CLINICAL VIGNETTES

SEPTIC PULMONARY EMBOLI DUE TO ANAEROBIC PHARYNGEAL INFECTION. P.Abtahi. The Reading Hospital and Medical Center, Reading, PA.
LEARNING OBJECTIVES: 1. Diagnose of Lemierre syndrome.
CASE: A previously healthy 15 year-old female presented to the ER with severe right-sided chest pain and respiratory distress. One week earlier, she developed a sore throat. She was evaluated in an outpatient clinic and was started on amoxicillin for pharyngitis. However after her first dose, she experienced a choking sensation and stopped taking the antibiotic. One day prior to admission she developed fever and jaundice with severe right-sided abdominal and chest pain. In the ER she was in severe respiratory distress, with vital signs T=36.8, P=120, RR=35 and BP=100/60. Her throat was erythematous. The neck exam was benign. Her chest and right upper abdomen were exquisitely tender to palpation. Arterial blood gas showed pH=7.42, pCO_2=32 mmHg, pO_2=58 mmHg with O_2 saturation 90% on room air. White blood cell count was 34,000 with 41% bands and 53% neutrophils, Hgb=12.9, platelets =40,000, and her liver function tests were markedly elevated. Chest x-ray revealed bibasilar atelectasis and a right pleural effusion. Computed tomography (CT) of the chest revealed bilateral septic emboli, a right pleural effusion and bilateral atelectasis. She was admitted to the ICU. After obtaining cultures, metronidazole, vancomycin and gentamicin were started. She subsequently required intubation for respiratory failure and ARDS. She soon required paralysis and sedation due to high pulmonary pressures and difficulty with oxygenation. During the first 24 hours she was hypoxic with pO_2 of 30-40 mmHg on FiO_2 of 100% on pressure controlled ventilation. She was initially hypotensive as well, requiring three pressor agents over the first 72 hours. A neck CT showed inflammation and thrombus in the left internal jugular vein with no evidence of an abscess. A right pleurocentesis was performed with placement of a chest tube. The fluid was sent for culture and analysis. After 48 hours her cultures were positive for *Fusibacterium necrophorum* and *Eikenella corrodens*. After penicillin allergy testing, treatment with ampicillin and sulbactam was started and the other antibiotics were discontinued. She defervesced after 20 days of antibiotics at which point she was weaned off the ventilator. She was discharged 5 weeks after her initial presentation with no residual end organ damage.
DISCUSSION: Oropharyngeal infection in healthy adolescents and adults may lead to an anaerobic sepsis leading to a suppurative thrombophlebitis of the internal jugular vein and septic emboli known as Lemierre syndrome. The diagnosis can be made based upon the following criteria: 1) primary infection in the oropharynx; 2) septicemia demonstrated by at least one positive blood culture for *Fusibacterium necrophorum*; and 3) evidence of thrombophlebitis of the internal jugular vein. The disease can be severe but with appropriate antimicrobial therapy and drainage of any purulent collections, cure can be expected in the majority of patients.

UNILATERAL GRAVE'S EXOPHTHALMOS
Amer Adam, Department of Medicine, Lehigh Valley Hospital, Allentown, Pennsylvania
LEARNING OBJECTIVE. 1) Diagnose subtle presentations of Grave's Disease
CASE. The patient is a 34-year-old white female presented to the hospital with the complaint of left sided eye pain, swelling, erythema, nausea, and sporadic blurry vision. She related having experienced a blow to the left temporal area approximately 3 weeks prior to admission and has since had a head CT scan that was read to be negative for fracture or bleed. She had no loss of vision, seizures or fever. She did however have a ten-pound weight loss and did experience double vision when looking left. Her physical exam revealed mild erythema and edema of the left eyelid and the soft tissues of the left eye. There was impairment of the left globe in all planes but no proptosis. Routine blood tests as well as thyroid function and antithyroid peroxidase antibodies were normal. A magnetic resonance imaging scan (MRI) of the orbits revealed swelling of the extraocular orbital muscles on the left and a mild prominence of two extraocular muscles on the right with sparing of the tendons and no evidence of mass.
DISCUSSION. The differential diagnosis of a patient with unilateral exophthalmos includes: Cavernous sinus thrombosis, retro-orbital cellulitis, sphenoid ridge meningioma, retro-bulbar tumors, pseudotumor oculi, myasthenia gravis, hypertension, chronic alcoholism, superior mediastinal obstruction, and Cushing's syndrome.
The key evidence in this case was the MRI revealed edema of several extraocular muscles with sparing of the tendinous insertions and showed no evidence of retro-orbital mass. Interestingly the patient was euthyroid by laboratory evaluation. The discrepancy between the clinical findings of ophthalmopathy in the absence of thyroid disease is due to the inability of current laboratory tests to detect subtle, subclinical dysfunction. Grave's Disease is the most common cause of hyperthyroidism. It is an autoimmune disorder in which an abnormal immunoglobulin G binds to receptors for thyroid stimulating hormones (TSH) on the thyroid follicular cells resulting in stimulation of thyroid hormone production. Grave's Disease has 3 major manifestations, which include hyperthyroidism, ophthalmopathy, and dermopathy. These manifestations may run independent courses. The ophthalmopathy may initially be unilateral but often becomes bilateral over time. The ophthalmopathy involves an inflammatory infiltrate of the orbital contents with sparing of the globe. The infiltration is primarily in the musculature and involves lymphocytes, plasma cells, and edema with mucopolysaccharides. In general, clinical manifestations include tremors, weight loss, sweating, tachycardia and heat intolerance. Ocular manifestations include widened palpebral fissures, blinking, failure to wrinkle the brow on upward gaze, lid lag and lid retraction, chemosis, periorbital swelling, and conjunctivitis. Ocular complications can include corneal ulcerations, optic neuritis and atrophy

DEALING WITH THE UNWANTED TEST. RT Ackermann, Department of Medicine, UTHSCSA, San Antonio, TX.
LEARNING OBJECTIVES. Develop a strategy to approach results of unwanted tests
CASE. MR is a 93 y/o with deficits of hearing and vision that require her to need minor help with daily activities. She was admitted from home to the coronary care unit with one week of dyspnea and productive cough. She had an irregular heart rate at 150, crackles at the right lung base, and diffuse wheezing. Mentation was grossly normal. EKG showed atrial fibrillation with no ischemia. Cardiomegally, mild perihilar congestion, a right suprahilar opacity, and a right lower lobe infiltrate were seen on CXR. She received diltiazem, nebulized beta agonists, and empiric antibiotic therapy for presumed pneumonia. Her wheezing resolved and heart rate slowed to 80. Repeat CXR was unchanged. The radiologist recommended a chest CT to further evaluate.

MR was transferred to the Medicine service for a malignancy work-up. She told me, the Internal Medicine resident, that her wish was to go home without such a work-up. She understood how she could very well have cancer but felt that even if a diagnosis were made, she would not want treatment. Upon exiting her room, my plan was to discharge her home the following morning, but the radiologist called with the results of her CT. Unknown to me, this scan was performed immediately prior to her transfer. Findings confirmed a 2x3cm right hilar mass, with multiple additional smaller bilateral nodules, 1.5cm subcarinal lymphadenopathy, and multiple liver hypodensities.

This CT did not significantly alter my impression of her likely diagnosis, yet I was frustrated with a test that I had not ordered and the patient did not want. Indoctrinated under the mantra of "diagnose and treat," I wondered whether I should now try to convince MR to proceed with further work-up. I elected to present the results to her for their prognostic value and to discuss together whether our care plan should change. MR understood the test's implications and reiterated her intent to simply go home. Her family welcomed my approach, which also served to help them understand her wishes.
DISCUSSION. As generalist clinicians, it is not uncommon that tests are ordered by others that we would not otherwise have requested and that the patient had simply not desired. Through this experience, MR has taught me the following:

1) Do not ignore an unwanted test. Discern the information that it provides. Does it really alter the probability of disease, the prognosis, or the treatment implications?
2) Do tell the patient the results and utilize the information provided by the test. Openly discuss whether or not the result should affect your original care plan.
3) Refrain from the temptation of letting the result lead you down a standard algorithmic path for diagnosis and treatment. Providing quality, satisfying care can often be more dependent upon consideration of a patient's personal wishes than merely upon diagnostic certainty.

ADDRESSING DOMESTIC VIOLENCE IN PRIMARY CARE.
E Alpert, Boston U. School of Medicine, Boston MA, C Albright, Stanford U. School of Medicine, Palo Alto CA, H Greene III, Massachusetts Medical Society, Waltham MA.
LEARNING OBJECTIVES: 1) Diagnose domestic violence (DV) by history and clinical manifestations, 2) Assess risk to patient, 3) Approach intervention and safety planning when DV is diagnosed, 4) Explore strategies for effective communication with at-risk patients.
CASE: A 32-year-old woman who has been your primary care patient for about 6 months presents for a scheduled routine health care visit. Although it is a hot summer day, you notice that she is wearing long sleeves. She is also wearing dark sunglasses that she does not remove, even though she is inside. Her only complaint is insomnia due to "worrying." On exam, BP is 130/94, HR is 90 reg, RR 20. There is a large fresh ecchymosis around the right eye, and bruises on both forearms. Diffuse, faded ecchymoses are noted about the ribs and abdomen. The remainder of the PE is unremarkable. You frame your questions carefully and ask about DV. After seeking reassurance that you will not disclose to her husband what you discuss, the patient begins to cry and tells you that she has been threatened and beaten repeatedly over the last 2 years by her husband. The worst time was six months ago when she sustained two broken ribs and a concussion (a colleague of yours treated these injuries but the patient did not report abuse at that time, nor was she asked). Four days ago, the patient's husband became angry about a high electric bill, accusing her of wasting money. He pushed her down and then kicked her in the ribs. He later apologized profusely and promised he would never hit her again, but yesterday he punched her in the eye and repeatedly slapped and punched her upper body. She tried to defend herself by holding up her arms to protect her face. The patient expressed relief at being able to finally share her "dirty little secret," but refused to take patient education materials about DV because her husband might find them. She feels she can "handle things" for now on her own.

ACUTE INTERMITTENT PORPHYRIA MASQUERADING AS RECURRENT PYELONEPHRITIS. A Altschul, D Muller, Department of Medicine/Pediatrics, Mount Sinai Medical Center, NY, NY.

LEARNING OBJECTIVES. 1) Recognize the manifestations of AIP, 2) Diagnose and treat AIP, 3) Recognize the importance of prior medical records in the evaluation of patients.

CASE. A 33 year old woman presented to the Emergency Room reporting a one week history of flank pain that radiated to her abdomen and suprapubic region. She also complained of nausea, vomiting, chest pain, palpitations, subjective fevers, and dysuria. She had a history of asthma and a two year history of multiple ER visits and hospitalizations for kidney stones and kidney infections. On physical exam she appeared lethargic but arousable, diaphoretic, and in moderate distress. The blood pressure was 70/30, HR 152 bpm and she was afebrile. There was suprapubic and lower quadrant tenderness with voluntary guarding. Labs were significant for WBC 15.7 with 18% bands, potassium 2.2mEq/L, glucose 204mg%, and the U/A revealed 2+ blood. Abdominal ultrasound and CT scan revealed a distended bladder without any evidence of nephrolithiasis or hydronephrosis. In the ER the patient was treated with normal saline, IV dopamine, and Ceftriaxone. When the patient was transferred to the floor three hours later she appeared alert and in no distress, with a bp of 100/60 and a HR 95 bpm off of dopamine. A careful review of her medical record revealed that each of her prior presentations included flank pain, fever, tachycardia, hypotension, nausea, vomiting, chest pain, leukocytosis with a left shift, hypokalemia, hyperglycemia, and microscopic hematuria. Urine cultures were never positive and plain films, ultrasound, CT scan, cystoscopy, and IVP were always unrevealing. The episodes always resolved within 12 hours to 3 days of starting IV fluids. The patient had also visited clinic on three separate occasions for right eye pain and blurry vision, left arm weakness and dysesthesias, and intermittent paresthesias with headache. Family history revealed that her mother had similar episodes of flank pain for which she never sought medical attention. Subsequent lab results included a lead level of 4micro mol/L and a spot urine delta-aminolevulinic acid (d-ALA) of 4.7mg%. Urine porphobilinogen (PBG) was sent but was not performed because of UV light exposure. The patient was subsequently lost to follow-up.

DISCUSSION. The diagnosis of AIP requires a high index of suspicion in the appropriate clinical setting. All of the patient's findings, including transient hyperglycemia, bladder distension, and transient neurologic findings, are manifestations of AIP. The gold standard for diagnosis is urine PBG collected during an acute attack or a 24 hour urine for d-ALA, neither of which could be obtained in this case.

DAMAGING WORDS: TELLING BAD NEWS AND RECOGNIZING DISTRESS. D. Arterburn. Department of Medicine, UTHSCSA, San Antonio, TX.

LEARNING OBJECTIVE: Recognize the potential consequences of acute emotional distress and improve my skills in telling bad news

CASE: I was called to the Emergency Room to see a previously healthy 64-year-old woman who presented with acute left hemiparesis and slurred speech. At the bedside was her 60-year-old husband, also apparently healthy. Her examination showed a blood pressure of 198/80, left central facial droop, left upper and lower extremity weakness, and a positive Babinski on the left. Her head CT scan revealed a right-sided, internal capsule, lacunar infarct.

The Emergency Room was filled to capacity, and critically ill patients were in neighboring beds. As a Resident, I was accustomed to the hectic ER setting, and I began to discuss the preliminary diagnosis and treatment plan for the patient and her husband. Recognizing that they were upset, I tried to strike a balance between informing and reassuring them about her condition. During this, the husband began "feeling weak," and he was moved to the waiting area. Within minutes, he slumped over, developed left-sided weakness, and became lethargic. Next, he vomited and became apneic. After intubation, his blood pressure reading was 300/180. His head CT scan revealed a large, right-sided, thalamic hemorrhage extending into the 3rd and 4th ventricles. Another physician ran to the patient's bedside where I was standing and cried out, "We just brought her husband back with a stroke!" The patient became immediately distraught, and her blood pressure rose to 205/100.

DISCUSSION: This case represents a dramatic example of the physical consequences of extreme emotional upset brought about by me breaking bad news. Since clinical work will always include telling bad news, I have identified several essential steps to improve my skills. I must pay close attention to the physical context of the situation. The timing and setting should be made as appropriate as possible. I must recognize the knowledge level of those involved and their emotional state and make adjustments for each. I should observe the patient's and family's reactions to increase my awareness of adverse physical or emotional responses. I must also be aware of the potential for miscommunication by other staff members. With these steps in mind, I hope to recognize distress and change my damaging words into therapeutic tools.

ROLE OF MEDICAL RESIDENTS IN IDENTIFYING MUNCHAUSEN'S SYNDROME BY PROXY. A Arshad, S Kaur, C Karmen, SJ Peterson. New York Medical College, Valhalla, NY

LEARNING OBJECTIVES: To identify a case of Munchausen's Syndrome by proxy.

CASE: An 18-year-old female presented with shortness of breath and wheezing. Past medical history was remarkable for intractable asthma exacerbated by high doses of NSAIDs and enthesopathy from Reiter's syndrome secondary to pelvic inflammatory disease. She was treated with intravenous steroids, bronchodilators and antibiotics. On the day of the planned discharge, following a conversation with her mother, she had an episode of generalized seizure-like activity with unresponsiveness and bed-wetting. Complete evaluation including laboratory evaluation, lumbar puncture, Magnetic Resonance Imaging of the head, and toxicology screen were all normal. On transfer to our Epilepsy monitoring unit, 72 hour video electroencephalogram (EEG) monitoring was remarkable for the presence of three clinical episodes characterized by a loss of consciousness, rhythmic jerky movements and confusion. EEG during these events as well as the "interictal" EEG was normal. The findings were supportive of non-epileptic events.

We explained the results to the patient and her mother and described these events as "pseudoseizures". The mother insisted on treatment, she was increasingly hostile and insisted on transfer to another medical facility. Consultation with the neurologist at that hospital confirmed a prior admission with a similar clinical history. When extensive work-up for seizures had been negative. The patient left the hospital against medical advice after recommendation for transfer to the Psychiatric Inpatient Unit. Further investigation revealed multiple admissions to local hospitals, all with the same conclusion. The patient had spent the better part of the last seven months in various hospitals. The father's defensive presence prompted a much more aggressive and confrontational approach by the mother. This was the patient's fourth episode of pelvic inflammatory disease, a fact which she and her mother admitted , but was vehemently denied by the father . Her mother worked for a lawyer experienced in medical malpractice and used her knowledge of the system to keep the medical team on the defensive. The multidisciplinary team composed of seasoned professionals arranged admission to a psychiatric facility with family counseling.

DISCUSSION: Munchausen's Syndome by proxy is a form of child abuse in which a proxy, usually the mother, consciously distorts the description of her child's symptoms to seek hospitalization for her child that may result in unnecessary and potentially harmful tests.

LICORICE ROOT FOR THE TREATMENT OF CHRONIC FATIGUE SYNDROME AND NEURALLY MEDIATED HYPOTENSION. B Ashar, Johns Hopkins University School of Medicine, Baltimore, MD.

LEARNING OBJECTIVE. Recognize the potential benefits of licorice in the treatment of chronic fatigue syndrome.

CASE. A previously healthy 23-year-old woman presented with a three-month history of progressive fatigue, low-grade fevers, generalized headaches, and muscle aches. Due to the intensity of her fatigue she had taken a leave of absence from work and had stopped entirely her intense 3 hour/day kickboxing workouts. She denied any additional symptoms of depression. She underwent an extensive hematologic, endocrinologic, and infectious evaluation which was unrevealing. An ACTH stimulation test was normal. Tilt table testing revealed evidence of neurally mediated hypotension and postural orthostatic tachycardia syndrome. She was begun on fludrocortisone acetate and potassium chloride. Salt and fluid intake was liberalized.

Less than two weeks later she developed a constant pain in her legs. The fludrocortisone was stopped and the pain resolved. She was subsequently started on licorice root and potassium chloride. The dose of licorice root was titrated to 900mg TID with significant improvement in her symptoms. Three weeks after beginning the licorice she has returned to work and is exercising 1-1 ½ hours/day.

DISCUSSION. Bou-Holaigah and colleagues (JAMA. 1995:274:961-967.) have suggested that chronic fatigue syndrome is associated with neurally mediated hypotension and that its symptoms may be improved with the use mineralocorticoid medication. Glycyrrhizinic acid found in licorice can inhibit renal 11 beta-hydroxysteroid dehydrogenase, which is the enzyme responsible for catalysing the inactivation of cortisol to cortisone. The accumulation of cortisol can, in turn, stimulate the mineralocorticoid receptors of the cells in the cortical collecting ducts. The administration of licorice may therefore be a "natural" alternative in the treatment of chronic fatigue syndrome.

ALL SKIN CANCERS ARE NOT PRIMARIES. FL Austin-Tolliver, P Haidet, Houston Veterans Administration Medical Center and Baylor College of Medicine, Houston, Texas.
LEARNING OBJECTIVES: 1) Recognize that skin lesions may be the presenting feature of internal malignancy. 2) Distinguish between primary skin cancer and matastitic disease.
CASE: A 67 year old male with a history of dementia and basal cell carcinoma was referred to dermatology clinic for a suspicious facial lesion. At the time of evaluation, the patient denied any prior facial trauma, pain, bleeding, or discharge from the lesion. He was unsure of the length of time that the lesion had been present and denied occupational or recreational history of prolonged sun exposure. Review of systems was remarkable for memory loss and dyspnea on exertion. On physical exam, there was a 1.5 cm flesh-colored raised nodule with overlying telangiectasias on the left temple. Auscultation of the lungs revealed decreased breath sounds which were more severe on the right. There were also right-sided decreased fremitus and dullness to percussion. Recent electrolyes, cell count, and liver function tests were all within normal limits. Shave biopsy of the lesion revealed adenocarcinoma. Chest roentgenogram revealed a large mass in the right upper lobe consistent with neoplasm. Computed tomography showed extension of the mass into the mediastinal area and lover. The patient was subsequently discharged to home hospice with a diagnosis of stage IV lung cancer.
DISCUSSION-Cutaneous manifestations of internal malignancies result from direct extension of an underlying tumor or from hematogenous or lymphatic dissemination. The prevalence of cutaneous metastases has been difficult to determine in the past because skin lesions are often overlooked or misdiagnosed. Several autopsy studies have shown that up to 9 percent of internal malignancies can have cutaneous manifestation. Skin involvement occurs in a small but significant proportion of common tumors originating in the bone marrow, lymph nodes, breasts, lungs and colon. Primary care physicians should utilize clues from the patient's history and physical examination and recognize that an important, but often overlooked, part of the differential diagnosis for a suspicious skin lesion is internal malignancy.

HYPERTENSIVE CRISIS IN THE SETTING OF CARCINOID TUMOR CHEMOEMBOLIZATION AND MEPERIDINE ADMINISTRATION, C. Beaver

LEARNING OBJECTIVES. 1) Recognize the clinical presentation of malignant carcinoid. 2) Identify the mechanism of the potentially dangerous drug-disease interaction of meperdine and malignant carcinoid.
CASE: A 70 year old female with malignant carnicoid tumor of the ileum was admitted chemoemobolization of multiple hepatic metastases. The procedure was complicated by persistent severe hypertension that was unresponsive to anti-hypertensives. The patient was receiving meperidine for analgesic control which is thought to have contributed to blood pressure elevation via this drug's effect on serotonin (5-HT) metabolism. Blood pressure normalized after the initiation of nitroprusside infusion and discontinuation of the meperidine. Laboratory values correlated the patient's hypertensive crisis with an elevated 5-HT level.
DISCUSSION: 5-HT is a neurotransmitter released from carcinoid tumors that can act peripherally on vascular smooth muscle receptors to cause vasoconstriction. Meperidine prevents 5-HT inactivation by inhibiting the presynaptic cellular reuptake. This can result in severe hypertension. This hypertensive effect is supported by a known drug-drug interaction of meperidine and MAO inhibitors. The combination of which is thought to cause hypertension by blockading 5-HT metabolism resulting in a markedly elevated 5-HT level. We hypothesize that the source of the initial elevation of 5-HT was the carcinoid tumor with prolonged elevation due to the continuous meperidine infusion, resulting in severe hypertension. Therefore, the administration of meperidine in a patient with malignant carcinoid could markedly enhance 5-HT activity and, ultimately, result in a hypertensive crisis.

INTRAVASCULAR LYMPHOMATOSIS: A RARE CAUSE FOR FEVER OF UNKNOWN ORIGIN. O Ayad, A Cheema, S Hirshman, B Bashey, J Geders, N Anandarao and M Krishnamurthy, Department of Medicine, New York Methodist Hospital, Brooklyn, NY.
LEARNINIG OBJECTIVE. Recognize an uncommon, yet fatal, cause for fever of unknown origin.
CASE. A fifty-year-old male presented with weakness and fever 102 °F for three weeks. In the past he had cervical spine surgery for disc prolapse & paraparesis. Examination revealed fever, pallor, paraparesis and moderate splenomegaly. Laboratory tests revealed anemia, thrombocytopenia and cold agglutinin-hemolysis. Chest and abdomen computed tomography detected small bilateral pleural effusion, moderate splenomegaly and a few small retroperitoneal lymph nodes. Abnormal uptake in the spleen, bone marrow and liver was seen on Tc-99m-sulfur colloid scan. Liver and bone marrow biopsies showed intravascular lymphoma cells of B-cell type. The patient received eight courses of CHOP (cyclophosphamide, doxorubicin, vincristine and prednisone). His symptoms and pleural effusion have resolved but spleen showed minimal reduction in size.
Discussion. Intravascular lymphomatosis is a rare variant of non-Hodgkin's lymphoma described in 1959. About 150 cases have been reported so far. Most cases are of B-cell origin. The most common presentation is fever with dementia and skin nodules. Other manifestations depend on the involved organ(s). Malignant cells are exceedingly rare in peripheral blood and bone marrow biopsy is unlikely to yield the diagnosis. No radiological picture is pathognomonic. Diagnosis was not reached till postmortem in the majority of reported cases. There is no deficiency of lymphocytes-homing antigen so the intravascular predilection of these malignant cells remains unexplained. Complete response rate to chemotherapy is about 30% with average follow up of 12 months but relapse is common. Median survival without treatment is only 5 months.

A PATIENT WITH SJOGRENS SYNDROME COMPLICATED BY MANDIBULAR OSTEOMYELITIS. Alain Bertoni. Department of Medicine, Johns Hopkins Hospital, Baltimore, MD.
LEARNING OBJECTIVES. Recognize the potential for dental complications in patients with Sjogren's syndrome (SS). Prevent such complications by attention to oral hygiene and prompt referral to dentistry.
CASE. A 56 year old woman presented to clinic with left facial swelling, jaw pain, and fever shortly after loosing a tooth. One year prior she had described symptoms of chronic heartburn, dry eyes, and dry mouth. Her erythrocyte sedimentation rate was 62, the antinuclear antibody titer was 1:640 and the La antibody was positive, supporting a diagnosis of SS. Her past medical history included hypothyroidism and acute on chronic interstitial nephritis for which she was receiving prednisone. Physical exam was notable for an oral cavity with exceedingly poor dentition, gum swelling, and an apparent tooth remnant on the left. The left jaw was tender to palpation. Urgent oral surgery consultation diagnosed dental abscess and the patient was admitted to the hospital. Computed tomography of neck and jaw revealed focal erosive changes involving the left mandibular arch adjacent to the dental roots suggestive of osteomyelitis. Blood cultures were negative but dental abscess aspirate grew Streptococcus anginosus and Streptococcus viridans. Intravenous clindamycin was administered for six weeks with good resolution of symptoms.
DISCUSSION. This patient's mandibular osteomyelitis was likely the result of the combination of severe dental caries due to SS and immunosuppression by prednisone. Systematic review of the literature did not find previously reported cases of SS and mandibular osteomyelitis. Internists should be aware that decreased saliva production often precedes the diagnosis of SS by many years and predisposes patients to multiple and aggressive dental caries. In contrast with the dental literature, most medical and rheumatology references on SS acknowledge but do not elaborate on the increased risks of dental caries and the importance of proper dental care. When SS is diagnosed, internists should advise their patients to intensify oral hygiene which includes brushing with fluoridated pastes after meals, flossing, fluoride rinses, and avoiding heavily sugared foods. Prompt referral to dentistry is crucial to evaluate and treat existing cavities. These measures may prevent oral complications of SS as illustrated by this case of mandibular osteomyelitis.

AN UNUSUAL CAUSE OF HEADACHE. Dionne J. Blackman, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD.
LEARNING OBJECTIVES: 1) Recognize benign coital cephalalgia as a cause of headache that can mimic subarachnoid hemorrhage and 2) Distinguish benign coital cephalalgia from subarachnoid hemorrhage.
CASE: A 43 year old man with well-controlled hypertension presented to his internist with a severe throbbing occipital headache and neck stiffness that persisted since sudden onset during sexual intercourse two days prior. He said the headache was the worst of his life and noted having a similar but less severe headache two weeks previous during intercourse. He denied fever, photophobia and vomiting. The physical examination was negative for meningeal signs, fundoscopic abnormalities or any neurologic signs. The patient was sent to the emergency room to be evaluated for possible subarachnoid hemorrhage. A computed tomographic (CT) scan of his head was normal. Lumbar puncture (LP) revealed cerebrospinal fluid (CSF) with 1,250 RBCs and 2 WBCs in the first tube and 323 RBCs and 1 WBC in the last tube. The CSF was non-xanthochromic. Neurology consultants concluded findings were consistent with a diagnosis of benign coital cephalalgia. Blood in the CSF was thought secondary to a traumatic LP given the lack of xanthochromia. The patient was discharged from the emergency room in stable condition and has not had a recurrence of headache during intercourse.
DISCUSSION: Though headache associated with sexual activity has been recognized since the time of Hippocrates, benign coital cephalalgia was first formally reported in 1970. It typically presents as acute onset of a severe headache during or immediately after sexual intercourse that localizes most often to the occipital region. Associated symptoms can include photophobia and nausea. The similarity of this entity to subarachnoid hemorrhage can confound the diagnosis, particularly since intercourse has been reported as the precipitating cause of subarachnoid hemorrhage in rupture of 3.8 - 12% of aneurysms and 4.1% of arteriovenous malformations. Despite its similarities with subarachnoid hemorrhage, benign coital cephalalgia can be distinguished from it by: 1) lack of neurologic symptoms or signs, 2) quick resolution of headache (usually within two hours) 3) negative neuroimaging studies and 4) absence of CSF xanthochromia. Clinical features suggestive of serious intracranial disease, such as subarachnoid hemorrhage, would be: 1) an altered level of consciousness, 2) stiff neck, 3) fever, 4) neurologic signs, 5) vomiting and 6) headache lasting longer than a day. Despite the worrisome history in the case patient of stiff neck and headache lasting longer than a day, the diagnosis of benign coital cephalalgia could be made based on the lack of neurologic abnormalities on physical examination, normal head CT scan, and non-xanthochromic CSF on lumbar puncture.

REMISSION OF GASTRIC MUCOSA-ASSOCIATED LYMPHOID TISSUE LYMPHOMA IN A PATIENT WITH HELICOBACTER PYLORI. Joyce Boccier-Burton , Jose Batlle, Department of Medicine, SUNY- HSC, Syracuse, NY.
LEARNING OBJECTIVES. 1) Recognize the strong correlation of *Helicobacter pylori* and mucosa-associated lymphoid tissue (MALT) lymphoma, and 2) Treatment of low-grade MALT is directed towards the eradication of *H. pylori.*
CASE. A 66 year old physics professor presented with syncope and upper GI bleed. There was no prior history of peptic ulcer disease nor of alcohol or tobacco use. His only medication included one aspirin daily. Physical examination revealed an anxious gentleman in no acute distress. He was not orthostatic and conjunctiva were pink. The cardiac exam was benign and he demonstrated no neurological deficits. A rectal exam performed showed heme negative stool but he produced emesis which was heme positive. The hematocrit was 28.7 and EKG was normal. The patient was admitted and an upper endoscopy with biopsy showed an ulcerated mass in the antrum of the stomach. No other lesions were visualized. The biopsy report was consistent with low-grade gastric MALT lymphoma and CT scans confirmed this was confined to the stomach. He had positive serology for *H. pylori* with a corresponding positive C-14 Urea breath test. Induction therapy was begun with triple drug therapy and omeprazole for 3 weeks. Interval restaging of the lymphoma with endoscopy and C-14 Urea breath testing was performed with the goal of eradicating *H. pylori.*
DISCUSSION. Gastric MALT lymphoma arises from chronic inflammatory and immunological responses. Infection with *H. pylori* activates inflammation, autoantibody formation and cell mediated epithelial damage, which can then induce a lymphofollicular infiltrate known as MALT. This immunologic defense is believed to be associated with *H. pylori* in 90% of cases. Consequently, lymphomas that arise from gastric MALT may represent genetic changes evolving from this infection. At least six studies have shown the eradication of *H. pylori* would result in complete remission of low-grade gastric MALT lymphoma. This unequivocal evidence led the European *Helicobacter pylori* Study Group to "strongly recommend" treatment of *H. pylori* in MALT lymphoma. The causality of *H. pylori* in the pathogenesis of other diseases, such as gastric adenocarcinoma, is suggestive but not yet definitive. With the availability of non-invasive diagnostic techniques, the role of the General Internist in screening and management will likely expand as other *H. pylori*-related diseases are clarified.

ADVERSE SULFA REACTION PRESENTTING AS FEVER AND ACUTE HEPATITIS IN A PATIENT WITH AIDS. A Bleistein, M Wright, Department of Internal Medicine/Pediatrics, Wright State University, Dayton, OH.
LEARNING OBJECTIVES. 1) Recognize the increased prevalence and the biochemical mechanism of acute drug reaction (ADR) in patients with AIDS, 2) Differentiate the etiology of neutropenic fever in an AIDS patient, and 3) Recognize the broad spectrum of clinical manifestations of a sulfa allergy.
CASE. A 24 year old black male with AIDS and a CD-4 count of 0 presents with a four day history of vomiting, diarrhea, and fever. Recent medical history is remarkable for community acquired pneumonia and refusal of all anti-retroviral therapy. Medications include only trimethoprim-sulfamethoxazole (TMP-SMX) and levofloxacin for the previous twelve days, resulting in complete resolution of his respiratory symptoms. On physical examination, temperature is 103.7 degrees Fahrenheit, heart rate 115, blood pressure 110/75, with marked orthostatic changes, and room air oxygen saturation is 96 percent. He presents with oral thrush but no focal pulmonary abnormalities, lymphadenopathy, rash or hepatosplenomegaly. Laboratory evaluation demonstrates a WBC of 0.6 with 29 neutorphils, 21 bands and 43 lymphocytes. Platelet count is 74,000, AST 317, ALT 167, bilirubin 0.8. Liver enzymes had been normal two weeks prior. Chest x-ray is normal. The patient is admitted for aggressive fluid resuscitation, and is started on broad-spectrum antibiotics for presumed sepsis and G-CSF for neutropenia. TMP-SMX is continued as well. Multiple cultures are obtained. Serum acetominophen level is found to be 12, viral hepatitis serologies negative and abdominal CT normal. By the third hospital day, the patient remains persistently febrile to 104.7 but hemodynamically stable; cultures are negative. Transaminases, however continue to climb, and TMP-SMX is stopped because of concern for sulfa drug reaction. Within 24 hours of stopping TMP-SMX, the patient's fever resolves. When the patient is no longer neutropenic, the antibiotics are discontinued. Liver enzymes peak on day five of hospitalization, but gradually return to normal, and the patient is discharged to a nursing home. He continues to refuse anti-retroviral therapy and dies five months later.
DISCUSSION. Patients with AIDS receiving TMP-SMX have a 50 percent incidence of ADR compared with seven percent incidence in other hospitalized patients. Increased production of interferon leading to slow acetylation has been suggested as a possible mechanism of ADR in these patients. Indeed, slow acetylation has been associated with increased incidence of ADR in other patient populations. Typical sulfa allergy consists of rash and fever; however, leukopenia, thrombocytopenia, and hepatitis may occur as well. This patient's sulfa reaction presented as fever and hepatitis in a patient known to have AIDS, but in fact, as ADR are so prevalent in AIDS patients, Infectious Disease specialists have suggested that such reactions in at-risk populations should alert the primary care physician to consider the diagnosis of AIDS in a previously undiagnosed patient.

COMPREHENSIVE CARDIOVASCULAR MANIFESTATIONS OF CARBON MONOXIDE POISONING. KC Braddy and JJ Lopez, Department of Medicine, University of Iowa Hospitals, Iowa City, IA.

LEARNING OBJECTIVES: 1) Recognize the cardiovascular effects of carbon monoxide, 2) Distinguish cardiac dysfunction secondary to carbon monoxide from intrinsic cardiac disease, and 3) Treat carbon monoxide induced cardiac disease.

CASE: A 64 year-old woman with hypertension and migraines sustained carbon monoxide poisoning due to a faulty water heater. On admission, she complained of headache and was found to be somnolent and confused with a carboxyhemoglobin (CHb) level of 19% (normal 1 - 3%). Hyperbaric oxygen was administered. After 4 hours of treatment, the patient developed substernal chest pain associated with shortness of breath despite a repeat CHb of 1%.

The patient rapidly deteriorated and had chest pain associated with anterolateral and inferior ST segment elevation as well as hypotension requiring inotrope administration. Monomorphic ventricular tachycardia occurred requiring defibrillation, lidocaine, and intubation. An emergent cardiac catheterization was performed with hemodynamic findings consistent with cardiogenic shock (RA 23 mm Hg, PCW 39 mm Hg, Ao 55/32 mm Hg, CO 1.86 L/min). Coronary angiography revealed a probable acute occlusion of a small, mid-posterior descending artery branch which supplied only a limited amount of left ventricular myocardium with no other significant coronary artery disease. Echocardiography demonstrated severe global biventricular dysfunction. An intra-aortic balloon pump (IABP) and intravenous pressor support were used to stabilize the patient.

Despite enzymatic evidence of myocardial infarction (CK 616 u/L; troponin I 12.8 ng/mL), the patient hemodynamically improved over 48 hours (RA 12 mm Hg, PCW 14 mm Hg, CO 5.53 L/min) with repeat echocardiography revealing near normal biventricular function. The IABP and intravenous pressors were weaned, and the patient was extubated. She returned to baseline function and was discharged home with physical rehabilitation.

DISCUSSION: Carbon monoxide is believed to exert cardiovascular effects through several mechanisms including tissue hypoxia, shifting of the oxygen hemoglobin desaturation curve, direct binding to myoglobin, alterations to mitochondrial enzymes, as well as myocardial hemorrhage and necrosis. Carbon monoxide poisoning can affect all components of the heart: the coronary vasculature, conduction system, and myocardium. Case reports have described isolated effects of carbon monoxide poisoning such as reversible myocardial dysfunction (without infarction), ventricular arrhythmias, or myocardial infarction. However, such comprehensive cardiovascular manifestations as described in the above case are unusual and have not been previously reported.

VENOUS THROMBOSIS IN THE SETTING OF THE FACTOR V LEIDEN MUTATION AND ORAL CONTRACEPTIVE USE. C. Brands. Department of Internal Medicine, Wright State University, Dayton, OH.
LEARNING OBJECTIVES: 1) Review the nature of this mutation and the resultant alterations in the anticoagulant pathways. 2) Recognize the relationship between the presence of the mutation and prothrombotic stimuli, particularly oral contraceptives.
CASE: A 19 year old female presented with a ten day history of headache. The headache had a sudden onset without aura, and was characterized by constant pain over the entire cranium since its onset. Photophobia, dizziness, and nausea were present since the onset of the headache. The patient denied loss of vision, but reported pain with extraocular movement on extremes of lateral gaze. Intermittent weakness was noted in the left upper and left lower extremity over the last three days. Medications: ibuprofen 600 mg po tid and a combination estrogen/progesterone oral contraceptive agent (levonorgestrel with ethinyl estradiol). Past medical history was remarkable for a left femoral deep venous thrombosis two years earlier while on an oral contraceptive agent. Family history was remarkable for a history of recurrent venous thrombosis in the patient's father. Physical examination revealed an ill-appearing white female. Vital signs were stable and the patient was afebrile. Pupils were equal and reactive to light with photophobia noted bilaterally. Disc margins were sharp. Horizontal nystagmus was noted on far lateral gaze to the left and right. Cardiopulmonary and abdominal exams were normal. The patient was alert and fully oriented. Cranial nerves II-XII were intact. Motor, sensory, reflex, and cerebellar exams were normal. The left lower extremity had trace edema to the level of the knee. MRI and MRV studies showed thrombus in the anterior aspect of the sagital sinus, the right transverse sinus, and the right internal jugular vein. The patient was treated with intravenous heparin which was continued for five days after the initiation of warfarin. The oral contraceptive agent was discontinued. The patient had complete resolution of her symptoms. A complete laboratory evaluation for hypercoagulability revealed activated protein C resistance and heterozygosity for the factor V Leiden mutation.
DISCUSSION: The factor V Leiden mutation (a point mutation transmitted in autosomal dominant fashion) renders activated factor V less susceptible to degradation by activated protein C, which is the laboratory phenomenon known as activated protein C resistance. The factor V Leiden mutation is now the most commonly identified predisposing condition in patients diagnosed with deep venous thrombosis. The risk of venous thromboembolism is greatly increased in women with the mutation who use oral contraceptive agents. Oral contraceptive agents may have an independent factor V Leiden-like effect with resultant resistance to activated protein C.

IDIOPATHIC HYPERSOMULENCE: AN UNUSUAL CAUSE OF FATIGUE IN A MEDICAL STUDENT. P. F. Bressoud, Division of General Internal Medicine, University of Louisville, Louisville, KY.
LEARNING OBJECTIVE: (1) To recognize sleep disorders (2) To understand the essential elements of a sleep history and evaluation
CASE: A 22 year old medical student presented to our office for evaluation of fatigue approximately 4 weeks after entering medical school. Despite increased sleep, he had remained fatigued. The student reported having difficulty staying awake in lectures and studying. He repeatedly found himself starting to fall asleep while driving. He reported that his symptoms of fatigue and non-restful sleep surfaced 3-4 months prior to the start of medical school about the time he was married. His marital relationship was reported as being good after living with the same woman for several years. He denied nocturnal arousals, brash water, or paroxysmal dyspnea. No episodes of thrashing, snoring, choking or respiratory pauses were noted by the wife. His weight was stable. He specifically denied any symptoms of depression. He was not taking any medications. His past medical history was unremarkable. He did not use caffeine, ethanol or tobacco products. Physical examination was unremarkable. Laboratories including CBC, metabolic panel, TSH were normal. Titers for EBV, CMV and Hepatitis C were ordered. All were undetectable with the exception of a detectable IgG for EBV indicating a prior infection. Re-evaluation four weeks later failed to demonstrate any improvement. A nocturnal sleep study followed by multiple sleep latencies was ordered to evaluate for possible narcolepsy. The sleep study demonstrated a rapid on set of sleep without the early onset of REM associated with narcolepsy. A diagnosis of idiopathic hypersomulence was made and he was referred to a sleep specialist for further medical management with stimulants.
CONCLUSIONS: Symptoms of severe fatigue and sleepiness in students of academically rigorous programs should not be dismissed casually. A detailed history and physical with a thorough metabolic workup and detailed sleep history must be undertaken to evaluate fatigue and restless sleep. Although idiopathic hypersomulence is a rare cause of sleepiness, other sleep disorders such as sleep apnea, narcolepsy, restless leg syndrome or poor sleep hygiene are relatively common causes of fatigue and sleepiness. Primary care practitioners must include sleep histories as part of their routine evaluations and learn to differentiate between the common sleep disorders.

HEMORRHAGIC PULMONARY EDEMA IN A PATIENT WITH RHEUMATIC HEART DISEASE. C. Brands. Department of Internal Medicine, Wright State University, Dayton, OH.
LEARNING OBJECTIVES: 1) Highlight atypical presentations of pulmonary edema 2) Classify pulmonary edema according to inciting cause 3) Review the pathophysiology of cardiogenic pulmonary edema 4) Recognize mitral regurgitation as a rare cause of hemorrhagic pulmonary edema
CASE: A 69 year old white male status post two coronary artery bypass grafting procedures for coronary artery disease presented with new onset dyspnea and hemoptysis. Past medical history was remarkable for essential hypertension, Type 2 Diabetes, mild renal insufficiency, and iron deficiency anemia. The patient had no history of myocardial infarctions and no history of angina since immediately prior to his second revascularization. Medications: glyburide 5 mg qd, amlodipine 10 mg qd, aspirin 325 mg qd, and atorvostatin 10 mg qd. Vital signs: HR 120/min. RR 32/min. BP 110/60. T 98.9. Cardiopulmonary examination revealed a patient in acute respiratory distress with bibasilar crackles, a holosystolic murmur at the apex radiating into the axilla and a diastolic blowing murmur at the right upper sternal border. Both murmurs had been noted previously. Chest x-ray showed pulmonary edema with a normal cardiac silhouette. The patient developed profound hypoxemia and was intubated and mechanically ventilated. Hemoglobin was 10 (baseline 10) and creatinine 2.0 (baseline 1.8). Electrocardiogram revealed sinus tachycardia with intraventricular conduction defect. Transthoracic echocardiography revealed normal left ventricular size and function with left ventricular hypertrophy, mild left atrial enlargement, a restricted posterior mitral leaflet with extensive calcifications, severe mitral regurgitation, and moderate aortic regurgitation. Previous studies had demonstrated mild mitral and aortic regurgitation. Cardiac catheterization revealed moderate pulmonary hypertension, moderate mitral and aortic regurgitation, and widely patent coronary grafts. Pulmonary angiogram revealed no emboli. Myocardial infarction was excluded using serial cardiac enzymes. Blood, sputum, and urine cultures for bacteria and fungi showed no growth. The patient's hemodynamic status improved with treatment for diastolic heart failure, but hemoptysis continued into the second week of hospitalization with persistent, bilateral perihilar infiltrates noted on chest x-rays. Thoracotomy was performed to obtain open lung biopsies from the right upper and middle lobes. Histopathology confirmed a diagnosis of focal pulmonary hemorrhages typical of changes seen with chronic congestive heart failure, with no evidence of vasculitis. Mitral valve replacement was performed using a porcine valve. Postoperative echocardiogram revealed the valve to be normally positioned in the mitral annulus with no evidence of regurgitation. Postoperative course was remarkable for atrial fibrillation. Surgical pathology of the patient's native valve revealed gross and histologic changes consistent with rheumatic heart disease.

