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YEARS OF  
EMERGENCY MEDICINE  
RESIDENCY



# ANNALS OF **B POD**

Fall 2021 (Volume XV Issue II)



*Leadership*

*Excellence*

*Opportunity*

# FOREWORD

 ANNALS OF **B POD**  
*Since 1970*

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Annals of B Pod is a resident-driven, evidence-based publication that was started by Dr. Aaron Bernard (Class of 2007) in the fall of 2002. Dr. Bernard began collecting interesting cases that were seen by interns and R4s exclusively in B Pod and producing a word document "publication" reviewing these cases, as well as the salient educational points. Each case included graphs, figures, pictures, and evidence. Dr. Bernard's primary goal was to not only highlight interesting cases and develop a unique educational tool, but also to dispel perceptions of lower acuity and less interesting cases in B pod. There is real pathology in ALL areas of the UC CEC!

In the last 19 years, Annals of B pod has grown from a monthly word document driven by one senior resident to a professionally produced, quarterly publication with resident and faculty editors. Annals has been supported and sponsored by the department, and it is embraced by faculty, residents, APPs, and even off-service residents. While the focus has grown and evolved based on the leadership of the resident editors to include a consultant's corner, ECG review, Air Care cases, and pediatric cases, the core mission has always been the patients seen and cared for in B Pod.

As we celebrate our 50th Anniversary as the first Emergency Medicine Residency in the country, it is appropriate to highlight and celebrate B Pod. B Pod is where most of the UC EM graduates in the country saw their first patients, wrote their first orders, and began their path in Emergency Medicine. This special Anniversary issue of Annals of B Pod celebrates that legacy, highlighting articles written by alumni editors over the past 19 years. We hope you enjoy this issue and appreciate the hard work of the residents in this unique educational initiative.

**William A Knight IV, MD**  
**UC EM Class of 2007**

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*by:*

*Seth Krupp MD '09*

*Originally published in  
2008 Volume III  
Issue III*



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*by:*

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MS '13*

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# ANTERIOR SHOULDER DISLOCATION



Seth Krupp MD

University of Cincinnati EM Alumnus '09

## Chief Complaint

Shoulder pain

## History of Present Illness

18 yo female presents after an altercation with another woman where she was pushed to the ground and had a sudden onset of inability to move her right arm and shoulder pain. Denies other injury with the exception of minor abrasions and contusions.

## Past Medical/Surgical History

None

## Medications/Allergies

None

## Vital Signs

Stable

## Physical exam

Musculoskeletal: Right arm held abducted with slight external rotation. No crepitation/deformity over clavicle. Patient refuses to internally rotate affected shoulder secondary to pain. Palpable radial/ulnar pulses with strength/sensation intact throughout radial/median/ulnar distribution.

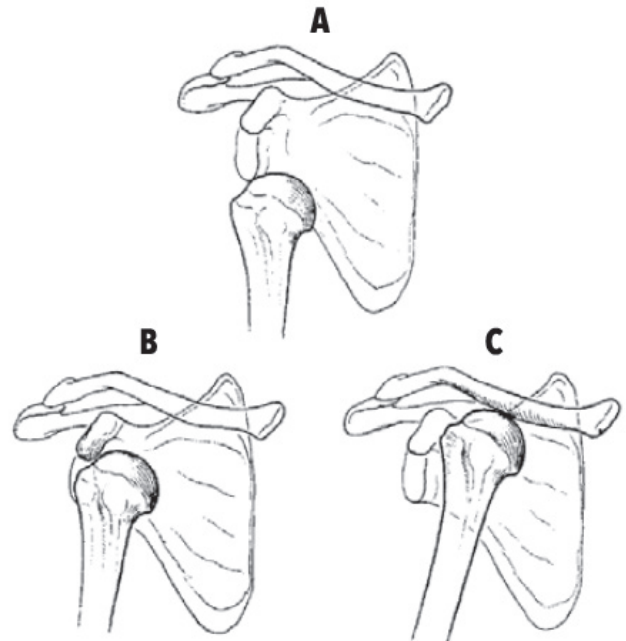
## Hospital Course

X-ray was performed and showed no acute fracture. Patient had 10cc of 1% lidocaine introduced into the shoulder joint from a lateral approach. Patient was then placed supine on the bed, had downward traction placed on the humerus, and the scapula was rotated medially with immediate reduction of the shoulder. Patient tolerated the procedure well, had a repeat X-ray performed that was also negative and was placed in a sling and discharged to home with Ortho follow-up.

## Discussion

### Background

Anterior shoulder dislocation is the most frequently encountered type of dislocation that we, as emergency physicians, will encounter in our careers. For patients, this can be an extremely painful injury that is fraught with uncommon but significant long-term consequences. For busy ED clinicians, optimal management of these injuries can increase both patient satisfaction and efficiency while avoiding missing a clinically significant in-



From, *Roberts and Hedges, Management of Common Dislocations*, p 959.

jury that could lead to long-term morbidity.

### Anatomy

Anterior dislocations are of 3 types, with management of each being the same:

- Subglenoid (rare)
- Subcoracoid (most common—approx. 75%)
- Subclavicular (rare)

Be aware of nerve injury—the axillary nerve, the nerve most often injured with shoulder dislocations, runs inferiorly to the humeral head and wraps around the surgical neck of the humerus.

### Pathophysiology

- Anterior dislocations comprise 95% of shoulder dislocations seen in the ED
- Anterior dislocations may result from seemingly minor mechanisms in patients with recurrent dislocations— such as reaching for an object on a shelf
- First-time dislocations are usually caused by a combination of abduction, extension, and external rotation applied during a fall or trauma

## Physical Exam

- Presentation is usually obvious—patient holds arm in abduction and slight external rotation and has an abrupt drop-off at the acromion process
- Patient will resist efforts to internal rotation
- Palpate radial/ulnar pulse and evaluate for any signs of an expanding hematoma in the axilla. This is done to assess for axillary artery injury
- Perform pre- and post-reduction neuro exams—axillary and radial nerves are most commonly affected. They both originate from the posterior brachial plexus which is most susceptible to injury
- Check “arm patch” (over the deltoid) sensation, abduction and external rotation to evaluate function of axillary nerve
- Check dorsal hand sensation and strength of wrist extension to evaluate the radial nerve

## Imaging

- Anterior dislocations are associated with fractures 15-35% of the time
- Some have advocated that pre-reduction films are not necessary in patients with obvious recurrent dislocations without history of blunt trauma, but data is limited to support this practice
- Post-reduction films ARE ALWAYS NECESSARY—as many as 37% of fractures can be missed without them.<sup>5</sup> Clinical significance of fractures found on post-reduction films is usually negligible, but they should still be obtained.

