing the production of aqueous humor (similar to the acute treatment of glaucoma), including beta blocker drops such as timolol and carbonic anhydrase inhibitors such as acetazolamide. Additionally, hyperosmotic agents such as mannitol may also be used to try to decrease edema contributing to increased IOP.[4] These patients require prompt ophthalmology evaluation and follow-up.

In summary, retrobulbar hemorrhage is an uncommon but time-sensitive, highly morbid condition that carries an approximately 50% chance of vision loss for the patient. Timely recognition and intervention at bedside with a lateral canthotomy with cantholysis can help prevent further ischemic damage to the nervous structures of the eye and help to mitigate vision loss for the patient.

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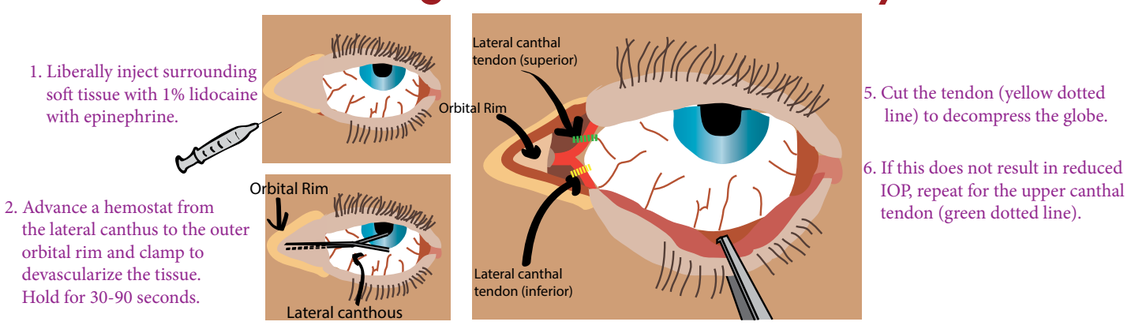
## Performing a Lateral Canthotomy



# Mastering Minor Care

Benjamin Ostro, MD
University of Cincinnati R4

Nosebleeds are a common, frustrating, and potentially life-threatening disease process. Sixty percent of the general population will experience a nosebleed at some point in their lifetime.[1] Despite its prevalence in emergency medicine, many providers approach this complaint with trepidation. As you walk into the room of a patient with epistaxis you may ask yourself what is my initial approach hemostasis? What do I do if/when my initial interventions fail? When should I suspect a posterior bleed and how do I treat it?

Here to shed light on these questions and share some tricks of the trade are our resident minor-care gurus Drs. Hooker and Trott, and new-comer to the column and co-creator of EM Lyceum, Dr. Bryant. Take it away!

**Annals of B-pod:** What is your initial approach to the patient with epistaxis?

**Dr. Trott:** The most important step is preparation. Gown, face mask, light source, suction from ENT tray, cocaine, cotton pledgets, bayonets, nasal speculum. Assuming the patient has expelled the clots and Afrin has been used, cocaine cotton ball or pledgets can be applied for 2-3 minutes to the antrum/septum. Remove the cotton and the septum should be dry and anesthetized. Then apply silver nitrate to the dry bleeding point and septum around it. This seems like a lot to do, but, in my experience it works and the patient is grateful not to go home with an obstructed naris.

**Dr. Hooker:** I think that the key is getting cocaine. Without it, you don't get adequate anesthesia nor vasoconstriction. I order up a neosynephrine and then inject the cocaine right into that bottle (through the top). Spray it up in the nose and directly at the septum using an atomizer. I really do not like pledgets. They absorb too much of the cocaine and the patient gets too little. Before spraying the cocaine mixture up in the nose, have the patient blow their nose really well. Otherwise, all the cocaine is on the clots.

Preparation is key. Get an ENT tray, make sure that you have good suction and that you are using the ENT suction tip. Make sure that you have the nasal speculums so that you get adequate visualization. You must use a head lamp. Being unprepared is a sure way to fail.

Dr. Bryant: I agree with clearing the clot and am an Afrin fan. I then put the plastic nose clip on and let the coagulation cascade do its thing for 15-20 minutes. This, honestly, is all that is required in most bleeds in people with normal anatomy, not post op, not traumatic, not on anticoagulants, normal platelet count, etc. Silver nitrate is a next line for me. I actually tend to avoid silver nitrate because it's painful and damages tissue.

**AoBP:** What about electrocautery?

**Dr. Hooker:** I never use electrocautery. It is too dangerous. You can burn right through the septum. I only use silver nitrate. You will need a bunch of them. I also make a PCA or a nurse be standing there to hand me more as I need. I will often use 10 or more sticks to get a septal bleeder stopped.