NOT EVERY RED'SWOLLEN DIABETIC FOOT IS INFECTED: RECOGNIZING AND MANAGING ACUTE NEUROGENIC ARTHROPATHY. RC Brooks. University of Pittsburgh and VA Pittsburgh Healthcare System, Pittsburgh, PA.
LEARNING OBJECTIVES: 1) Recognize the clinical features and differential diagnosis of acute neurogenic arthropathy. 2) Distinguish acute neurogenic arthropathy from osteomyelitis and cellulitis using laboratory and radiographic tests. 3) Manage acute neurogenic arthropathy to reduce long term disability and complications.
CASE: A 58 year old male with an 18 year history of insulin-requiring type II diabetes complained of one month of insidious onset of redness, swelling, and pain in his left foot. He had no recent trauma and denied fevers or chills. Prior foot exams had documented patchy bilateral sensory loss to monofilament testing and decreased pedal pulses, but no foot ulcers or infections. On examination, the dorsum of the left foot was erythematous, warm, swollen (with pitting), and tender from the metatarsophalangeal joints to the ankle, with the most severe findings over the dorsal mid-foot. There was abnormal bony prominence and tenderness to the medial mid-foot area. Laboratory tests showed a normal WBC (4,200), Hct, and ESR (16). Radiographs of the left foot demonstrated erosive changes involving the first through fourth tarsometatarsal joints, lateral subluxation of the second metatarsal, and mild periosteal reaction of the proximal third and fourth metatarsal shafts. A triple-phase technetium bone scan showed markedly increased left mid-foot uptake in blood flow, blood pool, and delayed bone images. An indium-labeled WBC scan showed only a slight area of left mid-foot accumulation, most consistent with an acute Charcot joint. The patient was treated with non-weightbearing status, followed by a total contact cast, with gradual improvement in inflammatory signs and moderate final foot deformity.
DISCUSSION: Diabetes is the leading cause of neurogenic arthropathy, which occurs in up to 7.5% of diabetics. Acute neurogenic arthropathy presents with an inflammatory clinical picture mimicking infection or crystal-induced arthritis. Rapid identification and management with reduction in weightbearing is essential for preventing progression to debilitating, limb-threatening deformity. Diagnosis requires a high clinical suspicion, along with appropriate use of imaging tests. Plain radiographs, indium-labeled WBC scans, and MRI are the most useful diagnostic studies.

CARDIAC COMPLICATIONS OF CHRONIC ALCOHOLISM.
Christopher L. Bryson. UT Southwestern. Dallas. Texas.
LEARNING OBJECTIVE: Recognize sequelae of chronic heavy alcohol use.
CASE: Mr. N. is a 45 year old white gentleman with a history of heavy alcoholism who presents to the emergency room complaining of shortness of breath. He states that his last drink was several days ago and that he stopped drinking because he had a cough. He notes that he had to sleep upright two days prior to admission. He also reports that he has been increasingly anxious over the past two days. He states that he has had subjective fevers and a mildly productive cough. He denies any melena, vomiting, or blackouts. At home the patient had been taking diltiazem SR 180 mg QD, captopril 50 mg TID, metoprolol 25 mg BID, furosemide 80 mg QD and potassium supplements. His past medical history is remarkable for long-standing atrial fibrillation and dilated cardiomyopathy.

His physical examination is notable for a somewhat disheveled dyspneic white male with an irregularly irregular pulse at a rate of 150 to 180. His blood pressure is 120/70 with a maximum temperature in the ER of 38.6. His head and neck exam reveals poor dentition and his neck reveals a jugular venous distention of 10 cm. The cardiovascular exam reveals a laterally displaced PMI, with an irregular and rapid heart rate. His pulmonary exam shows tubular breath sounds throughout and respiration in a Cheyne-Stoke pattern; there are crackles in the lower ¼ of the bases bilaterally with wheezes throughout. The abdominal exam reveals hepatosplenomegaly with a palpable left lobe. On the neurologic exam he is initially anxious and has a fine tremor; otherwise he has a nonfocal exam. His EKG reveals atrial fibrillation. His chest X-ray shows an enlarged cardiomediastinal silhouette and peripheral vascular redistribution.

He was felt to have rapid ventricular response due to alcohol withdrawal; this was addressed with aggressive benzodiazepine therapy. His tachycardia induced failure was addressed with intravenous diltiazem while being monitored in the CCU. Over the next day the patient was converted to oral therapy when his requirement for negative chronotropes decreased as his withdrawal attenuated. He was placed on his previous therapy as well as vitamin supplementation. He was noted to be well rate controlled at his follow-up appointment a week and a half after discharge.
DISCUSSION: This case illustrates several of the most important clinical sequelae of chronic heavy alcohol use and the necessity of making a unifying diagnosis when possible in order to treat the underlying cause. This gentleman's heavy alcoholism resulted in an alcoholic cardiomyopathy with dilated chambers as well as atrial fibrillation, and his withdrawal precipitated a rapid ventricular response which contributed to his failure exacerbation.

LARGE-CELL LYMPHOMA OF THE NASOPHARYNGEAL SINUSES AND ORBIT PRESENTING AS A HYPOGLOSSAL NERVE PALSY IN A PATIENT WITH ACQUIRED IMMUNODEFICIENCY SYNDROME (AIDS). D Burn, CL Karmen, D Kombert, and SJ Peterson, New York Medical College, Valhalla, New York.
LEARNING OBJECTIVE. To recognize the causes of hypoglossal nerve palsy in a patient with Acquired Immunodeficiency Syndrome (AIDS).
CASE. A heterosexual 42-yr-old Haitian male, previously diagnosed with AIDS, presented to the hospital complaining of discomfort in the right eye, and difficulty with speaking and chewing food. He had no history of headaches, seizures, fever, changes in visual acuity or any motor, sensory or gait abnormalities.

On physical examination, he had proptosis of the right eye but normal extra-ocular muscle movements, visual acuity and fundoscopic exam. He had left palatal swelling with deviation of the uvula to the right. On protrusion, the tongue deviated to the left. Taste sensation was intact. The remainder of the physical exam was within normal limits.

A CAT scan of the head, orbits, and sinuses revealed a very large nasopharyngeal mass, bulkier on the left than on the right, extending into the left antrum, destroying its medial wall. It also extended into the left ethmoid crossing over into the right, invading through the lamina propria with extension into the right orbit. The mass displaced the intraocular muscles to the right, but did not directly involve them. The mass also reached to the anterior aspect of the left hypoglossal canal. No intracranial extension was noted. Biopsy of the lesion revealed a large B-cell malignant non-Hodgkin's lymphoma. Radiation therapy significantly decreased the proptosis and afforded symptomatic relief to the patient. He is currently undergoing chemotherapy.
DISCUSSION. Lymphoma, tuberculosis or fungus can cause a large sinus mass in an immuno-compromised patient. To our knowledge, this is the first reported case of a large cell, non-Hodgkin's lymphoma causing isolated hypoglossal nerve palsy in a patient with AIDS.

MACROPHAGE ACTIVATION SYNDROME. Cheryl Buck, Caroline Mueller, Bihu Sandhir, University of Cincinnati, College of Medicine, Cincinnati, Ohio
LEARNING OBJECTIVES: 1. Recognize that Macrophage Activation Syndrome (MAS) is a serious complication of Systemic Juvenile Rheumatoid Arthritis (S-JRA). 2. Recognize the clinical features of MAS. 3. Diagnose and treat MAS in the setting of S-JRA.
CASE. A 26 year old white female with history of S-JRA (since age 5) on weekly Methotrexate and prednisone, s/p bilateral hip replacement and endometriosis presented to an outlying hospital with a two month history of fatigue and two day history of nausea, vomiting and low back pain. Laboratory data was most remarkable for pancytopenia (WBC 3.4, Hgb 11.3, Plt 87K). She received parenteral antibiotics, steroids and intravenous hydration. ECHO revealed a mild to moderate pericardial effusion without tamponade and abdominal CT showed mild splenomegaly. She developed fever, confusion and a macular rash on the dorsum of her feet, lumbar puncture was negative. Her pancytopenia worsened and she was transferred to a tertiary care center, high dose steroids were given and antibiotics continued. Lab abnormalities included alkaline phosphatase 338, AST 251, ALT 84, Tbili 6.1, Alb 2.5, PT 18.9, INR 1.8, PTT 36, WBC 1.8, Hgb. 11.2, Platelets 64K. Two days after hospital transfer coagulation studies revealed an elevated PT >75, INR >6.7, PTT 33, Fibrinogen 40, D-dimer 4 and peripheral smear showed no schistocytes. Cryoprecipitate and FFP corrected her coagulation abnormalities. Bone marrow aspirate showed maturation arrest in the myeloid series with hemophagocytosis. All bacterial and fungal blood cultures were negative however, the monospot test was positive. Steroids were weaned, antibiotics discontinued and her LFTs normalized.
DISCUSSION. Macrophage activation syndrome (MAS), previously known as virus-associated hemophagocytic syndrome is a serious and often fatal complication of systemic juvenile rheumatoid arthritis (S-JRA). This cytokine mediated syndrome results in fever, hepatosplenomegaly, pancytopenia, coagulopathy, elevated serum transaminases, hyperferritinemia, and hypertriglyceridemia. The pathognomonic feature of MAS is hemophagocytosis on bone marrow aspirate. Causative factors include viruses (EBV, Varicella, and Coxsackie), gold therapy and aspirin. Treatment consists of high dose parenteral steroids and cyclosporin A for steroid refractory cases. A rare syndrome, MAS is mostly seen in the pediatric population, but as demonstrated in this case it can also be seen in an adult population. Hemorrhage and opportunistic infection has been the major cause of death of patients with MAS. The potentially fatal nature of MAS emphasizes the importance of recognition of its constellation of signs and symptoms and prompt initiation of treatment.

MALIGNANT NEUROENDOCRINE CELL TUMOR OF THE PANCREAS PRESENTING AS SECRETORY DIARRHEA. D Burn, L Biddle, CL Karmen, A Mumtaz, and SJ Peterson, New York Medical College, Valhalla, NY.
LEARNING OBJECTIVE. To recognize islet cell tumors of the pancreas as a cause of secretory diarrhea.
CASE. A 30-year-old woman presented to the hospital with severe diarrhea of approximately two liters per day. The patient was dehydrated with a potassium of 1.9 and bicarbonate of 13. There was no gap between her calculated and measured stool osmolality, and the rest of her stool studies were normal. A diagnosis of secretory diarrhea was made.

The differential diagnosis included infection, gastrinoma, pancreatic tumor, medullary carcinoma of the thyroid, villous adenoma, and carcinoid tumor. After completing the evaluation for these possibilities, potassium was replaced in a monitored setting and diarrhea was controlled with intravenous octreotide.

Chemistry studies revealed vasoactive intestinal peptide of 233 (normal is less than 50), and calcitonin of 122 (normal is less than 4). Levels of gastrin, serotonin, histamine, parathyroid hormone, parathyroid hormone related protein, and urine 5-hydroxy indole acetic acid were all within normal limits.

A computerized tomography (CT) scan showed a 4 cm mass in the head of the pancreas, and multiple metastases in the liver. CT scan-directed biopsy of the masses in the liver and pancreas revealed malignant cells consistent with neuroendocrine tumor/islet cell tumor. The diarrhea started again after discontinuing octreotide, but resolved after chemotherapy.
DISCUSSION. Patients presenting with secretory diarrhea must be evaluated for the possibility of malignant tumors originating in the pancreas.

ISOLATED ANTI-PHOSPHOLIPID ANTIBODY SYNDROME (APLS) PRESENTING AS CENTRAL RETINAL VEIN OCCLUSION (CRVO). D Burn, M Burns, S Forman, L Osvath, CL Karmen, and SJ Peterson, New York Medical College, Valhalla, NY.

LEARNING OBJECTIVE. To recognize central retinal vein occlusion as a complication of antiphospholipid antibody syndrome.

CASE. A 48-year-old woman presented to the hospital complaining of shadows in front of the left eye on awakening. She gave no history of glaucoma, diabetes, hypertension, deep vein thrombosis, joint pains, rash or photosensitivity. Past history was significant for mycoplasma pneumonia complicated by Coomb's positive cold antibody autoimmune hemolytic anemia that resolved without treatment. Her twin sister has systemic lupus erythematosis (SLE) and a history of retinal artery thrombosis. Physical exam was within normal limits except for thrombosed central retinal veins with scattered posterior pole hemorrhages in the absence of other afferent and efferent disease. Her blood count, chemistries, sedimentation rate, protein C and S, anti-thrombin III, serum homocysteine, factor V-Leiden, serum protein electrophoresis and viscosity, lupus anti-coagulant, anti-nuclear antibody, anti-ds DNA, anti-Smith, anti SSA and SSB, were all within normal limits. Anti-cardiolipin antibody and cold agglutinin test were positive and she had a low complement level. Serum test for syphilis [veneral disease research lab (VDRL)] was positive, but fluorescent treponema antibody (FTA-ABS) was negative. An echocardiogram showed a small pericardial effusion. The patient was anti-coagulated. Her occular complaints disappeared.

DISCUSSION. To our knowledge, this is the first reported case of isolated central retinal vein occlusion presenting as a complication of antiphospholipid antibody syndrome. We recommend that all patients with retinal vein occlusion be evaluated for this syndrome.

IRON DEFICIENCY ANEMIA DUE TO SMALL BOWEL TUMOR

Authors: M-J Chang, M Goldstein, and G Tabas, UPMC Shadyside, Pittsburgh, PA

LEARNING OBJECTIVE: Recognize an important but unusual cause of iron deficiency anemia.

CASE: 77 yo w/m presented with 1 month history of abdominal discomfort, urinary retention and generalized fatigue. These symptoms became progressively worse a few days prior to the admission. He also complained mild exertional dyspnea but denied nausea and vomiting. His past medical history included coronary artery disease, hyperlipidemia, GERD, prostate cancer, status post radical prostatectomy and orchiectomy five years previously, and chronic constipation for which he was taking charcoal. He was also taking baby aspirin, verapamil, Lipitor, Imdur, Prilosec, Paxil and multivitamins. On admission, he was afebrile and BP was 168/74 mm Hg, which decreased on standing. Physical exam showed resting tachycardia with pulse 102. His abdomen was soft and nontender with active bowel sounds. There were no palpable masses or organomegaly and there was no stool in the rectum. He had 2+ pedal edema that had been chronic according to the patient. Initial laboratory tests revealed H/H of 7.3/23.4 with MCV 77, reticulocyte count 3%, ferritin level of 7. B_{12}, folate, BUN/Cr, serum electrolytes, PSA and U/A were all normal. He was transfused with PRBC and found to have intermittent heme positive stool. Endoscopy and colonoscopy showed no significant findings. Small bowel series revealed intussusception with elongated defect in the ileum suggesting small bowel tumor. Laparotomy disclosed a 10-cm long and 4-cm wide small bowel tumor in the jejunum that was believed to be responsible for the intussusception. Small bowel resection was performed after frozen section revealed benign tumor. Gross examination showed a polypoid lesion with an ulcerated tip and microscopic pathology confirmed an inflammatory fibroid polyp.

DISCUSSION: GI bleeding is one of the major etiologies of iron deficiency anemia in the geriatric population, however, small bowel tumors are not among the common causes. Although the small bowel represents 75% of the alimentary track, its tumors only account for 3-5% of GI tumors. Most of them are asymptomatic and without clinical significance. Fluctuating vague abdominal pain caused by intermittent partial obstruction is the most common presentation of the benign tumors that do become symptomatic. Intussusception is the most common cause of obstruction from small bowel tumors, which in severe cases can lead to GI bleeding and anemia. Fortunately, they do not present as a surgical emergency in adults and a thorough diagnostic evaluation can usually be done before intervention. It is important to consider small bowel tumors in the differential diagnosis of iron deficiency anemia.

HYPEREMESIS IN PREGNANCY-LINES IN LAST. Michael P. Carson (member) Departments of Medicine, Obstetrics, and Gynecology. UMDNJ-Robert Wood Johnson Medical School. New Brunswick, NJ

LEARNING OBJECTIVES. 1) Recognize the differential diagnosis of vomiting in pregnancy, 2) Manage hyperemesis gravidarum once medications have failed, 3) Recognize the risks of starting parenteral nutrition in a pregnant woman.

CASE. 19 year old female G1P0 was admitted to the hospital at 7 weeks gestation with nausea and vomiting without abdominal pain. She was only able to tolerate sips of juice. Physical exam revealed volume depletion, normal thyroid, and normal abdomen. Intrauterine pregnancy was documented by ultrasound. Her lab evaluation included a urinalysis, electrolytes, TSH, liver enzymes, and an ultrasound of the liver and gall bladder. Except for hypokalemia and kentonuria, the labs were normal. After excluding molar pregnancy, thyroid, parathyroid, liver, and gall bladder disease, she was diagnosed with hyperemesis gravidarum (HG). She was placed on appropriate intravenous fluids and a medical subspecialist was consulted. The consultant ordered metoclopramide 10mg I.V. QID, famotidine 20mg I.V. BID, had a peripherally inserted central catheter (PICC) placed, and started her on peripheral parenteral nutrition without a trial of nasogastric (NG) feeding. A month later our medical consult service was called because she presented to the hospital with left upper extremity swelling, and pleuritic left chest pain. An ultrasound revealed thrombosis of the axillary vein, so the PICC was removed and she was placed on heparin using a weight-based normogram. V/Q scan was normal. In the hospital she tolerated a full oral diet, so we converted her to subcutaneous adjusted dose Heparin every 12 hours and discharged her. After three months of treatment the heparin was discontinued to avoid heparin associated bone loss, and she was followed closely for clinical signs of thrombosis.

DISCUSSION. Nausea and vomiting transiently affect about 50% of pregnant women, with HG affecting 0.1-1.0%. Inability to swallow saliva (ptyalism) should be differentiated from true HG. Treatment of HG may include the use of prokinetic agents, H2-receptor antagonists, and antiemetics. The potential risks to the fetus of medication exposure are generally outweighed by the ability to facilitate adequate maternal nutrition. As internists are often asked to see these women when medications fail, we should be familiar with the treatment options. We should also remember that pregnancy is associated with a relative thrombophilia due to a decrease in free protein S, a functional resistance to activated protein C that develops in up to 60% of women, an increase in fibrinogen, and decreased fibrinolysis. When medications have failed, it is reasonable to follow the adage "if the guts-works use it" and consider placing a weighted NG tube, preferably into the duodenum. While a "low tech" solution, an NG tube is preferable to the risk of infection and thrombosis associated with a chronic intravascular device. This case illustrates a thrombotic complication of parenteral nutrition that could have been avoided if a feeding tube, with its lower morbidity rate, had been placed.

TO OBTAIN XRAYS OR NOT TO OBTAIN XRAYS OF THE KNEES: THAT IS THE QUESTION. A Chandra and M Stellini, Department of Medicine, Detroit Medical Center/Wayne State University, Detroit, MI.

LEARNING OBJECTIVES. 1) Review clinical decision rule on indications for x-rays of the knees, 2) review the components of the physical exam of the knee, and 3) review the differential diagnosis of patellar tumors/pain.

CASE. A 52 yo female presented with a complaint of left knee pain which started while walking down some steps at work. She gave a history of slipping at home approximately 1 week prior and hurting her knee such that she could not walk on it for several hours. Physical exam showed a swollen knee with pain. The patient was unable to tolerate a physical exam of the knee other that that her range of motion was limited and neurologic exam was normal. X-rays of the knee demonstrated a comminuted patella fracture with some honeycomb appearance to the bone. Surgical repair was performed and pathological evaluation of the knee demonstrated a non-Hodgkin's lymphoma. Staging studies did not find any other sites of involvement. Patient was treated with chemotherapy and did well. Literature search did not reveal any reports of isolated patellar lymphoma.

Discussion. Though patellar tumors are a rare entity, knee pain is a common complaint in primary care physicians' offices. Radiographs of the knee are mostly unrevealing and add up to a significant cost over time. Thus, it is important for physicians to select those patients in whom x-rays may have a higher diagnostic yield. This presentation will review the literature on indications for knee radiographs and demonstrate the various components of a knee physical exam.

CONTEMPORARY MANIFESTATIONS OF VITAMIN B12 DEFICENCY. A Chandra and M Stellini, Department of Internal Medicine, Detroit Medical Center Wayne State University, Detroit, MI.
LEARNING OBJECTIVES. 1) Make Internist's aware of diverse presentation of B12 deficency, 2) Demonstrate the signs and symptoms of B12 deficency, 3) Demonstrate microscopic characteristics of macrocytic anemia, 4) Discuss the use of homocysteine and methylmalonic acid levels in diagnosis of B12 deficency, and 5) Discuss the prevalence of B12 deficency in the geriatric population.
CASE I. A 67 yo female presented with exertional dyspnea and syncope. Physical exam was significant for pallor, beefy red tongue, and posterior column deficiencies of the lower extremities. Lab analysis showed macrocytic anemia from B12 deficency with subacute combined degeneration from pernicious anemia.
CASE II. A 42 yo male with HIV and progressive multifocal leukoencephalopathy developed a painful neuropathy of both feet. A physical exam showed proprioception, vibration, pinprick, temperature, and gait abnormalities. Lab analysis was unrevealing except for a normocytic anemia. A B12 level was found to be extremely low. Patients symptoms improved with B12 supplementation.
CASE III. A 42 yo female was brought by family for worsening depression, related to a recent death of her brother. Previously she had complained of paresthesias of her lower extremities. A lab analysis demonstrated macrocytic anemia secondary to B12 deficency. Patients paresthesias and depression improved with treatment.
DISCUSSION. This series of cases demonstrate a diverse presentation of B12 deficency. Signs and symptoms were key in helping make the diagnosis. A great deal of evidence exists that geriatric patients are at risk for nutritional deficiencies due to several mechanisms. Recent literature also demonstrates that earlier diagnosis of B12 deficency can be made using methylmalonic acid and homocysteine levels.

TRY NOT TO PANIC WHEN EVALUATING CHEST PAIN AND PALPITATIONS. B. Clyburn and D. Schuyler, Department of Medicine and Psychiatry, Medical University of South Carolina, Charleston SC. LEARNING OBJECTIVES: 1) Recognize the prevalence and common presentations of panic disorder in primary care, and 2) Distinguish the patient with panic disorder from patients who require more extensive diagnostic evaluation.CASE: A 26 year old female without significant past medical history presented with a 3 year history of episodic palpitations, shortness of breath, chest pain, and diaphoresis. The patient's major concern was the sense of impending doom and fear of dying which accompanied these episodes. She reported that her symptoms generally started with frequent palpitations followed by shortness of breath and intense anxiety. Her symptoms generally progressed to the point where she became diaphoretic and experienced non radiating substernal chest pain with a feeling of choking. The patient reported that her "attacks" seemed to be occurring more frequently and without warning. She was unaware of any inciting factors, though the episodes seemed to occur more often in public places such as the mall. She had limited her shopping significantly because of her fear of these "attacks." The patient had previously been evaluated by several medical subspecialists for this constellation of symptoms with a negative workup which included: a normal EKG, normal exercise stress test, normal thyroid function testing, an unremarkable cardiac event monitor, normal 24 hour urine for metanephrines, and normal pulmonary function testing. Social history and family history were unremarkable. The patient's physical exam was completely normal. She was subsequently diagnosed with panic disorder. Though she was doubtful about this diagnosis, the patient agreed to a trial of pharmacologic therapy. She was subsequently started on paroxetine therapy at 10 mg daily which was subsequently titrated up to 40 mg per day with a marked diminution of her symptoms.
DISCUSSION: Most studies estimate the prevalence of panic disorder in primary care patients at between 5% and 10%, though a recent study in our own clinic population identified a 27% incidence. Panic disorder is characterized by frequent, spontaneous episodes of extreme anxiety with prominent symptoms of sympathetic activation. These episodes are often accompanied by feelings of impending doom, fear of dying, or an impulse to flee. Common symptoms include palpitations, chest pain, dyspnea, a sensation of choking, diaphoresis, paresthesias, dizziness, tremulousness, and diarrhea. Patients may become disabled by their fear of subsequent episodes and subsequently avoid public places. Panic disorder is underdiagnosed in the primary care setting despite the fact that panic disorder markedly increases the utilization of primary care services. Patients often "doctor shop", and these patients often take more than 10 years to be correctly diagnosed. As in our case, this can result in expensive and potentially dangerous diagnostic evaluations. However, careful history taking and an appropriate index of suspicion can result in rapid diagnosis and successful treatment.

AN ALTERNATIVE CAUSE FOR—AND TREATMENT OF—HEMOLYSIS. Carol Chou, Division of General Internal Medicine, University of Pennsylvania School of Medicine, Philadelphia, PA.
LEARNING OBJECTIVES. Distinguish oxidative injury, particularly G6PD deficiency, from other causes of hemolytic anemia. Treat symptomatic severe anemia in patients who decline blood transfusions. Identify potential health hazards of alternative therapies.
CASE. A 57 year-old African-American female Jehovah's witness was transferred from an outside hospital with shortness of breath, fatigue, hematuria, and anemia. She denied taking any oral medications but revealed that she was receiving weekly chelation therapy initiated by her family physician 3 months prior to admission to "cleanse her system." As part of the therapy, she had received a "massive dose" of vitamin C intravenously one day prior to onset of symptoms. The next day she noticed the passage of dark urine and began to feel extremely fatigued, short of breath on exertion, and lightheaded on standing. Physical examination was significant for icterus, postural tachycardia, and no splenomegaly. Laboratory data revealed a hemoglobin of 6.7, LDH of 1240, and a reticulocyte count of 11.7%; a smear was prepared. She was begun on intravenous methylprednisolone, and since her religious beliefs prevented her from receiving a blood transfusion, she was also given daily subcutaneous injections of erythropoietin and a dose of intravenous iron. Subsequently, Coombs' test was negative; steroids were discontinued. Examination of the smear showed bite cells indicative of oxidative injury. She continued to receive daily erythropoietin injections and oral iron. Her hemoglobin rose to 8.7 by hospital day 4 and 9.9 by hospital day 7. Symptoms resolved with the rise in hemoglobin and she was discharged in good condition on daily oral iron and folate. 3 months after hospitalization her hemoglobin had returned to 12.4. G6PD assay performed at that time was 5 (low).
DISCUSSION. G6PD deficiency is the most prevalent inborn metabolic disorder of red cells. Oxidative injury is suspected when tests for autoimmune hemolysis are negative and by the presence of Heinz bodies and bite cells on the peripheral blood smear. Assay for G6PD must be performed after the hemolytic episode has resolved. Precipitants of hemolysis in patients with G6PD deficiency are numerous; some agents, such as ascorbic acid, that are harmless at low doses can in high doses cause hemolysis. It is thus important to recognize when patients are using alternative therapies and the potential dangers that may arise from those treatments. Recombinant human erythropoietin has been used to treat anemia in a variety of settings and can result in significant acceleration of hemoglobin recovery within the first week of administration. With this and other measures, it is possible to honor the wishes of severely anemic patients who refuse blood transfusion.

EMPYEMA AS INITIAL PRESENTATION OF MYOTONIC DYSTROPHY. J Cummings, S Wood, G Wickstrom, and T Koenig. Summa Health System, Akron, OH.
Learning Objective: 1) Recognize dysphagia as an insidious presentation of myotonic dystrophy (MD); 2) Recognize respiratory infections due to dysphagia as common complications of MD.
Case: A 30 y/o Caucasian woman presented with a two-week history of dyspnea, sharp left sided posterior chest pain, orthopnea, malaise, anorexia, non-productive cough, and hoarseness. Review of systems, past medical and surgical histories were unremarkable. Social history was negative for alcohol, tobacco, or drug abuse. Her only medication was Naprosyn. Family history was significant for MD affecting her father and brother; the patient had a negative neurologic evaluation 5 years prior to admission. On exam, she was afebrile, BP 110/60, pulse 95 and regular, respirations 22. She was 65 inches tall, weighed 108 pounds, and cachectic. She had bilateral temporal muscle wasting and a laterally wide skull. She had diminished breath sounds with increased tactile fremitus over the left lower lung field. Cardiac and abdominal exams were normal. Neurologic exam revealed bilateral ptosis, inability to elevate her left pharynx, and a "wet" quality of phonation. Muscle strength was diminished in her upper extremities and intrinsic hand muscles and was normal in lower extremities. She had no action or percussion myotonias in the hand muscles. Lab findings revealed: white blood count of 20,200 with 48% granulocytes, 40% bands, and 11% lymphocytes; hemoglobin of 11.6; normal electrolytes; normal liver enzymes except for alkaline phosphatase of 447; ABG on room air showed pH 7.47, pCO2 42, pO2 61. EKG was NSR. Chest X-ray showed a large left pleural effusion, defined as multiloculated by CT.
Hospital Course: Empiric IV ampicillin/sulbactam and doxycycline were started. Bacterial and fungal blood cultures remained negative. A PPD was non-reactive. Thoracentesis revealed an exudative effusion which grew *S. pneumoniae*. Therapeutic drainage by chest tube was unsuccessful necessitating a thoracotomy and decortication procedure that was successful. Pathology results were consistent with an infectious empyema. Prior to surgery, a diagnosis of MD was made, based on classic EMG findings. A cookie swallow revealed severe laryngeal penetration with direct aspiration into the left lung. The patient was discharged 19 days after admission with a PEG tube for nutrition as well as ongoing dysphagia therapy.
Follow up: Six months later, the patient has gained weight, safely enjoys small meals of mechanical soft foods, has returned to work part-time, and will return to college soon.
Discussion: MD, the most common form of muscular dystrophy in adults, has an incidence of 1/10,000. It is autosomal dominant with variable expression. According to Cecil's and Harrison's medicine textbooks, MD affects skeletal, cardiac, and smooth muscle fibers resulting in the hallmark complications of muscle wasting and weakness, myotonias, cardiac conduction defects, hypoventilation, and impaired gastric and intestinal motility. Although well-documented in the literature, recurrent respiratory infections as common complications of MD are not mentioned in the general medicine textbooks. Our patient's presentation of silent dysphagia and empyema represents a new contribution to the MD literature and also illustrates a life-threatening complication of MD that may be avoided by early diagnosis and appropriate therapy.

SARCOIDOSIS PRESENTING AS ISOLATED LIVER AND BONE DISEASE Gary A Daniel and Mukta Panda, University of Tennessee College of Medicine-Chattanooga Unit, Chattanooga, TN

LEARNING OBJECTIVES Diagnose sarcoidosis even in the absence of pulmonary manifestations. Recognize sarcoidosis in the differential of osteoblastic lesions. Obtain tissue for definitive diagnosis of sarcoidosis.

CASE A 47-year-old black female presented with right upper quadrant pain and dyspepsia. Laboratory tests revealed abnormal liver function tests and liver biopsy revealed non-caseating granulomas consistent with sarcoidosis. The patient had no pulmonary symptoms and her chest x-ray was normal. A gallium scan showed mild abnormal pulmonary uptake but no definite abnormal uptake involving the hilar, paratracheal, or mediastinal lymph nodes. Angiotensin converting enzyme level was 39U/L. The patient's pain resolved after a short course of corticosteroids. Four years later the patient presented with vague low back pain. X-rays of the pelvis revealed osteoblastic lesions. Subsequent bone scan showed increased uptake in the pelvis, upper thoracic spine, and left femur suspicious for metastases. Biopsy of the pelvic lesion showed non-caseating granulomas consistent with sarcoidosis Again the patent denied any pulmonary symptoms and had a normal chest x-ray. Pulmonary function tests showed only a slightly reduced vital capacity but normal total lung capacity and no definite restrictive pattern. Her low back pain resolved after a course of corticosteriods. Repeat bone scan done 6 months later showed resolution of the focus of increased uptake in the left femur.

DISCUSSION Osseous involvement by sarcoidosis is rare and involvement of liver and bone in the absence of pulmonary and dermatologic symptoms has not been previously reported. Patients can have remissions for long periods before reoccurrence in different sites. This case stresses the importance of including sarcoidosis in the differential diagnosis of a patient with radiographic osteoblastic lesions, even in the absence of pulmonary or skin involvement. The bone scan is useful both for the diagnosis of osseous sarcoid, and as an aid in locating potential biopsy sites. Our case re-emphasizes the value of obtaining tissue for definitive diagnosis.

RECURRENT PANCREATITIS MASQUERADING AS HYPEREMESIS GRAVIDARUM. PR DeVersa, M Panda, C Schmitt, and M Taslimi, Departments of Internal Medicine and Obstetrics and Gynecology, UT College of Medicine – Chattanooga Unit, Chattanooga, TN.

LEARNING OBJECTIVES. 1) To recognize the clinical features of pancreatitis in early pregnancy, 2) To distinguish the most common causes of gestational pancreatitis 3) To review the evaluation and management of gestational pancreatitis.

CASE. A 20 year-old G3P0020 black female developed acute abdominal pain, nausea and vomiting at 9 weeks gestation. She was admitted for intravenous hydration with the diagnosis of hyperemesis gravidarum. Similar complaints in the first trimester of her last 2 pregnancies resolved with termination of the pregnancies. Pancreatitis was confirmed in the second pregnancy. She had no history of abdominal trauma, hyperlipidemia, gallstones, medication use, or alcohol use. Laboratory data revealed calcium 8.2 mg/dl, triglycerides 51 mg/dl, amylase 134 u/l, and lipase 273 u/l. Intact parathyroid hormone and abdominal ultrasound were normal. In spite of conservative treatment, the amylase and lipase levels continued to rise with accompanying clinical deterioration. The patient elected to terminate the pregnancy, which led to complete resolution of symptoms within 24 hours. The pancreatitic enzymes normalized. Outpatient endoscopic retrograde cholangiopancreatography (ERCP) revealed no evidence of stones, crystals, strictures or other anatomic abnormality. Nineteen months later the patient presented at 6-7 weeks gestation with nausea, vomiting and mid-epigastric pain radiating to her back. Her amylase level was 146 u/l. She, again, had rapid resolution of symptoms after termination of this 4th pregnancy.

DISCUSSION. Nausea is common in early pregnancy. Most cases are transient and do not require intervention. Nausea associated with abdominal pain must be evaluated in pregnant females. Epigastric pain that radiates to the back is suggestive of pancreatitis. Pancreatitis occurs in less than 1% of all pregnancies. The majority of cases occur in the last trimester. It is usually caused by cholilithiasis. Usual treatment of pancreatitis in pregnancy is bowel rest, intravenous fluid administration, and pain control. When severe, endoscopic or surgical management may be required. Pancreatitis rapidly resolving after elective termination of pregnancy has not been described. We postulate that increased levels of human chorionic gonadatropin and/or human placental lactogen modulate pancreatic receptors, which lead to pancreatitis in predisposed individuals.

THE DIAGNOSIS AND MANAGEMENT OF PLASTIC BRONCHITIS. Richard M. Del Sesto, Robert P. Baughman, University of Cincinnati College of Medicine, Cincinnati, Ohio.

LEARNING OBJECTIVES.
Recognize the clinical features of plastic bronchitis.

CASE. DP is a 37 year old white male who presented with complaints of an intermittent cough with a progressive increase in shortness of breath, increased sputum production and coughing up mucus plugs. His review of systems was significant for sweats without chills and chronic right sided chest pain. His past medical history is significant for plastic bronchitis diagnosed by open lung biopsy in 1992, SLE, recurrent pneumonia, duodenal ulcer, chronic pain, and ½ ppd of tobacco. On admission, the patient was afebrile and hemodynamically stable with 92% 02 stats on room air. He appeared to be in mild respiratory distress. His physical exam was significant for scattered expiratory wheezed and crackles, and bilateral coarse breath sounds. Except for mild peripheral edema of the lower extremities, the remainder of his physical exam was unremarkable. Initial lab work was significant for a 14k WBC. Chest x-ray revealed bibasilar interstitial fibrotic changes and right upper lobe bullae. There were no significant changes on EKG. The patient was empirically started on broad spectrum antibiotics, solumedrol, and albuterol HHN. He underwent multiple bronchoscopies and had aggressive pulmonary toileting in effort to clear mucus plugs. His bronchoscopies revealed mucus plugs, increased secretions, and no endotracheal lesions. The patient's symptoms gradually improved and he was discharged.

DISCUSSION. Plastic bronchitis is a rare disorder characterized by the presence of inspissated long, branching bronchial casts that may be expectorated, found at bronchoscopy or in surgical specimens. The pathophysiology is unknown. It is unclear whether abnormalities in mucus, or clearance, or both allow airway secretions to solidify and form a cast. However, complications of their presence can include lobar or total lung collapse, secondary pneumonia, and life-threatening asphyxia. Clinically, patients usually have productive coughs, dyspnea, fever, wheezing, and occasionally chest pain and hemoptysis. Plastic bronchitis can be classified as a member of a group of diseases associated with large mucoid casts. Plastic bronchitis occurs at all levels of the bronchial tree, but is more common in the central airways and lower lobes. Therapy for plastic bronchitis consists of specific measures to treat the underlying pulmonary condition as well as maneuvers designed to frequently remove or facilitate the expectoration of bronchial casts. Plastic bronchitis is usually self-limited or responsive to medical therapy, with a good prognosis.

PHYSICAL RESTRAINTS IN THE NURSING HOME SETTING: HELPFUL OR HARMFUL? PR DeVersa and A Rybolt, Department of Internal Medicine, UT College of Medicine - Chattanooga Unit, Chattanooga, TN.

LEARNING OBJECTIVES. 1) Recognize the hazards of physical restraint use, 2) Recognize the circumstances that warrant physical restraint use, and 3) Recognize alternatives to physical restraint use.

CASE. An 81 year-old demented female was admitted to a nursing home after progressive confusion and falls. She required the use of a walker due to imbalance, had occasional urinary incontinence, and needed assistance with dressing and toileting. She scored 2 on the Mini-Mental State Examination. On the night of admission, she became more confused and was found to be climbing out of bed. She had a posey vest restraint applied and the bed side rails were raised to prevent her from falling out of the bed. She became combative on the 4th night of her stay and was moved to a different room on the 11th night. The next morning she was found dead with her head entrapped in the side rail and her feet and legs on the floor in a sitting position. The probable cause of death, as determined by the medical examiner, was neck compression.

DISCUSSION. Physical restraints do not reduce injuries. They are associated with many adverse effects, including skin breakdown, more frequent falls, increased incontinence, serious accidents and death. The use of physical restraints in the elderly has decreased in recent years. Their use should be reserved for circumstances in which they are required to treat medical symptoms. Alternatives to restraints are best delineated with a multidisciplinary team evaluation of the patient. These include positioning devices, medication reduction, physical therapy, scheduled toileting, and "alarm" devices that prompt staff when a patient is wandering off the unit, getting out of bed, or getting out of a wheelchair.

AMPHOTERICIN B-ASSOCIATED PULMONARY TOXICITY, S. Eapen

LEARNING OBJECTIVE: Recognize acute pulmonary toxicity as a side effect of intravenous (i.v.) infusion of Amphotericin B.

CASE: A 34 year old Hispanic male presented with a two week history of spiking fever, malaise and headache. His past history was significant for a seizure disorder and multiple sexual partners. Physical examination showed generalized lymphadenopathy and hepatomegaly. Routine laboratory investigations on admission were normal. Chest xray revealed bilateral hilar lymphadenopathy. A spinal fluid India ink preparation was positive for cryptococcus. Amphotericin B and Flucytosine were started. On day two, thirty minutes after starting the infusion of Amphotericin, the patient had a sudden onset of severe respiratory distress and hypoxia (pO2 was 36 mm of Hg.). Amphotericin was stopped and the patient had to be intubated. He was also given one dose of i.v. Solumedrol and Benadryl. Repeat chest xray showed bilateral diffuse infiltrates. His respiratory status improved quickly and he was extubated the next day. He was further treated with Diflucan. He had a prolonged hospital stay during which HIV infection was confirmed.

DISCUSSION: The most common side effects of Amphotericin B are fever and chills followed by nausea and vomiting. We could find only two published cases of acute pulmonary toxicity. This usually occurs within the first two hours of starting the infusion. In the present era of AIDS and AIDS-related fungal infections use of this drug is becoming more frequent. Hence clinicians should be aware that though uncommon, life threatening respiratory reactions can occur. These reactions appear to be reversible by stopping the drug promptly.

CHRONIC PANCREATITIS MIMICKING PANCREATIC CARCINOMA

Authors: A. Esho, R. Gregorio, and V. Bahl, UPMC Shadyside, Pittsburgh, PA

LEARNING OBJECTIVE: Recognize the clinical, radiologic and laboratory similarities between chronic pancreatitis and pancreatic cancer.

CASE: A 57 year-old man with a history of type 2 diabetes mellitus and peptic ulcer disease presented to our hospital with a 3 week history of severe epigastric abdominal pain, nausea and early satiety symptoms. He described his pain as dull and constant with radiation to the right upper quadrant area. He admitted to a 14 pound weight loss over the previous 6 months. He denied noticing any jaundice or change in the color of his urine or stool. He initially saw his PCP who treated him with antacids and H2 blockers with only minimal relief of his symptoms. Physical examination was unremarkable except for cachexia and marked epigastric tenderness. Laboratory investigations were notable for a glucose of 161, alkaline phosphatase of 201, amylase of 246, lipase of 580, and a CA 19-9 of 50 (0-37). A CT scan of the abdomen revealed a mass in the head of the pancreas with slight dilatation of biliary and pancreatic ductal structures. An attempted CT-guided needle biopsy of the pancreas was inconclusive. A selective angiography revealed subtle changes of neovascularity in the pancreatic head with effacement of the dorsal and transverse pancreatic arteries as well as slight compression of the portal vein, all suggestive of a pancreatic neoplasm. He then underwent a laparotomy where a pancreaticoduodenectomy was performed for a presumed pancreatic cancer. His histopathology however returned with the diagnosis of chronic sclerosing pancreatitis.

DISCUSSION: Epigastric pain and weight loss are two clinical features in our patient common to both chronic pancreatitis and pancreatic carcinoma. An elevated CA 19-9 and a mass in the head of the pancreas however are more typical of pancreatic cancer. Nevertheless, our patient's final diagnosis was chronic pancreatitis. Review of the literature reveals that while CT remains a useful test in evaluating a patient suspected of having a pancreatic lesion, it can be difficult in distinguishing between pancreatic cancer and chronic pancreatitis as both conditions can present with a focal mass. A focal mass is seen in about 80% of the former and about 30% of the latter. In addition, a positive CA 19-9 is found in about 80% of patients with pancreatic cancer and in 11% of patients with chronic pancreatitis. The above case highlights the clinical, radiologic and laboratory similarities between the two conditions. Therefore, the only reliable method of distinguishing them is by tissue examination.

BILATERAL EXTRACRANIAL CAROTID ARTERY DISSECTION COMPLICATED BY CRANIAL NERVE XII PALSY

Authors: R. Erlich, J. Quaye, M. Weinfeld, and A. Tulsky, UPMC Shadyside, Pittsburgh, PA

Learning Objectives: 1) Recognize a rare presentation of extracranial carotid artery dissection. 2) Review presentation, etiology, treatment and prognosis of extracranial carotid artery dissection. 3) Recognize carotid dissection as a possible cause of cranial nerve XII palsies.