- Routine films
  - AP
  - Scapular “Y” View—(see image next page)— displacement of humeral head towards the ribs indicates anterior dislocation
  - Axillary view—patient only needs to hold arm 10-15° away from body to get this image. A normal humeral head should sit on the glenoid like a golf ball on a tee.

Anterior/posterior displacement is most obvious in this view

## Complications

### Axillary Nerve Injury

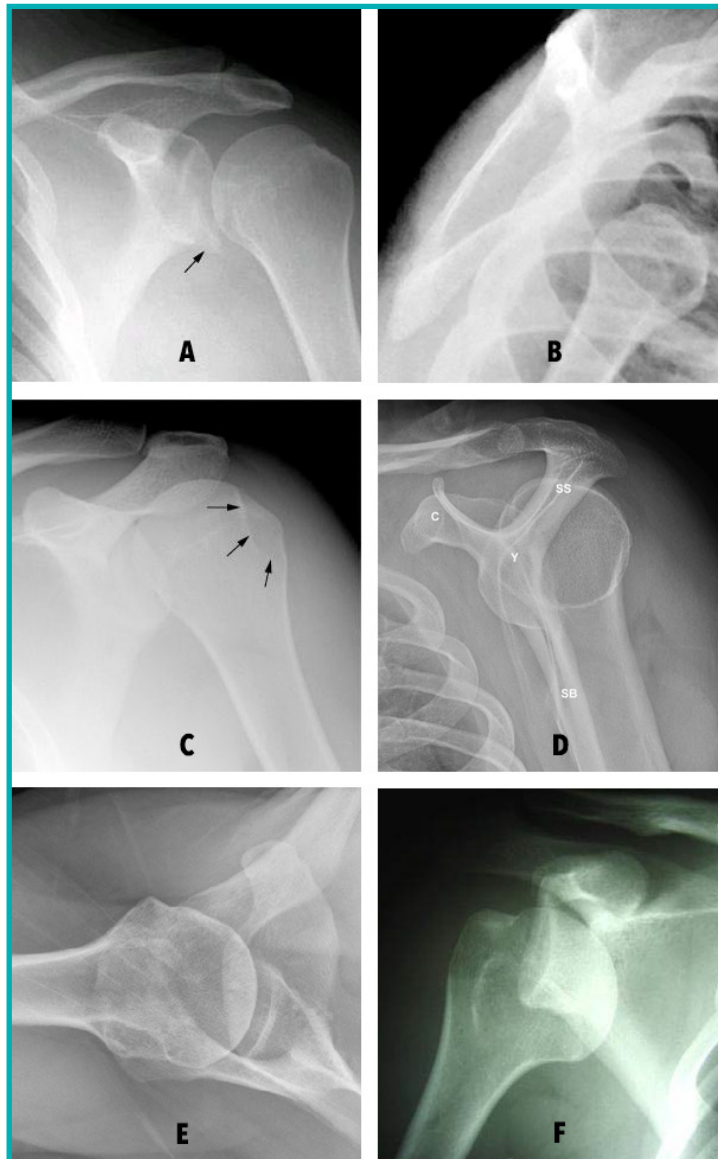
- Incidence varies among case series, but reported between 3-10%
- Management is conservative—physical therapy and EMG in 1 month
- Symptoms will usually improve over weeks, but in rare instances surgery is required. Surgery is usually deferred 4-6 months
- With any evidence of nerve injury, the least forceful method of reduction should be attempted and multiple attempts should be avoided

### Axillary Artery Injury

- Extremely rare
- Most common in the elderly
- Consider vascular studies in any elderly patient with a brachial plexus injury, especially if the onset of neurologic symptoms are delayed from the initial injury
- Vascular injuries are much more common in INFERIOR dislocations

### Hill-Sachs Deformity

- Usually develops after repeated dislocations
- Hill-Sachs lesions occur when the edge of the glenoid causes an impaction fracture in the posterolateral aspect of the humeral head during anterior dislocation
- Best visualized on a post-reduction film
- Identification of this fracture in the ED will not change



A. Bankart Lesion. B. X-ray showing an anterior dislocation in the 'Y' view. C. Hill-Sachs Deformity D. X-ray showing normal orientation in a 'Y' view. E. X-ray showing normal "golf ball on a tee" appearance of the humerus in the glenoid on axillary view. F. AP view showing humeral head outside the glenoid and below the coracoid. All images from www.uptodate.com

initial management

### Bankart Lesion

- Fracture of the anterior rim of the glenoid labrum associated with joint capsule rupture
- Significantly displaced anterior or posterior glenoid rim fractures require operative management and may complicate reduction

**Anterior Shoulder Dislocation**

Continued on page 8



# CLIMATE CHANGE & HUMAN HEALTH



Renee Salas MD, MPH, MS  
*University of Cincinnati EM Alumnus '13*

## The Intersection of Climate Change and Human Health

What does the Lancet call the “biggest global health threat of the 21st century”? You may be surprised to learn that it is a topic which very few physicians currently learn about during their training – climate change.<sup>1</sup> While there are still the ever shrinking collection of skeptics who state that climate change is not occurring, there is no lack of consensus among scientists. In fact, the Intergovernmental Panel of Climate Change (IPCC), which first released its reports in 1988, currently has thousands of authors and is possibly the “largest scientific assessment exercise in human history.”<sup>2,3</sup> The recent press that global warming has slowed below previously proposed predictions is due to the fact that the oceans are currently acting as a heat sink with potentially catastrophic warming of the world’s oceans as a result (Figure 1).<sup>4</sup> Thus, climate change is a reality which has, and will continue to, change the environment in which we live.

