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# MISSOURI HOSPITALIST

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## Hospitalist Update:

## Improving Primary Prevention of Coronary Heart Disease by Increasing Documentation of Framingham Risk Scores

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**BACKGROUND:**
Heart disease is the leading cause of death in both men and women, accounting for nearly one-fourth of the deaths in the USA in 2010 [1]. Coronary heart disease (CHD) is the most common type of heart disease with about 715,000 heart attacks occurring in the United States each year. Startlingly, nearly 75% of these heart attacks are in those without known coronary disease [2]. Thus, primary prevention of CHD is often the responsibility of primary care physicians in the outpatient setting. In 2001, the Adult Treatment Panel III (ATPIII) published clinical guidelines to aid in the primary prevention of CHD [3]. A critical component of these guidelines is the use of the Framingham Risk Score (FRS) to guide treatment. Recent data have shown that improving cholesterol primarily through reductions in

LDL reduces CHD mortality [4]. Aspirin has also shown similar benefits in primary prevention [5]. We observed that the primary prevention of CHD in the outpatient internal medicine resident clinic at the University of Missouri Healthcare System (UMHC) was compromised by inadequate documentation of CHD risk factors and FRS. Further, we hypothesized that increasing the documentation of FRS would improve management of CHD and thereby prevent future coronary events.

**AIM:**

To increase the documentation of the 10-year Framingham Hard CHD Risk Score for patients in the outpatient internal medicine resident clinic at UMHC from 3% in December 2011 to 50% by December 2012.

**METHODS:**

Following approval from the UMHC institutional review board, we performed baseline data collection through a chart review of 60 random patients from the outpatient internal medicine resident clinic at UMHC. Charts were reviewed for documentation of CHD risk factors and FRS in the preceding twelve months. Patients without CHD or CHD risk equivalents, according to ATPIII, were categorized as primary prevention and all others were categorized as secondary prevention. We then reviewed for the percentage of patients undergoing lipid monitoring and percentage with LDL at goal, again according to ATPIII guidelines. We also examined for aspirin prophylaxis according to the US Preventative Services Task Force (USPSTF) guidelines. We compared the incidence of these endpoints between the primary prevention and secondary prevention groups. We then identified stakeholders utilizing fishbone diagramming and developed interventions utilizing effort/yield study [6]. Based on these studies, we implemented a multi-faceted intervention to increase the documentation of FRS [6]. We placed ATPIII Quick Desk Reference handouts at each physician cubicle in the resident clinic and performed serial educational seminars for all internal medicine resident physicians on appropriate use of ATPIII guidelines and documentation of FRS and treatments. Four months after our intervention, we conducted follow-up data collection on 60 random patients in an identical manner as our baseline data collection. After follow-up data collection, we compared incidences between the pre- and post-intervention primary prevention groups. Data for each endpoint were presented as percentage of patients from each group positive for variable in question. Statistical analyses of our baseline and follow-up data collections were done using chi-square test with p value $<0.05$ considered significant.

**RESULTS:**

Our baseline data collection revealed that only 3% of primary prevention patients had documentation of their FRS and only 18% of patients had their CHD risk factors documented. Further baseline data analysis revealed that primary prevention patients were significantly less likely than secondary prevention patients to have lipid monitoring performed, 59% versus 83% respectively. Additionally, the primary prevention group had significantly fewer patients with LDL at goal, 61% compared to 77% respectively. Finally, primary prevention patients were significantly less likely to be on appropriate aspirin prophylaxis with only 55% of the primary prevention group on appropriate aspirin prophylaxis compared to 87% of the secondary prevention group.

After our intervention, we found significant improvement in the documentation of FRS with an increase from 3% at baseline to 34% of patient charts having FRS documentation (p value $<0.001$).

Documentation of CHD risk factors also showed an increased trend from 18% to 37% but this difference did not achieve statistical significance (p value 0.07). Importantly, the percentage of primary prevention patients who underwent lipid monitoring increased significantly from 59% to 90% (p value <0.001). Percentage of patients with LDL at goal increased to 78% and appropriate aspirin prophylaxis increased to 76% but neither result achieved statistical significance.