Case: 48 year old male with history of poliomyelitis as a child, developed sinus infection associated with strong and recurrent coughing spells two weeks prior tc admission. He was treated with antibiotics and steroids but, four days prior to arriving at our hospital, developed severe bilateral headache, not associated with nausea or vomiting or photophobia, but associated with neck "stiffness" and "feeling of a swollen tongue". The patient had no change in hearing, no tinnitus, no visual changes, no dysphagia. No history of diabetes, hypertension, cancer or connective tissue disease. No family history of neurologic disorders. **Physical Examination:** Vital signs were normal. Neurologic examination was only remarkable for tongue deviation to the left with atrophy. Rest of the physical examination was normal. Laboratory: CBC, differential, plts, PT, PTT, glucose, electrolytes, C-reactive protein, sed rate, ANA were all normal. Patient refused a lumbar puncture. MRI with gadolinium and MRA revealed bilateral carotid artery dissections at the skull base beginning at the mid cervical region, proximal to the narrowest portion of the left internal carotid artery with pseudoaneurysm. No signs of fibromuscular dysplasia, or brain stem infarct. Patient was placed on heparin followed by coumadin and was discharged on day four with improvement in neurologic symptoms. **Discussion:** 1) Cervical carotid artery dissection is predominant in the middle adult years, with only 14% of cases occurring bilaterally and only 4% affecting cranial nerve XII. 2) Major symptoms are headache (65%), tinnitus (30%), neck pain (20%). Major signs are partial Horner's syndrome, monocular blindness, and cervical bruit. 3) In the absence of fibromuscular dysplasia and connective tissue disease, coughing is the probable triggering factor in the case presented. 4) Anticoagulation is indicated to prevent transient ischemic attacks and stroke, with 70% of the patients with normal or almost normal artery after a median of six weeks. Recurrence rate is 3%. 5) Differential diagnosis include poliomyelitis, intramedullary tumor, motor neuron disease, diabetes, lesions of the basal meninges and occipital bones, base of skull mass, aneurysm, nasopharyngeal mass, carcinomatous meningitis and craniocervical anomaly.

POSTPERICARDIOTOMY SYNDROME: AN UNUSUAL PRESENTATION

Authors: A. Esho, K. Mansilla, I. Domat, and M. Roberts, UPMC Shadyside, Pittsburgh, PA

LEARNING OBJECTIVES: 1) Recognize that a late presentation of the postpericardiotomy syndrome (PPS) exists, and 2) recognize PPS in the absence of a fever and ECG findings of acute pericarditis.

CASE: A 67 year old man who underwent complex open heart surgery was admitted to our hospital, approximately 15 weeks after the surgery. He complained of a sudden onset of severe left shoulder pain of several hours in duration. The original surgery included aortic valve replacement, repair of an ascending aortic aneurysm and 5-vessel coronary artery by-pass surgery. He described the pain as pleuritic and radiating to the left chest area. His pain was not relieved by sitting or leaning forward. He denied shortness of breath, fever or chills. He denied evidence of a recent viral illness. Physical examination revealed that he was hemodynamically stable and in no apparent distress. He had a grade 3/6 systolic ejection murmur. A pericardial friction rub was not appreciated. Laboratory investigations were notable only for an ESR of 27. An ECG was remarkable only for an old inferior wall myocardial infarction. An ECHO showed the presence of a small pericardial effusion. He was diagnosed with PPS and was treated with indomethacin. He responded remarkably well and was discharged home asymptomatic within 3 days.

DISCUSSION: According to the literature, PPS typically occurs within 4 weeks of cardiac surgery and is characterized by chest pain, fever, and pericarditis(pericardial friction rub and/or pericardial effusion). The above case illustrates that PPS can be a late presentation. Our patient did not report a fever and classic ECG changes of acute pericarditis were not present. The diagnosis was made based on the presence of a pericardial effusion. Although the possibility of a post-viral pericarditis cannot be completely eliminated, the lack of a viral prodrome, the recent cardiac surgery, and the rapid response to an anti-inflammatory agent make this less likely.

THYROTOXIC GRAVES' DISEASE WITH PROFOUND ANEMIA, THROMBOCYTOPENIA AND LYMPHADENOPATHY. Dr. C. Fanning, Dr. M. Nyman, Dr. B. Clarke, Mayo Medical Foundation, Rochester, Minnesota.
LEARNING OBJECTIVES: 1) Recognize typical and atypical presentations of hyperthyroidism, and 2) Recognize thrombocytopenia and anemia as potential complications or associations of hyperthyroidism
CASE: A 19 year old male presented with a one month history of palpitations, tremor, fatigue, 15 pound weight loss and a two week history of dyspnea, diarrhea, increased sweating and heat intolerance. His past history was unremarkable. He denied any preceding illness or drug abuse. Family history was remarkable only for Graves' Disease with ophthalmopathy in his mother in her teenage years.

Examination revealed a resting tachycardia of 120, respiratory rate of 24; mild bilateral proptosis, chemosis, lid lag and retraction; smooth, non tender thyroid of approximately 80 gm, with bruit; tender lymphadenopathy in the cervical, posterior auricular, supra-clavicular, axillary and inguinal areas; ejection systolic murmur; mild tremor, symmetrical hyperreflexia and bipedal petechiae.

Investigations revealed: hemoglobin 4.5, MCV 89, absolute reticulocytes 7.1, leukocytes 10.1, platelet count 11 with MPV 8.7; TSH 0.027, total thyroxine 15, free thyroxine 6.3, triiodothyronine 582, thyroid stimulating antibodies 5.7 and thyroid peroxidase antibodies 1530; negative hemolytic and disseminated intravascular parameters. Blood smear revealed anisocytosis and hypochromic, microcytic erythrocytes; cytogenetic and molecular genetic studies were normal; bone marrow studies showed 25% cellularity, reduced erythrocyte precursors and marked reduction in megakaryocytes – suggestive of drug or toxic effect. Viral serology was negative. CT scans showed extensive thoracic, abdominal and pelvic lymphadenopathy. Graves' disease was diagnosed, with bone marrow suppression and reactive lymphadenopathy.

Treatment with propylthiouracil, prednisone, propranolol and blood products resulted in symptom improvement and marked resolution of thyromegaly and lymphadenopathy. At discharge thyroxine was total 10.0 and free 1.9. One month later, the patient discontinued his medications and was re-admitted with a recurrence of symptoms. Radioactive iodine study revealed uptake of 51.3% at 24 hours. The patient underwent radioiodine therapy with subsequent normalization of thyroid and blood indices.
DISCUSSION: Thrombocytopenia is seen in 3% to 14% of hyperthyroid patients. Suggested hypotheses include a similar autoimmune pathogenic mechanism, a thyrotoxic-induced decrease in platelet survival and a genetic predisposition for autoimmune disease. Bone marrow erythroid hyperplasia and mild erythrocytosis is usual in hyperthyroidism with mild anemia seen in 10% to 25% of cases, most commonly secondary to iron, B12 or folate deficiency. Thymus and mediastinal lymph node enlargement associated with hyperthyroidism has been rarely reported. There is no report to date of Graves' Disease associated with extensive lymphadenopathy and bone marrow suppression, resulting in severe anemia and thrombocytopenia.

SHE SWALLOWED A FORK?! AN UNUSUAL PRESENTATION OF A COMMON DISORDER. Stephanie Fein and Cindy Caffrey., Department of Medicine, UCLA- Olive View Medical Center, Sylmar, CA.
LEARNING OBJECTIVE. Recognize the signs and symptoms associated with Bulimia in the primary care setting.
CASE. A 22 year old female college student was admitted from the Emergency Department with sudden onset of sore throat, a choking sensation, and shortness of breath after accidentally swallowing a plastic fork. She denied hematemesis, fever, or chest pain. Upon further questioning, the patient admitted she had been using the utensil to induce vomiting. She admitted to an obsession with eating and weight control for several years as well as intermittent episodes of starvation, excessive exercise, diet pill and laxative abuse. Daily episodes of bingeing and purging had been increasing in frequency during the past several months. She stated her finger could no longer induce a gag reflex, so she began using objects that could reach further down her throat. On review of systems, the patient had 20 pound weight loss and irregular menses during the past year. Her physical exam was remarkable for a thin, mildly anxious female. Her dentition appeared normal. Her abdominal exam was significant for mild tenderness in the right upper quadrant. Small abrasions and a callus were noted on the dorsum of her right second metacarpophalangeal joint (MCP). Pertinent labs included sodium 144 mEq/L, potassium 3.7 mEq/L, chloride 101 mEq/L, and bicarbonate 31 mEq/L. Abdominal series showed no free air.
DISCUSSION. Bulimia Nervosa is defined by the DSM IV as binge eating and self-induced vomiting a minimum of two times per week for three consecutive months. Bulimia is most common in young women with a reported incidence between 1-10%. Although the morbidity associated with this disease is great, the mortality is small but significant. Some of the potentially fatal complications include esophageal or gastric rupture, Mallory-Weiss tear, and arrythmias secondary to an electrolyte abnormality. Bulimia Nervosa can be a difficult diagnosis to make. Unlike the cachexia associated with Anorexia Nervosa, patients with Bulimia are usually of normal weight. Russell's sign (abrasions and/or calluses on MCPs) is commonly seen in people who repetitively induce vomiting. Other physical findings which may lead physicians to suspect Bulimia include bilateral parotid gland swelling, dental erosions, deterioration of tooth enamel, and subconjuctival hemmorhages. In addition, patients with Bulimia often present to a primary care physician for a variety of seemingly unrelated nonspecific complaints. These can include fatigue, heartburn, sore throat, constipation, and abdominal bloating. It is imperative that physicians confronted with this constellation of symptoms and subtle physcial findings suspect Bulimia. Our case illustrates an unusual presentation of Bulimia and serves to remind us of the potential for serious complications associated with this disease.

A COMMON PRESENTING SYMPTOM WITH AN UNCOMMON BUT VERY SERIOUS CAUSE. B Favrat, A Pécoud, Medical Oupatient Clinic, University of Lausanne, Switzerland.
LEARNING OBJECTIVE. To recognize that presentation of very common diseases may be misleading and that even when the diagnosis seems to be straightforward, the clinician should remain vigilant to the possibility of other diagnosis.
CASE. A 71-year-old white man with complaints of two months' duration presented at our facility. He reported severe neck pain with neck stiffness and had even been hospitalized in another facility two months previously at the onset of the symptoms. Cervical spine X-rays and a cerebral CT scan were performed. He was discharged with the diagnosis of cervical spondylosis and was treated with non-steroidal anti-inflammatory drugs and muscle relaxants. Because his condition did not improve, he presented to two other physicians and physical therapy was prescribed with the same diagnosis. Pertinent aspects of his medical history included alcohol and tobacco abuse. Physical examination revealed severe neck stiffness and pain. Neck rotation was severely limited. Diffuse enlargement of the left side of the neck was noted. No adenopathy was found nor any abnormality of his oral cavity. He was afebrile and his white blood count was normal. Cervical spine X-rays showed cervical spondylosis with extensive degenerative changes but at C1 and C2 there were changes very suggestive of vertebral osteomyelitis. Magnetic resonance imaging clearly demonstrated a retropharyngeal abscess and findings consistent with osteomyelitis involving the C1 and C2 vertebral bodies. The diagnosis of retropharyngeal abscess was made. Because of the risk of cord compression, the patient underwent surgery. Culture of the abscess fluid revealed group G Beta-hemolytic streptococci. After surgery and antibiotics, neck pain improved and the patient was finally discharged with no trouble with his neck.
DISCUSSION. Our patient had been evaluated by several physicians and had been treated for cervical spondylosis. In this case, the presence of severe limitation of neck rotation should have alerted the physician to the possibility of more serious disease necessitating further testing. When confronted with common presenting symptoms, the physician should take atypical signs and symptoms into consideration.

AVASCULAR NECROSIS AS A CAUSE OF KNEE PAIN IN A PATIENT WITH AIDS. M Fingerhood, Department of Medicine, Johns Hopkins Bayview Medical Center, Baltimore, MD
LEARNING OBJECTIVES. Recognize avascular necrosis as a cause of joint pain in individuals with HIV, especially after treatment with steroids.
CASE. A 40 year old HIV positive man presented with a several weeks of right knee pain. There was no history of trauma. He had been HIV positive for 8 years and AIDS defined by a bout of pneumocystis pneumonia 7 months previous, treated with trimethoprim-sulfa and prednisone. His most recent CD4 count was 106. His medications were stavudine, lamivudine, nevirapine, azithromycin and trimethoprim/sulfa. He was not sexually active. Physical exam was remarkable for absence of fever and an abnormal right knee- diffuse swelling with obvious effusion, no erythema, mild warmth, no crepitance and no ligamentous instability. All other joints were normal. A knee tap of grossly serosanguinous fluid showed RBC count 618,000 and WBC count 330 (76% monos, 24% polys). Culture was negative and no crystals were seen. Knee x-ray showed no degenerative changes. Naprosyn was prescribed for pain relief. Despite some improvement in swelling, the knee continued to be very painful. Four months later, the patient presented with a petechial rash and a platelet count of 10K. A bone marrow aspirate showed adequate megakaryocytes and a presumptive diagnosis of thrombocytopenia secondary to trimethoprim-sulfa was made. Trimethoprim-sulfa was stopped and prednisone was started. Over the next month, his platelet count returned to normal and predisone was stopped. However, three weeks into the prednisone therapy, he complained of worsening right knee pain. Repeat x-ray of the knee was unchanged, but a MRI of the knee revealed extensive avascular necrosis with bone infarcts in the patella, and medial and lateral condyles of the tibia and femur.
DISCUSSION. Avascular necrosis presenting as joint pain may occur in HIV positive individuals after short exposures to prednisone. The diagnosis requires MRI and can be missed by plain x-ray.

DEEP VEIN THROMBOSIS DUE TO INFERIOR VENA CAVA OBSTRUCTION BY UTERINE LEIOMYOMAS. D Forman, D Muller, Division of General Internal Medicine, Mount Sinai Medical Center, NY, NY.

LEARNING OBJECTIVES. 1) Identify potential causes of deep vein thrombosis, 2) Assess the utility of hypercoagulability work-ups, and 3) Treat IVC obstruction in the face of acute thrombosis.

CASE. A 30 year old African American female presented to the Emergency Room with a 9 day history of progressive right leg swelling. Her past medical history was significant for uterine fibroids with subsequent meno/metrorrhagia and iron deficiency anemia. The patient had never been pregnant and was not using oral contraceptives. On physical examination her vital signs were unremarkable, lungs were clear, cardiac rate and rhythm were regular and no gallops were auscultated. The abdomen was remarkable for a non-tender 20 week uterus. There was 2+ pitting edema involving the right lower extremity extending up to the thigh without palpable cords or Homan's sign. Labs were remarkable for a microcytic hypochromic anemia, normal PT and PTT, and a negative pregnancy test. A duplex exam of the right leg revealed a deep venous thrombosis (DVT) from the common femoral vein to the popliteal vein. The patient was treated with intravenous heparin. Subsequent labs were remarkable for ANA 1:320 homogeneous, ESR 87, and free Protein S antigen 50 (70-140). Anti-cardiolipin antibody, Lupus anticoagulant, Anti-Thrombin Three, Factor V Leiden, Protein C, and homocysteine were normal. Pelvic ultrasound revealed a 20 week uterus with a large fundal myoma and normal ovaries. CT scan of the abdomen and pelvis revealed marked compression of the IVC by a 20x14x13cm heterogeneous pelvic mass, with a DVT that extended from the right common femoral vein to the distal IVC. Because of the high risk of peri-operative embolism, a Greenfield filter was inserted prior to performing a myomectomy. The procedure was uncomplicated and the patient was sent home to complete a six month course of oral anticoagulation.

DISCUSSION. Despite their prevalence, our review of the medical literature revealed only two reports of uterine fibroids as the underlying cause of a lower extremity DVT. In light of an obstructing lesion, it is unclear whether there is any utility in searching for an underlying hypercoagulable state.

ABDOMINAL PAIN IN PREGNANCY. Dorothy Graham, Raymond Powrie, Karen Rosene-Montella, Division of Obstetric and Consultative Medicine, Department of Medicine, Women and Infants' Hospital, Brown University, Providence, Rhode Island.

"LEARNING OBJECTIVES." 1) Recognize the unique differential diagnosis of abdominal pain in a pregnant woman, and 2) Recognize the presentation and management of HELLP (hemolysis, elevated liver function tests and low platelets) syndrome.

"CASE". A 33 year old woman 33 weeks into her third pregnancy presented with a 3 day history of intermittent severe epigastric pain which radiated to the right upper quadrant and back. The episodes lasted from 1-2 hours, had no exacerbating or relieving factors and were associated with nausea and on one occasion vomiting. Her previous pregnancies were unremarkable apart from intrauterine growth restriction in her first pregnancy, and she had a past history of renal calculi.

On physical examination she was afebrile, had a pulse rate of 68/min and a BP of 129/71. Her cardiac examination was normal. Respiratory examination revealed scattered wheezes. There was mild epigastric tenderness, her uterus was palpated at a height consistent with dates and there was moderate peripheral edema. Fundoscopy and neurological examination was normal. A urinalysis showed no protein or blood. A complete blood count revealed a hemoglobin of 13.3 g/dl, platelet count of $220x10^3$/ul and white cell count of $6.1x10^3$/ul. SGOT was 35 U/L, SGPT was 27 U/L, serum amylase was 77 U/L. Abdominal ultrasound showed normal liver, pancreas, spleen and kidneys. Chest X ray showed small bilateral pleural effusions. A differential diagnosis of peptic ulcer disease, biliary colic, pancreatitis and renal colic was considered. She was commenced on ranitidine and admitted for observation and analgesia. Her pain resolved that evening, but awoke her again at 4am. Her examination at that time remained unchanged, but a repeat CBC revealed that her platelet count had fallen precipitously to $28x10^3$/ul and her hemoglobin to 10.8 g/dl. The peripheral smear showed a few schistocytes. Repeat SGOT was 202 U/L, SGPT was 160 U/L, total bilirubin was 1.6 mg/dl and LDH was 432 units/mL. APTT was 26.6 seconds, INR was 1.0 and fibrinogen was 462 mg/dL. Uric acid was 5.0 mg/dL and creatinine was 0.6mg/dL. Repeat urinalysis remained normal. A diagnosis of HELLP syndrome was made and she was treated with betamethasone and a magnesium infusion. A live male infant was delivered by cesarian section. Her abdominal pain resolved rapidly postpartum. The transaminase and platelet levels normalized over the next 3 days.

"DISCUSSION". Preeclampsia, HELLP syndrome and acute fatty liver of pregnancy should always be considered in the differential diagnosis of pregnant women with abdominal pain. This case also highlights the importance of careful reassessment of patients in whom the diagnosis is not clear at initial presentation.

AN ELDERLY WOMAN WITH DYSPEPSIA AND WEIGHT LOSS. JM Fox, JM Watanabe, Department of Medicine, University of Washington, Seattle, WA.

LEARNING OBJECTIVE. Recognize an uncommon disease in a patient with a common constellation of symptoms refractory to empiric interventions.

CASE. A 76 year-old Champa female from Cambodia presented with a three month history of progressive abdominal pain and anorexia associated with 30 pound weight loss. Three months prior to her hospitalization, on her initial outpatient presentation, the patient was diagnosed with a viral gastroenteritis. Her initial symptoms waned but persisted and the patient subsequently failed a course of a histamine H_2-receptor antagonist with minimal relief. The patient's stool revealed no evidence of a parasitic infection. Her thyroid test, lipase level, liver enzyme tests, and viral hepatitis screen were negative. The patient was then initiated on a proton pump inhibitor, again with only minimal relief. With persistent symptoms, the patient underwent an esophagogastroduodenoscopy that revealed both erosive esophagitis and multiple gastric ulcerations. The patient's antacid regimen was increased. The patient's biopsies showed no evidence of malignancy or Helicobacter pylori infection. Two weeks prior to her hospitalization she developed a watery diarrhea occurring three or more times per day. Her past medical history was remarkable for type 2 diabetes mellitus, coronary artery disease, hypertension, and hypercholesterolemia. She did not smoke or drink alcohol and denied use of non-steroidal anti-inflammatory drugs other than her daily aspirin. On admission to the hospital, the patient was cachectic with evident orthostasis secondary to overt dehydration. Her physical exam was largely unremarkable. Her abdomen was soft with normoactive bowel sounds and no hepatosplenomegaly. The patient had mild epigastric tenderness. Her stool guaiac was negative.

DISCUSSION. Prior to her hospitalization, the patient had undergone and failed a progressive antacid regimen for multiple endoscopically identified gastric ulcerations which were not associated with NSAID use, a Helicobacter pylori infection or an evident malignancy. The multiple non-healing gastric ulcers, the chronic nausea, the intermittent diarrhea with significant weight loss and possible malabsorption suggested a unifying diagnosis of the Zollinger-Ellison Syndrome. An admission serum gastrin level was elevated to 1315 mg/dl (normal <100 mg/dl). Her gastric fluid pH was 2.0, off the proton pump inhibitor, ruling out achlorhydria as a cause of the elevated gastrin. Hence, a probable diagnosis of Zollinger-Ellison Syndrome was made. Abdominal imaging including CT scan and somatostatin receptor scintigraphy failed to localize the gastrinoma. The patient was started on high-dose proton-pump inhibition with improvement in her abdominal pain, appetite, and nausea, vomiting and diarrhea. This case illustrates the patient who presents with common but persistent and progressive symptoms which declared themselves as the manifestation of a relatively rare disease.

POSSIBLE S.bovis MENINGITIS AND ADENOCARCINOMA OF THE COLON. N.M.Guda, A.Affi, N.Vakil, Univ. of Wisconsin, Milwaukee clinical campus, Sinai Samaritan Med.Ctr, Milwaukee, WI.

LEARNING OBJECTIVES. 1) Streptococcus bovis bacteremia is associated with colonic malignancy.2) S.bovis is an uncommon pathogen causing meningitis. There is no definite data correlating a cause association between S.bovis meningitis and colonic malignancy. When meningitis is the initial presentation it is reasonable to look for occult GI malignancy.

CASE. An 88-yr. Old male was brought to the emergency room by his son who found him unresponsive. His past medical history was significant for recently diagnosed anemia for which he refused any further work up. On evaluation he was found to be lethargic and unresponsive to questions. He had nuchal rigidity and a temperature of 103 degrees Fahrenheit. He was anemic and fecal occult blood test was positive. There was no clinical evidence of endocarditis. Rest of his exam was normal. CSF analysis: 2950 WBC, glucose 37mg/dl, and protein 562mg/dl. Gram's stain revealed no organisms. Blood cultures were positive for S.bovis and CSF culture failed to grow any organisms. He was hemodynamically stable and he improved clinically with appropriate supportive therapy. A colonoscopy was later performed and he was noted to have a mass, which was histologically consistent with adenocarcinoma. He refused any intervention and was later discharged to hospice care.

DISCUSSION: S.bovis is an uncommon pathogen causing meningitis. There are only a few cases reported and review of literature shows that GI disease, endocarditis and oral lesions are predisposing factors. The neurological findings are often subtle and the organism is usually not seen on Gram's staining. Response to Penicillin G is very good. Currently there is no consensus on the need for a work up for GI malignancy in patients presenting with S.bovis meningitis, however it may be prudent to do so since atleast a fourth of the patients were found to have GI tumors.

CONSTRICTIVE PERICARDITIS: DIFFERENTIAL DIAGNOSIS BY CLINICAL, HEMODYNAMIC, ECHOCARDIOGRAPHIC, AND RADIOGRAPHIC FINDINGS. RJ Gumina[1], EA Pfeiffer[2], JK Oh[3], and MD McGoon[3]. Department of Internal Medicine[1], Division of Cardiology[3], and Department of Pathology[2], Mayo Clinic, Rochester MN.

LEARNING OBJECTIVES: Distinguish constrictive pericarditis from restrictive cardiomyopathy based upon clinical, hemodynamic, echocardiographic and radiologic findings.

CASE: A sixty-six year old female presented to Mayo Clinic with a fourteen month history of ankle swelling, and dyspnea which despite aggressive medical therapy for congestive heart failure progressed to NYHA Class III/IV. The physical exam revealed an elevated jugular venous pressure with distension to the angle of the jaw in a sitting position. Cardiac examination revealed a regular rate and rhythm, faint to normal S1 and S2, but no murmurs, gallops, or rubs. Crackles, egophony, and decreased vocal fremitus were appreciated on the right lung field. The abdomen was distended with shifting dullness and hepatomegaly. The legs were edematous. Anemia, elevated liver enzymes, elevated creatinine, and a right-sided pleural effusion on chest x-ray were noted. Pulmonary function tests revealed a moderate to severe restrictive pattern. ECG demonstrated nonspecific T wave inversion in the anterior leads with marginally decreased voltage. Cardiac catheterization from 4 months previously showed normal coronary arteries and elevated right atrial, right ventricular, pulmonary artery and pulmonary capillary wedge pressures. Echocardiography revealed a left ventricular ejection fraction of 60%, mild right ventricular enlargement, and changes of ventricular interdependence by 2D/Doppler. Computerized tomography of the chest demonstrated a dilated inferior vena cava and circumferential pericardial thickening causing compression of both the right and left ventricles. The patient underwent a pericardiectomy that revealed an adhesive dense fibrotic non-calcified pericardium over all aspects of heart. Tissue stains were negative for tubercle bacilli. Following surgery, the patient experienced multiple episodes of hypercapnic respiratory failure and expired. Autopsy demonstrated a remarkably thickened remaining pericardium, bilateral pleural fibrosis, and cardiac sclerosis of the liver; however, no specific etiologic factors or cause of death were determined.

DISCUSSION: Patients with non-calcific pericarditis may be difficult to distinguish from restrictive cardiomyopathy. Differentiation is crucial since pericardiectomy may improve hemodynamic and clinical status, whereas therapy for restrictive cardiomyopathy is limited to palliation or treatment of the underlying cause. Although surgical pericardiectomy is the only definitive management of constrictive pericarditis; a worse prognosis is associated with inadequate resection, myocardial involvement, progression to calcification, and a higher NYHA classification.

A WONDERFUL DRUG AND A WORRISOME SIDE EFFECT: TICLODIPINE AND THROMBOTIC THROMBOCYTOPENIC PURPURA (TTP) H.S Gurm, W.R ten Hove, B Pohlman. Department of Medicine and Hematology and Oncology, Cleveland Clinic Foundation, Cleveland, OH

LEARNING OBJECTIVES

1 Discuss the pharmacology and side effects of ticlodipine.
2 Review pathology and management of TTP
3 Review the literature on ticlodipine induced TTP

CASE

A 70-year-old white male presented to his local hospital with an inferior wall myocardial infarction. His other medical problems included hypertension and chronic obstructive airways disease. He had had rheumatic fever as a child without any long-term sequelae. Complete blood count, electrolytes, blood urea nitrogen and creatinine were normal. He was treated with beta-blockers, nitrates and Heparin. Seven days later he underwent cardiac cathetrization, balloon angioplasty and stenting of the right coronary artery. He was discharged home on ticlodipine, atenolol and enalapril. One month later he was readmitted to the same hospital with confusion and a rash on the extremities. A CT of the brain showed mild diffuse cortical atrophy. He was transferred to a tertiary care facility. Examination revealed a pale mildly confused elderly white male. Vital signs were normal. A non-palpable purpuric rash was present on all the extremities. There was no jaundice or lymphadenopathy. A right-sided carotid bruit was audible. Cardiac examination revealed normal heart sounds and a 2/6 systolic ejection murmur heard best at the base. Abdominal and chest examination was normal. There were no signs of meningismus and the neurological examination was non-focal. His laboratory studies showed hemoglobin of 6.4 g/dl, WBC 6400, platelets 10,000, Na 138 mmol/l, K 3.7 mmol/l, Cl 111 mmol/l, creatinine 1.2 mg/dl, glucose 104 mg/dl, bilirubin 1.7 mg/dl, PT 11.9 s, PTT 26.9 s and LDH 645. The blood smear showed marked RBC fragments and thrombocytopenia. The patient received emergent plasma exchange. He also received 4 units of packed RBC and was commenced on 80 mg of methyl-prednisone intravenously daily. He received a total of 6 treatments and his platelet count one month later was stable at 169,000.

DISCUSSION

TTP is a rare and potentially fatal disorder that usually presents as a triad of thrombocytopenia, microangiopathic hemolytic anemia and fluctuating neurological deficits but may also include fever and renal abnormalities. Ticlodipine administration has recently been implicated in the pathogenesis of some cases. Prompt diagnosis and discontinuation of the offending drug are vital. Most patients respond to a regimen of plasma exchange with or without glucocorticoids. Platelet transfusion is contraindicated.

TITLE: MEGESTROL ACETATE (MEGACE) ASSOCIATED CLINICAL ADRENAL INSUFFICIENCY IN A PATIENT ON DIALYSIS. A. Gupta, L. Merkle, J. Guzzo, J. Dupree, Department of Medicine, Lehigh Valley Hospital, Allentown, PA.

LEARNING OBJECTIVE: 1) To recognize the association of Megestrol Acetate to clinical adrenal insufficiency. 2) Awareness of this association is important because of the expanding use of this medication and the potential serious nature of this side effect.

CASE: Patient is a 76 year-old white female with history of end stage renal disease on peritoneal dialysis. She presented to her physician's office with the chief complaint of cloudy peritoneal dialysis exchanges, profound weakness, dizziness and fatigue. Vital signs were basically normal except for hypotension. Serum cortisol and ACTH levels were obtained and found to be markedly abnormal. Her cortisol level was <1.0ug/dl and ACTH was 0.7pg/ml. The patient had been taking Megace 40mg daily for the last several months as an appetite stimulant. An ACTH stimulation test was also abnormal with a basal cortisol of 0.19ug/dl which only stimualted to 5.66ug/dl after 250 ug of cosyntropin. Subsequently patient was started on hydrocortisone replacement therapy. She was discharged on 20mg of hydrocortisone each day with improved sense of well being.

DISCUSSION: The earlier reports suggest that clinically significant adrenal insufficiency may occur after discontinuation of therapy with the megace, but recent evidence indicates that this complication may also occur during therapy. In high doses progestational agents especially megace can bind for the intranuclear cortisol receptor and either cause some degree of Cushing's syndrome or suppress the CRH-ACTH- Cortisol axis. Awareness of this association can be very important since the patients with Chronic Renal Failure and cancer can frequently have similar complaints as those of adrenal insufficiency. An increase awareness of this potential fatal side effect is needed along with appropriate screening.

CATAPLEXY MISTAKEN FOR PRESYNCOPE: THE IMPORTANCE OF A CAREFUL HISTORY. D. Gutknecht, Section of General Internal Medicine, Penn State Geisinger Medical Center, Danville, PA.

LEARNING OBJECTIVES. 1) Recognize the clinical features of narcolepsy and cataplexy, 2) Distinguish cataplexy from presyncope, and 3) Appreciate the importance of listening to the patient.

CASE. A 72-year-old woman with hypertension complained of episodes of weakness in the legs, which would occur with emotional upset. Symptoms had occurred for years but had increased in frequency in the prior year. Spells were brief, and light-headedness and loss of consciousness were denied. Presyncope was suspected. An EKG showed sinus bradycardia at 56 beats per minute, and an echocardiogram showed aortic sclerosis and mitral annular calcification. Three months later the patient slumped at a clinic reception desk and was reevaluated. A similar history was recorded, with the patient reporting "I just can't control my legs...usually after something stressful." Carotid duplex examination was normal, and a 24-hour Holter monitor showed only rare APCs. The patient then consulted another physician who obtained the same history as well as a history of daytime somnolence. Cataplexy was suspected. A nocturnal polysomnogram showed no apnea, and a multiple sleep latency test showed short sleep latencies with early REM sleep during each of five naps. Fluoxetine was prescribed and three months later the patient reported dramatic relief from her spells.

DISCUSSION. Symptoms associated with narcolepsy include sleep paralysis, hypnogogic hallucinations, and cataplexy. The latter involves sudden loss of motor tone without impairment of consciousness, is often triggered by emotional stress, and as in this case, may be mistaken for presyncope. Early-onset REM sleep during multiple sleep latency testing is characteristic, but the history is the real key to the recognition and successful treatment of this unusual disorder.

RECURRENT SOFT TISSUE INFECTION OF THE WRIST DURING TREATMENT FOR MYASTHENIA GRAVIS. E Hagen and M Sheffield, Department of Medicine, University of Texas Southwestern, Dallas, TX.
LEARNING OBJECTIVES. 1) Diagnose the cause of recurrent or chronic cutaneous and joint space lesions, and 2) Manage Nocardia soft tissue infections.
CASE. An 84 year-old man with myasthenia gravis presented with swelling and decreased mobility of his L wrist. He had been well until 10 months ago when he developed generalized weakness, dysarthria and dysphagia. He was diagnosed with myasthenia gravis and was admitted for plasmapheresis. He was discharged on 80mg a day of prednisone that was slowly reduced to 60mg a day without progression of his myasthenia. His course remained uneventful until 5 months ago when he developed an ulcerative lesion over the dorsum of the L wrist after "bumping" his wrist on a doorjamb. A surgeon debrided the wound and placed him on a two-week course of trimethoprim-sulfamethoxazole (TMP/SMX). His wound healed completely over the next month with no residual deficits in the function of his wrist. Over the next 3 months, the patient experienced transient flares of his myasthenia and remained on prednisone at doses from 60 to 80mg a day. One month prior to his presentation, the patient began having swelling and slight pain in his L wrist. His symptoms progressed and a fluctuant mass appeared on the dorsum of his wrist leading him to present to his physician. He had no constitutional symptoms and, except for his myasthenia, he was otherwise well. Physical exam revealed a 3x3cm fluctuant mass with surrounding erythema and warmth over the radial head on the back of the wrist. Flexion and extension of the wrist were diminished and were slightly painful. Radiographs of the wrist showed changes of radial-carpal arthritis but no destructive lesions. Aspiration of the wrist was done but cultures were sterile. One week later, the patient was taken to the operating room for debridement of his wrist. He had marked synovial proliferation suggestive of rheumatoid synovitis. Histology confirmed chronic inflammatory changes without granuloma. Stains, including Gram's, acid-fast and Congo red, were all negative. Cultures of the material taken at surgery grew *Nocardia asteroides* that was sensitive to TMP/SMX.
DISCUSSION. Nocardiosis is caused by aerobic members of the actinomycetes, with *N. aesteroides* and *N. brasiliensis* as the most frequent causative organisms of disseminated and primary cutaneous disease, respectively. The majority of disease develops in immunocompromised patients, especially those with impaired cell-mediated immunity. Nocardial soft tissue infections are difficult to diagnose as skin biopsies and wound cultures frequently fail to yield *Nocardia spp.* as the causative organism. Effective management of soft tissue infections usually requires surgical debridement and prolonged antimicrobial therapy with sulfonamides. Despite adequate therapy, soft tissue infections may recur.

DELAYED PRESENTATION OF AN AORTIC DISSECTION. D Haskell, Department of Internal Medicine, Medical University of South Carolina.
LEARNING OBJECTIVES: Recognize the importance of distinguishing between clinical and radiographic presentations of aortic dissection.
CASE: A 65 year old African American female presented to the Emergency Department with a complaint of chest and back pain. Her previous medical history was significant for atrial fibrillation, hypertension, and deep venous thrombosis. Three days prior to presentation the patient awakened with sudden onset of sharp interscapular back pain. The pain has persisted, and is now associated with abdominal burning and bloating. She reports feeling weak since the pain began. Prior to presentation the patient was taking benazepril, and warfarin. Upon presentation to the Emergency Department she was found to have a blood pressure of 210/115, and a pulse of 118. Physical examination revealed a well developed, overweight female in no acute distress. Neck exam revealed no bruits or jugular venous distension. Auscultation revealed rales in both lower lung fields. Cardiac exam revealed an irregularly irregular pulse, with normal S1 and S2, and no murmurs, rubs, or gallops. Her PT was found to be 13.4 seconds, with a PTT of 23.2 seconds, and an INR of 1.25. A portable chest radiograph revealed a markedly enlarged cardiac silhouette, a sclerotic unfolded aorta, and no evidence of pneumonia or pulmonary edema. The patient received clonidine, nitroglycerin ointment, and diltiazem while in the Emergency Room. Reassessment raised concern for a widened mediastinum on the chest radiograph, and revealed a pressure of 175/106 in the right arm, and 127/95 in the left. At this point a CT of the chest with contrast was obtained which demonstrated a fluid collection around the aortic arch and descending aorta. The fluid density was compatible with blood, but no dissection was evident. This was followed by an aortogram which demonstrated an ectatic aorta without aneurysm or dissection. Seven days later the fluid collection was reassessed. Repeat CT of the chest revealed a Stanford classification type B aortic dissection.
DISCUSSION: Aortic dissection often presents with the acute onset of severe tearing interscapular pain. Other symptoms may include diaphoresis, syncope, dyspnea, and weakness. Physical findings may include hypotension, hypertension, loss of pulses, aortic regurgitation, and pulmonary edema. Vessels originating from the aorta, or neighboring structures may be compressed. Chest radiograph can reveal widened mediastinum, or a left sided pleural effusion. Diagnosis can be confirmed by aortography, or by noninvasive means such as two dimensional echo, CT scan, or MRI. Transesophageal echo has also proven highly sensitive and specific. The Stanford classification identifies type A dissection as those involving the ascending aorta, and type B as those involving the descending aorta. Type A dissections are treated surgically, while uncomplicated type B dissections may be managed medically. Medical therapy is directed at reducing shear stress. Unless contraindicated beta adrenergic blockers are recommended. This may be accompanied by nitroprusside. Labetolol and trimethaphan may also be used.

A NEW TWIST ON DRUG ABUSE. J Hariharan, C Murphy, K Dobmeyer, AB Nattinger. Division of GIM, Medical College of Wisconsin, Milwaukee, WI
LEARNING OBJECTIVES. 1) Recognize an unusual presentation of substance abuse with underlying psychiatric pathology, and 2) Manage the problem with a contractual approach.
CASE. We assumed the care of a 28-year-old female with a clinical diagnosis of asthma, new onset hypertension and previous history of polysubstance abuse, drug free for the previous 5 years. The patient had presented on multiple occasions to multiple providers with complaints of shortness of breath, cough, and fever. Physicians usually recorded pulmonary wheezing, with no focal findings and typically prescribed a steroid course and antibiotics. The patient had recently developed hypertension and diabetes. Review of the records revealed consistently normal workup (methacholine challenge, chest X-ray and CT of the sinuses), and normal peak flows during acute exacerbations. Many of her medication courses were prescribed in the ER, at urgent clinic visits, or by telephone after hours. In the previous calendar quarter (90 days), she had been given 6 different prescriptions for courses of prednisone, which represented 148 days of steroid therapy, and totaled 3220 mg of prednisone. Her total pharmacy costs that quarter (including 7 courses of antibiotics, antihypertensives, and hypoglycemics) were $2700.

Recognition of her extent of health care utilization and the lack of supporting evidence of asthma led to the implementation of a contractual approach. The key components of the written contract were: 1) limitation of care to one attending and one resident physician; 2) regularly scheduled non-symptom-dependent visits; 3) medications were prescribed only by the two physicians and filled at a single pharmacy; 4) she was required to obtain psychiatric care. Implementation of the contract was facilitated by the fact that her health care payor required her to use our facility and pharmacy. Following implementation of the contract, her use of the ER and urgent care clinics fell dramatically. In the following quarter, her use of prednisone fell by 50%, and within 10 months, she was weaned off steroids entirely. Her pharmacy costs fell by 60% to $1088. Her ER visits declined from 7-8 per quarter to 1 per quarter. Her phone calls to the clinic doubled for about 4 months, then fell to two per quarter. Her pulmonary status remained normal off of steroid therapy. A psychiatrist diagnosed dysthymia.
DISCUSSION. 1) The case emphasizes the early recognition of underlying psycho-social issues presenting with physical complaints and steroid abuse in an otherwise young healthy woman, 2) It is possible that her mood was elevated by the steroids, 3) The general principles of contract use can be applied to any overuse of system or medication, 4) The development of mutual trust is essential to successful management, and 5) Patients treated in large academic centers can easily experience fragmentation of care, and we should attempt to limit this.

POST-TRAUMATIC COMPLICATIONS FOR A GOOD SAMARITAN. Karen E. Hauer, University of California at San Francisco, San Francisco, CA.
LEARNING OBJECTIVES: 1) Evaluate the etiologies of hyponatremia. 2) Recognize the protean manifestations of acute adrenal insufficiency.
CASE: Medical consultation was requested to evaluate a 40 year old man with hyponatremia. The patient was admitted with right lower extremity trauma sustained while helping an elderly woman drive her car out of a ditch. She inadvertently reversed her car while the man was pushing, crushing his leg. After several unsuccessful debridements, the patient underwent above the knee amputation. Postoperatively, his course was notable for multiple non-specific complaints attributed to reactive depression, including mood changes, poor appetite, vague abdominal pains, fatigue, and myalgias. Medical consultation was called to evaluate persistent hyponatremia despite normal saline hydration, a possible contributor to his symptoms. Physical exam was notable for low-normal blood pressure without other evidence of dehydration, and the lower extremity amputation. Recent laboratories revealed a sodium of 128-130 mg/dl and potassium of 4 mg/dl. Etiologies of euvolemic hyponatremia were pursued, and the patient was found to have a serum cortisol level of 1 ug/dl, which did not significantly increase with adrenocorticotropic hormone (ACTH) stimulation. Thyroid studies were normal. Abdominal CT revealed bilateral adrenal hemorrhagic infarction. The patient was promptly treated with replacement doses of steroids, with rapid improvement in his mood, appetite and energy level, and increase in his blood pressure, allowing him to participate more productively in rehabilitation.
DISCUSSION: Evaluation of this patient's abnormal sodium based on his volume status led to the diagnosis of adrenal insufficiency. Acute adrenal insufficiency is a medical emergency easily confused with other disorders. This diagnosis should be suspected with a constellation of findings including abnormal electrolytes, hypotension, or abdominal pain in the appropriate clinical setting. Trauma and surgery are two well-recognized precipitants of hemorrhagic adrenal infarction. Prompt administration of glucocorticoids is necessary to prevent progression to shock.

HYPOGLYCEMIA MIMICKING SEIZURE DISORDER AND HEMIPLEGIA.
Karen E. Hauer, Division of General Internal Medicine, University of California at San Francisco, San Francisco, CA.
LEARNING OBJECTIVES: 1) Distinguish hypoglycemia-induced neurologic symptoms from other neurologic disorders. 2) Obtain a thorough history from a patient with a seizure disorder to exclude underlying treatable etiologies.
CASE: A 39 year old woman with a history of hyperparathyroidism and seizure disorder presented with the sudden onset of right sided hemiplegia. She was well until six months prior to admission, when she was evaluated for fatigue and seizures and noted to have hypercalcemia due to a parathyroid adenoma. The patient underwent removal of 3 of 4 parathyroids. Subsequently, the seizure frequency increased to daily and was refractory to multiple medications. The seizures were described as 5 to 60 minutes in duration, sometimes associated with movements of all 4 extremities, and complicated by prolonged postictal mental status changes. MRI of the head was normal. On the day of admission, the patient awoke with transient right sided weakness which recurred following an afternoon nap, with complete right-sided hemiplegia and aphasia. The paramedics were called, and noted a blood sugar of 40 mg/dl. After intravenous administration of 50 ml of 50% dextrose, the symptoms completely resolved. Further history revealed that the seizures typically occurred in the early morning and after naps when the patient had been fasting, and were associated with cravings for juice and food. The patient was admitted and maintained on a continuous intravenous dextrose solution. The diagnosis of multiple endocrine neoplasia type 1 (MEN1) was suspected, based on the pituitary adenoma and probable pancreatic insulinoma. During a supervised fast, symptomatic hypoglycemia developed within 2 hours, with an inappropriately elevated insulin level. Surreptitious ingestion of insulin or oral hypoglycemic agents, hypothyroidism, hypocortisolism, and family history of endocrine disorders were excluded. The patient underwent successful resection of a pancreatic insulinoma with complete resolution of her symptoms.
DISCUSSION: Clues from the history regarding new seizures and other transient neurological symptoms can suggest hypoglycemia, a rare but treatable cause of seizures in non-diabetic patients. In particular, with syndromes characterized by prominent mental status changes occurring after fasting, hypoglycemia must be ruled out. Hypoglycemic hemiplegia has been reported in patients with diabetes, insulinoma, and other etiologies of hypoglycemia, and similarly does not indicate an underlying neurological abnormality. The deficits are reversible with prompt therapy. The concurrent presentation of multiple endocrine disorders should prompt physicians to entertain the rare diagnosis of multiple endocrine neoplasia.