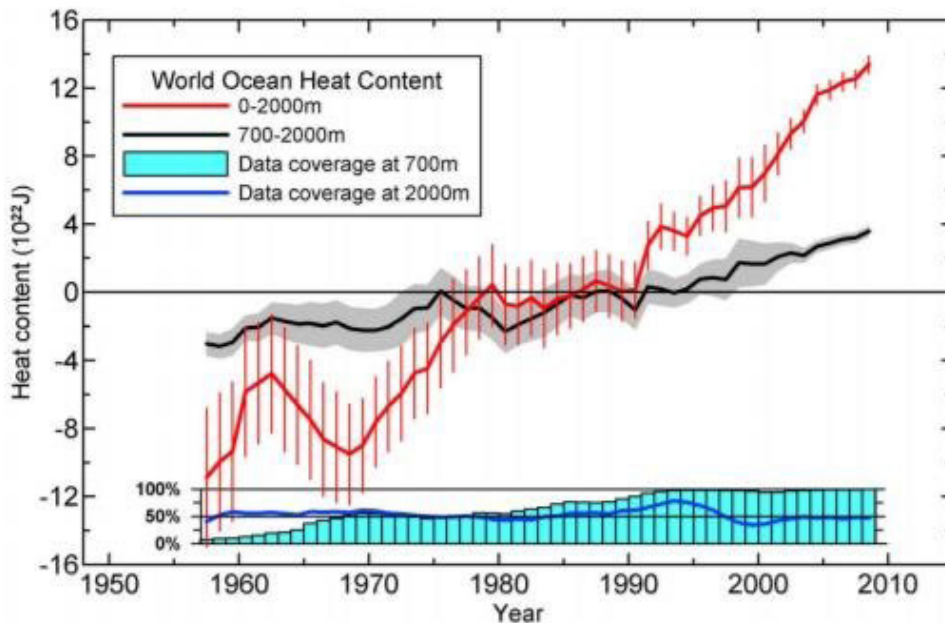


Figure 1: Time series for world ocean heat content

The effects of climate change on human health are numerous. The World Health Organization estimates that between 2030 and 2050, nearly 250,000 additional deaths per year will occur due to just four of the effects of climate change on human health – malnutrition, malaria, diarrhea, and heat stress.<sup>5</sup>

Aaron Bernstein, MD, MPH at the Harvard School of Public Health has created two diagrams which further exhibit some of the key effects on human health (Figure 2). While individuals may be able to predict some of the health effects, others are likely surprising. For example, increased ambient CO<sub>2</sub> decreases the nutrients of crops such as wheat, rice, and maize which can lead to malnutrition.

Our efforts should be most directed at those populations which will be most vulnerable. There is a stark contrast between the countries which have most contributed to CO<sub>2</sub> emissions and those which will suffer the worst of climate change health consequences. Thus, it is imperative that physicians in all countries, but especially those with abundant resources, begin to understand their possible roles in this constantly evolving issue.

Currently, the climate change/human health intersection is not well established within the house of medicine. Some place it under wilderness medicine not only because these practitioners have an innate love for the environment but also have the technical skills to reach and practice in the fragile ecosystems that are the front line. In addition, the clinical skill set is well suited to manage the climate change related health effects. Wilderness medicine inherently overlaps with the international and disaster sub-specialties which also play key roles in responding to the health effects of climate change.

With history as an indicator, it was physicians who organized and created the International Physicians for the Prevention of Nuclear War (IPPNW)

and were subsequently awarded the Nobel Peace Prize for their key influence in prevention of a nuclear holocaust. They felt it was a physician’s duty. The current role of a physician in the arena of climate change is nebulous. Two key leadership roles, as I currently see it, are for physicians to provide



the synthesis / advocacy or perform primary research. The goal of synthesis and advocacy is to re-frame climate change as a public health issue. Multidisciplinary efforts can be aimed at physicians through incorporation of this topic in educational venues and the creation of advocacy organizations similar to the IPPNW. In addition, I feel that every physician should take it upon themselves to learn the basics of the greatest global health threat of the 21st century. This is the nuclear holocaust of our generation, and the time to take action is now.

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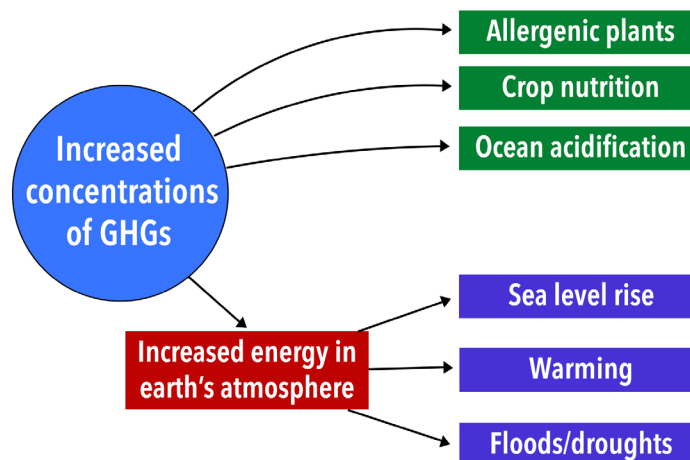


Figure 2: Effects of climate change and greenhouse gases (GHGs) on human health (courtesy of Aaron Bernstein, MD, MPH)

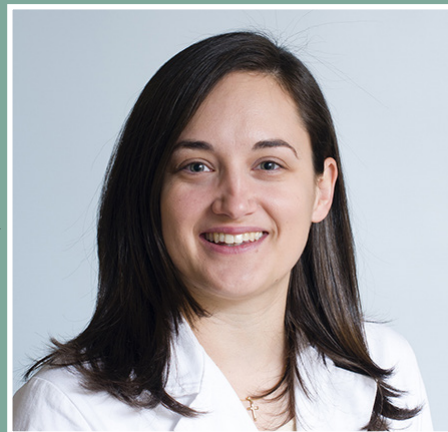
## Reflection

This article, published 7 years ago, outlines what, at the time, was a topic pushing the boundaries of discussion. Since then, our understanding has grown substantially and engagement on this issue has fallen squarely within the house of medicine with engagement of organizations like The New England Journal of Medicine and the National Academy of Medicine. Unfortunately, the lack of action on climate change has placed even more urgency on action occurring this decade. We must implement urgent and ambitious decarbonization to have any hope of minimizing the worst health impacts and health care system disruptions of climate change. There is much work to be done. In the next decade, may the Annals of B Pod also continue to push the boundaries of discussion.