**CONCLUSIONS:**

A quality improvement process to improve the primary prevention of CHD at the UMHC outpatient internal medicine resident clinic achieved a significant increase in FRS documentation through resident education and quick reference postings. This improvement significantly increased incidence of lipid monitoring and showed a trend towards increasing the percentage of patients with LDL at goal and on appropriate aspirin prophylaxis. Repeated cycles of quality improvement and hard changes to the electronic medical record are needed to achieve the stated goal of FRS documentation and significantly increase the adherence to ATPIII and USPSTF guidelines for CHD prevention.

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# Case Report

## 65-year-old male patient with left sided headache and orbital pain of 2-3 months duration


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**Introduction:**

Despite advancement of neurotechnology and neuroimaging, detailed history and examination remain the most important tools for diagnosis of unilateral headache and orbital pain. Headache is a common symptom among all age groups with a considerable array of differential diagnoses. Tolosa

Hunt syndrome is a rare cause of unilateral headache and orbital pain. High clinical suspicion based on clinical presentation, thorough history and physical examination is crucial for diagnosis.

**Case presentation:**

A 65-year-old male patient presented with 2-3 month history of excruciating, sharp, left-sided headache and orbital pain involving the ipsilateral forehead, eye and cheek above the upper jaw, which did not cross the midline. It had a gradual onset, starting as mild intermittent pain and progressing to a continuous pain with frequent spikes lasting 4-6 hours each. These attacks became more frequent and intense during the two weeks prior to presentation. The pain was not affected by posture and did not radiate. It was, however, associated with photophobia, phonophobia, ipsilateral conjunctival redness and tearing, nasal discharge and hypersensitivity to touch over the distribution of pain. There were no clear relieving factors. There was no history of recurrent headaches, fever, neck rigidity, jaw claudication, scalp necrosis, skin rash, blurring of vision, double vision, facial asymmetry, limb weakness or numbness or other localizing symptoms.

Physical examination revealed mild left upper eyelid ptosis, eyelid swelling, conjunctival erythema, and tearing. There was mild weakness of the lateral rectus muscle on abduction suggesting mild left abducens nerve palsy and hyperesthesia over the left ophthalmic and maxillary nerve distribution. The pupils were symmetric in size and reactivity. The examination was otherwise unremarkable, including funduscopic examination and remaining extraocular motility. Anhidrosis was not appreciated.

The differential diagnoses initially considered were daily persistent headache, ophthalmoplegic migraine, cluster headache, hemicrania continua, trigeminal neuralgia, temporal arthritis, early herpes zoster (HZV), diabetic neuropathy, Horner's syndrome secondary to mass or carotid dissection, and cavernous sinus thrombosis or tumor.

Laboratory studies including Complete Blood Count, Comprehensive Metabolic Panel, Thyroid Stimulating Hormone, Angiotensin-Converting Enzyme level, Hemoglobin A1C, HZV serology, C-Reactive Protein, and vasculitis screen including ANA were normal. The Westergren Erythrocyte Sedimentation Rate (ESR) was minimally elevated at 40 mm/hr (normal 0-20 mm/hr). Chest X-Ray, Head and Neck CT scan, CT angiography with venous phase and MRI also were normal. Temporal artery biopsy was declined by the patient.

The patient was treated with numerous analgesics including narcotics and Non-Steroidal Anti-Inflammatory Drugs, and additionally he received 100% oxygen which did not alleviate his pain.

Based on distribution of the pain, duration, evidence of involvement of cavernous sinus structures including hyperesthesia over left ophthalmic and maxillary nerve distribution, left abducens nerve palsy and ptosis likely secondary to involvement of oculomotor/sympathetic fibers around carotid artery and exclusion of other causes, Tolosa Hunt syndrome was proposed. High dose intravenous glucocorticoids were initiated and the patient improved dramatically within 12 hours of treatment.