HYPOGLYCEMIA MIMICKING SEIZURE DISORDER AND HEMIPLEGIA.
Karen E. Hauer, Division of General Internal Medicine, University of California at San Francisco, San Francisco, CA.
LEARNING OBJECTIVES: 1) Distinguish hypoglycemia-induced neurologic symptoms from other neurologic disorders. 2) Obtain a thorough history from a patient with a seizure disorder to exclude underlying treatable etiologies.
CASE: A 39 year old woman with a history of hyperparathyroidism and seizure disorder presented with the sudden onset of right sided hemiplegia. She was well until six months prior to admission, when she was evaluated for fatigue and seizures and noted to have hypercalcemia due to a parathyroid adenoma. The patient underwent removal of 3 of 4 parathyroids. Subsequently, the seizure frequency increased to daily and was refractory to multiple medications. The seizures were described as 5 to 60 minutes in duration, sometimes associated with movements of all 4 extremities, and complicated by prolonged postictal mental status changes. MRI of the head was normal. On the day of admission, the patient awoke with transient right sided weakness which recurred following an afternoon nap, with complete right-sided hemiplegia and aphasia. The paramedics were called, and noted a blood sugar of 40 mg/dl. After intravenous administration of 50 ml of 50% dextrose, the symptoms completely resolved. Further history revealed that the seizures typically occurred in the early morning and after naps when the patient had been fasting, and were associated with cravings for juice and food. The patient was admitted and maintained on a continuous intravenous dextrose solution. The diagnosis of multiple endocrine neoplasia type 1 (MEN1) was suspected, based on the pituitary adenoma and probable pancreatic insulinoma. During a supervised fast, symptomatic hypoglycemia developed within 2 hours, with an inappropriately elevated insulin level. Surreptitious ingestion of insulin or oral hypoglycemic agents, hypothyroidism, hypocortisolism, and family history of endocrine disorders were excluded. The patient underwent successful resection of a pancreatic insulinoma with complete resolution of her symptoms.
DISCUSSION: Clues from the history regarding new seizures and other transient neurological symptoms can suggest hypoglycemia, a rare but treatable cause of seizures in non-diabetic patients. In particular, with syndromes characterized by prominent mental status changes occurring after fasting, hypoglycemia must be ruled out. Hypoglycemic hemiplegia has been reported in patients with diabetes, insulinoma, and other etiologies of hypoglycemia, and similarly does not indicate an underlying neurological abnormality. The deficits are reversible with prompt therapy. The concurrent presentation of multiple endocrine disorders should prompt physicians to entertain the rare diagnosis of multiple endocrine neoplasia.

CEREBRAL VEIN THROMBOSIS ASSOCIATED WITH PROTEIN S DEFICIENCY AND FACTOR V LEIDEN MUTATION. Rob Hegeman and James Sebastian, Medical College of Wisconsin, Milwaukee, WI.
LEARNING OBJECTIVES. 1) Diagnose cerebral vein thrombosis (CVT) in the appropriate clinical setting, and 2) recognize the importance of identifying underlying thrombophilic disorders in patients with CVT.
CASE. A 23 year old man with a past history of deep venous thrombosis and protein S deficiency presented with a 3 week history of a sharp, throbbing occipital headache. The patient characterized the headache as different from his typical migraine and dated its onset to an episode of minor head trauma that he sustained 3 weeks ago. On the day of admission, he developed symptoms of left-sided weakness and numbness, light-headedness, and blurred vision. Physical examination showed the patient to be alert and oriented with normal vital signs. His speech pattern was slow but clear. Pupils were 4 millimeters each and briskly reactive. Funduscopic exam showed sharp disc margins. Muscle stretch reflexes were symmetrical and hypoactive. Pertinent positives on the neurologic exam included grade 3/5 left-sided weakness, multiple sensory deficits on the entire left hemibody, and a positive Babinski sign on the left. A head CT scan obtained shortly after admission showed no evidence of ischemia, hemorrhage, or subdural hematoma; a subsequent magnetic resonance (MR) image of the brain showed no specific abnormalities. Because of a strong clinical suspicion of CVT, MR angiography and venography were performed; these studies revealed thrombosis in the transverse and sigmoid portions of the right lateral sinus. Completion of the patient's hypercoagulable work-up detected a heterozygous mutation of Factor V Leiden.
DISCUSSION. CVT appears to be much more common than previous studies have indicated. Despite its broad spectrum of clinical presentations and its variable mode of onset, CVT should be strongly suspected in patients with underlying hypercoagulable states.

VIBRIO PARAHEMOLYTICUS BACTEREMIA IN A PATIENT WITH CIRRHOSIS WHO INGESTED RAW SHELLFISH David Hirsch, Paul Knoepflmacher and Mark Horowitz Department of Medicine, Mount Sinai Medical Center, New York, NY.
LEARNING OBJECTIVES 1) Recognize *Vibrio Parahemolyticus* as a cause of systemic illness in patients with cirrhosis. 2) Warn patients with cirrhosis to avoid ingesting raw shellfish. 3) Realize that the skin manifestations of essential mixed cryoglobulinemia may be amplified during acute infection.
CASE A 51 year old man with Hepatitis C cirrhosis presented with right leg pain, swelling and redness. Four days prior to admission he began vomiting and having diarrhea. Three days prior to admission his right leg became red and swollen. The next day he was unable to bear weight on this leg. He had fever with shaking chills and episodes of confusion. Physical exam revealed a lethargic, mildly ill appearing man. Temp.-38.5 C, BP-125/70, HR-95, RR-16. The sclera and skin were anicteric. The neck was supple. Lungs were clear. Spider angiomata and gynecomastia were present. Cardiac exam was unremarkable. The abdomen was non-tender with a small liver, an enlarged spleen and no detectable ascites. Stool was brown and negative for occult blood. The right lower extremity had erythema below the knee with scattered superimposed one cm. red papules on the anterior surface. WBC count was normal, Hgb-13.9, Platelets-28,000, PT-19.8, and total bilirubin-5.8. His urine, CXR, EKG and x-ray of the right lower extremity were unremarkable. The patient was started on IV cefazolin for a presumed cellulitis of the leg. By day two he was afebrile. By day three both the erythema and papules had regressed significantly. Gram negative rods were identified in aerobic and anaerobic blood culture bottles collected on admission. Assay for cryoglobulins was positive and biopsy of a skin lesion revealed leukocytoclastic vasculitis. On day four the organism was identified as *Vibrio Parahemolyticus*. The patient became more alert and recalled eating raw shellfish 48 hours prior to the onset of his symptoms.
DISCUSSION Patients with cirrhosis are susceptible to life-threatening illness from non-cholera vibrio organisms. *Vibrio Vulnificus* is a cause of septicemia, often with bullous skin lesions of the lower extremities and a high mortality rate. *Vibrio Parahemolyticus* usually causes a self-limited gastroenteritis. However, septicemia with fatal outcome has been reported in patients with end-stage liver disease. Our patient had cirrhosis and cryoglobulinemia and developed a febrile illness with GI symptoms, and vasculitic skin lesions in the setting of *Vibrio Parahemolyticus* bacteremia. He improved promptly with systemic antibiotics. While cellulitis may have been present, another possibility is that a systemic infection triggered the skin manifestations of cryoglobulinemia. This case illustrates the importance of considering *Vibrio Parahemolyticus* as a cause of serious illness in patients with cirrhosis, and admonishing such patients to avoid raw seafood ingestion.

A MIGRAINE HEADACHE AND ITS AFTERMATH

RAYMOND D. HOBBS, NANCY PAGE, TERRI BALA University of Michigan Hospitals, Ann Arbor, Michigan

LEARNING OBJECTIVES: To recognize the subtle ploys that may be used in drug seeking behavior.

CASE: A 30 year old female from Illinois, presented to an outpatient clinic in Michigan late on a Friday afternoon. She was erudite, well dressed, and believable as she complained of classic migraines with auras and unilateral pain as well as tension headaches caused by an impending divorce. She was an unemployed nurse who had not yet obtained her license in Michigan. She requested a specific brand of Hydrocodone. The exam was normal except for anxiety and stress.

A small amount of Hydrocodone was given, medical records were requested and she was referred to a counselor and a lawyer. She presented several times in the next month. She never saw the counselor or the attorney and did not apply for her nursing license. Considering these inconsistencies further evaluation was undertaken. She was found to have two different registration numbers differing only in birthdate. She was concurrently seeing three physicians within the same health care system, had given the same story to each and received Hydrocodone from pharmacies as far away as northern Ohio. Pharmacies in Illinois confirmed a history of drug seeking behavior. She was discharged from the practice.

DISCUSSION: This vignette illustrates a few of the common clues and ploys in subtle drug seeking behavior. These include the time of her presentation (late in the day when it is more difficult to check with pharmacies), the distance of the pharmacies from her home, the specific request for only one medication, noncompliance with other prescribed treatments and the 'textbook' presentation of her problem(especially in someone with medical training.). In broader terms it is a sobering reminder that even experienced physicians may be fooled by the drug seeker with a great story and a classic presentation.

ACUTE CHEST SYNDROME IN A HISPANIC ADULT

Lisa Humphreys, Rich Agajanian, Department of Medicine, UCLA Olive View Medical Center, Sylmar, CA.

LEARNING OBJECTIVES: 1) Recognize acute chest syndrome in sickle cell disease and, 2) increase awareness of the prevalence of sickle cell disease in Hispanic populations.

CASE: A 22 year old male from Acapulco, Mexico presented to the hospital with complaints of headache, neck pain, shortness of breath, and fever for one day. He also had photophobia, and pain in his shoulders, chest, and spine. Three years prior to this presentation, he was hospitalized with similar symptoms. At that time, he was kept overnight and discharged on daily folate tablets but was not given a diagnosis. He took no other medications and had no allergies. Social history was only notable for being in the U.S. for four months. Family history was significant for 2 siblings with occasional episodes of "bone pain" for which they had never been hospitalized. He denied any multi-ethnic heritage. Physical exam revealed BP 115/66, P 97, RR 22-30, T 38.6 C, pulse ox 85% on room air. He was well developed, well nourished, in mild respiratory distress. Heart: tachycardic without murmurs or gallops. Lungs: clear to auscultation. Abdomen: nondistended, nontender with normoactive bowel sounds, no hepatosplenomegaly. Kernig's sign was positive, Brudzinski's sign was equivocal. Lumbar puncture performed in the emergency room for presumptive meningitis revealed no cells in the CSF, glucose 86, protein 46. Later, CBC showed WBC 22.4, Hct 23.9, and RDW 25.9. Total bili was 2.9, direct bili was 0.1. Peripheral smear revealed sickle cells, target cells, and fragmented RBC's. Chest x-ray had diffuse interstitial changes with bilateral infiltrates. A subsequent hemoglobin electrophoresis showed Hb SS. His G6PD activity was normal.

DISCUSSION: Acute chest syndrome in sickle cell disease is characterized by fever, chest pain, and pulmonary infiltrates. It typically occurs in patients who are SS homozygotes. Early diagnosis is important as broad spectrum antibiotics should be initiated. Hemoglobinopathies such as sickle cell disease in African Americans, and thalassemias in people of Mediterranean and Southeast Asian descent are well recognized. However, hemoglobinopathies also occur in Hispanic populations and should not be overlooked. Recent California data estimate the overall prevalence of sickle cell disease in Hispanics to be 1 in 36,000 births and in African Americans to be 1 in 396 births [Gen Epi, 1996, 13(5):501-12]. This case illustrates the need to recognize presenting signs of acute chest syndrome and to consider hemoglobinopathies in various ethnic populations.

AN UNUSUAL ETIOLOGY OF GRANULOMATOUS ARTHRITIS: A CASE REPORT,

JASTI H, GONDI RK, LUNDSTROM T, DETROIT MEDICAL CENTER, WAYNE STATE UNIVERSITY, MI

LEARNING OBJECTIVE: Nocardia asteroides is a soil-born aerobic actinomycete, which infects humans most commonly through respiratory tract. It produces suppurative necrosis and abscess formation that is typical of pyogenic infection. As widely reported, nocardia is predominantly a pulmonary pathogen with occasional extrapulmonary infection including skin, soft tissue, mediastinum and central nervous system (CNS). But we are unaware of any prior report citing nocardia species as causative organism for granulomatous arthritis of a major joint.

CASE REPORT: A 35 year old male came with a three month history of recurrent episodes of swollen and painful right knee but without penile discharge, eye symptoms or diarrhea. He also complained of cough with yellow sputum, night sweats, fever, and weight loss for three weeks. Physical examination showed bilateral upper cervical and axillary lymph nodes. Extremity examination revealed swollen and tender right knee joint with decreased range of movements. Evidence of synovial thickening with joint effusion and without deformity was found. Patient was placed in isolation for respiratory symptoms and two days later laboratory tests revealed sputum acid fast bacilli 4+. Arthocentesis revealed protein of 6.1gms, RBC 480,000/cmm, WBC of 11,000/cmm with 89% lymphocytes. Synovial biopsy revealed evidence of chronic synovitis with multiple non-caseating granulomas, which are negative for acid fast and fungal stains. After 6 weeks, the synovial biopsy tissue grew Nocardia asteroides. Patient was initially started on cefotaxime and erythromycin for suspected community acquired pneumonia and the joint symptoms improved. Patient was also started on four drug regimen for pulmonary tuberculosis.

DISCUSSION: This case illustrates that Nocardia asteroides is capable of causing granulomatous arthritis and also demonstrates that Nocardia and Tuberculosis can coexist in the same patient. AIDS epidemic makes it more prudent for the clinicians to be aware of this infection. Even though nocardiosis is described predominantly in pulmonary, skin, and CNS infections, we believe that nocardia should be in the differential diagnosis of granulomatous arthritis, especially in immuno-compromised population.

INFLUENCE OF NELFINAVIR ON WARFARIN ANTICOAGULATION.

Ajay Israni, Sara Clay, Jennifer Chase, Department of Medicine, Boston University School of Medicine, Boston, MA.

LEARNING OBJECTIVES. Recognize the clinically relevant pharmacological effects of Protease Inhibitors on Warfarin.

CASE. A 37 year old HIV-positive man with history of recurrent deep venous thrombosis (DVT) of lower extremities started on antiretrovirals three months prior to admission. He was on Zidovudine, Lamivudine, Nelfinavir, Fluoxetine and Warfarin. As an outpatient his Warfarin dose was increased as high as 5mg daily except for 7.5 mg on Tuesday and Thursday with an INR no higher than 1.05. Patient regularly smoked 1pack per day and drank 3 drinks of alcohol four times a week. Patient did not take any vitamin K containing drugs, or any new medications. His liver function tests were normal as an outpatient and as an inpatient. Pt was admitted to the hospital with recurrent DVT with an INR of 1.05. While in the hospital, he received Warfarin daily under direct supervision along with intravenous heparin. Over the next 17 days his Warfarin dose was increased to 25 mg everyday with the highest daily INR of 1.42. On hospital day 18 his Nelfinavir was discontinued and on hospital day 20 the INR increased to 2.59 and the Warfarin was decreased to 5mg daily. The patient was discharged on hospital day 21 with an INR of 2.86.

DISCUSSION. Nelfinavir was expected to cause an increase the anticoagulant activity of Warfarin. Nelfinavir is primarily metabolized by the CYP3A4 isoenzyme of the cytochrome P450 system and it inhibits this isoenzyme. Warfarin is metabolized by several isoezymes including the CYP3A4 isoenzyme. Nelfinavir is also a known inducer of hepatic enyzmes. Our case report indicates that nelfinavir may decrease the anticoagulation effect of Warfarin with a mechanism of metabolic induction, even though other mechanisms cannot be excluded. There has been a case report of another protease inhibitor Ritonavir, a more potent inhibitor and inducer of the cytochrome P450 system, decreasing the anticoagulation effect of Warfarin. However with Nelfinavir this is the first such case. Since the pharmacological effect of Nelfinavir on Warfarin is unpredictable the INR needs to be monitored closely in patients on these medications.

LYMPHOCYTIC COLITIS AS A CAUSE OF CHRONIC DIARRHEA. J Jaeger, T Nickolas, Department of Medicine, University of Pennsylvania, Philadelphia, PA.
LEARNING OBJECTIVES: Recognize lymphocytic colitis as a cause of chronic diarrhea and distinguish this "atypical colitis" from other causes of chronic diarrhea.
CASE: A 60 year-old female presents with a chief complaint of weight loss and diarrhea. Her symptoms began following a left hemicolectomy and sigmoidectomy with colostomy performed 8 months prior for colon cancer. The diarrhea is copious, watery, and non-bloody. She has lost 20 pounds since the onset of symptoms. She denies anorexia, nausea, vomiting, fever, chills, night sweats, or melena. She reports that any p.o. intake causes worsening of the diarrhea. Past medical history is notable for colon cancer, Dukes C, stage unknown, for which she received no adjuvant therapy. She also has hypertension, non-specific arthritis, and a distant history of uterine cancer treated with radiation and surgery. She smokes one pack of cigarettes per day for the last 30 years. Medications: atenolol/chlorthalidone, KCl, FeSO4, and diphenoxylate/atropine. On physical exam she is severely cachectic. Blood pressure is 170/80 standing, pulse 80, temperature 98.6, weight 83 pounds, height 5'6". Her abdomen is scaphoid, with increased bowel sounds, no hepatomegaly and no masses. Remainder of exam is unremarkable. Laboratory studies show Hb 8.9, Hct 27. Magnesium 0.3 mg/dl, Calcium 7.1 mg/dl, Albumin 3.9 gm/dl. She is admitted to the hospital for further evaluation. Electrolytes are aggressively repleted and she receives IV rehydration. Stool cultures are negative. While on a normal diet, 48-hour fecal fat measurement is normal. While NPO, her diarrhea ceases. ANA is 1:80, ESR 34 mm/hr. An abdominal CT is unrevealing. CEA is within normal limits. An upper GI is notable for mildly dilated small bowel with effacement of the folds suggestive of celiac disease. Antibody studies for celiac disease are indeterminate. However, an upper endoscopy reveals normal appearing bowel mucosa, and biopsies are normal. Colonoscopy reveals normal appearing mucosa. Biopsies from the right colon reveal lymphocytic colitis. Treatment is instituted with mesalamine 800 mg three times a day. After four weeks, she has noted minimal improvement in symptoms and her weight has stabilized.
DISCUSSION: Lymphocytic colitis is a clinicopathologic entity characterized by profuse watery diarrhea, normal appearing mucosa on endoscopy, and normal imaging studies. Random colon biopsies show increased lymphocytes in surface and crypt epithelium, without crypt distortion. It is clinically similar but histologically distinct from collagenous colitis, another form of "atypical" inflammatory bowel disease. No specific etiology has been discovered for this entity; however, many patients have symptoms and studies suggestive of autoimmune disease, including polyarticular arthritis, thyroid disease, and abnormal ESR and ANA. As in this case, response to anti-inflammatory therapy is unpredictable. The onset of this condition following surgical treatment for cancer has not been reported and may warrant further investigation.

FORTY-SEVEN YEAR OLD WITH "A PAIN IN THE NECK." S Jocums, Department of Medicine, Vanderbilt University Medical Center, Nashville, TN.
LEARNING OBJECTIVES. 1) Recognize the value of a thorough physical examination of a new patient. 2) Review clinical features, natural history and epidemiology of syphilis in the United States.
CASE A previously healthy 47 year old garage attendant presented for the first time to his primary care provider one week after a motor vehicle accident involving a city bus in which two passengers were killed. He complained of pain and a lump in the left side of his neck. He denied a specific injury to the neck or shoulder, previous neck or shoulder problems, radicular symptoms, arthritis, stiff neck, headache, alterations of consciousness, fever, chills, rash, and ill contacts. A prior evaluation in the emergency room, including neck films, was unrevealing. Review of systems was significant for an episode of bloody ejaculate two weeks ago, followed by painless gross hematuria which resolved after several days. There was litigation pending. On examination, he was a thin black male in no distress. The neck was supple and minimally painful on flexion. He had palpable, nontender lymphadenopathy bilaterally in the anterior cervical, epitrochlear and inguinal regions with the largest node being 1.5 cm in diameter in the left neck. There were no abnormal gastrointestinal, cardiovascular, genitourinary, or skin findings. Laboratory evaluation revealed a serum albumin of 1.5 and 6,300 WBCs. Twenty four hour urine collection had 3.8 grams of protein with no monoclonal spike. EBV, CMV, and HIV tests were negative. RPR was 1:256. CSF examination, performed due to the discomfort on neck flexion, revealed 20 white cells with 54% lymphocytes, 18% polymorphonuclears, protein 43 mg/dl and glucose 68 mg/dl. CSF VDRL was negative. On further questioning, the patient recalled an unprotected sexual encounter three months prior and 15 months prior.
DISCUSSION The incubation period of syphilis is approximately three weeks. Primary syphilis lasts two to eight weeks. Secondary syphilis occurs six to eight weeks later and lasts two to six weeks. Associated nephrotic syndrome, mostly due to membranous nephropathy, is usually transient and resolves spontaneously. Spinal fluid abnormalities are seen in up to 40% of patients with secondary syphilis, and debate exists whether lumbar puncture is necessary in all cases. This case reminds us that syphilis is still the "great imitator," presenting here as neck pain, generalized lymphadenopathy and nephrotic syndrome after motor vehicle accident, and continues to present in our primary care patients. The South has the highest syphilis rates in the nation, with Tennessee third in the nation at 14 cases/100,000. This patient received intravenous penicillin for 10 days. His lymphadenopathy resolved and his lawsuit is still pending.

DOC, GOT TIC, CAN'T TOC, Maneesh Jain,MD, Stewart Babbott, MD, Baystate Medical Center, Springfield, MA.
LEARNING OBJECTIVE: Lyme disease and cranial neuropathy-An uncommon presentation of a common disease.
CASE: A 35 y/o man with history of medullary cystic kidney disease, s/p kidney transplant presented with progressive fatigue, diffuse weakness and right sided headache. He noted occasional lightheadedness, low grade fever and chills. Three weeks prior to admission the patient the noted intermittent dull headaches relieved with acetaminophen. These gradually increased in frequency and intensity, no longer resolved with medication, and became associated with episodes of fever and shaking chills. The patient also noted "fainting spells" (no syncope) and malaise. He subsequently sought evaluation at an ambulatory care center where he was diagnosed with viral illness. When symptoms continued worsen, he saw his primary care physician who referred him to a neurologist: workup included a normal cat-scan of the head and he was again diagnosed to have viral illness. One week prior to admission, patient noted difficulty with motor function of facial muscles exhibited by difficulty with smiling, incomplete closure of eye-lids, food-pocketing, and constant twitching of eyebrows. Medications taken regularly included cyclosporine and prednisone. Possible tick exposure included employment as a field surveyor in western MA and vacationing in Cape Cod during the onset of these complaints.
Admission physical exam was significant for temperature of 100 degrees F, erythema over face,ears, and throat, loss of facial creases including naso-labial folds bilaterally, inability to wrinkle forehead, difficulty with fully closing eyelids, and parting lips resulting in dysarthria. Remainder of cranial nerve exam was normal.
Extensive hematologic studies were unremarkable. Lumbar puncture revealed elevated protein level, lymphocytic pleocytosis, and negative Lyme antibody. Western blot analysis of CSF was negative for Lyme IgG, but positive for Lyme IgM. All other CSF studies were negative.
Patient was started on ceftriaxone and had progressive resolution of symptoms to baseline with discharge by hospital day four.
DISCUSSION: Lyme borreliosis is a spirochetal disease which usually presents with erythema migrans associated with constitutional symptoms. Here, an unusual presentation is reported manifested by bilateral facial nerve palsy with no history of erythema migrans. When encountering this uncommon cranial nerve affliction, borrelial infection needs to remain high on the clinician's differential diagnosis as prompt treatment leads to a successful outcome.

OF INTERLEUKINS, NEPHROTIC SYNDROME, AND LYMPH NODES: A 54-YEAR-OLD MAN WITH CASTLEMAN'S DISEASE. O Jones, H Gurm, A Lichtin, J Nally, Department of Internal Medicine, Cleveland Clinic Foundation, Cleveland, OH.
LEARNING OBJECTIVES. 1) Identify the pathology, course and management of Castleman's Disease, and 2) Recognize the renal manifestations of Castleman's Disease.
CASE. A 54-year-old male presented with chills, fatigue, and anorexia. He was being treated for pneumonia when a suspicious left lower lung nodule was incidentally found on a chest x-ray. Bronchoscopic biopsy and brushings were negative for malignancy or infection. He then developed extensive edema and peripheral lymphadenopathy. Excisional cervical lymph node biopsy revealed follicular and interfollicular hyperplasia. A pulmonary wedge resection was planned, but was deferred due to elevated alkaline phosphatase. An ERCP revealed a malignant-appearing distal common bile duct stricture. Biliary tract brushing cytology was negative. A CT scan was done for further evaluation of lymphadenopathy with an incidental finding of new bilateral renal vein thrombosis. Physical exam was significant for anasarca and numerous freely movable, nontender cervical and inguinal lymph nodes. Lung exam revealed bibasilar crackles. Laboratory studies confirmed nephrotic syndrome (serum albumin of 1.4 and a 24 hour urine protein of 14 grams) and a normochromic, normocytic anemia. Renal function was intact (BUN of 17 and serum creatinine of 1.1) with an initial blood pressure of 150/88. Extensive infectious and rheumatologic studies were negative. Bone marrow was normocellular with a single lymphoid aggregate without evidence for lymphoma. A CT was worrisome for mediastinal adenopathy suggestive of lymphangitic tumor spread. Biopsies of mediastinal and paratracheal lymph nodes revealed Castleman's Disease, plasma cell type. He is currently being treated with warfarin, prednisone, melphalan, and furosemide. His proteinuria is resolving, and he feels well, without further progression of his disease.
DISCUSSION. Castleman's Disease, a rare B cell proliferative disorder can be either localized or multicentric. It has three histologic variants: hyaline-vascular, plasma cell and mixed. The majority of cases represent hyaline-vascular disease, which is usually asymptomatic. Multicentric disease is usually of the plasma cell variety and may present with a variety of symptoms including: generalized lymphadenopathy, fever, weight loss, hemolytic anemia, hypergammaglobulinemia, thrombocytopenia, hepatosplenomegaly, nephrotic syndrome, and altered liver function tests. Renal pathology is most commonly related to amyloidosis. The etiology of Castleman's Disease is unclear, but some of the manifestations relate to an increase in interleukin-6 activity. Treatment consists of surgical excision for localized disease, and prednisone and/or chemotherapy for multicentric disease. There is risk of malignant transformation to Kaposi's sarcoma and malignant lymphoma in multicentric disease. Castleman's Disease should be included in the differential diagnosis of unexplained lymphadenopathy and nephrotic syndrome.

DIFFUSE FUNGAL MYOSITIS: DIAGNOSIS, TREATMENT, AND THE CLINICAL UTILITY OF TAGGED WHITE BOOD CELL SCANS. J Jorgensen, J Morris, Department of Medicine, Madigan Army Medical Center, Fort Lewis, Washington.

LEARNING OBJECTIVES. 1) Diagnose diffuse fungal myositis. 2) Assess treatment success using clinical and radiographic data. 3) Recognize the clinical utility and limitations of tagged white blood cell scans.

CASE. An otherwise healthy 36-year-old man was diagnosed with acute myelogenous leukemia and underwent induction chemotherapy. As anticipated he became neutropenic and subsequently developed fevers. He was initially treated with broad spectrum antibiotics and clinically improved. Vancomycin and later Amphotericin B were added when he became febrile again. Blood cultures revealed a yeast and S. hemolyticus. The patient developed a diffuse, papular, non-blanching rash and complained of muscle pain and weakness in his extremities. On exam he was areflexic and had diffusely decreased strength. A few days later the patient experienced respiratory failure and circulatory collapse requiring transfer to the ICU where he was intubated and treated with vasopressors. He continued to be febrile and demonstrate septic hemodynamic parameters, which prompted an aggressive search for an infectious foci. An abdominal CT was unrevealing. An indium labeled leukocyte study demonstrated diffuse uptake of both upper and lower extremities. This was consistent with either leukemic infiltration of skeletal muscle or a diffuse infectious myositis. Excisional biopsy of the posterior gastrocnemius demonstrated numerous budding yeasts and yeast in chains on special stains. No leukemic infiltrates were identified. Culture of fine needle aspirate of the muscle grew only S. hemolyticus. The patient was treated with vancomycin, amphotericin B, and fluconazole for an infectious myositis. A repeat indium labeled leukocyte scan was performed after 14 days of antimicrobial therapy and showed partial resolution. A third scan showed resolution of the fungal myositis.

DISCUSSION. Diffuse fungal myositis has been reported in the literature a few times. However, none of these patients survived and only one was diagnosed ante-mortum. The diagnosis of diffuse fungal myositis should be considered in neutropenic patients who present with the triad of diffuse myalgias, fever, and rash. Either positive blood cultures or characteristic muscle biopsies may confirm the diagnosis. The dilemma of how to determine if the infection has cleared so that chemotherapy may be resumed has not been addressed in the literature. We propose that a tagged white blood cell scan may be a helpful, less invasive method than repeat muscle biopsy of determining clearance of infectious myositis.

PERILYMPH FISTULA RESULTING FROM BAROTRAUMA: A CASE OF ACUTE SENSORINEURAL HEARING LOSS. R.M. Kaiser, Department of Medicine, Division of General Internal Medicine, University of Pennsylvania, Philadelphia, PA.

LEARNING OBJECTIVE: 1) Differential diagnosis of acute sensorineural hearing loss; 2) Recognition of circumstances requiring timely referral to a specialist.

CASE: A 39 year old white man presented to Medicine Clinic with acute onset of ringing in the right ear. The patient had been swimming laps for 45 minutes and afterward dove two times into nine feet of water to retrieve his goggles. He then noted ringing in his right ear. He denied head trauma, lightheadedness, vertigo, weakness, ataxia, cough, or nasal congestion. He was taking no medications. On physical examination, the patient was afebrile and blood pressure was 110/70 with a pulse of 70 and respirations of 20. Fundi and tympanic membranes were normal. Cardiac and lung exams were unremarkable. Neurologic exam showed intact sensory, motor, and cerebellar function. Weber test was equivocal, and Rinne test was normal bilaterally. Cranial nerves were otherwise normal except for decreased ability to hear a finger rub on the right side. The patient was subsequently referred to Otorhinolaryngology Clinic, and an audiogram was immediately ordered. It revealed severe sensorineural hearing loss in the right ear. The patient was diagnosed with a presumed perilymph fistula and admitted to the hospital. The patient was precribed on IV dexamethasone and oral hydrochlorothiazide/triamterene and placed on strict bedrest. An MRI of the brain was performed and was normal. CBC and sedimentation rate were normal. Serial audiograms showed progressive improvement in the hearing deficit over the next week with correction to normal.

DISCUSSION: Acute sensorineural hearing loss (ASNL) may result from drugs, infection, head trauma, acoustic neuroma, vasular occlusion, Meniere syndrome, or perilymph fistula. Patients suspected of ASNL require immediate referral for audiometry and evaluation by an otorhinolaryngologist. In this case, the patient presented with tinnitus and hearing loss, but not vertigo, which is part of the classical triad seen in patients with perilymph fistula. The history of barotrauma secondary to a rapid dive in the pool, with resulting tinnitus, suggested the diagnosis. The Rinne test can be normal in patients with sensorineural loss and can sometimes mislead clinicians. Prompt diagnosis and treatment of perilymph fistula can, as in this case, result in the recovery of normal hearing.

AN ELDERLY WOMAN WITH TETANUS. Paul Jung and Rachel Rogers. MetroHealth Medical Center, Cleveland, OH.

LEARNING OBJECTIVES. 1) Recognize the clinical presentation and course of tetanus, 2) Review the treatment and epidemiology of the disease.

CASE. A previously healthy healthy 85 year old woman presented to the emergency room after falling at home while gardening. Other than ecchymoses, there were no other physical findings or fractures. Mild trismus was noted as secondary to facial trauma. The patient presented to emergency two days later with a complaint of general stiffness and difficulty getting out of bed. While in emergency, she was noted to have mild trismus and generalized stiffness in all joints. Upon admission to the general medical service, we noted a deep puncture wound to her left lateral malleolus with some pus, severe stiffness, especially in the lower extremity joints, and a tensely flexed platysmus. The patient was immediately transferred to the MICU where she developed opisthotonos with severe trismus. The patient's mental status remained entirely intact. Treatment was initiated with diazepam, metronidazole, propranolol, and tetanus immune globulin. Gradually, the patient's stiffness resolved with opisthotonos absent by day 3. The patient was transferred from the MICU to the rehabilitation service on day 11 and discharged from the hospital on day 24. On follow-up, the patient had regained all her previous strength and resumed her usual activity.

DISCUSSION. There were 48 reported cases of tetanus in 1997. The majority of which were in patients aged 20-59, but of those patients greater than 60 years, the female-to-male ratio was 1.75, reflecting widespread childhood and military vaccinations. There is no diagnostic laboratory test for the disease, which must be diagnosed clinically. The onset of the disease typically follows 2-10 days after introduction of tetanus, typically through a puncture wound. Full onset to opisthotonos can happen rapidly. Airway maintenance and oxygenation are crucial during the hospital course. Additional treatment consists of muscle relaxants for spasm, beta-blockade for thyrotoxic-like activity of tetanus toxin, and metronidazole for residual infection, and tetanus immune globulin.

ENDOCARDITIS PRESENTING AS ENCEPHALOPATHY. A Kaplan and L Adler, Department of Medicine, Mount Sinai Hospital, New York, NY.

LEARNING OBJECTIVES. 1) Assess acute encephalopathy in healthy patients, 2) Recognize the neurologic manifestations of endocarditis.

CASE. A previously healthy 30-year-old man was brought to the Emergency Department by his wife who noted a change in his mental status. The patient complained of left-sided throbbing headaches lasting several hours daily for three weeks prior to admission. The headaches increased in intensity and frequency without relief. Four days prior to admission the patient's wife began noticing confusion, and forgetfulness. The following day the patient had a fever of 102°F, began feeling more lethargic and had night sweats. He was taken to a clinic, where he had 0/3 object recall at one minute and underwent a CT scan of the brain without contrast, which was normal. Two days prior to admission, the patient developed dyspnea with exertion and sharp left mid-clavicular chest pain located at the third to fourth intercostal space radiating to the abdomen, and unaffected by position or exertion. The review of systems revealed a five-pound weight loss over the preceding three weeks. He had no history of rheumatic fever, but had a dental procedure four months earlier. He denied history of smoking, drinking, drug use or HIV exposure. On exam the patient was noted to be a thin black male, who was somnolent and in no distress. The vital signs were blood pressure 110/60 mm Hg, pulse 104 beats per minute, respirations 18 per minute, temperature 37.3°C. Fundoscopic exam revealed sharp disc margins. Visual fields and extra-ocular movements were intact with several beats of fine end gaze nystagmus laterally to both sides. The neck was supple. The patient had a hyperdynamic precordium with a 3/6 systolic murmur at the apex radiating to the axilla. The lungs were clear to auscultation bilaterally. The patient had a constricted affect, was oriented to person and place but had difficulty remembering the year. He had 0/3 object recall at one minute and could not subtract serial 7's. His gait was normal, although he had a slight deviation to the right when he marched in place. The rest of the exam was normal. The laboratory examination revealed 8100 white blood cells/μL with a normal differential, and hemoglobin of 14.4 g/dL. Serum electrolytes and liver function tests were normal. The sedimentation rate was 35 mm/hr. A urine toxicology screen was negative. Urinalysis showed trace blood, no red cells, and 1+ bacteria. The patient had a CT of the brain without contrast, an MR of the brain with and without contrast and an EEG, all of which were normal. The results of a lumbar puncture were consistent with a traumatic procedure. Six sets of blood cultures were drawn, and the CSF was cultured. Five of the initial six sets of blood cultures grew Streptococcus Intermedus-Milleri one day after having been drawn. Transthoracic and transesophageal echocardiograms performed on days 2 and 3, respectively, showed ruptured chordae of the mitral and tricuspid valves, severe mitral regurgitation with a perforation of the posterior mitral leaflet, and mild tricuspid regurgitation. The patient's heart murmur intensified and he developed of a palpable thrill over the next several days. The patient was treated with penicillin and gentamicin for a total of four weeks and underwent mitral valve replacement on day 13 due to the development of congestive heart failure. His mental status and affect improved post-operatively.

DISCUSSION: Neurologic deficits can be seen in approximately 1/3 of cases of endocarditis. Awareness of the neurologic manifestations of endocarditis will facilitate early diagnosis and treatment.

UNCORRECTED TETRALOGY OF FALLOT IN AN ADULT. I. Kenoly, C. Kelly, B. Bialor, Wayne State University/Detroit Medical Center, Detroit, Michigan.
LEARNING OBJECTIVES: 1) Recognize that adults with uncorrected congenital heart disease (CHD) may be classified as New York Heart Association (NYHA) class I and live without significant morbity. 2) Recognize that an illness can cause significant decompensation in patients with uncorrected CHD resulting in significant morbidity and mortality 3) Recognize that correction of CHD is a viable option in adulthood with improved quality of life.
CASE: JH is a 56-year-old white male who was diagnosed with tetralogy of Fallot (Tof F) 1993 who initially presented to an outside hospital with a chief complaint of right sided chest pain, shortness of breath, diaphoresis, and productive cough. The patient was treated for pneumonia and decompensated acutely. He was intubated and transferred to Harper Hospital Cardiac Care Unit for further management of his condition. The patient worked as a real estate instructor and broker and had been doing well and was very active 7 months prior to the hospitalization. He also had a dental procedure followed by a viral illness 8 months prior to the admission. Since that time he has been less energetic. The patient also has had "blue spells" all of his life, which entails turning blue around the mouth and fingers on exertion ("Tet spells"). The patient was taking vitamin E.
On admission, the patient appeared older than stated age and had a bluish-gray discoloration. The patient was intubated with 0_2 saturations between 79-84 % on 100 % O_2, BP 121/60, HR 80. Pertinent findings on physical exam: kyphosis and scoliosis; grade III/VI systolic murmur heard loudest at the lower left sternal border, S_1 and S_2 were normal; marked cyanosis and clubbing of fingers and toes.
The patient had a transesophageal echocardiogram that showed vegetation on the pulmonary artery beyond the pulmonic valve, a large ventricular septal defect overriding the aorta and a dilated right ventricle with hypertrophy. Blood cultures were positive for Streptoccus viridans. The patient was treated for endocarditis with gentamicin and penicillin, stabilized and discharged home with O_2.
A pediatric cardiologist was involved with the case to discuss the possibility of correction of the T of F. The need for cardiac catheterization to review pressures as well as anatomy was deemed the first necessary step.
After discharge, the patient followed-up with the pediatric cardiologist, but he was unwilling to agree upon a date to perform the catheterization. We are currently waiting for him to commit to a date.
DISCUSSION: According to two unrelated studies done by Nollert and Rammohan respectively, the correction of tetralogy of Fallot in adulthood results in normal life expectancy. These studies showed that 100% of their selected patients were NYHA class II or III prior to corrective surgery and advanced to NYHA class I after surgery.

IMMUNOLOGIC ANALYSIS OF PLEURAL FLUID IN POST CARDIAC INJURY SYNDROME. D. Murali[1], M. Kesani[2], B. Pavlovich[1], P. Mikkilineni[1]. C. Karmen[2], S.J. Peterson.[2] [1]Illinois Masonic Medical Center, Chicago, IL. [2]New York Medical College, Valhalla, NY.
LEARNING OBJECTIVE: To demonstrate that low complement levels in pleural fluid has the potential to be diagnostic of post-cardiac injury syndrome.
CASE: A 60 year old man admitted 6 days after coronary artery bypass graft surgery with pleuritic chest pain, fever, and shortness of breath of one day duration. He had a history of coronary bypass graft surgery 10 years prior, complicated by post-cardiac injury syndrome (PCIS) requiring the use of steroids.
Findings on physical examination include temperature of 99.3, pulse rate of 96/min, blood pressure 120/70, respiratory rate 24. Neck was supple with no elevated jugular venous pressure was noted. Heart sounds were normal and regular, no S3 or S4 no gallops were present. Breath sounds were decreased at both bases, a pleural and pericardial rub were present. Chest x-ray showed moderate size bilateral pleural effusions. A ventilation perfusion scan showed low probability for pulmonary embolism. Other significant laboratory findings included a white cell count of 19,900 with 72% polymorphs and 2% bands. Sedimentation rate was elevated at 71. Echocardiogram showed small posterior pericardial effusion. A thoracentesis was done 1 day following admission. The right sided pleural fluid showed : pH 7.5, Glucose 172 (serum 243), Total protein 3.1 (serum 5.2), LDH 1282 (serum 230), RBC 40,000 WBC 4,200 with 87% polymorphs, 6% lymphocytes, and 7% monocytes. Routine cultures were negative. The anti myocardial antibody titers were <1:40 both in the serum and pleural fluid. C3 index was 0.57 and C4 index was 0.61. Patient showed improvement after starting indomethacin.
The laboratory findings from the pleural fluid are shown in the table 1.

	Pleural fluid	Serum
C3 mg/dl	60	176
C4 mg/dl	11	30
C1q Binding	Present	Absent

DISCUSSION: Post-cardiac injury syndrome is a diagnosis of exclusion. Kim et al first proposed a potential diagnostic test, which utilizes immunologic analysis of pleural fluid. In their case report they suggested that a reduced complement in pleural fluid or pleural fluid AMA: serum AMA ratio greater than one could serve to support a presumptive diagnosis which relies heavily on clinical pattern recognition. This case demonstrates that low complement levels in pleural fluid has the potential to be diagnostic of PCIS in the right clinical setting.

CONSIDER NUTRITIONAL DEFICIENCY IN THE HOMELESS PATIENT. S. Kertesz, Boston University School of Medicine, Boston, MA.
LEARNING OBJECTIVES. 1)Recognize when a diagnosis of pellagra should be considered, 2)Understand the utility of a dietary history in homeless and alcoholic persons presenting with multisystem illness, 3)Appreciate the impact of street-living on the dietary habits of homeless persons.
CASE. A 58-year-old homeless man was admitted to hospital for chest pain and complaints including fever, nightsweats, malaise,10-lb weight loss, muscular fatigue, mouth-burning, nausea, vomiting, diarrhea, cutaneous burning sensations, cough, increased skin pigmentation and moodswings. Past history was unremarkable, including negative PPD and HIV tests. The patient reported use of 2-3 alcoholic beverages per day as well as tobacco. He had lived on the street for the previous four months. Physical examination was notable for bronze-colored skin with rare scale, poor dentition and low-grade fever. Initial studies showed: Hct 33.8, MCV 104, normal WBC, platelets, renal function, ALT 76 U/l, AST 126 U/l, albumin 3.5 g/dl and ESR 23. Chest x-ray, EKG and serial CK determinations were normal. Work-up included the following normal tests: serum TSH, cortisol, B12, folate, iron studies and RPR; stool cultures and ova/parasite examinations; sequential blood cultures for bacteria, mycobacteria and fungi; serum histoplasma antigen assay, gastrin and vasoactive intestinal peptide levels; 24-hour urine 5-HIAA, catecholamines, VMA and metanephrines, CT scan of the torso and jaw. Two teeth were extracted. His symptoms improved slightly without specific intervention. He was discharged on a multivitamin, thiamine, and folate. At one-week follow-up he noted partial improvement in symptoms. Review of his discharge prescriptions prompted consideration of a nutritional deficiency. A dietary history was taken, revealing that he had subsisted on alcohol and a daily snack, usually corn chips, since leaving the municipal shelter system four months previously. A tentative diagnosis of pellagra led to the institution of niacin 100 mg po bid. All symptoms resolved within two weeks.
DISCUSSION. A dietary history is crucial to the evaluation of multisystem illness in homeless patients, particularly if they reside outside support systems where meals can be obtained. An exhaustive work-up could have been avoided in this patient. His diet of beer and corn chips is curiously instructive; classic reports of the pellagra epidemic in this country emphasized the vulnerability of alcoholics and populations subsisting on milled corn. Because the triad of "dermatitis, diarrhea, dementia" is present only in a minority of pellagra patients, the presence of any one symptom in the appropriate setting should prompt a thorough nutritional history.