## Biography

Renee N. Salas, MD, MPH, MS is an attending physician in the Department of Emergency Medicine and an Assistant Professor of Emergency Medicine at Harvard Medical School. She is also a Yerby Fellow at the Center for Climate, Health, and the Global

Environment at the Harvard T.H. Chan School of Public Health and Affiliated Faculty and a previous Burke Fellow at the Harvard Global Health Institute. Dr. Salas is a 2013 graduate of the University of Cincinnati Emergency Medicine Residency Program and completed a fellowship in wilderness medicine at Mass General Hospital. She is an expert on the intersection between climate change, health, and healthcare delivery and has dedicated her career to understand and share the impacts of climate change on our health. Her work has been featured in the New England Journal of Medicine, The Lancet, and has appeared in New York Times, NPR, Time, and the Associated Press.



## Anterior Shoulder Dislocation

*Continued from page 4*

- Most initial shoulder dislocations produce a Bankart lesion, particularly in younger patients

Greater tuberosity, acromion, coracoid, clavicle, and humeral neck fractures may also occur

Recurrent dislocation

- 80-94% of patients younger than 20 years at the time of the initial dislocation have a recurrence
- 26-48% of patients from 20-40 years develop recurrent dislocation
- 0-10% of patients older than 40 years have recurrent dislocation

## Treatment

- The first known reduction technique dates back to Egyptian hieroglyphs from 1200 B.C.

- Many different reduction techniques have been described—all available literature consists of case series and no RCTs have been performed to determine which method is best
- Success rates of the various methods range from 73-100%
- A particular method is usually chosen based on patient's level of discomfort, ability to cooperate, and body habitus
- The method used in this case will be described on the following page

## Sedation vs. Lidocaine

- Review showed similar success rates, decreased length of stay for intrarticular lidocaine, and increased rates of adverse events in IV sedation<sup>2</sup>
- Most studies used 20 mL of intraarticular 1% lidocaine

## Scapular Manipulation (figure 1,2)

- Success rate estimate at approximately 90%



- Gentle method of reduction with no reported complications
- Application of longitudinal traction and slight external rotation of the humerus in 90° of forward flexion favors successful reduction
- Reduction can be performed with patient sitting upright or lying prone, but the prone position is much easier
- Scapular manipulation is achieved by stabilizing the superior aspect of the scapula with one hand and pushing the inferior tip of the scapula medially towards the spine

## Dispo

- Displaced Bankart fractures, humeral neck fractures, greater tuberosity fractures, and shoulders unable to be reduced may need operative management
- Otherwise, patients successfully reduced should be put in a sling and have Ortho follow-up arranged for 1 week
- Some evidence that a sling with the patient in external rotation decreases the incidence of recurrent dislocation, but we do not have this type of sling in the CEC<sup>4</sup>

## Bottom Line

- You should get pre and post reduction films
- You should use the reduction technique that best suits the patient as none is shown to be definitively superior to another
- Intraarticular lidocaine works and can prevent you from doing a time-consuming sedation

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Figure 1: Patient lying prone, with arm at 90° of forward flexion and downward traction provided by hanging weights



Figure 2: While downward traction is being applied by weights or an assistant, the superior aspect of the scapula is stabilized while the inferior portion is rotated medially towards the spine

## Reflection

I was honored to be invited to reflect on my experience as a co-editor (with Dr. Laura Heitsch) of AOBP and look back on this experience as an important one in my training. Taking the time to do careful analysis of the literature helped me become both a better clinician and educator. I was glad you chose the shoulder reduction article—*intraarticular lidocaine for shoulder reductions* is a good example of a lasting practice I developed from my time at AOBP.

In honor of UC's 50th Anniversary, I would also like to submit a Haiku—probably my favorite part of AOBP 2008-2009.

*The first place teaching*

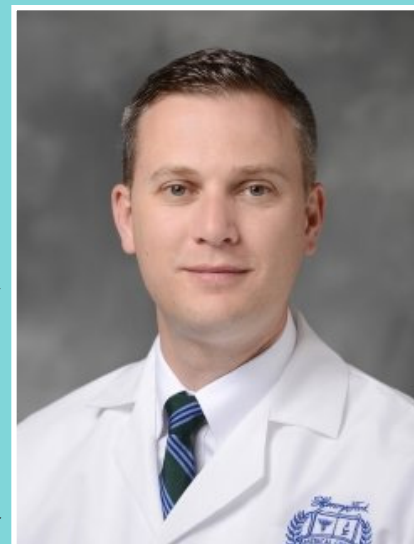
*A specialty saving lives*

*Millions and counting*

## Biography

Seth Krupp M.D.

I attended The Ohio State University for undergrad, University of Chicago for medical school and completed training at University of Cincinnati in EM in 2009. Currently, I am in my 12th year at Henry Ford Hospital in Detroit, MI and serve as the Vice Chair of Operations and Medical Director. I still have family in Cincinnati and love to visit whenever the opportunity arises!





# TRANSVENOUS PACING

David Silver MD

University of Cincinnati EM Alumnus '11

## Case Introduction

This patient has third degree heart block. Transcutaneous pacing is usually the first step in management. If transcutaneous pacing fails, however, transvenous pacing is indicated. This is a procedure rarely performed in the Emergency Department, but has life-saving implications. Every emergency medicine physician should know how to perform this procedure quickly and effectively.

## Pre-Procedure

### Indications

- Symptomatic bradyarrhythmias secondary to sinus node dysfunction (e.g. sick sinus syndrome, sinus arrest, or sinus bradycardia).
- Cardiac conduction abnormalities (e.g. type two second degree or third degree heart block).
- Overdrive pacing of tachyarrhythmias (e.g. SVT, atrial flutter, or v-tach).
- Failure of other pacing devices.

### Contraindications

- No absolute contraindications.
- Hypothermia - may lead to ventricular fibrillation when pacing is attempted.

### Equipment

- Universal precaution packet.
- Central line kit with a venous introducer system.
- Sterile sleeve.
- Transvenous bipolar pacing catheter.
- Pacemaker generator with battery and cable.
- Transcutaneous external pacer.
- Cardiac monitor.
- Crash cart with resuscitation equipment and defibrillator.
- Pacing generator.

### Anatomy

- The right internal jugular and left subclavian veins have the most direct anatomic pathway to the right ventricle and are the preferred routes.
- Femoral veins are associated with easy dislodgment, infection, and increased risk of thrombophlebitis.
- The brachial vein is seldom used in the emergency setting.

## Procedure

Transcutaneous pacing is usually initiated while the transvenous pacer is being set up. There are two components to transvenous pacer placement. First, a central venous introducer is needed. There are many different pacer wires, so make sure the proper size introducer is used for your institution's pacer wire. The introducer is placed via the traditional seldinger technique (like placing a central venous catheter) and will not be reviewed here.