**AoBP:** What do you do if your first approach fails to stop the bleeding?

Dr. Trott: If you have followed all steps correctly, it should work. If not, I pack the antrum with Vaseline gauze strip for 1 hour then come back and try again. If it still does not work, I repack and call or send to ENT the next day. Failure usually means there is a complication (ASA, anticoagulant use) or technique is not adequate.

**Dr. Hooker:** I don't fail. You can always (and I said always) stop an anterior bleed. I guess you could use Gelfoam, but I have never had to. The key is adequate suction while using the nitrate sticks. You may need someone to hold the nasal speculum while you hold the suction in one hand and the nitrate sticks in the other hand. I never use a rapid rhino. They are miserable and unnecessary.

Dr. Bryant: Gelfoam is a reasonable next step. I try another round of the above, and then use a small rapid rhino after some topical lidocaine (the best plan is to aerosolize it and spray like you're going to NP scope with the 4% lidocaine).

**AoBP:** Do you ever place bilateral packing?

**Dr. Trott:** Only if both sides can be demonstrated to be bleeding.

Continued on page 14



#bpodcase

# The Canary is in the spleen

Ryan LaFollette, MD
University of Cincinnati R4

## History of Present Illness

The patient is an African-American female in her mid-30s with a history of diabetes with diet control and metformin who presents with abdominal pain. The pain started last night without precipitating factors. It is mainly epigastric and radiates to her left flank. No relation to eating but laying down and movement makes it worse. She has had nausea and vomiting with the pain unable to take her metformin for the past 2 days.

## Physical Exam

T 37 HR 98 RR 18 BP 137/87
O2 Sat 100%

Exam revealed a comfortable appearing female, in no acute distress. HEENT, cardiovascular, neck, pulmonary, and neurological exams were unremarkable. Her abdomen is soft, non-distended with right upper quadrant and left flank tenderness to palpation. She has no edema in her lower extremities.

Laboratory testing included a CBC which was positive for a leukocytosis to 15.9 with 70% neutrophils. Unremarkable urinalysis and basic metabolic panel. Bedside ultrasonography of the right upper quadrant was obtained which showed a thin anterior gallbladder wall, no evidence of sludging, stones and normal common bile duct diameter without evidence of cholecystitis. CT Abdomen/Pelvic showed a hypoattenuated wedge consistent with splenic infarct (Image 1). Transthoracic Echocardiography showed a severely reduced ejection fraction of 20-25% with diffuse hypokinesis.

## Hospital Course

The patient was started on enoxaparin with a bridge to coumadin for presumed left ventricular thrombus that had embolized to cause her splenic infarction. She continues to be managed for her idiopathic heart failure and is doing well. The circuitous route to the diagnosis of heart failure in this otherwise healthy female highlights the diagnostic uncertainty inherent to our patients, and how vigilance of pursuing diagnostic results that do not fit the presentation, as in our patient's leukocytosis, can lead to unexpected results.

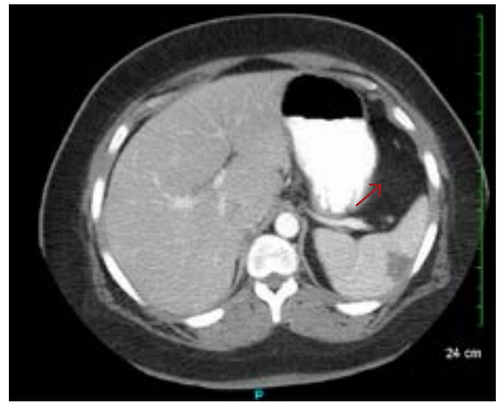



Image 1: The arrow points to the wedge shape of area of hypoattenuated spleen suggesting a likely embolic source of infarction

## Anticoagulation in Heart Failure

While left ventricular dilation causes a majority of the symptomatic presentations of heart failure of dyspnea, that dilation also predisposes to stasis. This increased stasis is combined with endothelial dysfunction and intrinsic changes in platelet and coagulation factors in heart failure creates a systemic prothrombotic state. Embolic phenomenon can manifest clinically in stroke, renal, bowel or splenic infarction as with our patient. In the Rotterdam study, after multivariate adjustment there was a 3 times increase in the risk of stroke in the first month after the diagnosis of heart failure[1].

The question of prophylactically anticoagulating patients in sinus rhythm with significant reduction in ejection fraction has been looked at extensively in the literature[2]. There has also been shown to be use of ACE inhibitors as they may reduce some of the components of the prothrombotic state and thromboembolic rate as compared to beta blockers alone.