A CASE OF MENINGOCOCCEMIA IN A JAIL INMATE WITH ADVANCED LIVER DISEASE. Muhammad Khalid, Martin Tobi, and Bruce Bialor, Department of Internal Medicine, Wayne State University School of Medicine, Detroit, MI.
LEARNING OBJECTIVES: (1) Recognize meningococcal disease as a potential complication of advanced liver disease, especially among patients living in overcrowded settings. (2) Recognize the importance of improving preventive services to persons who have been incarcerated.
CASE. A 47-year-old male jail inmate with a history of cirrhosis secondary to Hepatitis B, Hepatitis C, and alcohol abuse, presented with a 4-5 day history of altered mental status. He was supposed to be on lactulose and spironolactone, but was known to be non-compliant. There was no history of fever, constitutional symptoms, or gastrointestinal bleeding. The patient was known to have chronic liver disease, with many admissions for hepatic encephalopathy and spontaneous bacterial peritonitis (SBP) in the past. He was allergic to penicillin. On exam, he was afebrile and had stable vital signs. He was agitated and required four point restraints. He had scleral icterus, gynecomastia, tense ascites, and peripheral wasting. Paracentesis showed a WBC count of 8,500 /cmm with 80% neutrophils and an ascites serum albumin gradient of >1.1 gram. The peripheral WBC count was 8,800 /cmm with 91% neutrophils and 1% bands; albumin was 1.8 g/dl. The patient was treated with intravenous ceftriaxone for SBP. The patient remained afebrile throughout the hospital course. Routine blood cultures obtained on day 1 of admission were positive for meningococcus. Culture of ascitic fluid was negative. His mental status improved but fluctuated after admission. He had designated himself as a "no code" and subsequently refused all further blood draws and treatment, except for antibiotics. He died on day 9 of admission. There was no post-mortem.
Discussion: Carriage rates for meningococcus in jail inmates have been reported to be as high as 19%, compared to 1% in the community. The prison population of the United States has doubled in the last decade; therefore, one would expect meningococcal disease to become more prevalent in this population. Patients with chronic liver disease (which is very common in jail inmates due to alcohol abuse) are particularly susceptible to meningococcal infection due to acquired complement deficency, since the liver is the major site for complement synthesis (especially C5,C8, and factor I). The clinical picture may also be less fulminant as a result. In conclusion, communicable diseases like meningococcus are important public health problems in overcrowded conditions. While chemoprophylaxis and vaccination programs are not feasible for the majority of prison inmates, inmates with liver disease may benefit from prophylaxis for meningococcus. Doing so will improve the health of both inmates and the communities to which they return.

THROMBOTIC COMPLICATIONS OF AN HIV PATIENT WITH ANTIPHOSPHOLIPID ANTIBODIES.
S. Khan, S. Kessler, S.J. Peterson. New York Medical College, Valhalla, NY
LEARNING OBJECTIVE: To demonstrate that HIV patients with antiphospholipid antibodies can still develop thrombotic complications
CASE: A 41 year old male with no significant past medical history presented with complaints of fever and sinus congestion initially treated with oral antibiotics. The fever continued over the next three weeks with temperatures as high as 103 degrees, which prompted admission and workup. His physical exam did not reveal any obvious source of infection. He denied any risk factors for HIV. Laboratory data upon admission revealed a white blood count of 9000 and mild elevations of his liver function tests. An infectious etiology of his fever was considered and workup for Tuberculsosis, Leptospirosis, and Brucellosis was negative. CT scan of sinuses,abdominal ultrasound, and bone marrow biopsy were also unrevealing. An abdominal CT scan revealed a psoas muscle fluid collection which was aspirated and found to not be infectious. Three days after admission, the patient developed pain and swelling in his fingers on both hands which eventually progressed to gangrene. A skin biopsy done at that time revealed a leukocytoclastic vasculitis. A rheumatologic work up was sent and the patient was found to be anticardiolipin antibody positive. He was also found to be HIV positive with an absolute CD4 count of 10. Angiography revealed a completely occluded right radial artery and a partially occluded left radial artery. The patient was started on pulse dose steroids and was anticoagulated. His symptoms did not improve until plamapharesis was initiated.
DISCUSSION: Although it has been established that patients who are HIV positive can also have antiphospholipid/anticardiolipin antibodies but they have been of doubtful clinical significance. We found one other case reported in the literature of an HIV positive patient who had necrotic skin lesions and was found to have elevated antiphospholipid antibodies. Our case is another example of a patient with HIV and increased levels of anticardiolipin antibody who developed a thrombotic complication.

TITLE: MENINGIOMA MIMICKING A NONFUNCTIONING PITUITORY TUMOUR. P. Khanna, R. Ehrlich, V. Bahl, A. Tulsky. UPMC Shadyside, Pittsburgh, PA.
LEARNING OBJECTIVE; Recognize the importance of differentiating preoperatively meningioma from other sellar masses .
CASE; A 66 year old diabetic female presented with a six week history of vertigo, blurred vision and unstable gait. On examination she described subjective vertigo with change of head position but there was no nystagmus. Aleft temporal visual field cut and ataxic gait were noted. The rest of the neurological examination was normal. Magnetic resonance imaging with gadolinium revealed a lobulated homogenously enhancing intrasellar mass with suprasellar extension, compression and displacement of optic chiasm superiorly and lateral displacement of supraclinoid carotid artery. Laboratory work-up revealed normal luteinizing hormone, follicular stimulating hormone, growth hormone, prolactin, T3, T4, thyroid stimulating hormone and cortisol suggesting a nonfunctioning pituitory tumour. The patient underwent transsphenoidal resection and pathology revealed meningioma. Postoperatively visual symptoms persisted and repeat magnetic resonance imaging revealed 2 cm mass in the dorsum sella. Right frontal craniotomy with microexcision of the meningioma and decompression of optic chiasma was performed.
DISCUSSION; Differential diagnosis of intrasellar mass with suprasellar extension includes pituitory adenoma, suprasellar meningioma, craniopharyngioma, aneurysms, metastatic tumours, and chiasmal gliomas. These can be distinguished from one another noninterventionally except pituitory adenoma and meningiomas. There are some diagnostic studies which show promise. Enhanced magnetic resonance imaging with gadolinium can be helpful in differentiating them. Meningiomas are homogenously enhancing tumours with suprasellar epicenters,they can encase the carotids and cause truncation of the dorsum sella. Pituitory adenomas are heterogenous, have their epicenter in the sella, don't encase the carotid and don't truncate the dorsum sella. On Positron Emission Tomography using (11C)-L-Deprenyl adenomas show rapid and high uptake immediately after injection and a constant level later equal to or higher level than in brain tissue. Meningiomas show initial high uptake followed by marked decrease reaching level half that of brain tissue. When a homogenously enhancing mass with suprasellar extension is seen, there should be a high suspicion of meningioma and positron emmission tomography scan done to avoid the wrong surgical approach as occured in our patient The cause of vertigo in our patient remained unexplained but she found relief after surgery.

TITLE: GAVE: WHAT THE MIND DOES NOT KNOW THE EYES DON'T SEE
P. Khanna, A. Shapiro, S. Evans, J. Ward, A. Tulsky, UPMC Shadyside, Pittsburgh, PA
LEARNING OBJECTIVE; Recognize gastric antral vascular ectasia as a differential diagnosis in recurrent upper gastrointestinal bleed.
CASE; A 65 year old female with history of recurrent gastrointestinal bleed requiring 26 units of blood transfusion over six months was admitted with anemia and extreme weakness. She had undergone repeated sessions of endoscopic cauterizations with minimal improvement. Endoscopy confirmed appearances of gastric vascular ectasia. Failure of medical therapy prompted surgery which included antrectomy with a Roux-en-Y gastrojejunostomy. During surgery she was noted to have a liver nodule , pathology of which showed cirrhosis.
DISCUSSION: Gastric antral vascular ectasia or watermellon stomach is a clinicopathological condition characterized by severe gastrointestinal blood loss, iron deficiency anemia, endoscopic appearances of longitudinally prominent erythematous mucosal ridges converging on the pylorus and histological appearance of ectatic vascular channels, intravascular fibrin thrombi, and fibromuscular hyperplasia. Ettiologic factors include hypergastrinemia, slower gastric emptying, increased levels of prostaglandin E2, and increased proliferation of local neuroendocrine cells containing 5 hydroxytryptamine and vasointestinal peptide. Diseases which gastric antral vascular ectasia is associated with include portal hypertensive states resulting from hepatic cirrosis or venoocclusive diseases, cirrosis without portal hypertension, autoimmune diseases and chemotherapy and growth factor use in bone marrow transplant patients..Diagnosis of gastric antral vascular ectasia is delayed for an average of 5-6 years as both endoscopic and histologic findings can often missed or misinterpreted. Thus physicians should be aware of gastric antral vascular ectasia as a rare cause of gastrointestinal bleed.This presentation focuses on such a case with pitfalls in diagnosis and treatment. Therapy consists of medical management by endoscopic laser ablation. Pharmacotherapy with oestrogens, progesterone and calcitonin has been described. Antrectomy offers cure in patients who fail medical management. Awareness of gastric antral vascular ectasia can lead to early diagnosis by limitting the number of investigations, reducing morbidity associated with chronic blood loss and repeated blood transfusions. This review emphasizes gastric antral vasculare ectasia as an important cause of gastrointestinal bleeding, outlines management options and highlights the role of surgery in patients who fail medical management.

NEUROCARDIOGENIC SYNCOPE PRESENTING WITH COMPLETE HEART BLOCK. P. Khanna, G. Tabas. UPMC Shadyside, Pittsburgh, PA
LEARNING OBJECTIVE: Diagnose neurocardiogenic syncope in the presence of coexisting conduction abnormalities.
CASE Patient is a 70 year old white male with a history of three vessel coronary bypass surgery, rheumatic aortic stenosis (status post two aortic valve replacements), renal artery stenosis treated with stent placement and carotid endarterectomy had his last vascular surgery 8 months ago. Since then he has had multiple episodes of syncope which occurred with turning of his head, walking, sitting, during micturition, and on bending down. He described a "flash across my head" and passed out without any other premonitory symptoms. There was no associated chest pain, no palpitations, nausea or diaphoresis. Syncopal episodes usually lasted a few seconds , always less than a minute and on waking up, the patient noted that his pulse rate was always in the 40s.
Examination on admission showed no orthostatic blood pressure change, no significant murmurs and a normal neurological examination. Holter monitor showed a heart rate between 52-93 beats per minute and three asymptomatic sinus pauses, the longest lasting 3 seconds. Echocardiography revealed an ejection fraction of 45% and no thrombus. MRI of brain was normal and an electrophysiologic study showed normal conduction. Tilt table testing was positive. A diagnosis of neurocardiogenic syncope was made and the patient discharged on beta blockers. He returned after three days with increasing frequency of syncope and bradycardia. On admission beta blockers were discontinued and a subsequent presyncopal episode was associated with 6 beats of complete heart block. His syncope was now thought to be of cardiogenic origin and a pacemaker was inserted. The patient continued to have presyncopal episodes resulting in reinstitution of beta blockers followed by relief of his symptoms.
DISCUSSION: The pathogenesis of neurocardiogenic syncope is peripheral pooling of blood increasing the inotropic activity of the heart which stimulates the mechanoreceptor C fibers in the ventricles. This increases parasympathetic activity causing bradycardia. Beta blockers have proven to be effective in these patients. In our patient, beta blockers resulted in intolerable bradycardia with heart block necessitating pacemaker insertion. The diagnosis of neurocardiogenic syncope in this patient was confused by the short episode of heart block, but the normal electrophysiologic study, the positive tilt table test and the response to beta blocker therapy favor the diagnosis of neurocardiogenic syncope.

POSTPARTUM BLINDNESS: IT'S NOT JUST HYSTERIA R. Khurana, R. Powrie, L. Larson. Division of Obstetric and Consultative Medicine, Department of Medicine, Women & Infants' Hospital, Providence, Rhode Island.
LEARNING OBJECTIVES. 1) Recognize how preeclampsia can cause visual impairment 2) Manage cortical blindness secondary to preeclampsia.
CASE. A 13 year old female at 38 weeks gestation presented with a one week history of headache, scotomata and generalized edema. Physical examination was normal except for a BP of 144/84. (Her BP had been 100/60 on her first prenatal visit.) She was given a presumptive diagnosis of preeclampsia and admitted for observation and bed rest. Her uric acid was elevated at 6.9 mg/dL, but a CBC, AST, creatinine and 24 hour urine protein were normal. Her symptoms improved but a fetal ultrasound revealing intrauterine growth restriction lead to the decision to deliver her on hospital day 4. On her first postpartum day, she awoke with severe headache and complained of blindness. Her providers suspected either malingering or a conversion disorder so a consultation from psychiatry was requested. A general medicine consult was also requested. Her pupils at that time were found to be equal and reactive to light. She blinked to threat on the left but not on the right. Her funduscopic exam was normal with no papilledema, retinal detachment, retinal edema or vasospasm. Her neurologic examination was otherwise normal. She had no edema, clonus or epigastric tenderness. A CT Scan of the head was obtained and was normal. An MRI of her brain revealed bilateral occipital lobe edema. She was therefore diagnosed with cortical blindness presumed secondary to preeclampsia. The head of her bed was kept elevated and magnesium sulfate was given for seizure prophylaxis. By the next day her vision had improved so that she could see some color and shapes. Her vision was fully restored by the third day postpartum.
DISCUSSION. Visual disturbances (typically scotomata, scintillations and blurring) occur in 25% of women with preeclampsia. These disturbances are usually due to retinal artery vasospasm, retinal edema and serous retinal detachments. True cortical blindness occurring with severe preeclampsia is much less common. Management includes delivery, magnesium sulfate for seizure prophylaxis, elevation of the head, fluid restriction and control of severe hypertension. Visual complaints in the pregnant or postpartum woman always need to be taken seriously.

OUR EXPERIENCE ON THE USE A LOW-INTENSITY HELIUM-NEON LASER IN THE TREATMENT OF ACUTE MYOCARDIAL INFARCTION. NN. Kipshidze, Institute of Therapy, Tbilisi, Georgia

Intravenous blood irradiation with a Helium-Neon laser performed in 900 patients with acute myocardial infarction immediately after hospitalization (within first four hours after anginal attack) using the method developed by as. Laser procedures were done once a day during first five days of the disease. Our investigations showed that a He-Ne laser produced a broad therapeutic effect as manifested in alleviation of pain syndrome and limitation of ischemic area by the data of organ-specific enzymes (MB-CPK and CPK) monitoring and ECG mapping. The decrease in the injury area was confirmed by our earlier experimental studies. Antioxidant blood activity as well as blood and tissue oxygen contents under laser rose (catalase, by 42%; superoxide dismutase, by 45%; glutathione reductase, by 35%; pO_2, by 18%; tissue O_2, by 24%). Blood irradiation reduced the amount of drugs used, duration of stay in hospital decreased by 7 days, on the average, and mortality rate fell markedly (by 2.5 times). Holter ECG monitoring showed that the laser therapy had a high antiarrithmic efficacy: ventricular extrasystoles decreased by 77% and atrial ones by 70%. Ventricular fibrillation was observed in 1.2% vs. 6.9% in the control group given traditional therapy. The results of our studies showed that invasive laser therapy decreases the pain syndrome, normalizes electrical instability of the myocardium, limits ischemic damage area and accelerates scarring process.

MANAGING REFRACTORY DYSPNEA AND AGITATION IN A DYING PATIENT LA King, EL Krakauer, JA Billings, Palliative Care Service, Massachusetts General Hospital, Boston, MA
LEARNING OBJECTIVES: 1)Manage refractory dyspnea and terminal agitation in a patient unable to tolerate opioids 2)Explore institutional barriers to symptom management in end-of-life care
CASE: A 45 year-old woman with a history of advanced adenocarcinoma of the lung was admitted to the hospital with a two week history of worsening dyspnea. Chest computed tomography scan suggested lymphangitic spread of tumor as the likely etiology of her progessive symptoms. Despite treatment with corticosteroids, broad-spectrum antibiotics, oxygen, and benzodiazepines, she became increasingly dyspneic at rest and extremely anxious. All opioids caused severe nausea and vomiting despite aggressive anti-emetic regimens. Further options for palliative care, including terminal sedation, were discussed with the patient and her husband, who were relieved to learn that such options were available and were consistent with their religious beliefs. Her worsening symptoms were initially managed with escalating doses of intravenous benzodiazepines and neuroleptics (lorazepam and droperidol, both scheduled and as needed). However, despite massive doses of benzodiazepines (lorazepam titrated up to 45 mg/hr continuous infusion with bolus doses ranging from 5 - 10 mg as needed) and large doses of sedating neuroleptics (droperidol 2.5 - 5 mg up to hourly), the patient continued to awaken every 30 to 90 minutes over the next two days and to experience severe dyspnea, anxiety, and agitation. Intravenous propofol was proposed to control her symptoms, but hospital policy prohibited its use outside of the intensive care unit (ICU). The patient, her family, the nursing staff, and the ICU staff all were reluctant to move the patient. Finally, she became unresponsive and died eight hours later.
DISCUSSION: Dyspnea, terminal agitation, and anxiety occur commonly in patients during the last few days of life. These symptoms can usually be managed successfully with standard doses of opioids, benzodiazepines, and neuroleptics. Patients with refractory symptoms or side effects from standard therapies who view sedation as an acceptable side effect to achieve comfort may benefit from propofol therapy. Propofol, an intravenous sedative and hypnotic agent commonly used for induction and maintenance of anesthesia and for ICU sedation, has many features well-suited for providing terminal sedation and for managing anxiety, agitation, and other symptoms in terminally ill patients. Propofol has a rapid onset of action, is easily titrated, and lacks significant uncomfortable side effects. Nurses who care for dying patients can easily be trained in its use. Currently, hospital policies limit use of this agent in palliative care. Policies should be changed to permit its use outside of the ICU for patients whose care is focused on comfort and symptom control rather than on cure or life support.

HEPATITIS ON THE HALF SHELL: PRESENTATION AND PREVENTION OF HEPATITIS A. D. Krause, C. Smith, Department of Medicine, Beth Israel Deaconess Medical Center, Boston, MA.
LEARNING OBJECTIVES: 1) Recognize Hepatitis A as an increasing cause of fulminant hepatic failure and, 2) Recognize the importance of prevention through education and vaccination.
CASE: A 32-year-old man without significant past medical history presented with three days of malaise, nausea, and vomiting. He had a baseline alcohol intake of one case of beer per week, but had consumed one six pack of beer per day the week prior to admission. He denied any other drugs, toxin exposures, or family history of liver disease. Two weeks prior to admission he had consumed raw clams. On admission he was found to be jaundiced and dehydrated with mild right upper quadrant tenderness. His labs revealed WBC 16, PT 41, INR 9.5, ALT 6600 AST 6130, total bilirubin 9, direct bilirubin 5, LDH 3870, creatinine 4.9, BUN 25. Toxicology screen was negative, including alcohol and acetaminophen. Hepatitis A IgM was positive, HepBCore Antibody positive, Hep C negative, CMV IgG positive, EBV IgG positive, HSV1 IgG positive, negative ANA. Abdominal ultrasound revealed patent hepatic and portal veins. Over the initial 24 hours his PT rose to 50. He became agitated, then progressively more lethargic, necessitating intubation. Three days after his presentation he underwent orthotopic liver transplantation for fulminant hepatic failure, which was complicated by S.aureus sepsis and early rejection.
DISCUSSION: Hepatitis A (HAV) is usually a mild, and frequently subclinical condition in children; it is often a much more serious and prolonged disease in adults. Though it accounts for less than 1% of cases of fulminant viral hepatitis, the risk increases with age. In developing countries immunity to hepatitis A approaches 100% by the age of ten. However, in the United States, only 50% of those greater than 50 years old have detectable immunity. A substantial segment of the U.S. adult population is therefore susceptible to HAV infection, increasing the potential for large-scale outbreaks in urban areas. As this case illustrates, the consequences of this disease in adults can be devastating. Current vaccination recommendations are limited and should be expanded with investigations made regarding the utility of adding hepatitis A vaccine to routine immunization of children.

A REVERSIBLE CAUSE OF CARDIOMYOPATHY IN AN AIDS PATIENT

Ashok S. Krishnamurthy MD (Associate), Asher Tulsky MD, UPMC Shadyside, Pittsburgh, PA

LEARNING OBJECTIVES: 1) Cardiac diseases are a common cause of shortness of breath (SOB) in HIV patients. 2) AZT can induce a reversible cardiomyopathy, which is important to recognize.

CASE: A 22 year old male diagnosed as AIDS with PCP 18 months ago was admitted with increasing SOB and cough with scanty white sputum since 5 days. Eight weeks prior to admission he had a CD4 count of six, was started on AZT, Lamivudine, Indinavir in addition to bactrim and azithromycin for prophylaxis. On this regimen he was feeling well until a month prior to admission, gained weight, and had a normal PE, chest X-ray and Pulse oximetry. He is a seven pack-year smoker, occasional beer drinker and denied cocaine or IV drug use. Physical Examination: Vitals: PR-120/min, RR- 24/min, BP- 100/60, temp-38 c and jugular venous pulse raised. Chest exam revealed S3 gallop and bibasilar crackles. No pedal edema. ABG- pH 7.49, PaO2-82, PaCO2-21, Cxray – cardiomegaly with bilateral infiltrates in mid and lower zones. Echocardiogram – 4 chamber dilatation with MR, TR and Ejection Fraction 12%. CD4 count 26. SwanGanz - LVEDP – 30, Cardiac Index -2.1. Patient treated with diuretic, digoxin, captopril and anti HIV medications continued. Sputum, blood and urine cultures were negative for bacteria, fungi & mycobacteria. He was evaluated for myocarditis with serial cardiac enzymes, tests for Toxoplasma, Coxsackie A&B, Enteroviral cultures, Legionella antigen, CMV and Gallium scan all of which were normal. Cardiotoxicity due to AZT was then suspected & it was stopped on day 3. Patient started improving after cessation of AZT, LVEDP 14, and Cardiac Index 4.0. Patient discharged on day 11 on digoxin, lasix, captopril, bactrim, azithromycin, Saquinavir, Ritonavir and DMP 266.

DISCUSSION: Cardiac diseases occur commonly in AIDS patients presenting with SOB. Cardiomyopathy can be due to multiple causes but the only reversible causes are (1) Toxoplasma (2) Viral Myocarditis – spontaneous resolution and (3) Drug induced – AZT. AZT induced cardiomyopathy has been reported in 6 patients in 1992, and more recently in the pediatric population. It is thought that AZT induces changes in cardiac muscle mitochondria leading to a lower average fractional shortening of cardiac muscle. This type of cardiomyopathy is reversible (as in the above patient) it remits after withdrawal of the drug. Hence it is important to recognize this condition and take suitable steps during work-up of an AIDS patient with SOB – "bilateral chest infiltrates in AIDS is not always PCP!"

AN ATYPICAL COMPLICATION OF AN ATYPICAL PNEUMONIA, D. Liao

LEARNING OBJECTIVES: 1) Recognize the clinical features, diagnosis and treatment of bronchiolitis obliterans organizing pneumonia (BOOP). 2) Recognize conditions which may predispose to BOOP.

CASE: A previously healthy 63 y.o. man presented with a 3 week history of progressive shortness of breath and non-productive cough. Ten days prior to admission an outpatient chest x-ray showed bilateral infiltrates and clarithromycin therapy was initiated. After 4 days of treatment without symptomatic improvement, clarithromycin was discontinued and blood and sputum cultures obtained. The patient was treated empirically for congestive heart failure with furosemide and prinivil. The patient's dyspnea worsened and the day of admission he came to the emergency room where evaluation showed worsening infiltrates on chest x-ray and hypoxemia. He denied fever, chills, chest pain or symptoms of congestive heart failure. His past medical history was notable for controlled hypertension and excessive alcohol use. On examination he was afebrile with normal vital signs. Finding were notable for bilateral crackles in the lower 1/3 of the lung fields and purple discoloration of the fingertips. Laboratory evaluation was notable for positive Mycoplasma pneumoniae IgM antibodies and negative blood and sputum cultures. Chest x-ray showed bilateral multilobar infiltrates. CT scan of the chest showed alveolar infiltrates in all lobes and interstitial infiltrates in the upper lobes bilaterally. Transbronchial biopsy showed interstitial pneumonitis with areas of organizing felt to be consistent with BOOP. The patient was treated with a clarithromycin and prednisone and improved after several weeks.

DISCUSSION: Most cases of BOOP are idiopathic, but can be related to connective tissue disease, drugs and rarely infections such as Mycoplasma pneumonia. Transbronchial or open-lung biopsy is required for diagnosis, and most cases respond to prednisone therapy. BOOP should be considered in patients with diffuse abnormalities on chest x-ray suggestive of pneumonia who do not respond to antibiotic therapy.

HYPOPARATHROIDISM PRESENTING AS HYPERCALCEMIA: A CASE OF IATROGENIC VITAMIN D INTOXICATION. Neil Levin Division of General Internal Medicine, University of California, San Francisco, San Francisco, California

LEARNING OBJECTIVES: 1) Recognize the importance of obtaining an accurate history in patients, especially when language barriers exist 2) Assess patients for vitamin D intoxication and treat appropriately.

CASE: An 81 year old hispanic female with a history of diabetes, hypothyroidism (s/p thyroidectomy), and gout, presented for transfer of care to our clinic with c/o's weakness, anorexia, and mild confusion worsening over the past couple of months. She was spanish-speaking and lived with her children. As no translator was available during the clinic visists her daughter translated. Her current medications included glipizide, synthroid, vitamin D, and calcium, but the patient was unclear about her doses. Physical examination was unrevealing. Laboratory evaluation revealed a calcium level of 12.6 mg/dl (8.4-10.2), TSH of .3 mIU/L (.5-4.7) fT4 of 35 pmol/L (9-24), creatinine 1.5 mg/dl (.5-1.4). Subsequent work-up for hypercalcemia revealed a PTH < 10 ng/L (20-65), PTH-rp .5 pmol/L (<1.3), negative SPEP, normal mammogram and chest x-ray, and 25 OH vitamin D 92 ug/L (9-52). The daughter was asked to bring in all her mother's pill bottles which revealed that the patient was taking levothyroxine .2 mg per day, vitamin D (ergocalciferol) 50,000 U alternating with 100,000 U per day, and calcium carbonate 1500 mg per day. Diagnosés of iatrogenic vitamin D intoxication and hyperthyroidism were made and the patient was told to stop her vitamin D and calcium and lower her synthroid dose to .05 mg per day. Her symptoms improved over the next 1-2 weeks. The patient's prior physician was contacted and he revealed that she had a history of hypoparathyroidism requiring high doses of vitamin D for persistent hypocalcemia, as well as high doses of levothyroxine for persistent hypothyroidism. Upon further questioning the daughter then revealed that she had begun administering her mother's medications just prior to the onset of symptoms and transfer of care. The patient's 25 OH vitamin D level peaked at 301 ug/L (9-52) several weeks after she stopped taking vitamin D. Her 1,25 OH vitamin D level at that time was 80 ng/L (15-60). On follow-up visits her calcium level fell but remained slightly elevated (10.5 to 10.3) for almost three months before returning to the normal range.

DISCUSSION: 1) In this patient vitamin D intoxication resulted from changes in compliance with her medical regimen. The patient's inability to speak english and the lack of understanding regarding her medical problems by both the patient and her family made obtaining an accurate history a challenging endeavor, and inititally obscured the underlying diagnosis of hypoparathyroidism. 2) Vitamin D intoxication should be suspected in patients with hypercalcemia on any dose of vitamin D. The 25 OH vitamin D level should be elevated, while the 1,25 OH vitamin D level may be normal or only slightly high. Depending on the vitamin D analog used by the patient, the 25 OH level may rise for weeks after the medication is discontinued and as a result hypercalcemia can persist for weeks to months without treatment. The treatment of vitamin D intoxication begins with discontinuation of the vitamin D analog, and calcium supplementation. Rehydration with intravenous saline solution can acutely lower calcium levels. For severe hypercalcemia administration of a bisphosphonate has been shown to work better than steroids to normalize calcium levels.

A CASE OF A FALSE POSITIVE HIV VIRAL LOAD IN A MAN SUSPECTED OF PRIMARY HIV INFECTION. José A. Luchsinger. Wylie Hembree. Department of Medicine. Columbia-Presbyterian Medical Center. New York, NY.

LEARNING OBJECTIVES: To increase awareness of the potential for false negative HIV viral loads when used in the diagnosis of HIV infection.

CASE: A 35 year old homosexual man with a history of genital warts and no other significant medical history presented with a clinical picture of urticarial rash and migratory polyarthritis for 2 days; no fever, urethritis, or diarrhea. He denied use of prescription or over the counter medications. Recent exposure to an allergen was not apparent. There were no risk factors for Lyme disease. The patient denied sexual intercourse for 6 months prior to the presentation, but performed oral sex with men. The physical exam revealed normal vital signs, urticaria, mild left ankle swelling without evidence of synovitis, and normal genital and anal exam. In light of the clinical picture and the patient's risk factors, the following were considered the most likely differential diagnoses: disseminated gonococcemia, reactive arthritis, serum sickness, Still's disease, and primary HIV infection. Pharyngeal, penile, and anal samples for GC and Chlamydia were negative. CBC, Chemistries, ANA, RPR, and Rheumatoid factor, were unrevealing. A Chest roentgenogram was normal. An ophtalmological exam revealed no uveitis. A skin biopsy revealed perivascular infiltrates of mononuclear cells and eosinophils, suggestive of drug eruption or urticaria. An HIV ELISA test was negative. A course of prednisone was given with partial resolution of the clinical picture 7days after its onset. In light of the lingering concern for Primary HIV infection and a negative HIV ELISA test, a HIV Viral load test by PCR was obtained, which revealed 558 copies/ml. Given the unexpectedly low viral load, the diagnosis of Primary HIV infection was considered unlikely. The results of the test were disclosed to the patient, counseling about the possibility of a false positive result, and further testing was recommended. Lymphocyte subset counts were normal. The CD4/CD8 ratio was 2.2. Viral load testing was repeated in a different laboratory twice, and were negative. The patient was counseled that the first HIV viral load result was likely a false positive, and that the most likely explanation for his presentation was serum sickness, or an allergic reaction. Further HIV ELISA testing was also suggested after a few months.

DISCUSSION: Viral load testing is used to follow the response to antiretroviral treatment in known HIV infection. However, this is the 5th case of false positive HIV viral load testing that has been documented when using it for diagnosis of infection. A false positive diagnosis of HIV infection with improperly used viral load testing can lead to undue suffering on the part of patients.

ACUTE RESPIRATORY FAILURE AND CHARCOTMARIE TOOTH DISEASE
K Mahmood, J Kerr, Department of Medicine, Highland Hospital, Rochester, NY.
LEARNING OBJECTIVES. Recognize diaphragmatic paralysis as a cause of respiratory failure.
CASE. A 63 year old female with Charcot Marie Tooth disease (CMT) presented to the ER with a several day history of confusion and lethargy. She was wheel chair bound due to her advanced disease and lived at a chronic care facility.Exam was significant for a stuporous elderly woman . Pulse 100, BP 108/70, afebrile.Her lung exam showed poor air movement and basilar rales.Extremities showed severe wasting of both calf muscles and bilateral foot drop.Initial lab data was significant for an ABG on 100% nonreabreather : pH 7.4, pCO2 130.6, paO2 119. The chest x -ray showed small lung volumes, basilar atelactasis and elevated hemidiaphragms She was urgently intubated and transferred to ICU for ventilator support. The etiology of her acute respiratory failure was not immediately apparent. She had no past history of lung disease or smoking , nor drug abuse and no witnessed episodes of aspiration. Other labs including phosphate, magnesium and TSH were normal. Investigation of the elevated hemidiaphragms lead to no discernable EMG tracing. This was felt to be consistent with involvement of the phrenic nerve in her hereditary neuropathic process.
DISCUSSION. Although respiratory failure is often due to lung disease, its association with neuromuscular disorders including CMT disease should not be overlooked.This is a hereditary motor nuropathy whose classic presentation involves a slowly progressive degeneration of the somatic peripheral nerves. The disease predominantly affects the long axons of the popliteal and sural nerves and patients often report decades of calf muscle wasting , foot drop and unsteady gait. Occasionally , peripheral nerves with shorter axons such as the phrenic nerve are affected. This leads to bilateral diaphragmatic pralysis and respiratory failure. These patients invariably have advanced nuerologic disease, with both upper and lower extremity involvement impacting on the qualities of their lives. Patients are intubated in the setting of severe obtundation and CO2 retention which may assumed to be due to a primary lung problem with possible reversibility which in fact is an irreversible nuerological illness.
Early diagnosis would have lead to an oppurtunity to review with patients and their families wishes regarding chronic ventilator support.
The history (especially absence of previous lung disease and smoking and family history) and physical examination (evidence of neurological disease) are the key to diagnosis , confirmed by EMG and nerve conduction velocities.

CRYPTOGENIC CIRRHOSIS: CASE OF HYPERVITAMINOSIS A. UG Mason, L Whittaker, N Toribara, P Mehler, Divisions of General Internal Medicine and Gastroenterology, Denver Health Medical Center, Denver, Colorado.
LEARNING OBJECTIVE: Assess and recognize differential diagnosis of cirrhosis.
CASE. A 46 year old female presented with three months of diarrhea, increasing abdominal distention and lower extremity edema. She did not smoke, drink, or use illicit drugs; and there was no family history of inflammatory bowel disease. Physical exam was remarkable for a cachetic patient with alopecia, dental caries, chelosis, ascites, and lower extremity edema complicated by weeping ulcers. Thyroid, heart and lungs were normal. Laboratory studies revealed: albumin 2.2, alkaline phosphatase 205, AST 61, ALT 37, a normal ceruloplasmin, and negative hepatitis serologies, antinuclear antibody, antismooth muscle antibody, antimitochondrial antibody, and a normal alpha-1-antitrypsin level. An esophagoscopy/colonoscopy revealed 1+ varices with a normal mucosa. An abdominal CT showed cirrhosis and ascites. A liver biopsy was consistent with micronodular cirrhosis with fat storing cell hyperplasia and negative autoantibody stains. Upon further questioning the patient reported ingesting between 30,000 - 300,000 IU of vitamin A daily for the past ten years. Two months after stopping vitamin A, her health had improved: less ascites and healing of leg ulcers.
DISCUSSION. Hypervitaminosis A can cause liver failure. Since the initial report in 1944, there have been at least 140 cases described. Although the toxic dose of vitamin A is unknown, it appears that the total cumulative dose is of critical importance. The pathophysiology of vitamin A toxicity occurs from increased storage of the vitamin by fat storage cells leading to differentiation to a myofibroblast-like cell that secretes extracellular matrix components resulting in parenchymal obliteration and, ultimately, cirrhosis. Differential diagnoses of the cirrhosis include autoimmune hepatitis, captopril toxicity, alcohol-related injury, congestive heptopathy, viral hepatitis, and Wilson's disease. Promoted as having a role for a multitude of conditions, vitamin A is easily obtainable without prescription and bears no warning labels about potential hepatotoxicity. Internists should be aware of excessive vitamin A ingestion in patients being evaluated for liver failure.

HUMAN IMMUNODEFICIENCY VIRUS: WHOM DO WE TEST? WHEN DO WE TELL? Rajesh S. Mangrulkar, Jihn D. Han, Shelly A. McNeil. Department of Internal Medicine, University of Michigan Health System, Ann Arbor, MI.
LEARNING OBJECTIVES. 1) Recognize the clinical situations in which pursuing the diagnosis of underlying HIV infection is appropriate. 2) Manage the indeterminate HIV Western Blot. 3) Balance the complex issues of patient confidentiality against limiting potential spread of HIV, in the setting of an indeterminate Western Blot.
CASE. A previously healthy 42-year-old man presented with a rapidly progressive left upper extremity cellulitis. On the day prior to presentation, he struck his left elbow on an asphalt surface while changing a flat tire. Twenty-four hours later he noticed a large collection of fluid in his left elbow, accompanied by significant tenderness of his entire arm. On presentation, he was noted to have a fever of 102°F, a left olecranon bursa fluid collection with an overlying skin abrasion, extensive left arm erythema with bullae formation, and tender left axillary lymphadenopathy. His WBC was 12,500, with 73% neutrophils, 20% band forms and 3% lymphocytes. Total protein and albumin were both depressed at 5.1g/dl and 2.6 g/dl, respectively. Aspiration of his left olecranon bursa revealed frank pus, the cultures of which grew out Group A Streptococcus Pyogenes within 12 hours. All blood cultures were negative. He was placed on intravenous penicillin with no significant improvement after 2 days. Intravenous clindamycin was added for synergy against the exotoxin-producing streptococcal organism, resulting in a slow resolution over the ensuing 3 weeks. He was then changed to oral penicillin to complete a 6 week course.

The patient is a divorced man, with two consecutive 5-year monogamous sexual relationships since then. He denied intravenous drug abuse, previous blood transfusions, or homosexual activity. An HIV test was obtained and was positive for the ELISA component. He was informed of this result, but was told that a Western Blot was required to confirm the diagnosis of HIV infection. The patient stated that, pending this test, he would voluntarily abstain from sexual activity with his partner, but would not inform her about the reason. The Western Blot was indeterminate, positive only for the p24 band. The patient was uninsured at the time, having just applied for health insurance through his new employer. He was offered HIV PCR quantification and p24 antigen detection assays but refused, fearing rejection of his health insurance application. He will undergo a follow up Western Blot in 3 months.
DISCUSSION. The pretest likelihood of disease clearly should influence the decision to obtain an HIV test. Although this patient had no clear HIV risk factors, the rapidity and severity of his soft tissue infection, his hypoalbuminemia and his relative lymphopenia drove the decision to obtain the test. We will present a review of the evidence behind factors which influence HIV testing, including both patient and test characteristics. In addition, we will discuss the justification behind public policy decisions about HIV infection as a reportable disease.

CLOSTRIDIUM SEPTICUM SEPSIS IN METASTATIC COLON CANCER, R. McCormick and B. Petty, Department of Medicine, Johns Hopkins Hospital, Baltimore, MD
LEARNING OBJECTIVES: 1) Illustrate the association between Clostridium septicum sepsis and malignancy of the GI tract, 2) Expand the differential diagnosis of air in the abdomen, and 3) Demonstrate the proclivity of C. septicum for invading and destroying metastases while sparing surrounding healthy tissue, decreasing tumor burden.
CASE: E.J. is a 68-year-old man with known adenocarcinoma of the colon, metastatic to the lungs and liver, previously treated with 5-FU and leucovorin, who presented with a three-day history of worsening pleuritic right-sided chest and upper quadrant pain, fevers and nausea. On the evening of admission, the patient's temperature was 34.3, his blood pressure 90/60, and his pulse 138. His abdomen was distended and tender, particularly in the right upper quadrant, but without rebound. His white blood cell count was 20,600, with 36% bands; an abdominal x-ray revealed radiopacity in the liver and possible pneumoperitoneum. An abdominal CT scan showed air around the liver and within the two largest hepatic metastases, without any signs of colonic perforation. This was in contrast to a routine CT scan performed three days earlier as an outpatient, which revealed multiple liver metastases, but no air. At 24 hours, blood cultures grew Clostridium septicum, and the patient's broad spectrum antibiotics were replaced with penicillin, to which the bacterium was sensitive. The largest of the patient's hepatic masses, initially replaced with air, developed an air-fluid level and was percutaneously drained, but failed to grow Clostridium. He was gradually advanced to a regular diet and discharged on day 12 on oral penicillin. A follow-up CT scan 37 days later revealed partial resolution of the air within the hepatic masses. He continues to do well clinically two months later, and has avoided surgery or further hospitalization, taking penicillin four times a day.

A FRIGHTENING CASE OF A NORMAL PSA: PERPLEXING SYSTEMIC ADENOCARCINOMA. HF Mechaber*, A Linquist ˆ, AJ Mechaber*, *Department of Medicine, University of Miami, Miami, FL; ˆArlington, VA.
LEARNING OBJECTIVES: 1) Recognize the importance of yearly digital rectal exam (DRE) in screening for prostate cancer, and 2) Realize a normal prostate specific antigen (PSA) does not exclude presence of prostate carcinoma.
CASE:A previously healthy 55-year-old white male, with prostate cancer in three first-degree relatives, was receiving yearly PSA screening. While the results were always normal, he had not undergone a DRE in three years. He presented with acute cauda equina syndrome secondary to a sacral mass. He was treated emergently with dexamethasone, and follow-up CT revealed an invasive prostatic mass and a liver without lesions. His PSA was normal at 1.63ng/ml. Prostatic biopsy revealed adenocarcinoma with a Gleason score of 10. Therapy was initiated with bicalutamide, leuprolide, and radiation, without response.

Only two months after diagnosis, he noted worsening fatigue, dyspnea, right upper quadrant abdominal and sacral pain. Over the ensuing week, his transminases markedly increased. An emergent abdominal CT was performed. Frighteningly, the CT revealed multiple liver lesions consistent with metastatic disease. The patient was admitted to evaluate these lesions and to treat hypercalcemia and anemia. Physical exam revealed pallor, hepatomegaly, decreased rectal tone, and an enlarged, firm, irregular prostate with nodularity. At this time, his PSA was <1.0ng/ml.

Given the rarity of metastatic spread to the liver by prostate adenocarcinoma, the patient was evaluated for a second primary tumor. Work-up was remarkable only for a significantly elevated CEA. Liver biopsy demonstrated poorly differentiated adenocarcinoma histologically identical to the original prostate sample. The poorly differentiated regions of both the prostate and liver specimens stained positively for CEA confirming prostatic origin of the liver metastases. Given the patient's poor response to the initial hormone and radiation therapy, he was begun on cisplatin and paclitaxel.
DISCUSSION: Adenocarcinoma of the prostate is the most common internal malignancy and the second leading cause of cancer deaths in American men. While prostate cancer frequently presents as localized disease, 20-45% of newly diagnosed patients present with metastatic disease; most commonly, in the pelvic nodes, axial skeleton, and brain. There have been few reported cases of liver metastases from prostate cancer, indicative of aggressive disease.

While a yearly PSA was checked and was always normal, no DRE had been performed in this patient for three years. Although DRE alone has its limitations, it may have helped detect his aggressive and highly unusual carcinoma earlier, prior to its metastatic course.

NECROTIZING VASCULITIS WITH PREDOMINANT HEPATOSPLENIC INVOLVEMENT PRESENTING AS FEVER OF UNKNOWN ORIGIN (FUO) IN AN ADOLESCENT MALE
Authors: Hemlata Moturi, MD, Rodrigo Erlich, MD, Asher Tulsky, MD, Adrianna Selvaggio, MD and Nalini Rao, MD, UPMC Shadyside, Pittsburgh, PA
LEARNING OBJECTIVE: 1) To recognize an unusual presentation of necrotizing vasculitis as FUO. 2) To recognize vasculitis as a possible and treatable cause of FUO.
CASE: Patient is a 16 year old Afro-American male with past history of seizures & without any history of recent travel, presented with recurrent fevers up to 104°F for 5 weeks, with a 15 lb. weight loss associated with myalgias, arthralgias, generalized weakness, burning sensation in legs progressing to numbness and increasing frequency of seizures. Workup done two weeks after initiation of symptoms including cultures ANA, RF, ACE level, sickle cell prep, monospot test and PPD was negative.
Exam we remarkable for BP – 150/95, temp –39.5°, tachycardia, cervical micro-adenopathy, and absent deep tendon reflexes. No objective signs of neuropathy or organomegaly, and negative cardio pulmonary exam. Labs significant for normocytic-normochromic anemia, normal WBC & platelets, no eosinophilia. Elevated GGT, mild elevated transaminases, hypoalbuminemia, hypocomplementemia, increased acute phase reactants – ESR, CRP, Ferritin; Cryoglobulins were positive, ANA – 1:80. Microscopic hematuria and mild proteinuria were present. Bone marrow aspirate and biopsy were unremarkable. Imaging studies showed mild hepato-splenomegaly with splenic infarcts, and small wedge shaped defect in left kidney was suggestive of infarct. Echocardiography was negative for vegetations. Patient continued to spike fevers with negative cultures. Splenectomy performed two weeks later, revealed necrotizing vasculitis and focal granulomatous response in medium sized vessels with splenic infarcts. Liver biopsy showed prominent perivenular sclerosis, congestion, and chronic portal inflammation. He improved significantly, on steroids and cytoxan.
DISCUSSION: Presenting as FUO in a young age with predominant hepatosplenic involvement, features of polyarteritis nodosa (necrotizing vasculitis in medium sized vessels, infarcts, negative ANCA) and microscopic polyangiitis (venular involvement and granulomatous response) are unique features of this case.