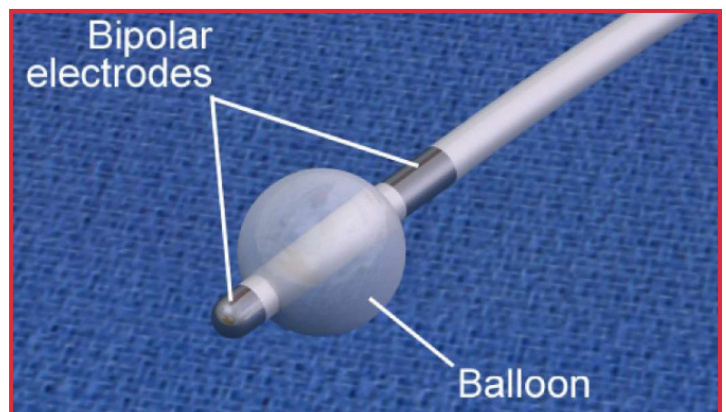
Once the introducer is in place, the pacer wire may be inserted. You will need an assistant to perform this procedure.

1. Attach the compressed sterile sleeve to the venous introducer.



2. Check the balloon by inflating and deflating with air before placing the pacer wire into the sleeve. The typical volume is 1.2-1.5 mL of air.

3. Have your assistant attach the pacer wires to the energy source.



- Use demand mode and turn to the highest level (rate around 80 bpm).
- With the balloon deflated, insert into the collapsed sterile sleeve.



- Inflate the balloon once the wire has passed through the end of the introducer.
- Advance the wire smoothly and watch the monitor for ECG capture. This appears as a pacer spike followed by a wide QRS complex.



- The pacer wire should be in the right ventricle at 15-20 cm. If no capture is seen by 25 cm, deflate the balloon and withdraw the wire. Advance with an inflated balloon and attempt again.
- Once captured, deflate the balloon and advance the wire another 1-2 cm to seat it within the endocardium.
- Extend the sterile sleeve to its full length and clamp down the valve to prevent subsequent movement of the wire.



- Turn the output from the energy source down to 10 mA in non-emergent cases and 15-20 mA in emergent cases, then slowly turn it back on to determine the pacing threshold.
- Pacing threshold is not constant over time because an endothelial sheath forms around the tip of the electrode. Set the output at two to three times the pacing threshold to prevent loss of capture.

13. If loss of capture occurs, assess the patient first, then check the connections and settings. If the patient is hemodynamically stable, test the pacing threshold again. If the patient is unstable, quickly increase the output amperage until capture occurs. Turning the patient to his/her left side may improve capture by increasing contact between the electrode and myocardial tissue.

## Post-Procedure

### Care

- Chest x-ray.
  - Assess proper placement of the pacing wire and any complications.
- 12-lead ECG.
  - Look for captured pacer spikes before every QRS.
  - The QRS should be in a LBBB pattern.
  - RBBB suggests coronary sinus pacing, or septal perforation.
- Admit to CCU or MICU.

### Complications

- Arrhythmias.
- Pacemaker malfunction:
  - Absence of stimulus.
  - Failure to capture.
  - Over-sensing (inappropriate inhibition).
  - Under-sensing (inappropriate pacing when there should be inhibition).
- Lead dislodgement.
- Pneumothorax.
- Cardiac perforation.
- Cardiac tamponade.
- Damage to vessel or valve structures.
- Arrhythmias.
- Thromboembolism.
- AV fistula formation.
- Endocarditis.
- Sepsis/Infection.

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## Reflection

I wrote this procedural review of transvenous pacing ten years ago while working as a senior resident in B pod. Since that point in time, I have worked as a solo coverage nocturnist in the community, in a community setting with multiple attendings, and as a teaching attending in a quaternary care center. However, my first transvenous pacer placement came during solo coverage at 3am when I was just two months out of residency. I had to think back to this exact article and in all honesty, I struggled. It was this experience that caused me to develop a practice that I have continued for the last ten years: each month I watch videos (often YouTube) on the four procedures that



# CEREBRAL VENOUS THROMBOSIS

Natalie Kreitzer MD

University of Cincinnati EM Alumnus '14

## History of Present Illness

The patient is a female in her 60s with a PMH significant for DM, HTN, CAD, and AAA who presented to the ED with a frontal headache for the past one and a half weeks. She describes the pain as moderate to severe, pressure-like, throbbing, and constant. She was recently admitted to an OSH and found to have increased intracranial pressure of 48 mmHg on lumbar puncture. She had 20cc of CSF removed at that time with resolution of her headache, and she was discharged with a normal MRI. She was told to follow up with a neurologist but was unable to do so due to lack of insurance. She states that she presented tonight because she is out of the Percocet prescribed to her for the headache and now the pain is unbearable. She denies any injury or trauma, fevers or chills, numbness or weakness, change in vision, confusion, neck pain or stiffness, chest pain, abdominal pain, recent weight loss, or instability with walking.

Secondarily, she also complains of chronic lower back pain that she has had for several months, without any red flags concerning for cauda equina or infectious etiology.

At the OSH, the following workup had been performed: CT head WO contrast, MRI W and WO contrast, UA, BMP, LFTs, CBC with Diff, CSF studies, troponin, and EKG. The only notable finding was on the MRI: FLAIR and T2 weighted imaging demonstrated a tiny 4 mm foci of T2 hyperintensity within the right basal ganglia-thalamus region; likely an area of old tiny lacunar infarct.

The patient's vital signs remained stable throughout her course with the exception of an elevated blood pressure to 181/99. A CBC showed a WBC of 11.7 with no left shift, and a BMP

showed hyperglycemia to 152 and hypochloremia of 97. She continued to complain of pain after the administration of 2 doses of 5-325mg Percocet, and the decision was made to order a CTV of the head with and without contrast and without contrast that demonstrated an internal jugular vein thrombus.

