

# Welcome to the Regions Hospital Emergency Medicine Residency

## Welcome Class of 2015

The Regions Emergency Medicine Residency is sponsored by the HealthPartners Institute for Medical Education (IME). The IME is the clinical learning arm of HealthPartners and consists of the Center for Continuing Professional Development, the Center for Graduate and Undergraduate Clinical Education which administers residencies at Regions Hospital, HealthPartners Simulation and Regions Hospital Medical Library. The executive director of the IME is Carl Patow, MD, MPH, who is also the Regions Hospital designated institution official.



From 2011 graduation dinner



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## Toxic Shock Syndrome — April 5, 2012

by STEPHANIE TAFT, MD on APRIL 5, 2012 · [LEAVE A COMMENT](#)

Toxic Shock Syndrome By Annalisa Rudser Diagnosis Fever, rash, hypotension, multi-organ dysfunction Specific Diagnostic Criteria Fever of 38.9 C (102 F) or higher Rash (diffuse macular erythema) resembles scarlet fever (Desquamation of skin 1 to 2 weeks after onset of disease ... Continue reading →

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## Severe Low Back Pain — March 22, 2012

on MARCH 22, 2012 by STEPHANIE TAFT, MD

From the presentation "Severe Low Back Pain" By Michael Goertz, MD, MPH Non-specific Low Back pain Lifetime occurrence 80-90% (majority of people experience severe low back pain at some point in life) 90% markedly improve or recover in 6 weeks ... Continue reading →

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## Spontaneous Retroperitoneal Hemorrhage — February 23, 2012

on MARCH 15, 2012 by STEPHANIE TAFT, MD

Critical Case Conference — Discussion by Dr. Kate Katzung Variety of causes, including: Post-procedure (any sort of vascular intervention from carotid to femorals) Coagulopathy Renal angiomyolipoma, renal cell carcinoma Trauma Dialysis Although rare, spontaneous retroperitoneal hemorrhage is a known complication ... Continue reading →

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## Spontaneous Esophageal Rupture — March 15, 2012

on MARCH 15, 2012 by STEPHANIE TAFT, MD

Boerhaave's (Spontaneous Esophageal Rupture) — Discussion led by Dr. Felix Ankel with discussion by Dr. Rob Bulander (HP Staff Surgeon) Additional information from reference below True esophageal rupture is uncommon Most times abnormalities (symptoms/chest x-ray findings) seen on left (e.g. ... Continue reading →

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## Abdominal Compartment Syndrome — February 9, 2012

on FEBRUARY 9, 2012 by STEPHANIE TAFT, MD

Abdominal Compartment Syndrome by Dr. Peter Baggenstos ACS — accumulation of fluid in abdominal space (from injury, surgery or massive fluid resuscitation/capillary leak) causing increased abdominal pressure, leading to end organ dysfunction [www.wsacs.org](http://www.wsacs.org) (World Society of the Abdominal Compartment Syndrome) ... Continue reading →

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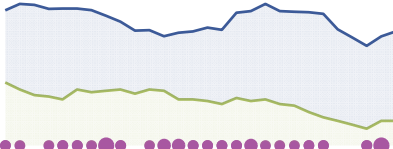
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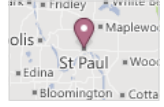
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**Inferior hyperacute T-waves and ST elevation. Angiogram is normal. What happened?**

I recently received this from a reader: "I recently saw this 31 year old male with central chest tightness that started at 530am and lasted 30 min. He came to ED pain free at 7 AM with no other symptoms. The initial ECG showed J point elevation in inferior leads. The morning doctors decided to do serial cardiac enzymes. The first trop I was negative and subsequently went up slightly. Patient was managed..."




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Regions Hospital Emergency Medicine Residency created the event [Regions EM Residency Advocacy & Ethics Day](#).