AN UNUSUAL DIAGNOSIS IN A CASE OF FATIGUE. David Mize, Department of Internal Medicine, Dartmouth Hitchcock Medical Center, Lebanon, NH.
LEARNING OBJECTIVES. 1) Recognize the symptoms of hypoglycemia, and 2) Diagnose and treat an insulinoma.
CASE. A 93 year-old woman had been complaining of fatigue and of feeling weak and shaky particularly in the early morning. Visiting nurses had checked her fingerstick blood glucose on two separate occasions when she was symptomatic and obtained readings of 72 and 108 mg/dl. On the morning of 4/3/98, a family member found her unresponsive and "frothing at the mouth". An ambulance was called and when it arrived at her house, her fingerstick was found to be 24 mg/dl. She was given 50% dextrose IV and taken to the local hospital. Her past medical history was significant for chromic complaints of fatigue for over 20 years, a pacemaker in 1972 for Mobitz II heart block, history of peptic ulcer disease, hypertension, and a hiatal hernia. Her only medicines were triamterene/hydrochlorothiazide and cimetidine. Following the administration of dextrose she rapidly regained consciousness. Her vital signs and physical exam were normal at the hospital. In the hospital her fasting blood sugars were consistently low and fasting insulin levels were consistently elevated. Factitious disease was ruled out. A CT scan of her abdomen revealed a large mass in the tail of the pancreas. The diagnosis of an insulin-secreting tumor was confirmed by biopsy of the mass. She failed medical management with diazoxide and later octreotide. She then underwent chemoembolization of her tumor which was also ineffective. Finally, she had a percutaneous injection of alcohol into her tumor with excellent results. Four months later she was seen in the endocrinology clinic and her blood glucose had remained in the normal range and her symptoms had resolved.
DISCUSSION. The symptoms of hypoglycemia can be divided into systemic symptoms and neuroglycopenic. The differential diagnosis of hypoglycemia includes among others, surreptitious insulin administration and insulin secreting tumors, which can be distinguished from each other on the basis of serum laboratory tests including C-peptide, pro-insulin, and sulfonylurea levels.

COMA INDUCED BY VALPROATE IN THE PRESENCE OF PHENOBARBITAL. R. O'Neil, C. Brands, Department of Medicine, Wright State University, Dayton, OH.
LEARNING OBJECTIVES: 1) Recognize valproate as a potential cause for hyperammonemia associated coma or lethargy, and 2) Recognize the interaction between valproate and other antiepileptic drugs leading to potentiation of hyperammonemia and mental status changes.
CASE: A 50 year old white female with a history of generalized tonic-clonic seizures was found in a lethargic state on the floor of her home. She had experienced many documented seizures within preceding months despite the use of phenobarbital (PB). Emergency room evaluation, including brain CT without contrast, electrolytes, CBC and blood and urine toxicology screens, was unremarkable. PB level was elevated at 61 mg/l (reference range 15-40). Her lethargy resolved and symptoms were attributed to a probable post-ictal state. Upon admission PB was discontinued and valproate (VPA) was started. Further laboratory evaluation showed normal thyroid, liver and renal function tests. By hospital day 3, the patient was observed to become progressively confused with fluctuating levels of responsiveness. EEG at this time showed diffuse, nonspecific irregular slowing and electrolytes remained normal. On the eighth day the patient was comatose. Liver and renal function tests remained normal. VPA level was 56 mcg/ml (reference range 50-125). PB level was 42 mg/l in spite of cessation of drug administration 8 days prior. Serum ammonia level was found to be markedly elevated at 338 micromol/L (reference range 11-32). EEG on hospital day 9 again demonstrated diffuse slowing without definitive epileptiform change consistent with toxic, metabolic encephalopathy. MRI revealed evidence of small vessel ischemic disease but no acute changes. On hospital day 10 she developed hyponatremia with a nadir of 122. Cerebral spinal fluid studies including cultures were unremarkable. VPA was discontinued on hospital day 12. Four days later the patient was noted to be alert and responsive with a return to her baseline mental status. At the time of recovery, her VPA level was <10 mcg/ml and PB was still within the therapeutic range at 17.4 mg/l. She was started on lamotrigine for seizure control.
DISCUSSION: Hyperammonemia associated lethargy and coma in the absence of liver disease has been described with the use of VPA, occurring most commonly when VPA is administered concomitantly with other antiepileptic drugs, most notably PB and phenytoin. VPA has also been shown to cause an increase in serum PB levels by impairing hepatic clearance. This patient demonstrated a considerable delay in clearance of PB (greater than 21 days, for an approximate elimination half-life of >200 hours) after VPA had been added. As has been the case in other reports, recovery was complete following discontinuation of VPA.

CETACAINE CAUSES LIFE-THREATENING METHEMOGLOBINEMIA. Jane E. O'Rorke M.D., Mark C. Henderson M.D., Department of Medicine, University of Texas Health Science Center, San Antonio, Texas

Methemoglobinemia (methb), the circulation of the oxidation product of hemoglobin in the blood, is a rare complication of topical anesthetic agents. Although potentially lethal, if recognized, this condition is easily reversible.

LEARNING OBJECTIVES: To be able to recognize and treat the clinical syndrome of methemoglobinemia.

CASE: We present a case of methb induced by Cetacaine spray used in preparation for a transesophogeal echocardiogram (TEE). Mr. P was an 81-year-old man undergoing TEE to evaluate mitral regurgitation. He was given 1 mg of intravenous midazolam and a liberal application of Cetacaine spray to his oropharynx. As the probe was introduced into his esophagus, Mr. P developed chest pain, hypotension and ventricular tachycardia. The study was aborted. One hour later he developed generalized cyanosis and pulse oximetry showed an oxygen saturation of 70% while breathing 100% oxygen by face mask. Electrocardiogram revealed acute ischemic changes. Mr. P was rushed to the cardiac catheterization lab for suspected evolving myocardial infarction. The patient was intubated, nitroglycerin and heparin were administered, and an intra-aortic balloon pump was placed. ABG revealed chocolate brown colored blood and a marked discrepancy between the measured pO_2 (partial pressure of oxygen) and the oxygen saturation. Methemoglobin level was 57% (normal <1%). Intravenous methylene blue, a cofactor that greatly increases the capacity of NADPH-methemoglobin reductase to reduce hemoglobin-bound iron back to the ferrous state (from the ferric state), was administered. The aim of this therapy is to restore hemoglobin's capacity to release oxygen to the tissues. A dramatic response to methylene blue treatment is shown in the table below:

TIME	pH	pO_2	O_2 sat	Methb level
Baseline	7.52	219	41%	57.0%
30 min	methylene blue administered			
50 min	7.52	386	N/A	49.0%
5 hrs	7.40	309	100%	5.4%

There was normalization of all hemodynamic parameters. The patient fully recovered.

DISCUSSION: Several therapeutic agents are capable of increasing the oxidation rate of heme, thereby overwhelming the capacity of red cells to maintain hemoglobin in the reduced state and thus impairing its capacity to carry oxygen. Medications associated with methb include nitrates, dapsone, phenacetin, lidocaine and antimalarial drugs. We report methb as a complication of the commonly used topical spray, Cetacaine, which contains benzocaine as the active ingredient. Methemoglobinemia should be a primary consideration in a patient with generalized cyanosis, chocolate brown-colored blood, and a normal or high pO_2 with a low O_2 saturation.

THYROTOXICOSIS DEVELOPING IN A PATIENT WITH ASYMPTOMATIC GRAVES' DISEASE AFTER AN UPPER RESPIRATORY TRACT INFECTION. JG Patterson and M Sheffield, Department of Medicine, University of Texas Southwestern, Dallas, TX.

LEARNING OBJECTIVES. 1) Distinguish between the clinical features of subacute thyroiditis and Graves' disease, 2) Diagnose the different types of thyroiditis, and 2) Manage the thyrotoxic patient.

CASE. A previously healthy 26-year-old Latin American woman presented with fever, sore throat and diarrhea to the emergency department (ED). Ten days prior to her presentation, the patient went to her physician complaining of sore throat and fever. She was diagnosed with pharyngitis and was given an injection of an unknown antibiotic. The patient did well for a week, but 2 days prior to admission she began to have throat pain on swallowing, fever, and diarrhea. Her symptoms progressed and she presented to the ED for further care. While in the ED, she had 6 watery, yellow stools. Her physical exam revealed fever to 38.8C, tachycardia with a rate of 130, a supine BP of 120/50, and no orthostatic changes. Generally, she was ill-appearing, anicteric and diffusely warm to the touch. She had no exophthalmos. Her oropharynx did not have either tonsilar enlargement or exudates. Her neck was exquisitely tender to touch over a diffusely enlarged thyroid that was firm and had bilateral bruits. Her exam, including abdominal and rectal exams, was normal. She was treated for pharyngitis and clostridium colitis with aggressive IV fluid resuscitation and appropriate antibiotics but did not improve. Subsequent laboratory evaluation showed a normal white cell count and differential, an elevated sedimentation rate at 40 and a TSH of <0.1 uIU/nl. Both T4 and T3 components were markedly elevated. A radioactive iodine uptake scan revealed 58% uptake, consistent with Graves disease. Her thyroglobulin level was measured and was normal. The patient's symptoms were controlled with propanolol and NSAIDS. She subsequently underwent radioactive iodine ablation.

DISCUSSION. Thyrotoxicosis can present in a patient as a mimic of more common diseases. Because of her fever, tachycardia and diarrhea, this patient was initially thought to have an infectious process. However, lab data revealed thyrotoxicosis. Her markedly tender thyroid suggested subacute thyroiditis as the cause, especially with her history of recent pharyngitis. However, her other clinical features and laboratory findings supported the diagnosis of Graves' disease. A literature search revealed two case reports of acute thyroiditis in the setting of Graves'. Additionally, a recent study reported the development of thyrotropin binding inhibitory antibodies in patients who had previously recovered from subacute thyroiditis suggesting a link between these immune mediated disease processes.

ALL THAT IS CAVITARY IS NOT TUBERCULOSIS. O. I. Ozkan, C. F. Maylath, Department of Internal Medicine, The Reading Hospital and Medical Center, Reading, PA

LEARNING OBJECTIVES: 1) Diagnose *Staphylococcus aureus* as a cause of pulmonary cavitary lesions in an immunocompromised patient. 2) Recognize the differential diagnosis of pulmonary cavitary nodules.

CASE: A 34 year-old obese, white female with a history of AIDS (CD4=133), intravenous drug abuse, asthma, and smoking presented with complaints of a productive cough with hemoptysis and fever for 5 days. Physical exam was remarkable for a temperature of 38.5°C, diffuse bilateral rhonchi, expiratory wheezing and multiple 2 cm by 2 cm skin abscesses on the abdomen. Chest roentgenogram revealed a right upper lobe thin-walled cavitary lesion. Computed tomogram of the chest confirmed two bilateral, thin-walled, upper lobe cavitary lesions. The patient was placed in respiratory isolation and empirically started on ampicillin/sulbactam and gentamicin. Acid-fast bacilli stain of the sputum was negative. The erythrocyte sedimentation rate was normal and the anti-nuclear antibody was negative. Non-invasive studies of the lower extremities were negative for thrombosis. However, 2 out of 2 blood cultures obtained on admission grew *Staphylococcus aureus*, sensitive to nafcillin. Trans-thoracic echocardiography revealed a tricuspid valve leaflet vegetation. On hospital day #4, the patient suffered a right pneumothorax felt to be caused by a rupture of one of the lung lesions; a chest tube was placed. Starting on hospital day #5, the patient temporarily required hemodialysis for acute renal failure secondary to infection and gentamicin toxicity. On hospital day #31, the patient was discharged to home to complete a 6-week course of intravenous nafcillin.

DISCUSSION: Secondary tuberculosis is the most common cause of apical cavitary lesions in the lung, especially in the immunocompromised patient. However, *S. aureus* causing pneumatoceles is the second most common infectious etiology of cavitary lesions. In addition, the complete differential diagnosis can be remembered using the mnemonic "CAVITY". Carcinoma (C) is most commonly bronchogenic and less commonly metastatic (2/3 of which are from the head and neck). Autoimmune (A) etiologies include collagen vascular diseases such as Wegener's granulomatosis, rheumatoid nodules as part of Caplan's syndrome, systemic lupus erythematosis, and sarcoidosis. Vascular (V) causes are most commonly embolic, both septic and non-infectious. In addition to the above infectious (I) etiologies, gram negative cocci (such as *Klebsiella spp.*), anaerobes, fungal etiologies (such as cryptococci, aspergillosis, coccidiomycosis), and parasitic causes are included in the differential. Traumatic (T) injury to the lungs, after hemorrhage or hydrocarbon ingestion, can also cause cavitary lesions. Finally, in young (Y) patients congenital abnormalities such as bronchogenic cysts must be considered.

HIV-ASSOCIATED PULMONARY HYPERTENSION N Peter, L Lynn, Department of Medicine, University of Pennsylvania, Philadelphia, PA.

LEARNING OBJECTIVES. 1) Diagnose pulmonary hypertension from its clinical manifestations and EKG findings. 2) Order the appropriate tests to look for an underlying cause of the disorder. 3) Recognize the association between pulmonary hypertension and HIV infection in women.

CASE. A 31 year old mother of two presented to the Emergency Department following a syncopal episode. She reported increasing exertional dyspnea over the preceding month, without paroxysmal nocturnal dyspnea or orthopnea, but did have intermittent pleuritic chest pain, lightheadedness, and a dry cough. She had not had fevers or weight loss. She had not had any prior significant illnesses; she did smoke ten cigarettes daily, take six alcoholic drinks weekly, and had used crack cocaine on two recent occasions. She had never used injection drugs and said that an HIV test a year earlier had been negative. Physical examination revealed a thin black woman in no distress. Blood pressure was 150/110. The pulmonic component of the second heart sound was loud, and she had a diastolic murmur at the base. Lungs were clear. The liver was enlarged and tender, but there was no lower extremity edema. Chest x-ray was normal. EKG showed right axis deviation, a large S-wave in lead I, and a Q-wave and inverted T-wave in lead III. She was admitted to the medical service, where an echocardiogram showed normal left ventricular function, right atrial dilatation, right ventricular hypertrophy, severe tricuspid regurgitation, and a pulmonary artery pressure of 80/30. A ventilation-perfusion scan was negative for emboli and shunt. ANA, complements, and rheumatoid factor were normal. A chest CT showed normal lung parenchyma, and hilar, mediastinal, and axillary lymphadenopathy. HIV antibody testing was repeated, and was positive. The patient was discharged on amlodipine, but became more dyspneic and was admitted to an intensive care unit for a trial of intravenous prostacycline. Despite aggressive treatment she suffered a respiratory arrest and died.

DISCUSSION: Patients with pulmonary hypertension (PH) generally present with exertional dyspnea, and syncope is reported in 15% of cases. EKG findings of right heart strain should prompt echocardiography. Chronic pulmonary emboli, collagen vascular diseases, and HIV infection should be considered as an underlying etiology. The course of the disease is more fulminant in HIV-positive patients, with a one-year survival rate of 51%, compared with 68% in HIV-negative patients. The virus itself is thought to be responsible for the vascular changes, but the mechanism has not yet been elucidated. PH may develop prior to any other complications of HIV. At this time, early vasodilator studies are indicated as drug therapy, oxygen and anticoagulation are the only treatments available. Further study is needed to determine whether or not treatment of HIV can alter outcome.

MALIGNANT HYPERTENSION AND ACUTE CARDIOMYOPATHY ASSOCIATED WITH PRESUMED IgA GLOMERULOPATHY. A. Peterson, Department of Medicine, Dartmouth-Hitchcock Medical Center, Lebanon, NH.
LEARNING OBJECTIVES. 1) Recognize the potential for acute glomerulopathy to cause malignant hypertension, and 2) Diagnose IgA nephropathy.
CASE. A twenty-seven year-old white male was transferred from a community hospital with hypertension, congestive heart failure, and hematuria. Two months prior to admission the patient presented with fatigue, malaise, and anterior chest "congestion" which was intermittent, non-exertional, and worse with lying flat. Exam revealed a temperature of 101 degrees, blood pressure (BP) 140/80, and clear lungs; a chest radiograph (CXR) was normal, and he was treated with azithromycin. He developed a productive cough; repeat CXR showed borderline cardiomegaly with increased interstitial markings, and he completed a second course of antibiotics. His dyspnea and orthopnea progressively worsened, and he began sleeping in a chair. Five days prior to admission the patient developed nausea, emesis, dark-colored urine, and confusion. On presentation he was afebrile with BP 199/130 and heart rate 108. Physical exam revealed retinal hemorrhages but normal mentation. The jugular venous pressure was normal but there were diffuse pulmonary rales. Laboratory studies revealed normal serum electrolytes, BUN of 14 milligrams per deciliter (mg/dL) and creatinine 1.3 mg/dL; the urine had 3+ protein, 5-10 RBC's per high-power field, and fine granular and hyaline casts. CXR was consistent with congestive heart failure, and electrocardiogram revealed left ventricular hypertrophy. Echocardiogram demonstrated a dilated and hypertrophic left ventricle with a systolic ejection fraction of 30%. A nitroglycerin drip was started and the patient was transferred to our institution. On arrival his BP was 163/91 and exam revealed an S4 with occasional bibasilar rales. 24-hour urine protein, plasma complement levels, and urinary metanephrines were all normal; antineutrophil cytoplasmic antibodies, anti-DNA antibodies, and antistreptolysin O titers were negative. The serum IgA was elevated at 424 mg/dL (normal, 69-309). Magnetic resonance angiogram revealed patent renal arteries. With aggressive medical management of his hypertension and cardiomyopathy, the patient's congestive heart failure resolved, and repeat echocardiogram revealed a significantly improved ejection fraction of 50%. Because of his normal renal function and the resolution of his proteinuria, renal biopsy was not performed.
DISCUSSION. The clinical course in this case is consistent with underlying essential hypertension accelerated to a malignant level by acute glomerulonephritis. The mechanisms by which renal parenchymal disease cause hypertension, while not fully understood, likely involve abnormal salt and water retention as well as activation of the renin-angiotensin system. IgA nephropathy is the most common form of glomerulonephropathy worldwide. While this patient's age, sex, associated respiratory tract symptoms, and elevated serum IgA are consistent with this disorder, formal diagnosis requires renal biopsy revealing diffuse mesangial deposition of IgA. Although this condition may be benign, it can lead to both acute renal dysfunction and slow progression to end-stage renal failure.

TUBERCULOUS MESENTERIC LYMPHADENITIS. Nancy Phifer and Edward N. Robinson, Jr. Internal Medicine Program, Moses Cone Hospital, Greensboro, NC.
LEARNING OBJECTIVES: Recognize that for individuals who present with fever or abdominal symptoms who originate from or frequently travel to regions of high tuberculosis prevalence, tuberculous mesenteric lymphadenitis should be considered in the differential diagnosis.
CASE: A 23 year-old woman with a viable 5 week intrauterine pregnancy presented with fever of three weeks duration. The patient was a native of India and had visited there two months prior to presentation. Past medical history was significant only for an appendectomy 5 years earlier. She was evaluated as an out-patient for fever and treated with Amoxicillin for a presumed urinary tract infection. A PPD was highly reactive with induration and vesicle formation. Her fevers persisted despite treatment and she was admitted to the hospital for further work-up. Her exam was remarkable for temperature of 103.4 but was otherwise normal. She had a microcytic anemia and mildly elevated AST and ALT (53 and 57 respectively) but normal white cell count and chemistries. Blood, urine and stool cultures, stool studies for ova and parasites and C. difficile, hepatitis serologies, HIV and malaria smears were all negative. CXR and abdominal ultrasound were normal. The patient was started on Ceftriaxone to cover the possibility of Salmonella typhi (typhoid fever). An MRI of her abdomen revealed a small area of fluid in the right paracolic gutter and retroperitoneal fat adjacent to the right psoas muscle consistent with an inflammatory process. The patient was transferred to Stanford University Hospital since she and her husband were in the process of relocating at the time of admission. Her fevers persisted despite Ceftriaxone. An abdominal ultrasound was repeated and a small mass was identified in the right lower quadrant. Aspiration of the mass revealed a necrotic lymph node with acid-fast bacilli. The patient was started on anti-tuberculosis medications.
DISCUSSION: Tuberculous mesenteric lymphadenitis is an unusual presentation of TB. Patients can present with symptoms that mimic gastrointestinal pathology such as peptic ulcer disease, appendicitis, and diverticulitis. This patient presented only with fever of unknown origin. Clinicians need to be aware of this entity and have a high index of suspicion in patients from areas where tuberculosis is endemic.

A CASE OF NEPHROLITHIASIS IN A PATIENT WITH ANURIA ON CONTINUOUS AMBULATORY PERITONEAL DIALYSIS PRESENTED WITH FLANK PAIN. A.Persaud, S.Swaminathan, F.Haddad, S.Reichert. Department of Medicine, Englewood Hospital and Medical Center, Englewood, NJ.

LEARNING OBJECTIVE(S): To recognize nephrolithiasis as a possible etiology of flank pain in anuric patients.
CASE: A 33 year old female patient with systemic lupus erythematosus (SLE) for 10 years and end stage renal disease (ESRD) on continuous ambulatory peritoneal dialysis (CAPD) for 7 years and anuria for 1 year presented to emergency department with intermittent flank pain for the past 2 months prior to admission. The pain was sharp in nature and radiating to the groin. She denied fever, nausea, vomiting, vaginal discharge, diarrhea or constipation. Patient was diagnosed with secondary hyperparathyoidism 1 year prior to admission that was treated by para-thryroidectomy. Subsequently she has remained normocalcemic. In the emergency department she was afebrile, had a pulse of 108 and blood pressure 115/70. Abdominal exam revealed right costophrenic tenderness. A peritoneal dialysis catheter was noted not to be erythematous or tender around the site of insertion. Pelvic and rectal exam was refused by the patient.
Initial laboratory results showed mild anemia, normal leukocyte count, amylase and lipase were normal. Serum pregnancy test was negative. Liver function tests were normal. Computed tomographic scan of the abdomen and pelvis showed a 9 mm right ureteropelvic junction calculus without any dilatation of right ureter.
Hospital course: Patient was admitted for pain control as well as for urological evaluation. On second day of hospitalization she underwent cystoscopic removal of the stone and placement of a stent. Postoperatively she remained pain free and did not require any further pain medications.
Subsequent analysis of the stone revealed Ca oxalate 95% and Ca phosphate 5%. The patient was followed up in primary doctor's office and since then she remained asymptomatic.
DISCUSSION: Nephrolithiasis should be considered in patients who present with flank pain even though they are anuric. Classic signs and symptoms should not be overlooked in this group of patients. Treatment should include aggressive pain management along with appropriate surgical intervention. In a study of 186 patients with ERSD who were treated with CAPD, 10 patients formed renal stones. Of the 10 patients who had renal stones, 7 of the stones were examined by x-ray diffraction and 5 of the 7 stones were composed of calcium oxalate monohydrate. From this study it was suggested that 2.7% of patients on CAPD are prone to renal stones.

PNEUMONIA WITH ACUTE HEMOLYSIS. GA Prasad, EL Lepgold , MA Gennis .Division of General Internal Medicine, Sinai Samaritan Medical Center, University of Wisconsin Medical School, Milwaukee, WI.

A 39 year old previously healthy Black female presented to the office with a 7 day history of dry cough, dyspnea and myalgias. After a chest X-ray revealed evidence of left lower lobe consolidation, she was started on oral Clarithromycin.4 days later, she presented to the office with increasing dyspnea, extreme weakness and fatigue. Examination revealed significant pallor, icterus, dry mucous membranes and orthostasis, without acrocyanosis or organomegaly. Laboratory evaluation revealed significant anemia (Hb:5 gm/dl), reticulocytosis (corrected reticulocyte count 4.5%), unconjugated hyperbilirubinemia (2.5 mg/dl),and an elevated LDH (3235 u/l).The haptoglobin level was low (< 6 mg/dl) and the Direct Coomb's Test was positive for complement (C3) but negative for Immunoglobulin G. The cold agglutinin titer was raised to 1:4096 (normal < 32).The patient was managed with transfusion of prewarmed red cells, maintaining a warm ambient temperature and antibiotics. Her hematocrit stabilized and hemolysis gradually resolved. Complement fixation antibody titer against M.pneumoniae was elevated.

Cold Agglutinin mediated hemolysis is a characteristic but uncommon feature of M.pneumoniae infection. Elevated titers of cold agglutinins have been reported in 33-76% of patients with M.pneumoniae infection, but clinically significant hemolysis is rare. Hemolysis usually is transient and coincides with recovery from the underlying pneumonia and the peak cold agglutinin titer. However significant intravascular hemolysis with hemoglobinuria and acute tubular necrosis leading to acute renal failure and death have been reported. Cold agglutinins are IgM antibodies which are active in low ambient temperatures (4-18 degrees Centigrade),and bind to red cell membrane antigens (I and i). They then activate the classic complement cascade and cause hemolysis. These antibodies cause complement activation in the peripheries, as the skin temperatures are lower than the core visceral temperatures. The low ambient temperatures in winter likely accelerated this process. These antibodies are polyclonal , unlike those associated with lymphoid neoplasms. Treatment is primarily supportive, with transfusion of prewarmed red cells, fluids, maintaining a warm ambient temperature. Steroids have been tried in some cases but no definitive studies have been conducted to assess their efficacy. Infectious Mononucleosis, Legionella ,and Cytomegalovirus infections can also lead to similar manifestations.

SHORTNESS OF BREATH IN A 43 YEAR-OLD WOMAN. AB Reisman, Johns Hopkins University School of Medicine, Baltimore MD
LEARNING OBJECTIVES: 1) Recognize clinical features of recurrent acute rheumatic fever (ARF), and 2) Educate ARF patients on the need for prompt treatment of sore throats.
CASE: A 43 year-old woman with hypertension and a childhood history of ARF presented with shortness of breath and ankle pain and swelling. Two months earlier she was briefly admitted with chest pain and dyspnea and was diagnosed with congestive heart failure due to diastolic dysfunction. Echocardiogram at that time was notable for a dilated left atrium and left ventricular hypertrophy. Over the next several months, she became unable to walk more than half a block without resting. During her second admission, she reported sharp chest pains that occurred both with exertion and at rest. Physical examination was notable for a fever, multiple cardiac murmurs, and joint warmth and swelling. Workup included negative blood cultures, positive stress thallium with lung uptake, restriction on pulmonary function tests, mild pulmonary edema on high-resolution computerized tomography, and a rheumatic panel notable for an ESR of 80. A repeat echocardiogram was significant for new multiple valvular abnormalities. Further history revealed a sore throat and fever three months earlier for which she did not seek medical attention. During the admission, the ankle arthritis abated while pain and swelling developed in the wrists, knees and shoulders. Antistreptolysin O and antihyaluronidase were elevated. Throat culture was positive for group A streptococcus. The diagnosis of recurrent rheumatic fever was made based on the Jones criteria. The patient was treated with high-dose salicylates and penicillin with prompt resolution of symptoms.
DISCUSSION: Patients with a history of ARF who are exposed to rheumatogenic strains of streptococcus have an increased risk of recurrence. Although the risk decreases with time, such patients should be educated on the need to seek medical attention for sore throats.

GENITAL ULCERS AND KNEE SWELLING-EXPANDING THE DIFFERENTIAL DIAGNOSIS. A Rothschild, D Muller, Division of General Internal Medicine, Mount Sinai Medical Center, New York, NY.
LEARNING OBJECTIVES. 1) Diagnose and treat chondrocalcinosis, 2) Diagnose and treat Behcet's, 3) Distinguish among the different etiologies for joint swelling and mucosal ulcerations.
CASE. A 68 year old woman presented to the Emergency Room with bilateral knee pain and oral and genital sores. The patient noted knee pain and swelling three weeks prior to admission, followed one week later by the onset of painful ulcerations in her mouth and genitalia. She denied any history of fever, rash, or trauma, had not been sexually active in 25 years and had no prior history of sexually transmitted disease. She had experienced a similar episode in the past that had resolved spontaneously. Her past medical history was significant for hypertension, coronary atherosclerosis, congestive heart failure and a remote deep vein thrombosis. On presentation the vital signs were stable and the patient was afebrile. There were cotton wool spots bilaterally on ophthalmologic exam. Several 2-3 cm ulcers were noted on the buccal mucosa and tongue. There was no lymphadenopathy, rash or hepatosplenomegaly. The knees were warm and tender, with bilateral effusions and exquisite pain limiting range of motion. There was a 0.5 cm ulcer on the left labia minorum. Lab values were significant for a Hgb 10.2 gm%, WBC 11,200, Plt 512,000, and an ESR 129 mm/hr. The patient initially refused arthrocentesis and was treated with Nafcillin and Ceftriaxone. On hospital day (HD) #2 arthrocentesis yielded 25cc of turbid yellow fluid with intracellular positively birefringent crystals. Cell counts were WBC45,000 (93% PMNs) and RBC 7,500, with a glucose of 49 mg% and protein 4.7 gm%. Antibiotics were stopped and the patient was treated with subcutaneous ACTH and oral colchicine. Lab evaluation included TSH, PTH, RPR, ACA IgG and IgM, ANA, Anti-DS DNA, Rheumatoid Factor and complement levels, all of which were normal. Chlamydia antigen, Gc probe and HSV cultures of the vaginal ulcers were negative. The C reactive protein was 13.8 mg% (0-0.8). Knee swelling and pain resolved by HD #2 but the oral and genital ulcers persisted, with new oral ulcers forming on HD #5. Prednisone was started, with improvement in the ulcers noted by HD #9.
DISCUSSION. A review of the literature from 1966 – 1998 revealed only one case report of chondrocalcinosis and Behcet's. Despite that, Behcet's should still be considered in the differential diagnosis of mucosal ulcers that accompany joint swelling. Ophthalmologic exam, culture of lesions, and biopsy can help differentiate it from other causes.

RECURRENT SYNCOPE: WHEN THE ROUTINE WORKUP IS NOT DIAGNOSTIC. L. Rucker, Department of Medicine Jacobi Medical Center, Bronx, NY
LEARNING OBJECTIVES: 1. Prioritize diagnostic testing after recurrent syncope, based on physical exam and results of preliminary testing. 2. Explore new diagnostic modalities when current strategies are not helpful.
CASE: An 87 year-old woman with a history of coronary artery bypass surgery, hypertension, Type II diabetes and hyperlipidemia presented with her fifth episode of syncope in two years. She felt light-headed for 30 seconds while crossing the street and then lost consciousness, lacerating her scalp when she fell. No tonic-clonic movements, incontinence, or tongue-biting occurred. Medications included hydrochlorothiazide, glipizide, simvastatin, aspirin and atenolol (prescribed after a previous syncopal event). Physical exam was significant for no orthostatic changes, bilateral carotid bruits, 2/6 systolic murmur at the base, and a non-focal neurologic exam. Electrocardiogram (ECG) showed left ventricular hypertrophy. Chemistries were normal except for glucose of 263. Previous workup at two hospitals showed normal head computerized tomography and magnetic resonance imaging, normal electroencephalogram, and high grade left carotid stenosis. She had a few premature atrial and ventricular beats on Holter monitor. Echocardiogram showed normal ventricular function, tricuspid regurgitation, a small atrial septal defect and a pulmonary arterial pressure of 80 millimeters mercury. Cardiology and neurology consultation failed to define the etiology. She remained on 24-hour telemetry for six days without syncope or dysrhythmia. She was a poor candidate for tilt table testing, programmed electrical stimulation (PES) and patient activated transtelephonic ECG recording without memory loop. A patient-activated intermittent loop recorder was implanted. She had a syncopal episode hours later and third degree heart block was diagnosed.
DISCUSSION: Potential causes of syncope can be identified by history and physical exam in more than 50% of patients. This patient had previous coronary artery bypass and had a systolic murmur. These findings, added to her normal neurologic evaluation and absence of likely causative drugs, suggested a cardiac cause of syncope. Attributing syncope to dysrhythmia is difficult because often symptoms resolve prior to testing. Rhythm abnormalities found on testing can confirm a diagnosis only if coincident with syncope. Her carotid stenosis precluded tilt testing and PES, and patient activated non-loop monitoring was deemed too complicated and low yield. A new diagnostic modality was pursued. Implantable patient activated loop recorder demonstrated the causative dysrhythmia. She had a pacemaker inserted and remains gratefully conscious.

DIAGNOSTIC DILEMMA: PANCREATIC CANCER VERSUS CHRONIC PANCREATITIS. J. Sahni. University of Pittsburgh Medical Center Shadyside. Pittsburgh, PA. LEARNING OBJECTIVE: To understand the difficulty in differentiating between pancreatic cancer and chronic pancreatitis. CASE: A 72 year-old woman presented with jaundice, abdominal pain, frequent loose light colored bowel movements, generalized pruritus, and malaise. On questioning, she admitted to a 15 pound weight loss over the past two months. Two months ago she had also been diagnosed with new onset type 2 diabetes mellitus. Social history was significant for 50-pack year cigarette use and for heavy alcohol use, although she had abstained for 20 years. Family history was significant for brain tumor and lung cancer in her sister and brother, respectively. Exam revealed a thin, markedly jaundiced woman without lymphadenopathy or abdominal findings. Her laboratory values included: alkaline phosphatase 677 U/L, AST 296 U/L, ALT 354 U/L, total bilirubin 7.9 mg/dL, direct bilirubin 5.1 mg/dL. An abdominal CT scan revealed biliary and pancreatic duct dilatation with slight inhomogeniety noted in the pancreatic head. To further clarify, an ERCP was performed and showed a common bile duct stricture consistent with extrinsic compression by a pancreatic mass. A subsequent pancreatic angiogram demonstrated a slight irregularity of the gastroduodenal artery. Laparotomy was performed and multiple pancreatic biopsies were taken as well as cholecystectomy and choledochojejunostomy undertaken. Pathology revealed no malignancy, but instead extensive parenchymal fibrosis consistent with chronic pancreatitis. DISCUSSION: The differentiation of chromic pancreatitis from pancreatic cancer can be difficult as many clinical features overlap. In 5-10% of patients who are operated on for presumed pancreatic cancer, chronic pancreatitis is found instead. Future research should be aimed at finding better ways to differentiate between these two entities.

LATERAL MEDULLARY SYNDROME PRESENTING AS ACUTE LABYRINTHITIS J. Sahni, Gary Tabas. University of Pittsburgh Medical Center Shadyside. Pittsburgh, PA. LEARNING OBJECTIVE: To recognize brainstem pathology presenting as peripheral disease. CASE: A 61 year-old man presented with nausea, dizziness, and vertigo exacerbated by change of position. Exam revealed only right gaze horizontal nystagmus and mild right dysmetria on finger-to-nose testing. Right pupil was irregular since cataract surgery. CT and MRI of the head revealed old bilateral cerebellar infarcts; bilateral PICA vessels were poorly visualized by MRA. Transcranial dopplers revealed mildly decreased flow in both vertebral arteries. With obvious pathology excluded, the patient was discharged on meclizine and compazine for symptomatic relief with the presumptive diagnosis of acute labyrinthitis versus benign positional vertigo. An outpatient otolaryngology exam was unrevealing. The patient returned to the hospital five days later with continued vertigo, and new complaints of headache, facial paresthesias, hiccups, hoarseness, dysphagia, and ataxia. Exam demonstrated an incomplete Horner's syndrome, horizontal nystagmus, facial numbness, vocal cord paralysis, dropped palate, and dysmetria on the right. He also had upper extremity sensory loss on the left as well as a markedly ataxic gait. Repeat MRI showed a new right dorsal and lateral medullary infarct. The patient was started on a heparin drip and clopidogrel. Over the course of three days, he greatly improved and was discharged to a rehabilitative facility. DISCUSSION: Lateral medullary syndrome, also known as Wallenburg syndrome, is usually due to occlusion of the vertebral artery, or less often, the posterior inferior cerebellar artery as was likely the case with this patient. When it results in an infarct of the inferior part of the cerebellum, the patient may experience sudden vertigo, nausea, vomiting, ataxia, and nystagmus–a picture that mimics acute labyrinthine disorder–thereby challenging the clinician's ability to differentiate from central and peripheral neuropathology.

TETANUS: KNOWING THE RISKS AND HALLMARKS TO MAKE THE CLINICAL DIAGNOSIS. S. Samii, Department of Medicine, University of Pittsburgh Medical Center, Pittsburgh, PA. LEARNING OBJECTIVES: 1) To recognize the clinical clues in making the diagnosis of tetanus, and 2) To recognize populations at risk for tetanus. Case: A 53 year old intravenous drug user presented to the emergency room with complaints of bilateral neck pain and stiffness for the past 10 hours. The pain started after injecting cocaine into her right deltoid muscle earlier that day. She had been using this site, along with her trapezius and neck muscles for the past four months. Acetaminophen and ibuprofen provided no relief of pain. She denied fever, chills, other muscle aches, photophobia or headaches. On physical exam vitals were normal with the exception of a blood pressure of 180/84. She had mild decreased range of motion of her neck in all directions but most prominent with flexion forward. Initially these physical findings were treated with orphenadrine 60 mg intramuscularly with no relief in symptoms. Diphenhydramine, ketorolac and morphine sulfate were later used intravenously, also with no improvement in symptoms. A few hours later the patient was found to be in moderate distress secondary to the neck stiffness and pain. Physical exam at this time was remarkable for trismis (rigidity of her masseter muscles bilaterally). Her oral aperture was less than 2 cm.
Discussion: Tetanus is a clinical diagnosis. The classic findings include trismus, dysphagia, and sustained contracion of the facial muscles causing risus sardonicus or grimace. The differential diagnosis includes strychnine poisoning, dystonic drug reactions, hypocalcemic tetany, meningitis, and abscess. Tetanus is usually thought to be a preventable infection given the widely available vaccine and booster. At obvious risk are those not having exposure to childhood vaccinations such as populations from developing countries and relatively isolated population in the U.S. such as the Amish. However, there has been 2 classes of people that are showing increased risk of tetanus infection in the United States as well as worldwide: the elderly, and intravenous drug users.

WHEN LARGE FINGERS MEAN MORE THAN LARGE GLOVES: AN UNUSUAL CASE OF HYPERTROPHIC OSTEOARTHROPATHY. A. Schriber, Department of Medicine, University of Chicago Hospitals, Chicago, IL.
LEARNING OBJECTIVES. 1) Recognize the clinical presentation of hypertrophic osteoarthropathy (HOA), 2) Understand how laboratory and radiographic studies confirm the clinical diagnosis, and 3) Learn how cirrhosis causes HOA.
CASE. A 24 year-old woman presented in September 1998 with progressive lower extremity pain. She had a history of autoimmune hepatitis that required orthotopic liver transplantation in 1992. This was complicated by chronic rejection refractory to intense immunosuppression. She had no previous arthritis. Examination showed upper extremity clubbing, and diffuse distal lower extremity tenderness with bilateral knee effusions. Synovial fluid analysis showed 240 RBCs/ml and 12 WBCs/ml with no crystals and negative culture. Radiographs of the legs showed periostosis of both tibial diaphyses and normal joints. Chest radiography was normal.
DISCUSSION. HOA is a syndrome consisting of clubbing and periostosis, and usually is manifested by pain in the lower limbs. Joint effusions are common and are noninflammatory. Radiographs show periosteal reaction, and bone scans can be confirmatory. Recent research implicates right-to-left shunting of blood as the cause of HOA. In the normal state megakaryocytes and platelet clumps produced in bone marrow are processed as they pass through the lungs such that fewer are delivered to the systemic arterial circulation. In states associated with HOA, shunted blood bypasses the lungs and these blood elements are delivered to the systemic circulation, where in the extremities they induce clubbing and periosteal new bone formation. In cirrhosis, blood shunting occurs through small pulmonary A-V malformations. Treatment options for HOA range from analgesics to radiation therapy but in cases associated with cirrhosis, liver transplantation is curative. Less than five case reports describe HOA in liver transplant recipients.

RIFAMPIN INDUCED ACUTE RENAL FAILURE. Erik Schuls, Department of Medicine, Medical University of South Carolina, Charleston, SC.
LEARNING OBJECTIVE: The recognition and diagnosis of rifampin induced acute renal failure.
CASE: A 25-year old male presented with an acute onset of fever, chills, and rigors thirty minutes after a single discontinuous dose of rifampin. Five months prior, he was found to have a positive tuberculin skin test. Having failed therapy with isoniazid because of gastrointestinal intolerance, for the past four months he had been maintained on a twice weekly regimen of rifampin. Three weeks prior to presentation, he stopped taking the rifampin because of flu-like symptoms. On the day of presentation, he was seen by his physician who suggested that he restart the rifampin; blood work that morning revealed WBC 7.8, Hgb 16, Hct 48, Plt 226. Later that day, he ingested a single 300 mg dose of rifampin, and within thirty minutes developed fever, chills, rigors, and felt severely ill. In the ER, he was febrile at T105, BP 110/60, P90, R18. Physical examination was unremarkable. Labs revealed WBC 1.3 with 75 polys, and 20 bands, Hgb 11, Hct 33, Plt 111, with 2+ RBC fragments on manual smear. SMA-7 was normal, Tbili 2.5, LDH 3581, D-dimer >8, PT 13, PTT 26. Urinalysis revealed 9 RBC, 100+ proteinuria, and a benign sediment. Blood and urine cultures were negative. CSF examination was normal. Within 48 hours, oliguric renal failure ensued. Workup revealed no urine eosinophils, a fractional excretion of sodium of 4.2, and normal ASO, ANA, and ANCA titers. Serum creatinine peaked at 11. Renal ultrasound showed mildly enlarged kidneys without hydronephrosis. Gallium scan demonstrated abnormal radiotracer in both kidneys consistent with interstitial nephritis. He required one dialysis treatment. Recovery of renal funciton took place after ten days of oliguria.
DISCUSSION: Rifampin induced acute renal failure (RIARF) manifests abruptly, usually within an hour of reinstitution of prior rifampin therapy. The rifampin-free period needed can be as little as two weeks, and as long as one year. The mechanism of renal failure is thought to be the result of a type II hypersensitivity reaction involving anti-rifampin antibodies with complement-fixing capability, which upon re-exposure to rifampin, cause autoimmune intravascular hemolysis. Oliguric renal failure ensues as a result of immune complex deposition in the kidneys. The diagnosis should be suspected in a patient taking rifampin who presents with hemolysis and renal failure. Although renal biopsy usually reveals tubulointerstitial nephritis, the triad of fever, rash, and eosinophilia or eosinophiluria is uncommon in RIARF. Anti-rifampin antibodies may be detected in the serum but is not necessary to make the diagnosis. Renal function recovers upon withdrawal of rifampin after a short period of oligoanuria.