**Discussion:**

Tolosa Hunt Syndrome (THS) is a rare cause of painful ophthalmoplegia caused by idiopathic noncaseating granulamatous or nongranulomatous inflammation of the cavernous sinus or superior

orbital fissure.[1,2] It is a part of a continuum of idiopathic orbital inflammation, also known as idiopathic orbital pseudotumor, with which it shares histopathological and clinical features, and is distinguished only by its unique anatomic localization to the cavernous sinus. THS has an estimated incidence of 1 per million per year, affecting any age group, but is rare before the age of 20.[1,2]

Clinically, THS is characterized by acute onset unilateral, rarely bilateral, periorbital pain that is described as "severe," "intense," or "lancinating." It often resolves spontaneously but tends to have episodes of relapse and remission.[3]

The pain frequently extends into retro-orbital, frontal and temporal regions and features of cavernous sinus structure involvement may provide clues to diagnosis. Key signs include ophthalmic and maxillary nerve involvement manifesting as facial paresthesia and loss of corneal reflex, cranial nerves III, IV, VI involvement evidenced by ophthalmoplegia, diplopia, miosis and ptosis, sympathetic fiber involvement appearing as ptosis with or without features of orbital involvement such as lid swelling, proptosis, orbital pain and vision loss. There is no pathognomonic feature of THS and all these signs can be seen with other cavernous sinus pathology such as thrombosis, tumor, fistula or infections. Furthermore, THS can mimic temporal arteritis especially if the pain occurs before cranial nerve involvement and ESR is elevated (which is seen in 45% of THS cases). On the other hand, eye pain, diplopia and cranial nerve involvement are rare in temporal arteritis.[9] Accordingly, THS is a diagnosis of exclusion.[1,4] Extracavernous extension of inflammation has been infrequently reported in the form of mandibular and facial nerve involvement, but no systemic involvement has been reported.[2,3,5]

While THS is considered a benign condition, permanent neurologic deficits like vision loss and cranial nerve palsies do occur, and relapses are common, often requiring prolonged immunosuppressive therapy.[1,2,3]

The International Journal of Headache/International Classification of Headache Disorders 2nd edition (ICHD-2) criteria for THS include:[4]

- One or more episodes of unilateral orbital pain persisting for weeks if left untreated.
- Paresis of one or more of the third, forth, and/or sixth cranial nerves and/or demonstration of granuloma by MRI or biopsy.
- Paresis coincides with the onset of pain or follows it within 2 weeks.
- Pain and paresis resolve within 72 hours when treated adequately with glucocorticoid.
- Other causes have been excluded by appropriate investigations.

Although painful ophthalmoplegia is a feature of THS, most patients (over 75 percent) who present with painful ophthalmoplegia do not have THS.[2] Diagnostic testing, including neuroimaging by angiography and MRI brain and orbit should be performed to rule out other causes.[1,2] Thin-slice high –magnetic field MRI of the cavernous sinus with fat-suppressed cuts of the orbits is the modality of choice looking for typical inflammatory changes in the cavernous sinus, superior orbital fissure, and/ or orbital apex. MRI findings of THS include enlargement of the cavernous sinus with abnormal tissue, abnormal convexity of the wall of the cavernous sinus and focal narrowing of the intracavernous internal carotid artery.[2] However, these findings are not specific to THS and normal images do not exclude the diagnosis.[1,2,3] Based on MRI findings, THS can be classified as benign with normal MRI scan, and inflammatory with classic inflammatory features on MRI scans.[6] In a meta-

analysis, approximately one-third of clinically defined THS had normal MRI findings, but a defined MR protocol has yet to be determined.[7] Reliable detection is complicated by the fact that differentiation of the normally enhancing venous space in the sinus from the contrast enhancement of granulomas may be limited with the conventional spin echo MRI and thus requires specific imaging modalities.[8]

Biopsy of the lesion may be required to confirm the diagnosis; however, the technical difficulty of cavernous sinus region biopsies limits its usefulness. Instead, a trial of glucocorticoids is usually performed and rapid response with dramatic decrease in pain occurs. More aggressive testing, including biopsy and CSF evaluation, to exclude other causes is recommended if symptoms are progressing, atypical or recurrent. This is particularly the case if the patient has failed treatment with glucocorticoids or if glucocorticoid efficacy is lost soon after an initial response.[1,2] Even with careful adherence to clinical criteria and diagnostic evaluation, misdiagnosis can still occur; consequently, follow-up is required to exclude other causes of painful ophthalmoplegia.[2,4]