So which patients with heart failure would benefit from oral anticoagulation (OAC)? There are contradictory recommendations in the literature about who needs OAC in HF. The WARCEF trial was a double blinded trial of aspirin vs warfarin in 2305 patients and demonstrated no significant difference between groups at primary endpoint of death, ischemic stroke or ICH at mean follow-up of 42 months[3]. There was a decrease in ischemic strokes in the warfarin group (4.7% vs 2.5%) and an increase in major bleeding (2.7% vs 5.8%).

Without definitive benefit in the evidence, society recommendations (Figure 1) and practice pattens vary and will be individualized to the clinical context . In this case, the patient had stigmata of systemic embolus and therefore was a candidate for systemic anticoagulation. Interestingly, there are recommendations as well that combined ASA and warfarin therapy has significant increase of bleeding events without clear improvement in HF with chronic CAD.
Heart failure patients are a significant portion of our ED population and it is important to recognize their potential thromboembolic morbidity.

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**Recommendations for oral anticoagulants in heart failure**
2010 Heart Failure Society of America[4]
- History of systemic or pulmonary emboli
- Recent history of large anterior MI or MI with LV thrombus
- Consider in patients with LV thrombus without embolus depending on thrombus characteristics
- Consider in patients with dilated cardiomyopathy and EF < 35%

VZV Meningitis
Continued from page 8

virus without the characteristic rash is possible (zoster sine herpete), although typically CNS involvement is accompanied by dermatologic findings. In most cases, the zoster rash appears several days into the patient's course, as it did in this case.

Laboratory findings in viral meningitis will typically show a pleocytosis (25-500 cells/μL) with a lymphocytic predominance, a normal or slightly elevated protein concentration (20-80 mg/dL) a normal glucose and a normal or slightly elevated opening pressure. Patients with HSV-2 or VZV infections may have slightly higher protein levels and WBC levels. If there is any clinical uncertainty, patients may be treated with broad-spectrum antibiotics until CSF PCR results return. The recommended treatment for VZV meningitis or encephalitis is acyclovir for 10-14 days, as was done in this patient.

The long-term outcome of patients with VZV meningitis is varied. In general, the outcome is favorable, although case reports exist describing poor neurologic outcomes. VZV encephalitis is thought to have a worse prognosis than meningitis. Patients who are immunocompromised (e.g., HIV patients) tend to have a worse neurologic outcome, as do patients who develop VZV CNS infection without the classical rash. As the varicella virus is one of the few viral etiologies of meningitis that requires prompt antiviral treatment, clinicians should maintain a high level of suspicion in any patient with viral meningitis, with or without the characteristic rash.

### Preventing Post Lumbar Puncture Headaches

By Tim Murphy, MD UCEM R1

1. **Use a thinner needle:** Rate of post-LP headache using needle under 20 gauge dropped incidence by >30%
2. **Use an atraumatic needle:** 26% absolute risk reduction of post-LP headache when using an atraumatic needle (ie Whitacre)
3. **Insert needle with bevel parallel to dural fibers:** Cuts post-LP headaches in half (10.2% to 5.5%)
4. **Replace the stylet before removing needle:** Decreases incidence of post-LP headache (5% v 16%)

Bed rest following the procedure or administration of IV fluids prior to the procedure have not been shown to decrease the incidence of post-LP headaches.

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MCM: Epistaxis
Continued from page 12

**Dr. Hooker:** I perform bilateral nasal packing only when I have a posterior or unidentifiable source bleed. I use bilateral foley catheters and 6 feet of gauze ribbons in each side. I then admit these patients to the ICU for observation.

Dr. Bryant: For the higher risk people (post op, traumatic, on anticoagulants, low platelet count) I have done the miserable thing of putting a rhino in both sides to get it stopped.This can involve an admission for pain control and anxiolysis, though. I have only done gauze ribbons twice because I think it's too time consuming and the result is no more effective, based on the literature, than the rapid rhino.[2]

**AoBP:** When should I suspect a posterior bleed?

**Dr. Trott:** If the patient is sitting straight up, then a posterior bleed will be seen active bleeding in the posterior pharynx without anterior bleeding. Also, elderly patients tend to bleed more often posteriorly and younger patients anteriorly. I use a device with a posterior balloon, although I have had success with a foley catheter.

Dr. Bryant: (Taken from emlyceum.com) "Delayed (5 days – 9 weeks), massive epistaxis after head and neck surgery, or trauma may point more towards a posterior nosebleed from an internal carotid artery pseudoaneurysm.[3] All other causes of nosebleeds, including those caused by anti-platelet and anticoagulant agents, hereditary telangiectasia, platelet disorders, nasal neoplasms, and hypertension, may cause either an anterior or a posterior nosebleed.