March 15



Regions Hospital Emergency Medicine Residency

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**Best Of: Flying After Pneumothorax**

Patients who have sustained a traumatic pneumothorax occasionally ask how soon they can fly in an airplane after they are discharged. What's the right answer? The basic problem has to do with Boyle's Law (remember that from high school?). The volume of a gas varies inversely with the

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Regions Emerg Med @regionsem 27 Mar  
ED Conference 3/29/12, 7:30-8:30 Ankle Fractures, Pfannestien, Auditorium 8:30-10:00 Critical Case,... <fb.me/1By9Zwwgz>

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**emshorts***Just another WordPress.com site***Toxic Shock Syndrome — April 5, 2012**Posted on [April 6, 2012](#)**Toxic Shock Syndrome**

By Annalisa Rudser

**Diagnosis**

Fever, rash, hypotension, multi-organ dysfunction

**Specific Diagnostic Criteria**

Fever of 38.9 C (102 F) or higher    Rash (diffuse macular erythema) resembles  
 of skin 1 to 2 weeks after onset of disease — not helpful to    diagnosis)    Hypo  
 <90 mm Hg, orthostatic drop of 15 mm Hg    or more, or orthostatic dizziness;  
 laboratory abnormalities in at least three organ systems:

Gastrointestinal: nausea and vomiting, diarrhea    Muscular: myalgia or CK >  
 membrane: vaginal, oropharyngeal (strawberry tongue), or    conjunctival l  
 conjunctivitis)    Renal: BUN or Cr level > twice normal or pyuria > 5 cells  
 transaminases at least twice normal level    Hematologic: thrombocytopenia,  
 Neurologic: disorientation or altered consciousness without focal findings    R  
 of other cause of illness

Results from colonization of mucous membranes or wounds

Mediated by toxins

&lt; 50% related to menses (i.e. results from other types of infection)

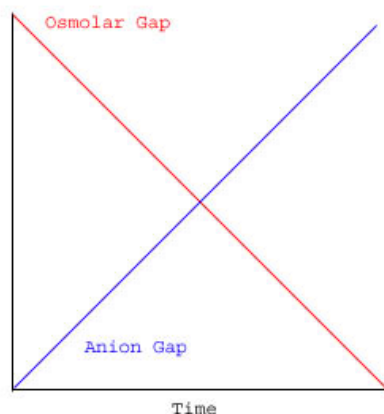
Abrupt onset (consider when there is a sudden worsening after another illness)

Clindamycin

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# Twin Cities Toxicology



I apologize for the gap between my last post and now. I'm in the middle of exploring some other potential blog formats/hosts. Speaking of gaps...

A recent emergency department consult brought up an interesting and controversial topic within the realm of medical toxicology. The case, in brief, was a middle aged patient with metabolic acidosis, elevated anion gap, and elevated serum osm gap. He presented to the ED with suicidal thoughts, admitting to heavy recent EtOH use and consistently denied even the chance of ingesting any other liquid or alcohol. The primary history takers in the ED thought his history was consistent and believable. The emergency providers were admitting the patient with diagnoses of alcoholic ketoacidosis, lactic acidosis, agitation, hypertension, tachycardia, and dehydration. The patient had a history of heavy alcohol abuse, a present-but-low serum EtOH concentration and normal renal function. The question that came up during the discussion between the admitting physician and the ED was about whether or not a "toxic alcohol" screen (I put this in quotes because it always annoys me that according to that test name apparently ethylene glycol and methanol are toxic but ethanol isn't) should be done in this context of metabolic acidosis, elevated anion gap, and osm gap. The list of core questions that need to be answered here are (and this list is not all-inclusive):

- 1) Why is there a lactic acidosis?
- 2) Is the anion gap explained by the lactate and AKA?
- 3) Is the osm gap explained by the lactate and AKA?
- 4) Is an osm gap reasonable and accurate enough to use to make decisions?
- 5) Finally, in the case of a middle-aged patient with AKA, lactic acidosis, elevated anion gap, elevated osm gap, do you need to rule out ethylene glycol and methanol as causes, and do you need to order a "toxic alcohol" screen in order to do that?

The following is a short (and again not by any means all inclusive) list of articles that I've collected and used to help answer questions in this setting in the past. Check them out, or just keep them somewhere for yourself, and I'll come back to give my version of how to approach this issue in the next post.

<http://www.ncbi.nlm.nih.gov/pubmed/2400167>

<http://www.ncbi.nlm.nih.gov/pubmed/17389437.1>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1950181/?tool=pubmed>

<http://www.cmaj.ca/content/177/5/489.2.full>

<http://www.ncbi.nlm.nih.gov/pubmed/8215742>

<http://www.ncbi.nlm.nih.gov/pubmed/9247780>

<http://www.ncbi.nlm.nih.gov/pubmed/21255564>

<http://www.ncbi.nlm.nih.gov/pubmed/15862086>



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## New Broslow Tape Changes

Posted on [April 5, 2012](#)

**Karen Manor RN, CEN, CPEN**

Regions Hospital Emergency Medical Services Critical Care Education Specialist gives us an update on the recently released 2012 Braselow tape.