Glucocorticoids have altered the course of the disease by providing significant pain relief within 24-72 hours of therapy initiation. However, there are no clinical data as to whether glucocorticoids hasten recovery of associated cranial nerve palsies.[1,3] Additionally, no standard dose or duration has been indicated in literature. A dose of 1mg/kg/day of methylprednisone tapered slowly was well received.[5]

Although the glucocorticoid trial is instructive, an initial clinical or MRI response is not diagnostic since other entities, such as lymphoma and vasculitis, also may respond clinically and radiographically to glucocorticoid therapy.[2,3]

Ophthalmoparesis usually requires weeks to months for resolution.[6] In some cases, the ophthalmoparesis may not completely resolve depending on the degree of inflammation and the aggressiveness of therapy. For refractory cases, azathioprine, methotrexate, or radiation therapy has been employed.[1]

Recurrences occur in about one-half of reported patients over an interval of months to years.[2] Ipsilateral, contralateral, and bilateral relapses have been reported. Relapses require repeated investigations to rule out inflammatory and neoplastic disorders such as sarcoidosis, Wegener's granulomatosis, and lymphoma.[2]

**Conclusion:**

THS is a rare cause of relapsing remitting painful ophthalmoplegia and unilateral headache secondary to idiopathic inflammation of cavernous sinus, superior orbital fissure, or both. THS can mimic many other conditions. A dramatic response to systemic glucocorticoids is helpful in the diagnosis. An MRI scan is the modality of choice for investigation, but the absence of MRI findings does not exclude THS. High index of suspicion and close follow up are important for accurate diagnosis.

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# High Vitamin B12 Level

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Vitamin B12/cobalamin functions as an important co-enzyme in the human body and is essential for purine and pyrimidine synthesis. Vitamin B12 deficiency is a very well recognized clinical entity but the conditions leading to elevated levels of vitamin B12 are generally not; hence the purpose of this article is to discuss such etiologies. Understanding the mechanisms will necessitate a brief review of vitamin B12 metabolism, presented below.

Under normal physiological conditions, dietary cobalamin binds to haptocorrin (HC) in saliva to be transported to the duodenum where free cobalamin is released. The free cobalamin in the duodenum binds to intrinsic factor (IF) forming a complex which is taken up by the intestinal mucosa to be ultimately released into the circulation. The majority of cobalamin in circulation is bound to haptocorrin (HC) and only a small portion (5-20%) is bound to transcobalamin (TC II). The plasma half -life (t ½) of TC II is short, only 40min-5hrs, whereas it is a long, 6-9 days for HC. When transcobalamin passes through enterohepatic circulation it is taken up by the liver for storage and is later released into bile.

| Bound protein/form | Transcobalamin (TCII) | Haptocorrin (HC) |
|---|---|---|
| Plasma t ½ | 40min-5hrs | 6-9 days |
| Site of synthesis | Liver, intestine, endothelium | Salivary glands, gastric mucosa |

The reference range for vitamin B12 levels is 200-900 pg/mL (picograms per milliliter) and elevated cobalamin levels can be seen in various malignancies including hematological conditions like myeloproliferative disorders, renal and liver disorders.

| | |
|---|---|
| Hematological etiologies | Myeloproliferative conditions<br>-Chronic Myeloid Leukemia (CML), Polycythemia Vera (PV), Hypereosinophilic syndromes (HES), Chronic myelomonocytic leukemia (CMML)<br>Myelofibrosis<br>Acute Promyelocytic leukemia (APL)- less commonly other types of Acute myeloid leukemia (AML) |
| Malignancy | Solid tumors like Hepatocellular carcinoma (HCC), Breast ca, Renal cell ca, Colon ca, Gastric ca & any cancer with liver metastases |
| Hepatic etiologies | Hepatitis, Cirrhosis, Hepatocellular carcinoma |
| Others | Chronic kidney disease, Cystic Fibrosis (due to liver injury) & Iatrogenic - VitB12 administration |

Mechanisms of elevated vitamin B12 levels in the above conditions include:

1. Excess Haptocorrin production

Markedly elevated B12 levels (up to 10x!) are often seen with myeloproliferative disorders as listed in the table above. The expanded myeloid cell population in myeloproliferative conditions is thought to be leading to an increase in HC level thereby causing elevated B12 levels. Elevated B12 levels are also more commonly seen in Acute Promyelocytic leukemia (APL) than other types of Acute myeloid leukemia (AML).