If history does not provide the clear answer, proper inspection with insertion of a nasal speculum can often help to define the origin when a bleeding vessel is visualized.

If both history and physical examination still leave you in the dust, anterior nosebleed treatment failure may be the only method of differentiating the two. Although there is a paucity of evidence on this topic, it is reasonable to conclude that if brisk bleeding continues, especially into the oropharynx, despite placement of bilateral anterior nasal packing, a posterior source of bleeding is likely."

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Fitz-Hugh-Curtis
Continued from page 6

ment as well as gallbladder wall thickening.[4,5] Ultrasound is a noninvasive diagnostic tool that may be able to visualize perihepatic inflammation and adhesions, potentially leading to earlier diagnosis.[7] Ultimately, FHC is a diagnosis of exclusion.

Clinical features of PID are not always present. Complications of FHC include those of PID such as chronic pelvic pain, sterility, tubo-ovarian abscess, and ectopic pregnancy. In addition, "violin-string" like adhesions may subsequently develop between the liver capsule and the peritoneum leading to chronic right upper quadrant pain and which may require adhesiolysis surgery.[6]

Given the presence of the patient's pain being isolated to above her umbilicus, and her initial denial of genito-urinary symptoms and lower abdominal pain, a pelvic exam was not performed. In retrospect, this was an error which delayed diagnosis and increased the patient's ED length of stay. The "bilateral tubular high attenuation structures" read on the CT pelvis were likely inflamed fallopian tubes from PID.

In summary, this was a common presentation of an uncommon disease. We should suspect Fitz-Hugh-Curtis in any sexually active female presenting with RUQ pain and have a low threshold to perform a pelvic exam early. Furthermore, in any female with abdominal pain for whom advanced imaging is being considered, a pelvic exam should be strongly considered, especially if there is clinical uncertainty or any doubt as to the reliability of the patient. Also remember that patients may downplay their symptoms, especially those involving sensitive issues or with potentially upsetting psychosocial ramifications.

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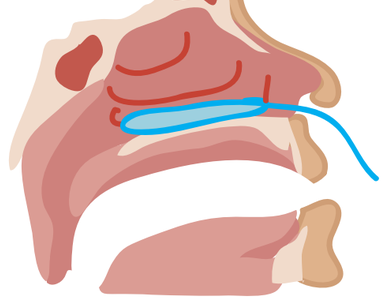
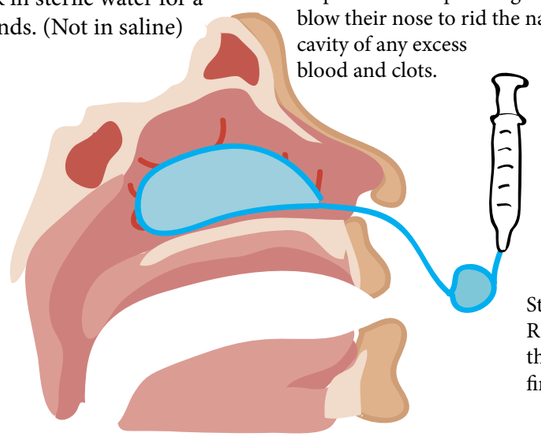
## Inserting a Rapid Rhino

Step 1: Soak in sterile water for a full 30 seconds. (Not in saline)



Step 3: Insert the Rapid Rhino into the nasal cavity along the septal floor and parallel to the hard palate until the plastic proximal fabric ring is well within the nares.

Step 2: Have the patient gently blow their nose to rid the nasal cavity of any excess blood and clots.



Step 4: Using a 20ml syringe, inflate the Rapid Rhino with air. Stop inflation when the pilot cuff becomes rounded and feels firm when squeezed.