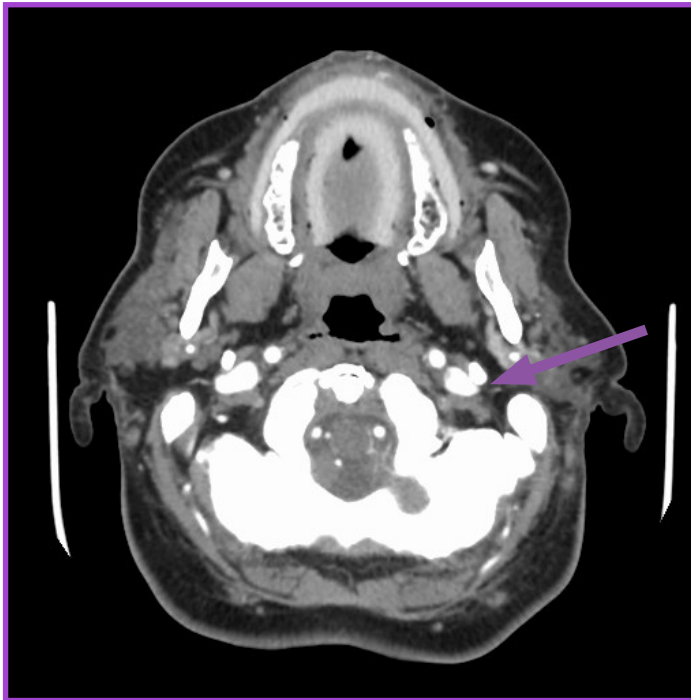


Figure 1: CT Venogram. There is a nonocclusive thrombus within the visualized portion of the left internal jugular vein extending into the jugular bulb with a trace amount within the left sigmoid sinus.

The patient was started on a heparin drip in the ED and admitted to neurology on step-down status. A hypercoagulable workup was done including Anticardiolipin, Factor V Leiden, prothrombin, lupus anticoagulation panel, ATIII deficiency, homocysteine, APCR, and PC/PS deficiency. Fibrinogen and FVIII were elevated, but heme-onc thought this was likely a stress response. A PET scan was even performed as the patient had a known lung nodule, but this was unrevealing. She was bridged to warfarin and will remain on life-long anticoagulation per heme-onc.

## Discussion

### Definitions

Internal jugular vein thrombosis refers to an intraluminal thrombus that can occur anywhere from the intracranial IJ vein to the junction of the IJ and the subclavian where it forms the brachiocephalic vein.<sup>2</sup> Those that occur in the intracranial portion of the IJ vein are also referred to as cerebral venous thrombosis (CVT). Dural (cerebral) venous sinus thrombosis is actually a subset of CVT and often coexists with deep or cortical vein thrombosis.<sup>3</sup>

### Epidemiology

Overall, internal jugular vein thrombosis, CVT, and dural venous sinus thrombosis are underdiagnosed. In large teaching hospitals in the US, only 5 to 10 patients with CVT are admitted annually. However, an autopsy study in the 1970s found a prevalence rate of 9%, but this is likely an overestimate. The

major risk factors include: head injury and mechanical precipitants, OCPs, malignancy, infection, pregnancy/peripartum, and pro-thrombotic conditions (either congenital or acquired). No underlying risk factor for CVT is found in 13% of patients.<sup>2</sup>

### Clinical Manifestations

The symptoms can be subtle and vary greatly by location. CVT most frequently presents as headache, but also often presents as hemorrhagic infarction in areas that are atypical for arterial vascular distribution. The symptoms, however, vary as widely from headaches, nausea, vomiting, and seizures to pseudotumor cerebri with increased ICP and headache to cranial nerve syndromes like vestibular neuropathy, tinnitus, double vision, unilateral deafness, facial weakness, and other visual disturbances.<sup>2-4</sup>

### Diagnosis

MRI and MRV are the studies of choice for diagnosing CVT. CT and CTV can also lead to the diagnosis. The cause of coagulopathy, when not readily apparent, also deserves investigation.<sup>3</sup>

### Treatment

Medically, begin anticoagulation therapy, though no large studies are available to substantiate the necessity of heparin bridging to Coumadin or Lovenox.

Since the condition is underdiagnosed, this seems to suggest that patients will do well regardless of anticoagulation, but ultimately the risk of PE is unknown. The number quoted from a relatively small retrospective study performed 25 years ago was 5%. Some

### Reflection

In the years since 2013, I have had the opportunity to complete a Neurocritical Care Fellowship, and I have spent a few years working as an attending in the UC Neuroscience Intensive Care Unit (NSICU). During that time, I've seen quite a few cases of venous sinus thrombosis. Throughout those years, landmark trials demonstrated substantial benefit to patients who receive endovascular therapy for acute ischemic stroke. The same cannot be said for the treatment of venous sinus thrombosis. Indeed, in 2020, the TO-ACT trial demonstrated no significant difference in endovascular therapy (thrombectomy or localized thrombolysis) when compared to anticoagulation, although this trial was criticized because of its small sample size (n=67).<sup>1</sup> Although the treatment has not changed much, I am much less hesitant to begin therapeutic anticoagulation as fast as possible, even in the setting of a venous infarct hemorrhage.

As a resident, I was always an avid reader of *Annals of B Pod*. In particular, as an intern, I felt a bit overwhelmed reading textbooks or journal articles, so I appreciated the *Annals of B Pod* approach to learning. I was a co-editor my R4 year, so I had the opportunity to learn from the cases, as well as become a better writer. Over time, *Annals of B Pod* has evolved, and is now an impressive layout with artwork, images, and online content, while still maintaining the “feel” of being an intern in *B Pod*.

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case reports describe the use of thrombolytics, but only in patients with extensive clot extending into the sigmoid sinus, which resulted in few complications. When infection is suspected, antibiotics are indicated. IJ vein thrombosis rarely requires surgical intervention.<sup>3,4</sup>

### Take Away Points

Knowledge of the venous system of the cerebrum is important to evaluate patient with CVT because symptoms associated with the condition are related to the area of thrombosis. For example, infarction can occur with sagittal sinus or cortical vein thrombosis secondary to tissue congestion with obstruction. However, lateral sinus thrombosis can present with headache and pseudotumor cerebri-like picture. Cavernous sinus thrombosis alternatively can present as cranial nerve palsies with compression of the nerves as they pass through the sinus. Studies have failed to prove, however, that location of the headache correlates with location of the thrombosis.<sup>2-4</sup>

### Things We Learned

Many cerebral venous thromboses present as a hemorrhagic stroke, but if not, they can be highly variable in presentation and difficult to detect. Thus, headache and isolated elevated ICP should involve a more extensive differential diagnosis than just pseudotumor.