2. Release from hepatocytes and reduced uptake

In cases of acute hepatitis, cirrhosis, HCC or metastatic disease of the liver, pathogenesis is thought to be related to both hepatocyte cell injury causing the release of stored vitamin B12 into the circulation along with decreased ability of the damaged hepatocytes to take up the circulating vitamin B12.

3. Excess of transport proteins—both transcobalamin and haptocorrin (TCII and HC)

In several solid tumors as listed above, excess transport levels were thought to be the culprit for elevated B12 levels. The excess transport proteins were both as a result of exogenous production by the tumor directly or by the indirect effects of tumor through stimulation of leukocytes to produce TCII and HC.

As per this overview, we conclude that a thorough medical investigation is necessary for patients with elevated B12 levels including a careful history & physical exam, basic labs including CBC with manual differential count, review of peripheral smear and CMP to carefully look at liver function as well as further work up as guided by these initial steps.

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# Diagnostic Dilemma

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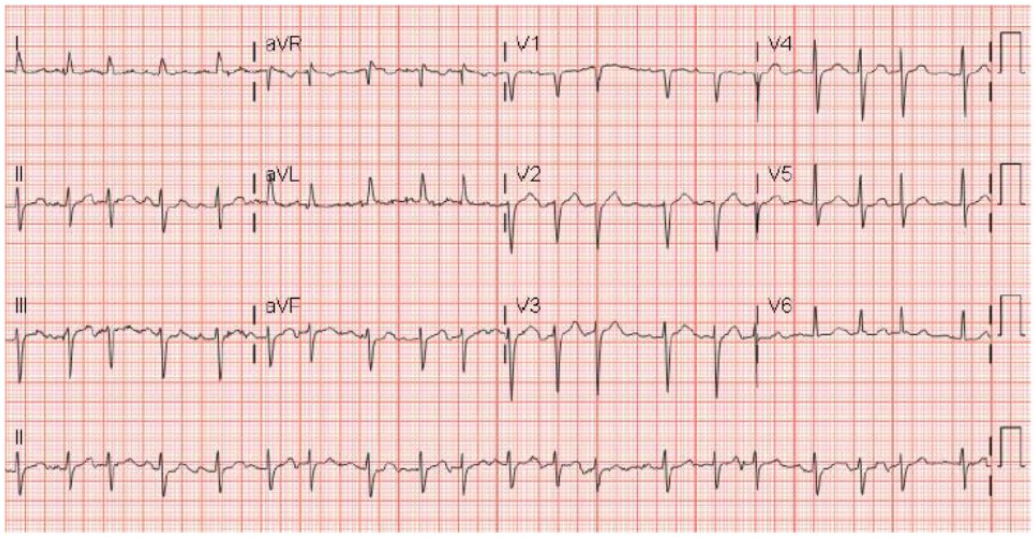
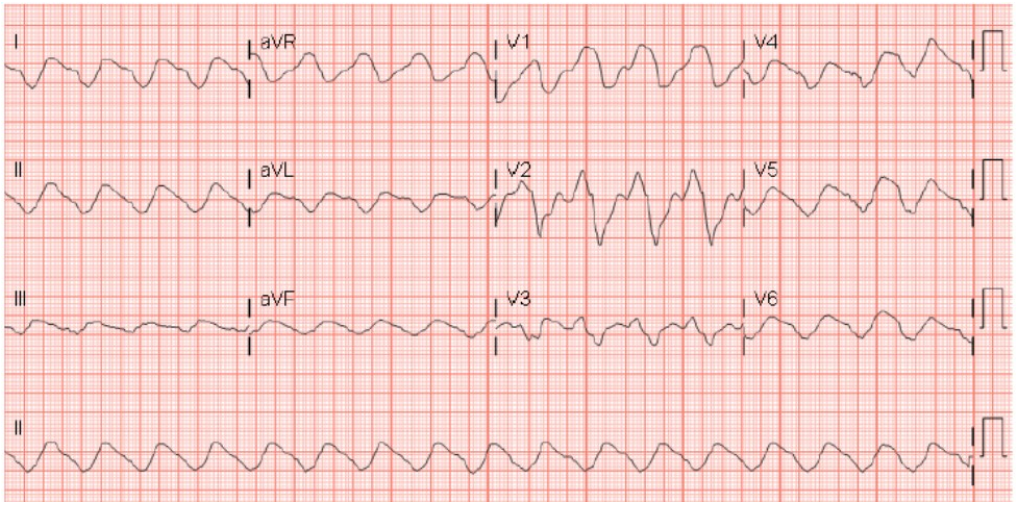
**Questions:**