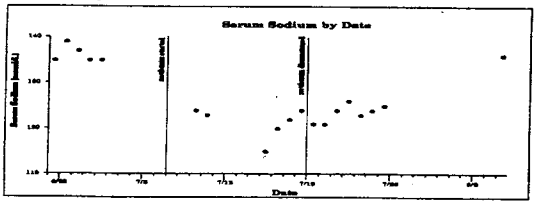
HOW DRY I AM: NEUROLEPTIC MALIGNANT SYNDROME (NMS) LEADING TO SEVERE HYPERNATREMIA. C Seibert, Dept of Medicine, University of Wisconsin, Madison.
LEARNING OBJECTIVES: 1) Identify the common features of NMS, and 2) Recognize the importance of fluid intake in cognitively impaired patients who are ill.
CASE: A 43 year-old man with a history of traumatic brain injury, depression, and delusional disorder was admitted to the psychiatric service with 2 weeks of increasing somnolence and worsening urinary incontinence. Admission medications included an antipsychotic agent, Olanzapine, which had been initiated 1 month prior to admission. A medicine consult was obtained due to increasing lethargy and fever.
On examination, the patient was difficult to arouse and diaphoretic. Temp was 100.9, BP was 180/87. Mucous membranes were dry and neck veins were flat. Neurologic exam was significant for extremity rigidity and hyperflexia. Laboratories revealed Na 176, K 3.1, Creatinine 1.8, CK 1951 (normal MB fraction) and WBC 12.3. Head CT showed no acute changes and LP was normal. EKG showed no ischemic changes.
The patient was diagnosed with NMS. Psychiatric medications were discontinued. Calculated free water deficit was 12L. Saline solutions were used to replete volume deficits. Na dropped from 176 to 165 over the first 48 hours and returned to normal range within the next 5 days. Bromcriptine was initiated. At discharge, more than 2 weeks later, the patient's mental status had improved to baseline, vital signs were normal, and muscular rigidity and all laboratory abnormalities had resolved. With significant prompting, the patient maintained his fluid intake to keep his Na in the normal range. After short-term rehabilitation, he returned to his group home.
DISCUSSION: NMS is characterized by autonomic dysfunction (including labile blood pressure, diaphoresis and urinary incontinence), muscular rigidity, hyperthermia, an elevated CK and leukocytosis. Elevated blood pressures may mask profound volume deficits. Insensible water losses should be considered and, if found, aggressively repleted in order to prevent hypernatremic CNS complications.

NEUTROPENIC ENTERCOLITIS ASSOCIATED WITH THE USE OF AZATHIOPRINE FOR RHEUMATOID ARTHRITIS. G. Sharma, D. Sweet, Department of Internal Medicine, University of Kansas School of Medicine-Wichita, Wichita, KS.
LEARNING OBJECTIVES. 1) Recognize the clinical presentation of neutropenic enterocolitis in a patient on immunosuppressive therapy, and 2) Learn about newly recognized complication of azathioprine.
CASE. This was a fifty- eight-year old man with a five-year history of rheumatoid arthritis. He was being treated with Azathioprine and Plaquenil. He was admitted to the hospital with painless bleeding per rectum. On the following day, he developed severe right lower quadrant pain. His temperature was 101°. The abdominal examination revealed generalized tenderness with guarding in the right lower quadrant and diminished bowel sounds. CT scan of the abdomen showed marked thickening of the ascending colonic wall. Some free fluid was seen in the right pelvis. On admission his WBC count was 0.4, Hb 7 gm, and platelets 27,000. Blood cultures showed growth of Streptococcus salivarius. Azathioprine and Plaquenil were stopped. The patient was treated with steroids, granulocyte colony stimulating factor, and antibiotics. Subsequently, he developed respiratory distress and hypoxia. His chest X-ray showed an infiltrate on the left side. His respiratory status worsened; he died on the 5th hospital day. On autopsy, he was found to have marked edema of the bowel wall and hemorrhagic infarction of the caecum. Inclusion bodies were present in the lungs; tissue culture was positive for cytomegalovirus. The colon culture was negative for the virus.
DISCUSSION. Neutropenic enterocolitis has previously been reported as a complication of chemotherapy for childhood leukemia and of solid and hematologic malignancies in adults. A case of enterocolitis associated with use of sulfasalazine for rheumatoid arthritis has been reported. To our knowledge, this is the first case report of neutropenic enterocolitis resulting from Azathioprine-induced bone marrow suppression. The diagnosis should be suspected when a neutropenic patient complains of abdominal pain. CT Scan findings of bowel wall thickness, especially on the right side and free fluid in the pelvis are suggestive of enterocolitis. Early diagnosis, withdrawal of the offending agent and medical management may allow resolution of this potentially life threatening condition.

ACUTE PULMONARY EMBOLUS PRESENTING AS HEPATIC FAILURE. H David Seidel, Eric Warm. University of Cincinnati College of Medicine, Cincinnati, Ohio.
LEARNING OBJECTIVES. 1) Recognize ischemic hepatitis as a cause liver failure in the acute setting. 2) Expand the differential of causes of ischemic hepatitis to include pulmonary embolus, in addition to the more traditional etiologies.
CASE. Ischemic hepatitis, also known as "shock liver," is described as an acute hemodynamic event that causes abnormalities in liver histology and function. Despite many small studies, the incidence is unknown and thought to be underdiagnosed and underestimated. Many causes have been described that can lead to this type of liver dysfunction. We present a case of ischemic hepatitis believed to be the result of an acute pulmonary embolus. A 78 year old male with a past medical history of type 2 diabetes mellitus presented with four days shortness of breath and lower extremity edema. He had also been experiencing a cough with clear sputum, difficulty laying flat, and darkened urine. In the emergency department the patient was oriented only to self. Physical exam revealed a normotensive but tachycardic male with anasarca, 10cm of jugular venous pressure above the sternal angle, coarse rales, hepatosplenomegaly, and asterixis. Initial labs were significant for leukocytosis, thrombocytopenia, acute renal failure, and coagulopathy. His hepatic profile showed an alkaline phosphatase of 422, t/d bilirubin 3.5/6.8, AST 6918, ALT 2792. Baseline labs one week prior were normal. A RUQ ultrasound showed normal liver and spleen, and normal hepatic and portal systems by Doppler exam. Other studies ruled out viral, alcoholic, cholestatic, or drug-induced causes of the hepatitis. An echocardiogram demonstrating evidence of right heart failure led to a V/Q scan which showed a high probability of pulmonary emboli in the right apex and left upper lobe as well as infarcted bases. The patient received supportive care and routine therapy for thromboembolic disease, and recovered.
DISCUSSION. Ischemic hepatitis has been described as the rapid elevation of the serum transaminases to more than 20 times normal secondary to decreased cellular perfusion. The ALT and AST levels, as well as the alkaline phosphatase and total bilirubin levels, can rise as rapidly as 30 minutes after the "shock" episode, and can be associated with coagulopathy, acute renal failure, and mental status changes, as was the case with our patient. This process can be reversible, with patient morbidity and mortality related more to the underlying cause of the ischemic event rather than the severity of the liver dysfunction. The hemodynamic insult resulting in ischemic hepatitis can have many etiologies, but in a review of the literature no cases could be found documenting pulmonary embolus (in the absence of hypotension) as the antecedent event.

INTRACTABLE HICCUPS. A Shaw, Department of Medicine, University of Louisville, Louisville, KY.
LEARNING OBJECTIVE. 1) Recognize that hiccups may indicate an underlying pathologic condition.
CASE. A previously healthy 54 year old male presented to the clinic complaining of hiccups for one year. He described them as persistent, occuring both day and night, and relieved by vomiting. However, several hours after vomiting, the hiccups would return. He also reported a 50 pound weight loss over the last year and was unable to work. Hiccups had not been responsive to Chlorpromazine. Initial work-up included a comprehensive metabolic panel, CBC, chest x-ray, and EKG - all within normal limits. Further tests included a CAT scan of the chest which showed a markedly distended stomach and esophagus with apparent thickening of the posterior wall of the esophagus. No mediastinal mass or lymph nodes were seen. CAT scan of the abdomen revealed increased intestinal gas pattern with no evidence of obstruction. No free air or free fluid was seen. An upper gastrointestinal series showed moderate gastroesophageal reflux and mild dilatation of the small bowel and stomach. A trial of Cisapride was given. The patient reported that the medicine helped but the hiccups came back two weeks later. He was also sent for hypnotherapy without benefit. MRI of the brain was obtained but the study was limited secondary to motion artifact; however, no gross abnormalities were seen.
DISCUSSION. Hiccups serve no known physiologic function. Prolonged episodes of hiccups deserve a careful assessment to evaluate for an underlying pathologic disorder. Causes to consider include organic, psychogenic, and idiopathic disorders. Treatment options include both nonpharmalogic and pharmalogic measures.

RECOGNIZING METFORMIN INDUCED HYPONATREMIA
Scott Sheldon, Mukta Panda, University of Tennessee, Chattanooga Unit, Department of Medicine.
LEARNING OBJECTIVE: Recognize hyponatremia as a side effect of metformin. Assess patients at high risk of developing hyponatremia.
Case: A 43-year-old male with insulin-requiring diabetes mellitus of uncertain duration required relatively high doses of insulin as an outpatient. His TSH was elevated at 9.86 uu/ml; electrolyte and lipid panels were within normal limits. Corrected for hyperglycemia, the patient's serum sodium was 139 mmol/L. The patient was started on metformin 500 mg twice daily in an effort to minimize his insulin requirements. Eight days into the metformin therapy, the patient's BUN and creatinine were stable at 11 mg/dL and 0.5 mg/dL, respectively. He was found to have euvolemic hyponatremia. Urine osmolarity was 356 mos/L and serum osmolarity was 255. The metformin was discontinued and within 16 days the serum sodium was 136 mmol/L.
Discussion: Metformin was recently released in the US. Katsuki and Ito were the first to describe the antidiuretic effect of metformin in several patients with diabetes insipidus. Gin et al. reported the only case of hyponatremia following the introduction of metformin. In this case, when metformin was discontinued the serum sodium returned to a normal level. They theorized that biguanides might inhibit the hepatic catabolism of antidiuretic hormone (ADH).
We speculate that our patient probably had mildly elevated endogenous ADH levels secondary to his subclinical hypothyroidism. Addition of metformin may have potentiated the action of ADH and aggravated the hyponatremia. Only when metformin was discontinued did the serum sodium return to baseline. This case serves to warn clinicians about hyponatremia as a potential side effect of metformin. Clinicians need to be aware that the use of metformin may produce a precipitous drop in serum sodium levels.



MICTURITION-INDUCED CORONARY VASOSPASM; Stacey L. Sheridan; Dartmouth Hitchcock Medical Center; Lebanon, NH.
LEARNING OBJECTIVES: (1) Recognize the clinical features of coronary vasospasm (2) Recognize the etiologic agents of coronary vasospasm
CASE: A 68-year old male with multiple medical problems including hypercholesterolemia, chronic obstructive pulmonary disease, and metastatic small cell lung cancer treated with lobectomy and chemotherapy, presented complaining of a 10 day history of chest pain following micturition. The pain was described as squeezing and substernal without radiation or associated symptoms. It occurred immediately after urinating, but at no other time, and lasted approximately 10 minutes before abating without intervention. It had been increasing in intensity and frequency, but still did not occur with every episode of micturition. The patient denied any prior history of rest or exertional chest pain although he had undergone an exercise treadmill test approximately 9 months earlier for lateral ST depressions on his baseline electrocardiogram (EKG). This was terminated at 5 mets secondary to fatigue and shortness of breath, but showed no EKG changes.

On exam, the patient was a plethoric male with central obesity who had a pulse of 83 and a blood pressure of 126/70. His exam was remarkable for xanthelasmas; a normal jugular venous pressure; a distant, but regular, heart exam without fourth heart sound or murmurs; and some chronic changes at the right lung base near the site of his prior surgery. He had no peripheral edema. A resting EKG showed normal sinus rhythm with lateral ST depressions and inverted T waves. Creatine kinase and troponin were normal, and electrolytes were normal except for a low magnesium at 0.6.

The patient was admitted for telemetry monitoring and had three episodes of chest pain following micturition. Each time, the patient developed ectopy or ventricular tachycardia followed by chest pain. The first two episodes resolved before full assessment or treatment, but an EKG with the third, showed acute anterior ST elevation, and the pain resolved immediately with one sublingual nitroglycerin. Echocardiogram revealed anterior hypokinesis. Exercise thallium showed small septal ischemia. Chest computed tomography showed enlarged mediastinal nodes and liver metastases, but no disruption of or impingement on the pericardium. The patient was diagnosed with variant angina, and was started on heparin, nitropaste, diltiazem, and magnesium with no recurrence of symptoms.
DISCUSSION: Variant or Printzmetal's angina is distinctly different from the common demand-induced ischemia which occurs with exertion. It occurs predominantly at rest, results from focal hyperdynamic vasoconstriction, often at the site of a fixed obstruction, and is more often associated with ST elevation than depression. Multiple mediators have been proposed including stress, magnesium deficiency, and acetylcholine. Due to its release of acetylcholine in the heart, the parasympathetic nervous system has also been targeted as a potential mediator. Vasospastic angina with micturition, and its vasovagal associations, seems to argue for this role.

WHEN IT RAINS, IT POURS: CHOLESTEROL EMBOLI SYNDROME; Stacey L. Sheridan; Dartmouth Hitchcock Medical Center; Lebanon, NH.
LEARNING OBJECTIVES: (1) Recognize risk factors for cholesterol emboli syndrome (CES). (2) Diagnose CES
CASE: A 70-year old female with multiple medical problems including hypertension, hypercholesterolemia, atherosclerotic coronary vascular disease (ASCVD), ascending aortic aneurysm, and chronic renal insufficiency presented complaining of a 1 month history of a severe, cramping postprandial abdominal pain with radiation to the back. It started approximately one week following treatment with thrombolytics, anticoagulation, and cardiac catheterization for a myocardial infarction, and was accompanied by nausea, vomiting, and a 16 pound weight loss secondary to fear of eating. She had already undergone an extensive negative work-up for liver, pancreatic, and biliary disease, and had had an upper endoscopy revealing a small H. pylori-negative duodenal ulcer. Despite adequate treatment with lansoprazole, symptoms had continued.

On exam, the patient was a thin comfortable elderly female with a blood pressure of 160/105 and a pulse of 70. Cardiovascular exam was unremarkable except for cool, bluish toes on the left foot despite normal and symmetrical pulses and the absence of bruits throughout. Abdominal exam revealed normoactive bowel sounds, no bruits, and mild epigastric and umbilical tenderness with no guarding or rebound. Stool on rectal exam was without blood. Initial laboratories revealed normal electrolytes, a newly elevated creatinine from 1.1 to 5.1, a new peripheral eosinophilia, and normal liver function tests.

With growing concern for mesenteric ischemia secondary to embolization or aortic dissection, magnetic resonance imaging was obtained which showed a 3.7 cm infrarenal abdominal aortic aneurysm; there was no dissection, and no involvement of the renal or mesenteric vessels. Mesenteric and renal duplexes similarly showed no dissection. An opthalmology consult was obtained and revealed Hollenhorst plaques in the right eye. Given the spectrum of disease, a diagnosis of CES was made and the patient was treated with supportive therapy including pain medications, anti-emetics, anti-hypertensives, and lipid-lowering agents. Nephrotoxins were avoided.
DISCUSSION: CES is a condition characterized by the dislodgement of separate cholesterol crystals or atheromatous plaques which travel downstream and occlude arteries or arterioles causing ischemia and infarct. Any organ can be affected although the kidneys, gastrointestinal tract, spleen, and skin are most common in histologic studies. Lab studies are nonspecific, but peripheral eosinophilia should raise suspicion. Similarly, the presence of multiple risk factors, including ASCVD, hypertension, angiography, anticoagulant or thrombolytic therapy, smoking, diabetes, and cardiopulmonary resuscitation, should raise suspicion; the presence of 4 or more risk factors, alone, increases the probability of diagnosis to as high as 66%. Definitive diagnosis requires biopsy, but may not be needed with a shower of clinical evidence.

RECURRENT ABDOMINAL PAIN, ASCITES, AND WEIGHT LOSS: TB OR NOT TB?. W Shindy, Sepulveda VA/ San Fernando Valley Program, Department of Internal Medicine, Sepulveda, Ca.
LEARNING OBJECTIVES: 1) To recognize M. Tuberculosis peritonitis as a cause of new onset ascites. 2) To review the diagnostic workup for M. Tuberculosis.
CASE. An 18-year-old African American female presented with three months of weight loss, increasing abdominal distention and right lower quadrant pain. Two days prior to admission, she had nausea, vomiting, dysuria, and loose stools. Previous work-up from an outside facility included an abdominal ultrasound, which revealed ascites and a right ovary with a simple cyst. A CT scan of the abdomen and pelvis showed massive ascites, two right-sided simple cystic ovarian masses and no abdominal masses. A pregnancy test was negative. The patient was single, US-born, had no recent travel history, no blood transfusions, and denied tobacco, alcohol and intravenous drug use. She had never been pregnant, and her last menstrual period was 3 weeks prior to admission. On physical exam the patient was afebrile. Her abdominal exam was remarkable for distention, shifting dullness and right lower quadrant tenderness. Initial labs included a urinalysis with WBC=110 and RBC=9, serum CA-125=124, LDH= 464 and albumin=3.8. A paracentesis was performed and the ascitic fluid revealed albumin=3.3, WBC=1100 (11%N/81%L/8%M), RBC=2000 with negative AFB smear and culture (to date). Cytology was negative for malignancy. A chest X-ray was negative. A PPD test was placed which showed 40 mm of induration. A laproscopic exam and biopsy were performed which showed multiple peritoneal plaques and studding. Biopsies identified necrotizing granulomas, but were AFB negative. A presumptive diagnosis of M. Tuberculosis peritonitis was made and the patient empirically treated with rifampin, INH, PZA, ethambutol and pyridoxine.
DISCUSSION. The differential diagnosis of ascites often includes chronic liver disease, cardiac failure, and nephrotic syndrome. However, less common causes such as tuberculosis and malignancy related ascites should also be considered. Tuberculosis has been found to account for up to two percent of cases of ascites. The diagnosis of peritoneal tuberculosis is a difficult one to make due the low yield of conventional tests such as AFB smear, culture and biopsy. Although not done in this case tests are such as adenosine deaminase activity and auramine O fluorochrome are both sensitive and highly specific. This case highlights the difficulty establishing the diagnosis of peritoneal tuberculosis and the availability of various tests to confirm the diagnosis.

TITLE: HIV IN THE OLDER POPULATION, CHERYL SMITH, JOSEPH KESSLER, MOUNT SINAI MEDICAL CENTER, NEW YORK, NY

LEARNING OBJECTIVES:

1. Assess HIV high risk behavior in the older population.

CASE: D.O. is a 80 year old man with a history of hypertension, colon cancer, Benign Prostatic Hyperplasia, Type II Diabetes, and Depression who had recurrent complaints of hematochezia. The patient requested a second opinion at the Mount Sinai Coffey Geriatric Center where a sexual history was taken for the first time. D.O. identified himself as bisexual. A gastrointestinal evaluation was negative and the hematochezia was presumed secondary to trauma. An HIV test was recommended and D.O. subsequently tested positive. His initial CD4 count and viral load were 498 and 367,870 k copies.

DISCUSSION: A growing number of older people have been diagnosed with AIDS; recent data reveals that 6% of all new AIDS cases in the United States are in persons 55 years or older. Older persons with HIV are likely to live for a shorter period of time; this is felt to be secondary to their delayed diagnosis. D.O. was followed by a general internist and cardiologist over several years, neither of whom took a sexual history. Several months prior to OD's initial assessment the Geriatric Department had a lecture about HIV in the older population which helped OD's physician identify and inquire about high risk behavior. This case illustrates that health care providers are less likely to ask their older patients about sexual behavior and provide education about HIV prevention. There is a need for education of health care providers about HIV in the older population.

ATYPICAL PRESENTATION OF TUBERCULOSIS M. Solomon, Medical University of South Carolina, Charleston, SC

LEARNING OBJECTIVE: Recognize the clinical and radiographical manifestations of Tuberculosis

CASE: A 53 year old male with a previous history of a right tonsillar squamous cell cancer status-post right radical neck dissection and partial pharyngectomy in 1994 and a history of left colon cancer status-post hemi-colectomy in 1996 presented for further evaluation of worsening dyspnea both with exertion and with rest. He also complained of a cough productive of yellow-brown sputum for approximately two weeks. He had no complaints of fever, chills or night sweats. He did complain of anorexia and reported about a 25 pound weight loss over a questionable period of time. He denied chest pain, orthopnea or paroxysmal nocturnal dyspnea. He denied any tuberculosis exposures. He had a 60 pack year history of smoking. On presentation, the patient was afebrile and was in mild respiratory distress with a respiratory rate of 28. His exam was significant for bilateral temporal wasting, no lymphadenopathy or jugular venous distention, tachycardia, and diffuse rhonchi over the right hemi-thorax and left upper lung zone. His admission lab work revealed a sodium of 126 with the other serum electrolytes normal. His initial leukocyte count was 14,200 with a mild bandemia, platelets of 523,000. Initial blood gas on 2 liters of oxygen revealed a pH of 7.6, pCO2 of 25 and a pO2 of 117. His HIV was negative and his Carcinoembryonic antigen was normal at 1.8. The admission chest x-ray had a diffuse interstitial and alveolar infiltrative pattern with sparing of the left base. Initially, the patient was started on broad spectrum antibiotics to cover for a community acquired pneumonia. The pulmonary service was consulted who felt that the differential diagnosis included malignancy (alveolar cell cancer or lymphoma), bacterial infection, fungal infection, or granulomatous disease. Initial studies were recommended by the pulmonologist including placing the patient in isolation and collecting sputum samples for acid fast bacilli (AFB), fungal cultures and cytology and obtaining a high resolution CT scan of the chest. If these studies were unrevealing then they planned to do a bronchoscopy. The CT scan showed extensive bilateral patchy airspace consolidation, a thick walled cavity in the right lung apex and areas of cavitation within the left lung. There were enlarged mediastinal, paratracheal and precarinal lymph nodes as well as small bilateral pleural effusions. At this time all three of the patient's sputum samples were growing 3-4 plus AFB. The patient was then started on the four drug treatment for tuberculosis. Since then the AFB in the sputum has been speciated as M. Tuberculosis.

VERTEBRAL ARTERY DISSECTION DIAGNOSED BY MAGNETIC RESONANCE ANGIOGRAPHY. D Smith, T Lane, M Kowalik, University of Connecticut Primary Care Internal Medicine Residency, Farmington, CT.

LEARNING OBJECTIVES. 1) Identify the features of vertebral artery dissection (VAD), 2) Optimize sensitivity of radiological studies via interdisciplinary discussion of clinical details, and 3) Recognize chiropractic manipulation as a potential risk factor for VAD.

CASE. A 26 year old female with intractable headaches, multifocal in location though never involving the neck, was seen for excruciating pain radiating from her left posterior neck to left eye. She was diagnosed with tension headache and prescribed analgesics. The next day a chiropractor diagnosed misalignment of the neck. In the afternoon of her 9th day of treatment she had her 4th manipulation. At 2:00 the following morning she was awakened by left face tingling, vertigo, and profound nausea. She progressed to have bilateral arm tingling, photophobia and vomiting. She used oral contraceptives, was a non-smoker, had 2-3 drinks a weekend and used no illicit drugs. Her mother had migraines, and two great aunts had cerebral aneurysms. Emergency department evaluation one hour after symptom onset revealed vitals: T 99.3, P 55, BP 127/68. General exam was remarkable for mild neck tenderness. Heart lungs and abdomen were normal. She was without meningismus or carotid bruit. Neurologic exam revealed spontaneous clockwise rotary nystagmus, fundoscopic exam impossible; right pupil 4mm, left 5mm; diminished light touch on left face; tongue deviation to left; bilateral dysmetria on finger to nose and heel to shin; and severely ataxic gait with tendency to fall to the left. Labs were normal. Head CT was normal. MRI was performed. Discussion with the neuroradiologist of the possibility of vertebral artery dissection led to identification of irregularities on the MRI. MRA and repeat MRI, with reorientation of the cut sequence to maximally expose the suspicious area, were performed. These clearly demonstrated flow void of the left vertebral artery at the level of the skull base consistent with dissection. The left PICA could not be visualized. The patient was promptly started on IV heparin and then converted to warfarin. Over the next 5 days she had gradual improvement of her symptoms, especially of her ataxia and dysmetria. Mild vertigo persisted.

DISCUSSION. Presentation of VAD usually includes unilateral occipital headache with ipsilateral neck pain and features of lateral medullary syndrome: vertigo, cerebellar ataxia, and facial hypalgesia. The cause is disruption of flow in one vertebral or posterior inferior cerebellar artery. Without strong clinical suspicion, the repeat MRI and MRA might not have been performed in as timely a manner, and appropriate therapy could have been delayed. The high-quality images in this clinical setting made angiography unnecessary. 30% of the approximately 125 million annual chiropractic visits involve cervical manipulation, with a reported estimated incidence of cervicocephalic artery dissection of 2-3 per million treatments (75 per year). VAD is thought to be caused by shearing injury to vessel walls at the alanto-axial joint where the vessel briefly travels horizontally. Although a prospective study is unlikely, the etiologic implication is important because dissection causes 5-20% of strokes among young people. More common dissection risks are hypertension, migraine, birth control pills and smoking.

SUBCLINICAL PRESENTATION OF HYPOTHYROIDISM L. Stallworth, C. Kelly, E. Ayers, Wayne State University/Detroit Medical Center, Detroit, Michigan.

LEARNING OBJECTIVES: 1) Assess a difficult patient for treatable causes of altered sensorium 2) Recognize subtle signs of changes in a difficult patient and make a logical diagnosis

CASE: The patient is a 23-year-old Hispanic male with a history of Down's Syndrome, severe mental impairment, seizure disorder and eczema. He has a history of combativeness during physical examinations, which makes assessment of the patient challenging. Sedation is required to complete his physical exam and draw his blood. He initially presented with the complaint of nasal discharge and was described as having difficulty in breathing while sleeping and snoring. His mother also stated that there was a history of a fall at school, which was witnessed and there was no history of head trauma and no witnessed seizures. On physical exam vital signs were stable and he had a weight gain of 14 lbs. Physical exam was difficult to obtain secondary to combativeness. The patient was treated for presumptive sinusitis and his mother was asked to keep a record of sleep pattern and the patient followed-up in one month. At the next visit, the upper respiratory symptoms resolved. The mother had reported that he had fallen at school. The patient's mother was told that his teachers had noticed that his legs were weak and he had fallen on his buttocks. The family had also noticed that he was sleeping more than usual. On physical exam, vital signs were stable, weight 253 lbs. (20-lb increase in 4 months). Patient was sitting in a chair sleeping, easily arousable, but falls back to sleep during exam with snoring. HEENT: down's faces, otherwise unremarkable. Neck: supple, no thyroid enlargement. Lungs: clear; Heart: unremarkable; ABD: unremarkable; Ext: no edema, good femoral pulses; Skin: dry, eczematous patches. LABS: TSH 356.65, Dilantin therapeutic, electrolytes within normal limits.

DISCUSSION: In a Down's syndrome patient with mental impairment and seizure disorder it is difficult to illicit a good history and physical. We ruled out infectious causes for the patient's altered sensorium, then we considered dilantin toxicity and subclinical seizures. Since the literature has shown that thyroid dysfunction is exceedingly common in adults with Down's Syndrome, we also considered hypothyroidism. The weight gain, weakness, and increased somnolence also supported the diagnosis of hypothyroidism which was supported by the laboratory data.

A 36 YEAR OLD WOMAN WITH HIRSUTISM AND AMENORRHEA. Bruce E. Strober and Navneet Kathuria, Department of Medicine, New York University School of Medicine, New York, NY.

LEARNING OBJECTIVE. Assess the etiology of recent-onset hirsutism by both a focused clinical history and appropriate laboratory studies.

CASE. A 36 year-old female presented to the clinic with the chief complaint of hirsutism, a 20 pound weight gain over the past year, and secondary amenorrhea for the past 18 months. She denied acne, any change in her appetite, and other constitutional symptoms. She described her increased hair growth as localized to her pubic area and face. She denied deepening of her voice, increased muscle mass, and male pattern baldness. The patient was not taking any medications, and denies any prior history of hypertension or diabetes. There is no family history of hirsutism.

The patient was a mildly obese female. Vitals were notable for a blood pressure of 150/110. She displayed dark facial hair limited to the upper lip, periumbilical coarse, dark hair that extended into the pubic area and involved the inner thigh and inguinal regions. There was an increase in supraclavicular fullness. There was no clitoromegaly, and no hyperpigmented striae.

Laboratory studies demonstrated normal electrolytes and CBC profiles with a normal fasting glucose. Liver function tests were remarkable for a mild elevation of AST and ALT values. The following hormonal studies were normal: TSH, β-HCG, LH, FSH, prolactin, dehydroepiandrosterone (DHEA), urinary 17-ketosteroids, and 17-hydroxyprogesterone. Both free and total serum testosterone are pending. An overnight dexamethasone suppression test was normal. A progesterone challenge test resulted in withdrawal menses with the subsequent resumption of normal menstrual periods. An abdominal ultrasound demonstrated normal appearing ovaries, with no other abnormalities.

DISCUSSION. Approximately 5% of women have hirsutism. Most cases involve androgen-dependent hair growth occurring in areas where boys typically grow hair at puberty. More than 95% of androgen-dependent hirsutism results from polycystic ovarian syndrome and idiopathic hirsutism. Less common causes include severe insulin resistance, congenital adrenal hyperplasia, androgen-secreting tumors, and Cushing's syndrome. Androgen-independent hirsutism can be inherited or can be drug-induced. The appropriate historical evaluation of hirsutism should assess for amenorrhea, the rapidity of onset of hair growth, medications, and any family history of hirsutism. Hirsutism, especially when associated with virilization, necessitates the measurement of serum testosterone, serum DHEA, and urinary 17-ketosteroids levels to assess both adrenal and ovarian androgen production. A serum 17-hydroxyprogesterone measurement should also be part of the initial evaluation to rule-out late-onset congenital adrenal hyperplasia. The above patient most likely has idiopathic hirsutism given her clinical history, exam, and laboratory findings.

PRIMARY LOCALIZED AMYLOIDOSIS OF THE URINARY BLADDER-A RARE CAUSE OF GROSS HEMATURIA. O. Tirzaman, D. L. Wahner-Roedler, R. Malek, Mayo Clinic, Rochester, Minnesota

LEARNING OBJECTIVES: To recognize that 1) primary localized amyloidosis of the urinary bladder is unlikely to progress to systemic amyloidosis and repeated search for systemic disease is unnecessary, 2) regular cystoscopic follow-up is mandatory because of a high local recurrence rate, 3) treatment consists of early eradication with fulguration or laser therapy.

CASE. A 59-year-old white male presented in 1986 with gross hematuria. Physical examination was unremarkable. Screening laboratory tests were normal except for urinalysis, which revealed more than 100 red cells per high power field. The patient underwent an excretory urogram and cystoscopy. Cystoscopy showed a lesion at the left posterolateral wall consistent with a bladder tumor. Biopsy revealed amyloid. The patient underwent a work-up for systemic amyloidosis consisting of a normal serum protein electrophoresis and special protein studies of serum and urine and a negative fat aspirate for amyloid. Fulguration of the bladder lesion was performed. The patient was followed on a six to twelve month basis and between 1986-1995 required treatment with fulguration and laser therapy on eight different occasions. In 1995 he received intra-vesicular DMSO (dimethylsulfoxide) every two weeks over a one-year period. He was last seen in July 1998, 12 years after the diagnosis of primary localized amyloid of the bladder was made, when he again presented with gross hematuria due to recurrent disease which was treated with laser therapy.

DISCUSSION. Primary localized amyloidosis of the urinary bladder is a rare disorder. It is of interest to the general internist as well as the urologist because clinical presentation (gross painless hematuria and/or irritative lower urinary tract symptoms) and cystoscopic findings are similar to that of an infiltrating neoplasm. Based on about 150 cases in the literature and a review of 30 cases seen at our institution, this condition does not progress to systemic disease, and systemic amyloidosis rarely manifests itself with urinary symptoms. Therefore, repeated search for systemic disease is unnecessary. As far as treatment is concerned, early eradication with fulguration or laser therapy appears to be the treatment of choice when possible, but for larger lesions transurethral resection or partial cystectomy might be necessary. Successful local therapy with DMSO has been described in the literature (Tokunaka et. al. J. Urol. 135:580, 1986). Regular cystoscopic follow-up is mandatory since there is a high local recurrence rate, and this condition behaves just like a low grade noninvasive and nonmetastatic malignancy.

Dermatomyositis and Malignancy: Role of Internist

Rewati Teeparti, Uma Karnam, Ernie Yodder, Department of Medicine, Wayne State University Detroit, MI

Learning Objective: 1) To diagnose dermatomyositis from it's clinical manifestations, 2) To follow the patients with dermatomyositis carefully to unravel underlying malignancy.

Case: 59-year-old man presented with fatigue pain and weakness in arms and legs for 3 months. He had weakness in thighs and arms with difficulty in climbing upstairs, shaving, combing and getting up from chair. Subsequently he noticed erythematous macular rash on his face and nape of the neck extending on to his chest. He had bluish rash on his eyes with puffiness. On physical exam in addition to above-mentioned findings patient also had muscle tenderness on palpation, 'V' sign of neck and periungual telangectasia. Relevant lab data revealed CPK=5500, LDH=1250, Aldolase=41.5. The skin biopsy of the rash was consistent with dermatomyositis and muscle biopsy showed inflammatory changes. Patient was diagnosed with dermatomyositis and was treated with steroids, cyclophosphamide and intravenous immunoglobulins. Patient did not have any evidence of malignancy anywhere at that time. Patient was followed carefully with periodic physical exams and one year after his initial presentation with dermatomyositis he was diagnosed with non-small cell carcinoma of the lung.

Dermatomyositis is an uncommon inflammatory myopathy of unknown cause associated with characteristic skin rash. In adults the disease may herald or be associated with underlying malignancy. The proportional association of dermatomyositis is reported to be in the range of 15-35%. The associated malignancy can precede follow or occur simultaneously with dermatomyositis. The relative risk is highest in the first year after the diagnosis of dermatomyositis. The cancer and dermatomyositis are causally not coincidentally associated. The common denominator is thought to be underlying immune dysfunction. An internist is recommended to evaluate a patient with dermatomyositis for underlying occult malignancy. The routine methods like complete history, careful physical examination and appropriate age related evaluation is suggested rather than extensive non-directed search.

CHRONIC PELVIC PAIN IN MEN, J Tjia and S Dev, Section of General Internal Medicine, Boston Medical Center, Boston University School of Medicine, Boston, MA

LEARNING OBJECTIVE: Recognize the need to include a sexual trauma history in the evaluation of men with chronic pelvic pain.

CASE: A 52 yo Cape Verdian man presented to the urgent care center with intermittent, recurrent left lower quadrant/perineal pain and white penile discharge after recent unprotected sexual intercourse with a woman. He expressed concern about sexually transmitted diseases. He had been treated numerous times for presumptive gonococcal and chlamydial urethritis over the past 3 years. He had no previous surgeries. Abdominal and rectal exam were unremarkable; genital exam revealed no penile discharge or focal tenderness. He was again empirically treated for gonorrheal and chlamydial urethritis. Penile cultures for gonorrhea and chlamydia and urine cultures were negative. Urologic evaluation resulted in a diagnosis of nonbacterial prostatitis and he was given instructions on altering his ejaculatory pattern to avoid prostatic congestion. On follow-up in primary care he noted persistent pelvic discomfort and was treated for chronic prostatitis with 1 month of Azithromycin. Follow-up revealed no relief. Further investigation included abdominal computerized tomography (CT) that was negative for diverticulosis and intra-abdominal pathology. On further questioning, the patient revealed that his symptoms began 18 years prior to this presentation after an unprotected non-consensual sexual encounter with a woman that his friends arranged during a drunken state -"just take her". The patient expressed tremendous guilt about this encounter and was concerned that he harbored a persistent infection from that contact. After some discussion, he agreed to psychological consultation to further address this issue.

DISCUSSION: Sexual trauma is a well recognized potential etiology of chronic pelvic pain in women with a lack of organic findings. Similar men are more commonly given the diagnosis of nonbacterial prostatitis and treated with antibiotics despite negative cultures. In such patients, a thorough sexual history may address the potential underlying etiology, afford a non-pharmacologic treatment modality, and possibly avoid the overuse of antibiotics.

A 64 YEAR-OLD WOMAN WITH ACUTE RENAL FAILURE AND METFORMIN-ASSOCIATED LACTIC ACIDOSIS. Michael Tracy, Paul Bernstein, and Joseph Cappuccio, Rochester General Hospital, University of Rochester, Rochester, NY.
LEARNING OBJECTIVES: 1. Recognize metformin as a cause of lactic acidosis. 2. Recognize the potential harm of metformin in the setting of renal insufficiency.
CASE A 64 year-old woman complained of left lower quadrant abdominal pain with no nausea, vomiting or diarrhea five days prior to hospitalization. Ciprofloxacin was prescribed for presumed diverticulitis with some improvement. She complained of increasing fatigue for two days, but was able to continue working. On the morning of admission she was noted to have slurred speech and later, a family member found her unresponsive. An initial fingerstick blood glucose of 20mg/dL was obtained in the home. Mental status improved with intravenous dextrose. Her past medical history was significant for diverticulitis and adult onset diabetes mellitus. Medications included ciprofloxacin, metformin and glipizide. There was no known over-the-counter medication or alcohol use. In the emergency department she was agitated and complained of abdominal pain. Vital signs revealed a rectal temperature of 34.9C, blood pressure of 80/66 mm Hg, pulse of 100/min and respirations deep and labored at 24/minute. Physical exam showed clear breath sounds, no cardiovascular abnormalities, a normal abdomen, no peripheral edema and no focal motor abnormalities. Laboratory data included: ABG:pH 6.71, pCO2 15 mm Hg, pO2 173 mm Hg on 4 liters oxygen; serum sodium 144meq/L, potassium 5.2 meq/L, chloride 105 meq/L, CO2 6 meq/L, BUN 77 mg/dL, creatinine 10.2 mg/dL, glucose 213 mg/dL, anion gap 33, lactate 11.4mmol/L and WBC 23,300/mL. A catheterized urine sample showed 20-40 RBCs/hpf, 2-5 WBCs/hpf, no casts and 300 mg/dL protein. Two weeks prior to admission BUN was 12mg/dL with a creatinine of 0.7mg/dL. Hemodialysis and intravenous bicarbonate normalized the anion gap by hospital day four. She was mechanically ventilated for three days and extubated. Ischemia of the left foot, a non-Q wave myocardial infarction, upper gastrointestinal bleeding, rhabdomyolysis and thrombocytopenia complicated the ICU course. She showed steady clinical improvement upon transfer to the medical floors on day five. Sudden death occurred on hospital day twelve.
DISCUSSION: This is a case of metformin-associated lactic acidosis in the setting of acute renal failure of unclear etiology. While existing renal insufficiency is a known contraindication to metformin use, one must recognize the potential for iatrogenic renal insufficiency leading to metformin-associated lactic acidosis. Although ciprofloxacin is a rarely described cause of acute renal failure, there are many prescription and over-the-counter medications that could lead to such a situation. One must also consider the effect of intercurrent illness on renal function in those patients on metformin.

GONOCOCCAL ENDOCARDITIS PRESENTING IN 18 YEAR OLD FEMALE AS THIGH PAIN, Don J. Tynes, C. Kelly, J Hopper, M. Turner, Detroit Medical Center, Wayne State University School of Medicine, Detroit, Michigan.
LEARNING OBJECTIVES: 1) To recognize important information from a complete history and physical. 2) To use this data to develop a broad differential. 3) To use a limited number of tests to illicit the diagnosis.
CASE: An 18 year old African-American female developed acute right thigh pain and difficulty in ambulating. She was diagnosed and treated for a musculoskeletal strain. The patient returned ten days later with excruciating pain in both thighs and unable to bear weight on her right leg. The patient's examination showed an obese female with bilateral thigh edema, a decreased left dorsalis pedis pulse, a non palpable right dorsalis pedis pulse and a vaginal discharge. Laboratory findings showed renal insufficiency, elevated white blood cell count, anemia, thrombocytopenia, hematuria and proteinuria. An arterial flow study showed that the right common iliac and bilateral popliteal arteries were occluded. The patient underwent a bilateral thrombo-embolectomy of the right common iliac and popliteal arteries. The patient had hemodialysis and blood cultures drawn were positive for Neisseria gonorrhea. The patient received a seven day course of IV antibiotics. Upon re-evaluation, a loud systolic ejection murmur was detected. A transesophageal echocardiogram showed mitral and aortic regurgitant murmurs, vegetations on the aortic and mitral valves. The patient was treated for endocarditis and a St. Jude valve was placed.
DISCUSSION: Gonococcal endocarditis is an uncommon (1-2%) but often fatal manifestation of disseminated gonococcal infection. Syphilis, gonorrhea and chlamydia are still prevalent and should be included as a part of a thorough differential diagnosis for endocarditis. Today a conservative work up is encouraged, a thorough history (including a sexual history) and physical exam and limited number of test should be done to assist with the differential diagnosis.

WHEN ESSENTIAL HYPERTENSION ISN'T. R Trotter, B Witzburg, S Borkan, Department of Medicine, Boston Medical Center, Boston, Ma.
LEARNING OBJECTIVES: 1) Recognize patients at risk for primary hyperaldosteronism, and 2) Systematically evaluate a patient suspected of primary hyperaldosteronism.
CASE. A 75-year-old Haitian female with a 10-year history of hypertension presented to primary care clinic with a blood pressure of 290/140. She took no medications and reported no chest pain, shortness of breath, change in vision, change in urinary habits, headache, use of chewing tobacco, consumption of licorice or any herbal medicines. Pulse was 80, respiratory rate was 16 and she had a normal body habitus. The lungs were clear, heart sounds regular with S4 but without S3 gallop, and there was no papilledema. Electrocardiogram showed no ischemic changes, but did show evidence of left ventricular hypertrophy with strain pattern. Potassium was 3.0 mmol/L and the blood urea nitrogen and creatinine were normal. Plasma aldosterone: renin ratio was 46.5 ng/dl / 0.6 ng/ml = 77.5. Serum 18 hydroxycorticosterone (18-OH CS) level was 86ng/dl. Magnetic resonance imaging and angiography showed no significant adrenal, renal or vascular abnormalities. The patient has declined invasive testing for diagnosis (adrenal vein sampling) because she will not accept surgical treatment. She is currently being managed medically with a combination of amiloride, verapamil, terazosin, hydralazine, and lisinopril with blood pressure 210-240/90-110.
DISCUSSION: Despite the relatively low prevalence of secondary forms of hypertension (primary hyperaldosteronism is estimated to be present in 1% of hypertensives), the high prevalence of hypertension insures that thousands of patients with secondary forms of hypertension will be seen in general medical practice in the U.S. Although most patients with hyperaldosteronism will have hypokalemia up to 20% will not. Severe diuretic induced hypokalemia is a clue to the diagnosis in normokalemic patients. Some hypertensive populations have a higher incidence of primary hyperaldosteronism: urban blacks 2-12%, spontaneous hypokalemia 50%, diastolic blood pressure >100 3%, resistant hypertension and extremes of age.
The most useful next screening test for primary hyperaldosteronism is the serum aldosterone: renin ratio. A ratio >30 is estimated to be 80% sensitive and a ratio > 50 almost 100% specific for primary hyperaldosteronism. A ratio > 30 with aldosterone > 20ng/dl is 90% sensitive and 91% specific for an adrenal adenoma as the cause. The addition of serum 18-OH CS level, abdominal CT scan, and adrenal vein sampling serves to further differentiate adrenal adenoma from other causes of primary hyperaldosteronism. Because high-risk groups can be identified and because greater than 50% of people diagnosed with primary hyperaldosteronism will have an aldosterone producing adenoma, curable in 69%, at risk patients should be identified and tested.