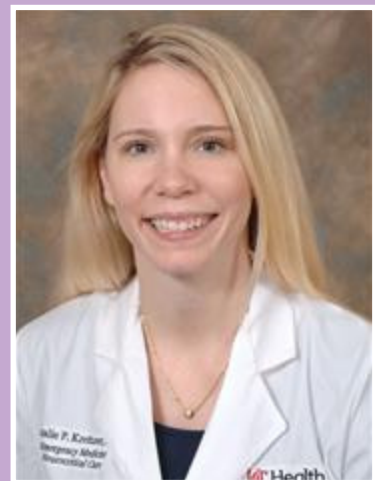
### References

1. Actual patient images  
2. Up-to-date “Cerebral Venous Thrombosis”  
3. Medscape “Cerebral Venous Thrombosis”  
4. Medscape “Venous Sinus Thrombosis”

### Biography

Natalie Kreitzer is originally from Logan, West Virginia, and attended West Virginia University, where she had undergraduate degrees in Piano Performance and Biology. She then attended medical school at West Virginia University, where she was elected to Alpha Omega Alpha. After that, she completed an Emergency Medicine residency at the University of Cincinnati. As a fourth-year resident, she was an editor of *Annals of B Pod*. Her residency was followed by a three-year combined T32 Neurocritical Care and Neurovascular Emergencies fellowship, where she earned a master's degree in Clinical and Translational Research. She was recently awarded a K23 award, titled, “Caregiver Wellness after Traumatic Brain Injury (CG-Well): An Intervention Designed to Promote Well-being in Caregivers of Acute Moderate to Severe Traumatic Brain Injury.”

Dr. Kreitzer is currently an associate professor of emergency medicine at the University of Cincinnati and works clinically in the emergency department as well as the Neuroscience Intensive Care Unit and the University of Cincinnati Stroke Team.



# HETEROTOPIC PREGNANCY



Caitlin Schaninger MD

University of Cincinnati EM Alumnus '14

## History of Present Illness

The patient is a 32-year-old female who presents with abdominal pain and vaginal bleeding. She is currently 7 weeks pregnant based on her last menstrual period. She states that she has had right lower quadrant pain and vaginal bleeding for the past two hours. The patient describes her abdominal pain as sharp and radiating to her right groin. She admits to one episode of emesis. The patient denies diarrhea, dysuria, frequency, hematuria, headache, or dizziness.

This is the patient's fourth pregnancy. Her previous three pregnancies resulted in two full term live births and one premature live birth. All three children are living.

## Past Medical History

Polycystic ovarian syndrome  
Kidney stones  
Pre-eclampsia

## Past Surgical History

C-Section x 2

## Medications

Multivitamin

## Allergies

None

## Physical Exam

General: Alert, appears to be in pain and looks uncomfortable






HEENT: Conjunctivae pink, anicteric, mucous membranes moist

Neck: Supple with full range of motion

Pulmonary: Clear to auscultation bilaterally without wheezing or crackles. Non-labored breathing.

Cardiac: Regular rate and rhythm, no appreciable murmurs or rubs.

Abdomen: Soft, non-distended. Tender to palpation in the right lower quadrant and right groin with voluntary guarding. No organomegaly or masses.

	T 97.2 F
	HR 97
	BP 141/86
	RR 18
	SpO2 100% on RA

Genital urinary: No costovertebral angle tenderness bilaterally. Normal appearing external female genitalia. A small amount of scant brown discharge is visualized in the posterior fornix. The cervical os is closed.

There is no cervical motion tenderness, adnexal fullness or masses. Patient had a tender uterus on bimanual exam that did not lateralize.

Musculoskeletal: Moving all extremities spontaneously

Skin: Warm and dry

## Hospital Course

The patient presented with abdominal pain and vaginal bleeding in the setting of pregnancy but without a confirmed intrauterine pregnancy (IUP). She then had a bedside transvaginal ultrasound performed that showed an IUP as demonstrated by the presence of a yolk sac within a gestational sac. A mild amount of trace free fluid was also noted in the Pouch of Douglas. Threatened abortion was initially considered as the etiology for the patient's symptoms however serial abdominal exams in the emergency department were significant for increasing point tenderness in the right lower quadrant localizing to McBurney's point. This raised concern for possible appendicitis as an etiology for the patient's symptoms. An MRI of the abdomen and pelvis was performed which showed a right adnexal mass with free fluid in the pelvis consistent with a ruptured ectopic pregnancy. A formal transvaginal ultrasound was performed which confirmed the presence of both an intrauterine pregnancy and an ectopic pregnancy. The patient was transferred to an OSH for definitive management of heterotopic pregnancy with ruptured ectopic pregnancy.

## Discussion

The patient presented with abdominal pain and vaginal bleeding in pregnancy with a confirmed IUP based on bedside transvaginal ultrasound. Her evolving abdominal exam led her practitioner to discover a heterotopic pregnancy with a ruptured ectopic pregnancy on advanced imaging.

Heterotopic pregnancy is the simultaneous existence of an intrauterine and an extrauterine, or ectopic, pregnancy. In natural pregnancies, which originate without the use of assisted reproductive technologies (ART), heterotopic pregnancies are very rare, and occur in 1 in 30,000 pregnancies. With ART, the incidence of heterotopic pregnancies is between 1 in 100 and 1 in 500 pregnancies.<sup>3,5</sup>

Risk factors for heterotopic pregnancy in a naturally conceived pregnancy are the same as those for ectopic pregnancies: tubal damage due to pelvic inflammatory disease, endometriosis or tubal surgery.<sup>4</sup> Heterotopic pregnancies are most often located