1) A 68-year-old male was admitted for increasing shortness of breath. Past medical history was significant for COPD, rheumatoid arthritis and coronary artery disease. A few hours after admission he became increasingly short of breath. An EKG obtained at that time is shown below. HR was 140 bpm, BP 100/70 mmHg, pulse oximetry sat was 88% on 6 liters of oxygen via nasal cannula. What is the next best step in patient management?




A) Administer adenosine
B) Perform DC cardioversion
C) Administer amiodarone
D) Improve oxygenation

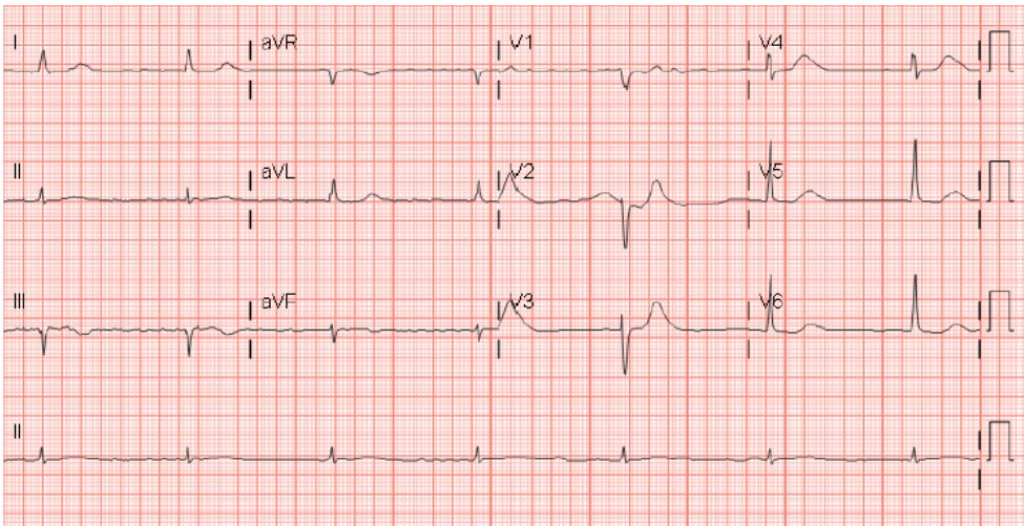
2) A 45-year-old African American male presents to ER with increasing weakness. He has CKD stage V on chronic hemodialysis. His medications include lisinopril, amlodipine and clonidine for hypertension. EKG obtained at presentation is shown below.



What is the next best step in patient management?

A) Obtain electrolytes
B) Administer calcium gluconate
C) Emergent hemodialysis
D) IV magnesium

3) A 55-year-old male with history of congestive heart failure, hypertension and ischemic cardiomyopathy is admitted for nausea, vomiting and diarrhea of 2 days duration. EKG was obtained and is shown below.




What is the most likely cause of the EKG findings?

A) Electrolyte imbalance
B) Drug related
C) Myocardial ischemia
D) Pericardial effusion

Answers on page: 12

# ASK A PATHOLOGIST

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**QUESTION:** My patient has a palpable neck mass that is suspicious for malignancy, and I am considering ordering a fine needle aspiration (FNA) versus a core needle or open biopsy. Which type of biopsy will have the fastest result from pathology?