# EKG focus

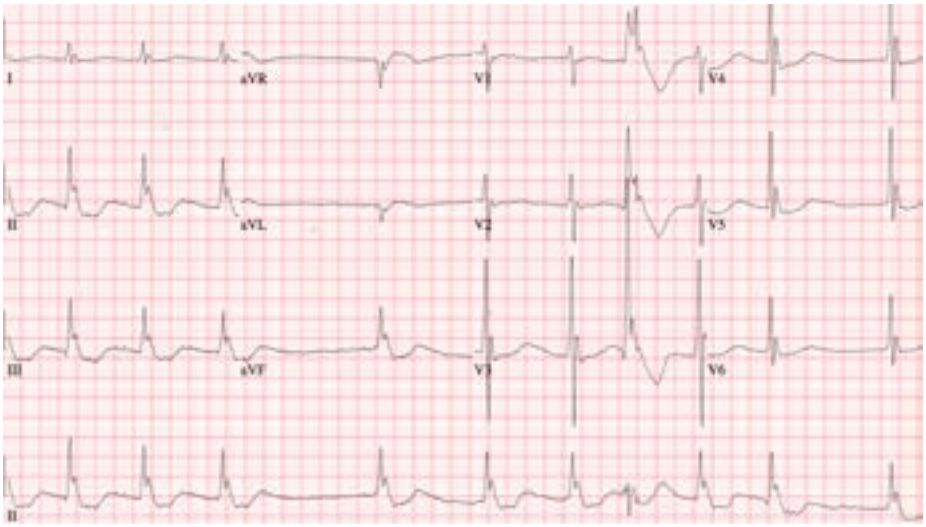
Grace Lagasse, MD
University of Cincinnati R2

## History of Present Illness

The patient is a man in his 40's with unknown past medical history who was found down and unresponsive by EMS. Upon arrival to the ED his rectal temperature was found to be 31.3°C (88.4°F).

## Patient Outcome

The patient required intubation in the ED for airway protection and initial GCS of 3. He was rewarmed with warm IV fluids and a Bair Hugger warming unit. After 24 hours the patient's temperature improved to 36.9°C (98.5°F). After rewarming the patient's mental status improved and he was discharged on hospital day 5.

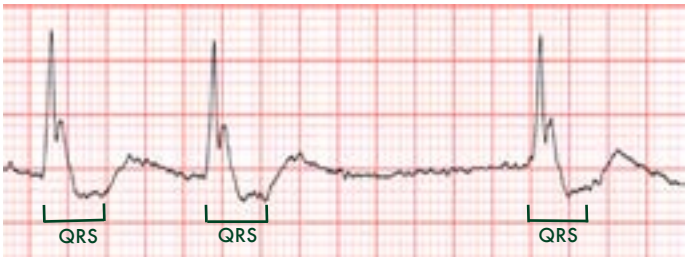



This EKG highlights many abnormalitites that can be seen in hypothermic patients. 1. Atrial fibrillation with slow ventricular response 2. QT interval prolongation 3. Osborn waves

## EKG Changes Associated with Hypothermia

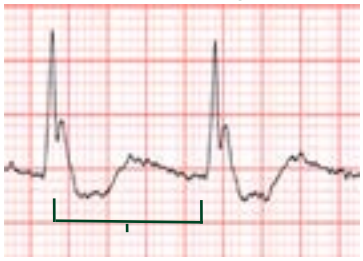
1. Sinus bradycardia or atrial fibrillation with slow ventricular response. This is thought to precede more ominous arrhythmias such as ventricular fibrillation and asystole.
2. Interval prolongation; typically PR, QRS, and QT. This is due to slowing of myocardial conduction and prolongation of the cardiac cycle.
3. Presence of Osborn or "J" waves. The amplitude of the "J" wave is inversely related to the degree of hypothermia. They are commonly seen in the anterior and lateral precordial leads and lead II.

### Atrial Fibrillation with Slow Ventricular Response



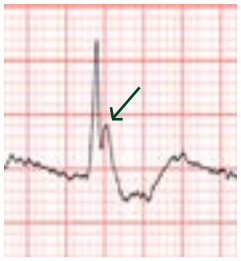

Irregular ventricular rate without P-waves consistent with atrial fibrillation. The heart rate in this patient is 61 bpm, showing a slow ventricular response.

### QT Interval Prolongation



The QT interval in this patient was 612ms. As heart rate slows the QT interval lengthens.

### Osborn or "J" Wave



Osborn waves are a positive deflection in the terminal QRS.

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*EKG and Case referred by*

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*Annals of B Pod is looking for YOU to submit your interesting cases of B Pod - There is a composition book at the R4 desk - please make sure to include the R1/R4 involved in the case, a brief synopsis and a patient sticker*
annalseditors@gmail.com

## List of Submitted B Pod Cases

| Case | Case Physicians |
|---|---|
| VZV Menginitis | Ventura (M4)/Moellman |
| Nicrobiosis Lipoidia Diabeticorum | Whitford/Mann |
| INH overdose with seizures | Boyer/Locasto |
| Ruptured ectopic with +FAST | Loftus/Nelson |
| Acute Ischemic Stroke | Bernardoni/Latimer |
| Severe DKA | Shaw/Betz |
| Traumatic Hyphema & Open Globe | Shaw/Selvam |
| Electrocution w/ shoulder dislocation | Colmer/Doerning |
| Intraparenchymal mass | Merriam/Doerning |