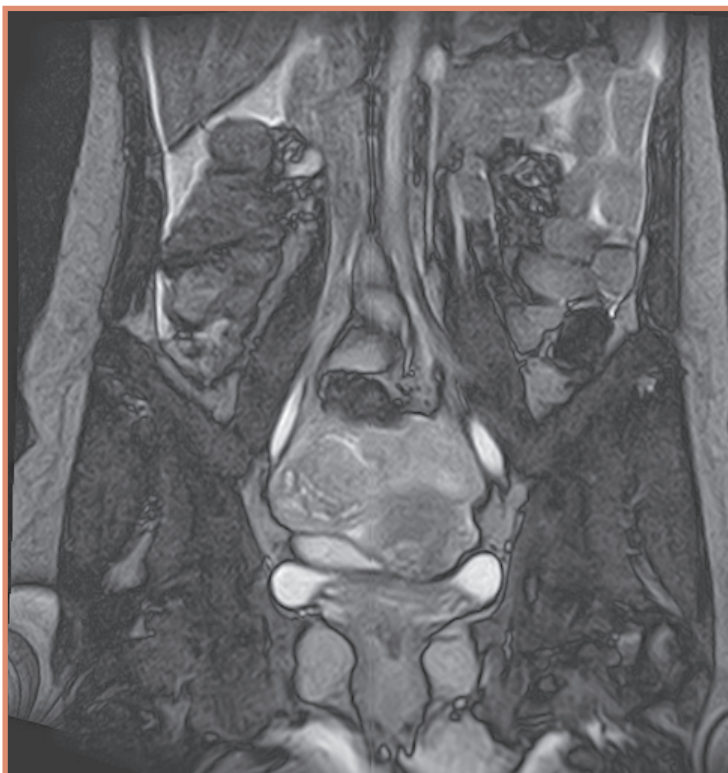


in the fallopian tubes but can also implant in the corneal region, cervix, ovary, or on old cesarean scars. The median gestation at time of diagnosis for heterotopic pregnancy is between 6-9 weeks.<sup>3</sup> Patients with heterotopic pregnancy commonly present with abdominal pain, vaginal bleeding, or hemodynamic instability.

Heterotopic pregnancy is a diagnostic dilemma in women who have naturally conceived pregnancies because they have a confirmed intrauterine pregnancy on imaging that has the potential to modify future workup. In the emergency department, the diagnosis of ectopic pregnancy is considered a diagnosis of exclusion, which precariously leaves the opportunity for a missed heterotopic pregnancy.<sup>2,3</sup> In patients with confirmed intrauterine pregnancies the diagnostic pathway for abdominal pain and or vaginal bleeding in the presence of a closed cervical os includes threatened abortion versus other acute abdominal pathology such as appendicitis, ovarian torsion, and heterotopic pregnancy.<sup>3,4</sup>

Heterotopic pregnancies are managed differently than ectopic

pregnancies because of the co-existence of an intrauterine pregnancy. A ruptured ectopic pregnancy is treated with emergent surgical intervention by OBGYN. Often, the intrauterine pregnancy can be carried to term. If the ectopic pregnancy is not ruptured, the options for treatment expand depending on the mother's wishes regarding her intrauterine pregnancy.<sup>3</sup>



*MRI Abdomen - Complex cystic abnormality in the right adnexa suspicious for a ruptured ovarian cyst or a ruptured ectopic pregnancy and extensive complex ascites suggesting possible hemorrhagic ascites.. Nonvisualization of the appendix. Appendicitis cannot be excluded.*

### Lessons Learned

Although the practitioner in this case did not initially suspect a heterotopic pregnancy, she maintained clinical suspicion for an acute abdominal process as the etiology for the patient's persistent symptoms and pursued advanced imaging which led to the correct diagnosis. Her #lessonlearned was to always pay attention to the "spidey sense" that accompanies certain patients. If you think there may be something else going on with a patient, it is important to investigate further or observe them for a longer amount of time!

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## Reflection

I still remember working to convince radiology to get an MRI on that overnight shift. The more seasoned I get in my career, the more I listen to any nagging gut feeling that tells me to dig a little deeper into complicated patients or something that doesn't quite fit that I have been thinking about. I have less of an internal monologue talking myself out of doing a test or procedure than I used to. It is less of a "spidey sense," but rather converting system one thinking into system two thinking whenever I'm wavering, whereas in residency it was the opposite. Emergency medicine is a constant balance between being methodical and trusting your gut.

Among the many magazines one receives as a physician, AOBP was always one of my favorites to peruse. I was impressed with the innovations my colleagues had come up with, including fellow alumni Dr. Steuerwald's trans-tracheal ventilation kit that is still somewhere in the trunk of my car. I always learned something new from the teaching pearls based on cases submitted. It's like a miniature Grand Rounds that you can complete at your own pace. Thank you to the AOBP editing team for continuing to carry the torch so that others can continue to learn.

## Biography

Dr. Schaninger is a partner with The Permanente Medical Group (TPMG) in the Bay Area, California, where she serves as the Assistant Chief of Efficiency and Flow for the Greater Southern Alameda Area and the Assistant Chair of Chiefs of Emergency Medicine for Northern California, TPMG. She uses her operations experience to build relationships between teams and make processes less frustrating or slow.



► **Transvenous Pacing**  
*Continued from page 11*

are extremely rare in Emergency Medicine yet are lifesaving in the moment (transvenous pacing, thoracotomy, surgical and seldinger cricothyrotomy, and umbilical lines). Without a cadaver lab or simulation center that are available to you as trainees during residency, this is the closest thing I can do to retain memory and it has paid dividends.

Another piece of advice that I wish I had paid more attention to during residency is the importance of learning the full extent of resuscitation possibilities, rather than being distracted by the glamor of procedures. Speaking from experience, when working solo coverage in the community, the last thing you want is to get stuck putting in a central line. No matter how fast you are, you will still come out to at least five new patients and five more to dispo.

In the end, trust in the training you are getting over your four years at UC. You will come out ready to face whatever comes your way, as I did.

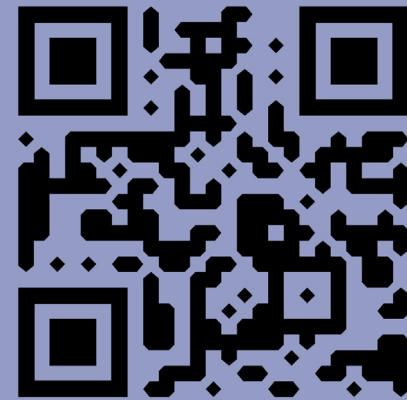
**Biography**

After graduating residency, Dr. Silver returned to New York where he has worked for the past 10 years as a nocturnist at Northwell Health. He is currently an assistant professor of Emergency Medicine at Zucker School of Medicine/Hofstra University. He also continues his interest in EMS by giving continuing medical education lectures for local volunteer fire departments. He has two children, 5-year-old Brynn and 3-year-old Gavin, with Mindy, his wife of 6 years.

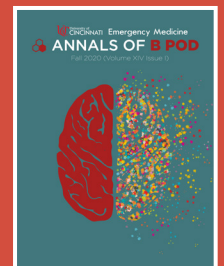
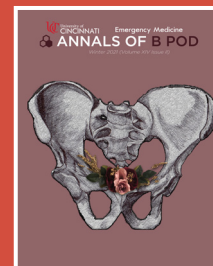


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