**ANSWER:** Fine Needle Aspiration (FNA) is the most rapid method of obtaining a tissue diagnosis. FNAs are performed manually by pathologists, clinicians or surgeons for the assessment of palpable masses, and may also be performed under ultrasound or CT guidance for non-palpable lesions. FNA biopsy can be useful in the diagnosis malignancies, certain infections (fungal, viral, protozoal), inflammation (granulomas, sarcoidosis) or infiltration (amyloidosis). The advantages of FNA biopsy can be summed up in the acronym **SAFE**: **S**imple; **A**ccurate; **F**ast; **E**conomic.

For palpable lesions, FNA can be performed on an outpatient basis or at the patient's bedside in the hospital. It has the best safety record of any method of procuring tissue for a morphologic diagnosis. No other biopsy can be processed as rapidly as an FNA biopsy—open tissue or core needle biopsies must be fixed in formalin and embedded in paraffin for several hours and even intra-operative frozen sections take several minutes to freeze and stain with H&E. FNA samples are air-dried and stained with Diff Quick stain which requires about one minute, and slides can then be examined wet without a cover slip at the time of FNA. Microscopic evaluation by a pathologist at the time of FNA is referred to as **R**apid **O**n-**S**ite **E**valuation or **ROSE**, and can be used to confirm that the obtained sample is adequate/diagnostic to rapidly triage additional testing that may be needed for that specimen (for example, a portion might be sent for flow cytometry if the specimen suggests a lymphoproliferative process), and to give the patient and clinician a preliminary diagnosis at the time of FNA.

Risks of FNA include pain and anxiety, hematoma or minor bleeding, vasovagal reaction, nerve damage, infection, and tumor necrosis. The needles used for FNA are usually 23 gauge or smaller, the same or smaller than needles used for routine phlebotomy. Anesthetic is generally not required. Non-image guided FNA should not be performed if the patient has a bleeding disorder, has a skin infection at the FNA site, is extremely uncooperative or agitated, or if the mass is not palpable.

Send your questions to coberlye@health.missouri.edu to be published in future editions of the Missouri Hospitalist.

# ID Corner

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## Laboratory Diagnosis of Infections

Ever wonder what type of culture or test to order to diagnose an infection? Well, the IDSA has published guidelines which are arranged by anatomical site, with lots of tables that tell you what culture, Smear, PCR or serologic test to obtain to optimally make a diagnosis of an infection:

Baron EJ et al. A guide to the utilization of the microbiology laboratory for diagnosis of infectious diseases: 2013 recommendations by the Infectious Diseases Society of America (IDSA) and the American Society for Microbiology (ASM). Clin Infect Dis 2013; 57:485.

http://www.idsociety.org/uploadedFiles/IDSA/Guidelines-Patient_Care/PDF_Library/Laboratory%20Diagnosis%20of%20Infectious%20Diseases%20Guideline.pdf

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# Diagnostic Dilemma

**Answers:**

**1) D**
ECG reveals multifocal atrial tachycardia. Multifocal atrial tachycardia is due to increased automaticity. It is diagnosed with the following criteria: heart rate >100 bpm and 3 different morphology of P waves. Varying PP, PR and RR intervals are also noted. Treatment includes treating underlying cause of hypoxia.

**2) B**
Sine wave pattern suggestive of hyperkalemia is seen on EKG. Sine wave pattern indicates potassium level > 8 meq/dl. Although options A and C are reasonable, the first step would be to administer calcium gluconate to stabilize membrane potential followed by emergent hemodialysis.

3) **B**
This EKG shows atrial fibrillation with junctional or AV nodal escape rhythm. Digitalis toxicity should be suspected whenever a patient with atrial fibrillation has regular bradycardia. Digitalis increases the refractoriness of AV node and in digitalis toxicity can cause a complete heart (AV nodal) block and AV nodal escape rhythm.

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# CONFERENCE CALENDAR

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Mayo Clinic Hospital Medicine: Managing Complex Patients
Dates: November 6 - 9, 2013
Venue: Loews Ventana Canyon Resort, Tucson, Arizona

14th Annual Southern Hospital Medicine Conference
Dates: November 7 - 9, 2013
Venue: Hyatt Regency New Orleans, New Orleans, Louisiana

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