The new Braselow tape has now been released. I have looked it over a bit.

It has the new dosing for defibrillation that go along with the new AHA guidelines. Other changes include some small changes in medications. The biggest I see is the change from 15mg/kg/dose to 20 mg/kg/dose for Fosphenytoin and Phenytoin. I think using it as is for reference is fine.

### Recent Posts

- [New Broslow Tape Changes](#)
- [Immediate Trial Results](#)  
Published in JAMA
- [EM Resident Conference 2/23](#)
- [EM Resident Conferences](#)
- [Toxicology Grand Rounds!](#)

### Archives

- [April 2012](#)
- [March 2012](#)
- [February 2012](#)
- [January 2012](#)
- [December 2011](#)
- [November 2011](#)
- [October 2011](#)
- [September 2011](#)
- [July 2011](#)
- [June 2011](#)
- [May 2011](#)

# The Trauma Professional's Blog

## Best Of: Flying After Pneumothorax

**Patients who have sustained a traumatic pneumothorax occasionally ask how soon they can fly in an airplane after they are discharged. What's the right answer?**

The basic problem has to do with Boyle's Law (remember that from high school?). The volume of a gas varies inversely with the barometric pressure. So the lower the pressure, the larger a volume of gas becomes. Most of us hang out pretty close to sea level, so this is not an issue.

However, flying in a commercial airliner is different. Even though the aircraft may cruise at 30,000+ feet, the inside of the cabin remains considerably lower though not at sea level. Typically, the cabin altitude goes up to about 8,000 to 9,000 feet. **Using Boyle's law, any volume of gas (say, a pneumothorax in your chest), will increase by about a third on a commercial flight.**

The physiologic effect of this increase depends upon the patient. If they are young and fit, they may never know anything is happening. But if they are elderly and/or have a limited pulmonary reserve, it may compromise enough lung function to make them symptomatic.

Commercial guidelines for travel after pneumothorax range from 2-6 weeks. The Aerospace Medical Association published guidelines that state that 2-3 weeks is acceptable. The Orlando Regional Medical Center reviewed the literature and devised a practice guideline that has a single Level 2 recommendation that commercial air travel is safe 2 weeks after resolution of the pneumothorax, and that a chest xray should be obtained immediately prior to travel to confirm resolution.

**Bottom line: Patients can safely travel on commercial aircraft 2 weeks after resolution of pneumothorax. Ideally, a chest xray should be obtained shortly before travel to confirm that it is gone. Helicopter travel is okay at any time, since they typically fly at 1,500 feet or less.**

### References:

- Practice Guideline, Orlando Regional Medical Center. Air travel following traumatic pneumothorax. October 2009.
- Medical Guidelines for Airline Travel, 2nd edition. Aerospace Medical Association. Aviation, Space, and Environmental Medicine 74(5) Section II Supplement, May 2003.

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The Trauma Professional's Blog provides information on injury-related topics to trauma professionals. It is written by Michael McGonigal MD, the Director of Trauma Services at Regions Hospital in St. Paul, MN. Regions is a Level I Adult Trauma Center, and has partnered with Gillette Children's Specialty Hospital to become the first Level I Pediatric Trauma Center in the Upper Midwest.

Want to see a post on a specific topic? [Click here or go to www.regionstraumapro.com/ask.](#) You can also email me at [Michael.D.McGonigal@HealthPartners.com](mailto:Michael.D.McGonigal@HealthPartners.com)

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## Best Of: The Downside Of Not Taking Your Anticoagulant

We've all been faced with injured patients who are taking some kind of anticoagulant, and it complicates their care. Many trauma professionals just say, "they just shouldn't take this stuff any more." **Why can't we just stop them in patients at risk for injury (e.g. an elderly patient who falls frequently)?**

Two major risk groups come to mind: those taking the meds who have DVT (or a propensity to get it), and patients with atrial fibrillation who take them to decrease stroke risk. I was not able to find much info (yet) on the former category. But there is a series of nicely done studies based on work from the Framingham Heart Study.

The Framingham study started in 1948, and has been following over 5,000 people for the development of cardiovascular disease. In this particular analysis, 5070 patients who were initially free of disease were analyzed for development of atrial fib and occurrence of stroke. Anticoagulants were seldom used in this group.

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