RHYTHM FROM THE RIGHT, S. M. Uppal, H. S. Gurm, P. V. Maroo, Department of Internal Medicine, Fairview Hospital, Cleveland OH. LEARNING OBJECTIVES. 1) Describe a case of right ventricular dysplasia, 2) Discuss causes of ventricular arrhythmia's in young, and 3) Role of MRI in evaluation of cardiac disease. CASE.A 48 years old female presents with episodes of palpitations gradually worsening over the past 5 months.
These were occurring 10 to 15 times a day and were associated with lightheadedness, near syncope and flushing. Only significant past medical problem was Asthma for which she used inhaled bronchodilator on prn basis. Physical examination, routine laboratory studies including CBC, Renal function, Electrolytes were normal. EKG showed normal rate, axis and rhythm.
The holter study revealed 6000 beats in ventricular ectopy, including 10 ventricular runs, the longest being 65 beats and the fastest at 230 beats per minute. An echocardiogram was normal. Cardiac catheterization revealed completely normal coronary arteries with normal systolic function. Ventricular Tachycardia was not inducible on electrophysiological studies. A cardiac MRI revealed a band of focal thinning with systolic dysfunction of anterior wall of right ventricle involving lower infundibulum with extension toward interventricular septum.The myocardial characteristics showed fibrous replacement consistent with right ventricular dysplasia.
DISCUSSION. Arrhythmogenic right ventricular dysplasia is a cardiomyopathy of unknown etiology characterized by cardiac electrical instability due to fibrofatty infiltration, which begins in the right ventricle and progresses to involve the left ventricle. It presents most commonly in the ages 15-50 as a) clinically concealed disease which is localized to the right ventricle causing sudden death, b) overt electrical disease with right ventricular structural changes and left ventricular histological involvement causing symptomatic arrhythmia's c) endstage biventricular cardiomyopathy leading to progressive heart failure. Cardiac MRI or biopsies of the myocardium are diagnostic.

Title: HEMOPTYSIS IN AN ANTICOAGULATED PATIENT WITH A NON-LOCALIZING CHEST X-RAY Jerome Van Ruiswyk, Zablocki VA Medical Center, Medical College of Wisconsin, Milwaukee, WI

LEARNING OBJECTIVES. 1) Recognize significance of hemoptysis in anticoagulated patients, 2) Assess hemoptysis in patients with non-localizing chest x-rays, and 3) Diagnose and manage occult lung cancers.

CASE. A 70 year old male presented with a two week history of hemoptysis. His past medical history included atrial fibrillation, peripheral vascular disease, s/p right carotid endarterectomy, hypertension, COPD, s/p right middle lobe resection for lung cancer [20 years prior] and monoclonal gammopathy of unknown significance. His medications were warfarin, digoxin, verapamil and maxzide. He smoked 1.5 packs per day for 50 years. Physical examination was unremarkable; labs showed an INR=3.3. The hemoptysis resolved when his repeat INR=2.6, but returned later when INR=3.0. Initial evaluation showed normal chest x-ray, normal chest CT, normal bronchscopy, normal ENT examination but sputum cytology positive for squamous cell cancer. Repeat chest CT at 4 months was again normal, but at 7 months showed a left upper lobe nodule. Repeat bronchoscopy then showed a large endobronchial tumor at the bifurcation of the left upper and left lower lobes. Pulmonary function tests showed FEV1=1.6L, abg on room air: 7.44/pCO2=40/pO2=68/88% saturation. Quantitative ventilation/perfusion scanning showed less than ten percent of ventilation and perfusion to the left upper lobe. Mediastinal lymph node sampling at the time of surgery showed no regional spread. The left upper lobe tumor was removed with a sleeve resection and a synchronous left lower lobe tumor was removed by wedge resection.

DISCUSSION. Although minor bleeding occurs frequently in anticoagulated patients, hemoptysis is uncommon. Yields of diagnostic evaluations are inversely related to the level of anticoagulation at the time of the bleeding. In patients with hemoptysis and non-localizing chest x-rays the etiology of the hemoptysis is often not found, the prevalence of occult cancer is low, the diagnostic yields of CT and bronchoscopy are similar and complementary, and the limited data available suggests that there is no additional cancer yield from sputum cytologies. However, false positive sputum cytologies are more uncommon than false negative bronchscopies. Therefore, repeated bronchscopies may be necessary to localize chest x-ray [and bronchoscopic] occult cancers which usually become apparent within 6 months after initial symptoms. Patients with initially occult lung cancer usually have a good prognosis but a high rate of second primary lung cancers. Therefore, definitive treatment should preserve remaining lung function.

A NO-PRESSURE DIAGNOSIS OF PAPILLEDEMA. E. Vargo, Johns Hopkins Medical Services Corporation, Baltimore, MD. OBJECTIVES. 1) Evaluate a patient with clinical findings consistent with papilledema; 2) Review conditions which may mimic the appearance of papilledema. CASE. A 25-year old woman presented to request a referral to optometry clinic. She stated that for the past week she had noted increased "straining" of both of her eyes and felt that she needed to have a stronger prescription for eyeglasses. Otherwise, the patient was without complaints. Her past medical history was significant for mild obesity. She had for a short time taken phentiramine, prescribed by an outside physician, but had ceased taking this two weeks earlier. On examination, she appeared to be in no distress. Visual acuity of both eyes was 20/20. The lids, conjunctivae and sclerae were within normal limits. Extraocular movements were intact. Visual fields were normal to gross confrontation. The pupils were normal in size and shape and reacted appropriately to light and consensual response. An undilated funduscopic examination revealed obscuration of both disc margins consistent with papilledema. The neurologic examination was normal. An noncontrasted computed tomography scan of the brain revealed normal parenchyma with no evidence of increased intracranial pressure. A lumbar puncture was performed; the opening pressure was within normal limits. The cerebrospinal fluid studies were within normal limits. Formal visual fields were normal. Ultrasound of the optic nerves revealed elevated optic discs with no evidence of fluid in the optic nerve sheaths. Examination by an ophthalmologist revealed no other optic nerve abnormalities. Two months later, the obscuration of the optic disc margins was found to be persistent. No pathologic etiology for the finding could be established; it was concluded that this patient had elevation of the optic discs as a normal anatomic variant. DISCUSSION. "Pseudopapilledema" is a term reserved for patients found to have elevation of the optic discs without increased intracranial pressure. When confronted with a patient manifesting obscuration of the disc margins, imaging of the brain and lumbar puncture establishing normal cerebrospinal fluid opening pressure and indices are essential. A variety of conditions may produce funduscopic findings consistent with papilledema; some are benign, such as congenital anomalies, while others are vision-threatening or indicative of a serious systemic disease. Awareness of these conditions may assist the primary care physician in directing the correct diagnostic and therapeutic plan for these patients.

A "CLEAR" CASE OF PINK EYE: RECOGNIZING THE SIDE EFFECTS OF OVER-THE-COUNTER OPHTHALMIC PREPARATIONS. E. Vargo, Johns Hopkins Medical Services Corporation, Baltimore, MD. LEARNING OBJECTIVES. 1) Evaluate a patient with anisocoria; 2) Recognize side effects of over-the-counter ophthalmic preparations; 3) Recognize the importance of taking a non-prescription drug use history. CASE. A 29-year old woman presented with a chief complaint of "pink eye," noting a five-day history of redness, itching and foreign body sensation in her right eye. She denied antecedent upper respiratory infection or contact with individuals with conjunctivitis. The eye had a clear discharge which caused the lids to be stuck together in the mornings. She noted some blurring of the vision in the eye as well as photophobia in bright sunlight. Her past medical history was significant for latent syphilis which was treated at age 17. Her only medication was an over-the-counter eye drop used to decrease the itching and redness. On examination, she was a healthy-appearing woman who could tolerate the ambient light of the examination room. Visual acuity was 20/25 in the right eye and 20/20 in the left eye without correction. The extraocular movements were intact and the lids were normal. The conjunctiva of the right eye was minimally erythematous with no discharge or exudate. The sclera was clear. The right pupil measured 7mm and was non-reactive; the left pupil measured 4mm and constricted to 2mm with light. There was no consensual reaction of the right pupil. Funduscopic examination was normal. Fluorescein staining of the right eye did not reveal any corneal defects. The neurologic examination was within normal limits. Of note, the patient had not noticed a difference in the sizes of her pupils. A non-contrast computed tomography scan of the brain was normal. Ophthalmology consultation was obtained. Tonometry of the right eye revealed normal intraocular pressure. Slit-lamp examination of the right eye revealed no inflammation in the anterior or posterior chambers. Instillation of topical 0.125% and then 1% pilocarpine solution resulted in no constriction of the right pupil. She was discharged with the diagnosis of pharmacologic pupil and her anisocoria resolved within a few days' time. DISCUSSION. When a patient is found to have anisocoria, the first priority is to eliminate life-threatening or vision-threatening causes. Therefore, establishing that the remainder of the neurologic examination and brain imaging are normal is essential. The differential diagnosis for this patient included trauma to the iris from rubbing the eye, inflammation of the iris from an infectious or autoimmune disease, neurologic events such as cerebral aneurysm rupture, or pharmacologic paralysis of the iris. Use of over-the-counter agents for decreasing eye redness and itching is common. Inquiry into the history of use of these agents should be part of the evaluation of patients with eye complaints. In addition to pharmacologic agents, there are other agents which patients may come into contact with which can produce ocular signs and symptoms. A logical step-wise approach will assist the clinician in making the correct diagnosis.

RHABDOMYOLYSIS AS A COMPLICATION OF INFLUENZA. A Vasiliadis, Department of Medicine, Mount Sinai Hospital, New York, NY.

LEARNING OBJECTIVES: Recognize rhabdomyolysis as a complication of Influenza viral infection.

CASE: A 21- year- old male presented to his medical doctor with flu-like symptoms, including fever, headache, chills, mild cough, rhinorrhea. He had no significant prior medical history. The patient was given symptomatic treatment with acetaminophen. Several days after the initial presentation the patient noted the development of bilateral thigh pain. The severity of the pain resulted in difficulty ambulating. The patient also noted dark urine. Upon laboratory examination, he was noted to have an elevated CPK (Creatine Phosphokinase) level > 99,000 U/L. The patient was referred for inpatient hospitalization and treatment of rhabdomyolysis. The patient had no history of trauma, seizures or strenuous activity. He denied the use of any high protein drinks, anabolic steroids, and had no history of drug or alcohol abuse. His drug and alcohol toxicology screen was negative. The patient denied any joint pain and rashes. On physical examination the patient was noted to be alert and in no distress. He had a temperature to 37.8 degrees Celsius, heart rate at 107 and regular, blood pressure 130/70, respiratory rate of 18. The patient's neck was supple with no cervical lymphadenopathy. Examination of the oropharynx , heart, lungs, and abdomen was within normal limits. Costovertebral angle tenderness was not elicited. His extremities did not have edema or cyanosis. There was no tenderness on palpation of the extremities. The neurologic examination was normal. The deep tendon reflexes were normal. There were no motor or sensory deficits. The EKG was normal. The CXR revealed clear lungs. The laboratory examination revealed serum electrolytes within normal range. The BUN was 12 mg/dl; creatinine, 0.8 mg/dl. The maximum CPK was greater than 99,000 U/L with an LDH level at 10,500, AST level at 2881, alkaline phosphatase was 84 IU/L. Urinalysis showed 3+ protein and 3+ occult blood with no red blood cells. The TSH was within normal range. The CBC was within normal range. The patient was not anemic and did not have a leukocytosis. The Mycoplasma AB was negative. Influenza AB was positive. EMG revealed changes consistent with an irritative myopathic process. The patient was treated with vigorous intravenous hydration with ½ normal saline with 2 amps of sodium bicarbonate per liter of fluid to maintain alkalinization of the urine to a pH > 8.0. The patient clinically improved with treatment. He had resolution of the myalgia and was able to ambulate. The CPK level at time of discharge was 502 U/L. The patient's BUN and creatinine remained within normal range.

DISCUSSION: Rhabdomyolysis can be caused by a number of factors. Infectious diseases including influenza may also be the etiology. Rhabdomyolysis is a rare complication of influenza viral infection. Prompt identification and treatment may prevent its complications.

TITLE: ATYPICAL SHOULDER PAIN, C M Verzara, Hartford Hospital, Hartford,CT
LEARNING OBJECTIVES: 1)Distinguish shoulder pain due to primary shoulder disease from referred shoulder pain 2) Perform a systematic shoulder examination
CASE: Mr. J is a 62 year old African-American male with no medical history who had new-onset, persistent right(R) shoulder pain for the last 3 months. After multiple emergency room visits, the pain remains unresponsive to topical and systemic analgesics. He denied any recent or past trauma or heavy exertion. The pain is exacerbated when he removes or puts on his shirt. He states the pain keeps him awake at night. He prefers to sleep on his right shoulder because the movement of the bed sheets over his right shoulder worsens the pain. He admits to a long tobacco history. Review of systems is significant for an unintentional weight loss of 20 pounds during the past 3 months. Examination reveals a man in moderate discomfort with normal head & neck, chest, abdominal and cardiac exam. Shoulder examination revealed the following: (a)Inspection: Normal symmetry without muscle atrophy or winging of the scapula .(b)Palpation: No tenderness over acromioclavicular joint (ACJ), biceps, sternoclavicular joint (SCJ) or deltoid. The skin of the posterior aspect of the R shoulder and upper back was markedly anhidrotic, cooler in temperature and hyperesthetic. (c) Active Range of Motion (ROM): Supinated Abduction(AB) to 180 degrees with extended arms was intact. He was also able to perform External Rotation (ER) and Internal Rotation(IR) without difficulty. There was no hitch or painful arc when asked to touch the contralateral shoulder (This maneuvers tests for adduction and/or the ACJ). Forward flexion and extension was intact. (d) Passive ROM was intact (e) Resisted ROM: Resisted AB(supraspinatus), IR (subscapularis), ER(infraspinatus), adduction and supination (biceps) with elbows flexed resulted in no pain exacerbation. Flexion/extension of the neck did not worsen the shoulder pain.
DISCUSSION: Pain from primary shoulder disease arises from either the glenohumerol joint(GHJ) or extracapsular structures such as the ACJ, rotator cuff muscles (RCM), subacromial bursa or biceps. GHJ pathology results in impaired active and passive ROM. Extracapsular shoulder pathology (e.g.,RCM tear) typically spares passive ROM, in the absence of muscle spasms. Moreover, the patient's preference of lying on the affected side argues against primary shoulder disease. The pain is most likely referred from the chest, abdomen or neck. A GI cause is unlikely due to his lack of GI symptoms. Cardiac involvement typically refers pain to the left shoulder and would not be continuous for 3 months. The findings of hyperesthesia,anhidrosis, and focal hypothermia on shoulder palpation suggests peripheral nerve(s) involvement along the C6-T1 dermatome. The absence of pain exacerbation with neck movement argues against cervical spine disease. His long tobacco history and recent weight loss suggest the presence of occult malignancy. Work up revealed a lung mass and lesions on the C6 and T1 vertebrae.

SYNCOPE AS INITIAL MANIFESTATION OF SILENT CORONARY ISCHEMIA, V. Viyarthi
LEARNING OBJECTIVES: Recognize silent ischemia as an unusual cause of AV block.
CASE: A 68 year old man with history of hypertension, past myocardial infarction, hyperlipidemia and smoking presented with one syncopal episode after three days of dizziness. In examination he had bradycardia with HR 25 beats/minute and BP 120/70. The remainder of his examination was unremarkable. His ECG showed third degree AV block and no ischemic changes. His cardiac enzymes were normal. A temporary transvenous pacemaker was inserted. Non cardiac causes of bradycardia were ruled out. He developed two fleeting episodes of retrosternal chest pain and underwent cardiac catheterization which revealed a dominant right coronary artery with significant occlusion at the distal end of the AV nodal branch. A coronary stent was placed at this site with complete resolution of the AV block, obviating the need for permanent pacemaker placement.
DISCUSSION: The cause of syncope in the setting of AV block usually is attributable to degenerative disease of the conducting system and these patients are usually treated with permanent pacemakers. The AV node has a rich arterial supply, therefore pure ischemia as a cause of AV nodal block is rare even in the setting of acute myocardial infarctions. In acute inferior myocardial infarction, the cause of AV block is secondary to increased vagal tone. In anterior myocardial infarctions extensive myocardial damage or transient reversible ischemia is necessary to cause AV block. This is the second reported case in the literature of AV block secondary to silent ischemia, successfully reversed by coronary stenting.

LOBULAR CARCINOMA IN SITU-A MARKER OF A POPULATION AT INCREASED RISK FOR INVASIVE BREAST CANCER. D. L. Wahner-Roedler, Department of Internal Medicine, Mayo Clinic, Rochester, Minnesota
LEARNING OBJECTIVES: To recognize that 1) lobular carcinoma in situ (LCIS) is an incidental pathologic finding 2) LCIS is frequently multicentric and bilateral 3) LCIS is a marker of a population at increased risk for invasive breast cancer 4) the risk for invasive cancer is similar in either breast and infiltrating ductal carcinoma is more common than infiltrating lobular carcinoma 5) treatment options are close surveillance, chemoprevention or bilateral prophylactic mastectomies.
CASE: A 52-year-old woman presented for routine breast examination and her first screening mammogram in August 1993. Breast examination was unremarkable. A mammogram revealed a 2 cm radiographically indeterminate nodule in the upper inner quadrant of the left breast which was solid on ultrasonographic examination. A stereotactic biopsy was done which showed findings consistent with a fibroadenoma. In addition to that, terminal ductal and lobular hyperplasia with mild cytologic atypia was noted. The patient therefore underwent an excisional biopsy, which revealed a fibroadenoma and multiple foci of atypical lobular hyperplasia and LCIS. The margins were not free of tumor. The following treatment options were discussed with the patient: close surveillance (professional breast examination every four to six months, yearly mammogram), participation in a chemoprevention trial, and prophylactic bilateral mastectomies. The patient decided for close surveillance. Her last breast examination and mammogram four years later were normal.
DISCUSSION: LCIS is an incidental pathologic finding. It is nonpalpable and not visible on mammogram. It is usually discovered only after biopsy of a palpable or mammographic visible lesion such as a fibroadenoma. The mean age of presentation is before menopause. The disease is frequently multicentric and bilateral. LCIS is not a direct precursor of invasive cancer, but a marker of a population at increased risk for invasive breast cancer. Several studies have shown that the risk for development of an invasive breast carcinoma is approximately seven to ten times that of the index population. The risk of subsequent cancer development is equal in both breasts. Most carcinomas that develop in women with LCIS are infiltrating ductal, not infiltrating lobular carcinomas. Treatment options for patients with LCIS consist of close follow-up (biannual breast examination, annual mammogram), chemoprevention (Tamoxifen, Raloxifene), and bilateral mastectomies.

OF DYING MUSCLES AND DYING MEN; GAS GANGRENE IN A 68 YEAR OLD MALE. Wazni O, Gurm H, Kranitz D, Departments of Internal Medicine and Emergency Medicine, Cleveland Clinic Foundation, Cleveland Ohio
Learning Objectives;
1 Recognize the presentation of Gas Gangrene.
2 Review the complications of Gas gangrene.
3 Review the descriptions and treatments of gas gangrene in history.
CASE
A 68 year old male presented to the emergency room with knee pain that had been present for about 12 hours. There was no history of antecedent trauma. He was known to have myelodysplasia with chronic pancytopenia.He had had multiple pulmonary infections and was on Home Intravenous Antibiotic Therapy (HIVAT). His other medical problems included insulin requiring diabetes mellitus, chronic obstructive pulmonary disease and congestive heart failure. He was also receiving desferoxamine as chelation therapy for transfusion associated iron overload. His initial examination was unremarkable. The patient was re-examined 15 minutes later as he continued to complain of severe pain. Examination of his right thigh revealed crepitus and marked tenderness. An X-ray confirmed gas along tissue plains. Intra-operatively severe myonecrosis was noticed and he underwent a hip disarticulation. Gram stain revealed multiple gram positive bacilli with a few neutrophils and cultures were consistent with Clostridium *perfringens*. The patient developed shock and acute renal failure and died approximately 36 hours later inspite of aggressive supportive measures.
DISCUSSION
Gas gangrene due to anaerobic bacteria is generally associated with contaminated traumatic injuries. As a complication of 5% of traumatic injuries, crepitus was a much-feared sign in World War I. Spontaneous gas gangrene has been described in association with colonic neoplasia, leukemia and diabetes. The hallmark of the infection is pain,usually localized to an extremity, edema, local pallor and tenderness. Hemmorrhagic bullae often appear with a dirty brown sero- sanguineous discharge. The fluid often stains for clostridia but neutrophils are characteristically absent. Myonecrosis, intravascular hemolysis, renal failure and shock complicate uncontrolled infection. Aggressive surgical debridement and antibiotics are the cornerstone of therapy.

TOO OLD FOR INTRAVENOUS DRUG ABUSE?: AN UNUSUAL CAUSE OF SEPSIS IN THE ELDERLY O.Wazni, H.Gurm, and C. Nielsen. Department of General Internal Medicine, Cleveland Clinic Foundation, Cleveland, Ohio.
LEARNING OBJECTIVES. 1) Recognize the presentation of septic arthritis, 2)Evaluate the cause of Staphylococcus aureus sepsis, and 3) Recognize the social stigmata associated with intravenous drug abuse (IVDA). CASE. A 64-year-old female presented to the emergency department for worsening low back pain. She had presented to the emergency department for chronic low back pain on numerous occasions, for which she usually received narcotic therapy. She had had a laminectomy twenty years ago. Her medications included nifedipine for hypertension and oral codeine for pain .The patient denied the use of tobacco, alcohol, or illicit drugs. She had a temperature of 36.1 degrees C, pulse rate of 130/min,blood pressure of 110/60 mmhg and a respiratory rate of 26/min. A III/VI holosystolic murmur was heard on chest exam. A distended urinary bladder was found on abdominal examination. There was left arm cellulitis and inflammation of the left sternoclavicular joint, the proximal interphalangeal joint (PIP) and the distal interphalangeal joint (DIP) of the right 5th finger and the PIP of the left 3rd finger. Percussion of the lower spine revealed mild tenderness. Neurologic exam revealed 4/5 motor power in the right leg. The range of motion of both lower extremities was normal. Laboratory tests disclosed a white blood cell count of 19,600 with 95% neutrophils and a serum creatinine level of 1.6 mg/dl. A urinalysis revealed >25 white blood cells. Blood cultures and urine cultures were also obtained. The patient was started on intravenous hydration and antimicrobial therapy with vancomycin. On admission a transthoracic echocardiogram revealed 2-3 +mitral regurgitation but no mitral valve vegetations. There were echodensities on the aortic valve consistent with either vegetations or calcifications. Rifampicin and gentamicin were added to the regimen to treat probable endocarditis.Fluid aspirated from the affected joints, blood cultures and urine cultures all grew methicillin sensitive Staphylococcus aureus. Vancomycin was replaced by oxacillin. MRI of the back revealed L5-S1 discitis and a noncompressive epidural abscess. Blood cultures became sterile on the third day of admission. The cause of this patient's sepsis remained elusive until two days prior to discharge when a family member informed us that the patient was in fact an active heroin abuser. The patient was discharged eleven days after admission to a skilled nursing facility for a prolonged course of antibiotics and rehabilitation.
DISCUSSION. Intravenous drug abuse may not be suspected in the elderly as a cause of sepsis. However, some social and clinical findings hinted towards IVDA.The patient lived among drug abusers. She also had frequent ED presentations through which she sought narcotics. Clinically the patient had multiple septic joints including the sternoclavicular joint that is classically noted as being involved in septic arthritis secondary to IVDA. The treatment of Staphylococcal sepsis is founded on early aggressive antibiotic treatment and drainage of abscesses when clinically feasible.

ANTIFREEZE SUICIDE ATTEMPT IN AN HIV POSITIVE MALE. T. WOLFMAN, Department of Medicine, Medical University of South Carolina, Charleston, SC.
LEARNING OBJECTIVE: 1) Recognize clinical features of ethylene glycol ingestion. 2) Diagnose and manage acute ethylene glycol overdose.
CASE: A 33 year old male with HIV for 6 years admitted to ingesting $^1/_2$ gallon of antifreeze (ethylene glycol) in a suicide attempt. Within one hour of the ingestion, he experienced headache, diarrhea, vomiting, diplopia and dyspnea. He was brought to an outside emergency room and received nasogastric lavage with charcoal. Initial arterial blood gas (ABG) was normal, but within one hour the chemistries demonstrated an anion gap of 29 metabolic acidosis with respiratory compensation (CO_2 = 13 mmol/l). The patient (pt.) was transferred to the medical intensive care unit. Physical exam was remarkable for pulse of 106, blood pressure 129/100, oral thrush, cervical lymph nodes, kussmaul's respiration, negative abdomen. Neuro exam demonstrated slurred speech and diplopia, otherwise non-focal. Ethylene glycol level was 248.5 mg /dl. Serum osmolality was 405 mosm/k with an osmolar gap of 108 (normal <10). Urine was positive for oxalate crystals. The pt. was loaded with 10% ethanol intravenously and maintained on a drip. He was emergently hemodialyzed for severe metabolic acidosis. He experienced an episode of seizure-like activity with respiratory arrest and was intubated. EEG and head CT were negative and etiology was thought to be toxic encephalopathy from ethylene glycol ingestion. Once the serum ethylene glycol level reached zero, the ethanol drip was weaned. The pt. was extubated and he made a full recovery. He did, however, continue to express suicidal ideations and was voluntarily transferred to an inpatient psychiatric unit for further evaluation.
DISCUSSION: Ethylene glycol is used in paints, cleaning solvent and anitfreeze. Peak serum levels are 2-3 hours after ingestion and the half-life is 3-8 hours. Ethylene glycol is metabolized into substances that can cause metabolic acidosis, nephrotoxicity, hypocalemia, calcium oxalate crystal deposition, profound central nervous system depression and death. Ethylene glycol and ethanol are both oxidized by alcohol dehydrogenase and when both are present, the alcohol dehydrogenase will preferentially metabolize the ethanol, leaving the ethylene glycol to be excreted unchanged in the urine. For an unresponsive pt., consider ethylene glycol overdose if there is an anion gap acidosis, an elevated osmolar gap, and urine oxalate crystals. Diagnosis can be established by serum ethylene glycol levels and a history of ingestion. Treatment should begin immediately with gastric lavage and charcoal. Use ethanol therapy and bicarbonate for acidosis and keep serum ethanol levels at 100mg/dl until ethylene glycol levels are zero. Hemodialysis is indicated for ethylene glycol levels greater than 50 mg/dl, acute renal failure or intractable acidosis.

COLLEGE FEMALE WITH SYNCOPE AND ANOREXIA NERVOSA. E Whittenburg, C Brands, Wright State University, Dayton, OH.
LEARNING OBJECTIVES. 1) Recognize eating disorders as important causes of syncope in the adolescent and young adult, 2) Recognize diagnostic criteria and multisystem manifestations of anorexia nervosa, and 3) Implement a systematic multidisciplinary approach to outpatient treatment emphasizing the internist's role in monitoring medical stability.
CASE. A 20-year-old local college female presented to the office with five months of lightheadedness, syncope on standing, and palpitations. She admitted to eight months of intentional weight loss through calorie restriction, prolonged daily exercise and occasional laxative and diet pill use. Although she was at 80 per cent of her ideal body weight, she still believed she was overweight. She was otherwise healthy and denied substance abuse. Review of systems revealed amenorrhea for eight months, constipation, bloating, cold intolerance and dry mouth. Exam revealed a 65-inch tall female weighing 100 pounds. Heart rate was 60 beats per minute. Blood pressure was 90/68 mmHg supine and 88/60 mmHg standing. Positive findings included a I/VI systolic murmur, epigastric tenderness, depressed deep tendon reflexes, and brittle hair. The patient failed to gain weight and proved orthostatic during outpatient management and was admitted. On discharge she followed weekly with a psychiatrist who used a cognitive therapy approach and fluoxetine. She also had weekly medical follow-up. She gained weight adequately until final exam time when she became depressed. She began restricting herself to 350 calories per day and became obsessed with weighing herself up to 20 times daily. Symptoms of lightheadedness, palpitations and chest pain returned, and she developed an S4 gallop on cardiac exam. Her electrocardiogram showed marked bradycardia with borderline normal intervals. She also began self-mutilating and was finally readmitted for suicidal ideation.
DISCUSSION. Adolescents and young adults are generally considered to be healthy individuals who, when presenting with illness, may be discounted as having benign disease. Careful history and physical may uncover diagnostic criteria for anorexia nervosa to account for presenting symptoms. These criteria include 1) refusal to maintain body weight at 85 per cent expected, 2) intense fear of becoming fat even though underweight, 3) disturbed body image and 4) absence of three consecutive menstrual cycles. Internists who encounter young adults and adolescents must understand the relapsing and potentially fatal course of this disorder as well as the gastrointestinal, cardiac, endocrine and psychiatric complications that may arise. Internists must also be prepared to provide firm but reasonable guidelines regarding weight gain and nutrition in order to contribute meaningfully to the medical and psychiatric therapeutic plan.

PRIMARY SCLEROSING CHOLANGITIS AND PYODERMA GANGRENOSUM IN A COLLEGE STUDENT WITHOUT INFLAMMATORY BOWEL DISEASE Maria Wright, Department of Internal Medicine, Wright State University, Dayton, OH
LEARNING OBJECTIVES. 1) Evaluate jaundice in a young adult patient 2) Recognize the clinical features of primary sclerosing cholangitis and pyoderma gangrenosum 3) Recognize common extraintestinal manifestations of inflammatory bowel disease.
CASE. A 22-year-old male presented with a one-month history of jaundice. He denied fever, change in weight, pruritus, gastrointestinal symptoms, or risk factors for sexually transmitted diseases. He reported fatigue, easy bruising and frequent epistaxis for six months. He denied prescription or over-the-counter medication use. Family history is negative for liver and gastrointestinal disease. Physical examination demonstrated icteric sclera, jaundiced skin, and hepatosplenomegaly. Stool was brown and hemoccult negative. Laboratory studies revealed a platelet count of 40,000, PT 15.8 seconds with INR 1.9, albumin 2.4, AST 220, ALT 164 and alkaline phosphatase of 465. Iron, copper and autoimmune studies were normal. Viral hepatitis serologies were negative. Abdominal ultrasound and CT scan demonstrated hepatosplenomegaly without cholelithiasis. The patient was referred for endoscopic retrograde cholangio-pancreatography, which revealed beading abnormalities of the intra-and extrahepatic biliary tree, consistent with primary sclerosing cholangitis. Esophagogastroduo-denoscopy and colonoscopy revealed esophageal varices, but no gross or microscopic evidence of inflammatory bowel disease. The patient underwent evaluation for orthotopic liver transplantation and was stabilized on medical therapy. Six months later, he returned for evaluation of lower extremity ulcers. He denied trauma to the area as well as gastrointestinal and systemic symptoms. Physical exam revealed two 2-cm ulcers on his lower extremity with well-defined violaceous borders. Diagnosis of pyoderma gangrenosum was suspected and confirmed by dermatologic consultation. He currently is without gastrointestinal symptoms, awaiting liver transplantation.
DISCUSSION. Primary sclerosing cholangitis (PSC) is an unusual cause of jaundice in a young adult. Patients with PSC characteristically present with pruritus, fatigue and jaundice with a marked elevation of the serum alkaline phosphatase level. The diagnosis of PSC is made by cholangiography, as hepatic histologic biopsy findings can be nonspecific. Three-fourths of patients with PSC have associated inflammatory bowel disease (IBD), which usually precedes the diagnosis of PSC. Pyoderma gangrenosum is likewise closely associated with IBD, and it may precede or coincide with colitis. The skin lesion begins as an erythematous papulopustule, which progresses to an ulcer with violaceous edges and a halo of erythema. Primary care physicians should recognize that PSC, pyoderma gangrenosum and other extraintestinal complications of IBD may run a clinical course independent of the gastrointestinal disease, and may even precede the diagnosis of IBD. This patient's clinical course demonstrates that these entities may even occur without clinical or histologic evidence of IBD.

DISSEMINATED ASPERGILLOSIS IN AN IMMUNOCOMPETENT HOST
Jia-Yia Liu and Gigi Nickas, Harbor-UCLA Medical Center, Torrance, CA
LEARNING OBJECTIVE: Recognize that disseminated aspergillosis can occur in immunocompetent hosts.
CASE: A 68-year-old woman with a history of heart failure, mild renal insufficiency and hyperthyroidism presents with three days of weakness and one week of diarrhea and decreasing urination. Her temperature was 95.2°F, blood pressure 67/33, heart rate 83 and respiration 18. She was thin and somnolent with mild epigastric tenderness. Her blood urea nitrogen was 173mg/dL, creatinine 9.3 mg/dL, and white count was 5.7/mm^3 with 65% neutrophils and 13% lymphocytes. The initial chest x-ray showed normal lung fields, but she developed acute respiratory distress syndrome with worsening gas exchange within a few hours. Broad spectrum antibiotics were started. Hydrocortisone was given for an inappropriately low baseline cortisol with a normal response to cosyntropin stimulation. On day 4 she was febrile and intermittently hypotensive. She developed Candiuria on day 8 and fluconazole was started. HIV test and serial blood cultures were negative for bacteria and fungi. Fluconazole was replaced with amphotericin on day 14. Aspergillus grew from sputum culture on day 21. She developed multi-organ system failure and expired on day 25. Postmortem examination revealed invasive aspergillus in the brain, lung, pericardium, and myocardium.
DISCUSSION: In a review of Medline since 1990, there have been only two reported cases of disseminated aspergillosis in immunocompetent hosts. Aspergillus is a ubiquitous, dimorphic fungus, which is inhaled as spores that germinate into invasive hyphae. Lymphocytes, macrophages, neutrophils and complement are vital host defenses. Predisposing conditions include hematological malignancies, sinus surgeries, HIV infection, organ transplantation and immunosuppressive therapy. The sensitivity of blood cultures is low, while sputum cultures in patients known to have invasive pulmonary aspergillosis are positive less than 10% of the time. Currently the most reliable serological studies are available only as research tools. Chest radiographs may reveal a focal or diffuse process. Definitive diagnosis is by tissue biopsy showing vasculature with invasive hyphae.
Our patient was considered to be immunocompetent, although she received hydrocortisone for 25 days. Her chronic diseases are not usually thought of as predisposing factors for disseminated aspergillosis. Aspergillus fungemia may have been the primary etiology of sepsis or subsequent to treatment with hydrocortisone and broad spectrum antibiotics. She most likely had an immunocompromising factor not identified with routine work-up. This case illustrates the importance of suspecting fungemia in any septic patient failing to improve on broad spectrum antibiotics and without an infection source. Clinicians need to be aware immunocompetent hosts can develop aspergillosis since early diagnosis is vital for successful therapy with amphotericin B.

ACUTE ONSET ALBINISM AND HYPOPITUITARISM IN AN ADULT: A CASE REPORT

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LEARNING OBJECTIVE: To demonstrate that acute onset albinism in an adult may be correlated with systemic disease.
CASE: A 42 year old female was admitted with community-acquired pneumonia. The patient had recently been diagnosed to have 'decreased hormone levels'. She also reported lightening of her skin and hair over the last year.
On examination, the patient was noted to be a thin, pale woman with marked, diffuse hypopigmentation of her skin and irises. She also had snow-white scalp hair, eyebrows and eyelashes. This was in contrast to her photographs from young adulthood in which she was a brunette. Endocrine work-up and lab data revealed that the patient had hypopituitarism with low ACTH (< 1 pg/ml) levels and low FSH and LH levels(4.8 and 3.5 mU/ml respectively). Dynamic testing revealed no growth hormone response to hypoglycemia. However, her TSH and thyroid hormone levels were normal. The patient also had an extensive work up of her anemia that was later diagnosed to be auto-immune hemolytic anemia. No clinical or biochemical evidence of lupus or other rheumatological disease was found. Genetic testing failed to reveal any chromosomal abnormality.
Pituitary imaging revealed changes consistent with an old destructive process of the pituitary with only a sliver of preserved pituitary tissue. The patient was treated with hormone supplementation including cortisone, growth hormone, estrogen and progesterone and antibiotics. With the extensive hormone therapy, her clinical status improved and she progressed from being completely dependent on her family to an independent individual.
DISCUSSION: Vitiligo or localized hypopigmentation may be associated with a variety of systemic diseases including several auto-immune conditions. However, literature search failed to reveal any cases of adult onset, diffuse pigment loss or albinism in a patient with co-existing endocrine deficiency. We present this case to illustrate that in this patient, the syndrome of hypopituitarism and albinism may have a common pathogenetic basis, probably, auto-immune in nature. Also, pigment loss, both localized and diffuse, may be a marker for serious underlying systemic disease.

MULTIPLE LEFT ATRIAL MYXOMAS PRESENTING WITH RIGHT HAND WEAKNESS: FROM DIAGNOSIS TO MANAGEMENT.

R Yuan, S Agha, S Bose, L Blum, J Cleary, C Gruss, Department of Medicine, Norwalk Hospital (CT), Yale University School of Medicine.
LEARNING OBJECTIVES. 1) Consider the diagnosis of atrial myxoma in patients presenting with stroke, and 2) Understand options in the management of multiple atrial myxomas.
CASE. A 63-year-old apparently healthy man suddenly developed right hand weakness while watching television. He had no other symptoms and denied taking any medications. There were no previous medical illnesses except for a detached retina which had been surgically repaired without complication 5 years ago. He did not obtain any routine medical care but denied hypertension, tobacco use, diabetes, hyperlipidemia or other risk factors for stroke. He had never been told of a heart murmur and did not have one at the time of presentation. There were no signs of congestive heart failure on his examination. In fact, his physical exam was unremarkable, except for right hand muscle weakness. His laboratory evaluation was normal. Evaluation of the cause of the stroke included two-dimensional echocardiography which revealed one large left atrial mass. The patient underwent surgery and two separate left atrial masses were found and excised. Pathology confirmed the diagnosis of myxoma.
DISCUSSION. Cardiac myxoma is a rare benign tumor of the heart with a predilection for the left atrium. It usually presents as a classic triad of valvar obstruction, constitutional symptoms and embolism. Rarely, it may be asymptomatic. Although the incidence of left atrial myxoma is very rare, it should be included in the differential diagnosis of stroke in patients without cardiovascular or cerebrovascular risk factors. Diagnosis is often initiated with an echocardiogram. Cardiac myxomas should be surgically removed in order to prevent cardiac and noncardiac complications. Recurrence is not uncommon and regular follow-up should be advised.

A RARE CAUSE FOR HYPOTENSION IN A SARCOID PATIENT
Mary A. Zaky, and Michael P. Carson. Department of Medicine. UMDNJ-Robert Wood Johnson Medical School. New Brunswick, NJ
LEARNING OBJECTIVES. (1) Remember the differential diagnosis for hypotension in sarcoidosis patients. (2) Recognize the presentation of secondary adrenal insufficiency. (3) Manage panhypopituitarism secondary to sarcoidosis.
CASE. The patient is a 37 y.o. male who presented to the emergency room with weakness, fatigue and hypotension. He had been diagnosed with sarcoidosis six months previously after a chest x-ray revealed hilar adenopathy. At that time, pulmonary function studies were normal and transbronchial biopsy revealed noncaseating granulomas. On this admission, he was afebrile, had a BP of 78/63 and an orthostatic rise in his pulse rate of 20 bpm. Exam revealed a mucosal erosion within the left nare. There was no hyper-pigmentation, visual field deficits, thyroid abnormalities or peripheral edema. The lungs were clear and reflexes were normal. Laboratory evaluation revealed normal serum electrolytes, anion gap, and creatinine. Erythrocyte sedimentation rate was 107 mm/hr. Cultures and echocardiogram were normal. The morning cortisol was less than 5 mcg/dl, and an ACTH stimulation test confirmed the diagnosis of adrenal insufficiency. Dexamethasone 4 mg q 6 hours was begun, and was eventually decreased to physiologic replacement doses. Interestingly, the patient promptly developed polyuria with production of 9 liters of urine over a 24 hr period, and his weight decreased by 2 kg over a 36 hr period. A water deprivation test demonstrated that he was unable to maximally concentrate his urine during dehydration (8 hr rise in urine osmolarity from 218 to only 377 mosm/kg with the serum osmolarity remaining stable at 320). With administration of DDAVP, the urine osmolarity rose to 595, and the serum osmolarity fell to 294 mosm/kg. The diagnosis of central diabetes insipidus (DI) was made, and DDAVP therapy was started. We suspected pituitary sarcoid, and assessment of pituitary function revealed a borderline low TSH, low FSH, LH and testosterone levels. MRI of the brain revealed focal thickening and enhancement of the superior pituitary stalk, and scattered foci of abnormal signal intensity consistent with neurosarcoidosis.
DISCUSSION. Hypotension in sarcoidosis patients can be secondary to infection, cardiomyopathy, pericardial disease and primary or secondary adrenal insufficiency. In this patient, neither infectious nor cardiac diseases were found. Therefore, an endocrine etiology was sought. This case demonstrates that patients who have secondary adrenal insufficiency do not always present with frank electrolyte disturbances as they do with primary adrenal insufficiency. Administration of corticosteroids to this patient unmasked central DI. This phenomenon is rare, but has been reported in association with other causes of panhypopituitarism. Ultimately, treatment consisted of thyroid hormone replacement, DDAVP, transdermal testosterone replacement, and high dose corticosteroids for neurosarcoidosis with gradual taper to replacement doses.

NEPHROTIC SYNDROME: DIAGNOSIS AND TREATMENT - B Zimmerman, C Brands, Wright State University Department of Internal Medicine, Dayton, Ohio.
LEARNING OBJECTIVES: 1)Recognize the clinical features of the nephrotic syndrome 2)Highlight causes of this syndrome 3)Review pathophysiology of this process 4)Understand the current recommendations for management of this syndrome
CASE: A 44 year old white male presented to the emergency room complaining of intermittent headaches and disequilibrium. The patient reported dizziness upon standing with increased frequency over the past two weeks. Two months previously the patient reported having two syncopal episodes upon standing. The patient stated that approximately ten years ago he was told he had hypertension and borderline diabetes which were never evaluated or treated. His past medical history was remarkable for a ten year history of low back pain which had been treated with various pain medications including hydrocodone, acetaminophen and ibuprofen. He had a 40 pack year history of tobacco use but denied alcohol or drug use. The patient denied any dyspnea, polyuria, polydipsia, nocturia or hematuria. Vitals: T 99.7, P 74, R 14, BP 200/110. Blood pressure measurements showed no evidence of orthostatic changes. Head and neck exam was unremarkable. Lungs were clear. Cardiovascular exam revealed a regular rate and rhythm with no murmurs, rubs or gallops. No carotid or renal bruits were appreciated. Neurological exam was normal. The lower extremities showed pitting edema to mid-tibia bilaterally. CBC showed a white count of 9.0, hemoglobin 13.5, hematocrit 41.0 & platelets 139. Renal panel showed Na 139, K 3.6, Cl 102, CO2 27, BUN 18, Cr 1.5, glucose 239. UA was remarkable for albumin>100 mg/dl with glucosuria and 6-10 hyaline casts/lpf. Serum total protein was 4.9 and albumin was 2.5. CXR, ECG & head CT were normal. The patient was started on glyburide and captopril. The patient's creatinine rose to 3.1 over 16 weeks. Further evaluation included a 24 hour urine protein of 22 grams, HbA1c 11.5. Total cholesterol was 306. Serum complement levels, hepatitis profile, serum/urine electrophoresis were normal. Ultrasound studies showed no evidence of renal vein thrombosis or renal artery stenosis. A formal opthamalogic exam revealed bilateral diabetic papillitis with moderately severe background diabetic retinopathy.
DISCUSSION: Clinical features of the nephrotic syndrome include sodium and water retention, edema formation and hyperlipidemia. Nephrotic syndrome is a common endpoint of many disease processes. Although the specific etiology is still not fully characterized, it involves a defect in the glomerular basement membrane. Current management of nephrotic syndrome involves establishing a negative sodium balance, judicious use of loop diuretics, ACE inhibitors and steroids. Other more controversial treatments can be offered in refractory cases.