

The LAB Report

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Have you checked your iron lately? Results may be surprising.

Exciting new equipment: Immulite 2000, i-STAT

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LERC: Honoring deserving Lab employees



Managing Editor

Martin Renaldi, mrenaldi@mgh.org

Associate Editor

Michele Toyras, mtoyras@mgh.org

HYPERCOAGULABILITY EVALUATION

By SueAnn Hampton, MT (Section Head)

Thromboembolic disease is a serious and potentially fatal disorder affecting five to six million people per year, and is one of the most common causes of death in North America.

Genetic, acquired and physiologic risk factors can all interact to increase a patient's risk of developing thrombosis. A hypercoagulable state is one in which, due to an inherited or acquired disorder, there's an increased propensity to form



SueAnn Hampton

thrombosis. There is no single test for diagnosing hypercoagulability, rather a panel of selective tests is used.

Genetic risk factors include deficiencies of protein C, protein S, antithrombin, plasminogen, and protein C resistance (factor V Leiden) as well as prothrombin G20210A mutation, and hyperhomocysteinemia. Factor V Leiden causes >95 percent protein C-resistant cases and is the most common known hereditary predisposition to venous thrombosis. Protein C, protein S, and antithrombin deficiencies are not common, but the risk associated with them can be high, particularly for antithrombin.

Hyperhomocysteinemia results in a three- to five-fold increase in the risk of coronary artery disease. Severe thrombophlebitis and recurrent thrombosis have been associated with a congenital deficiency of plasminogen. Antiphospholipid antibodies include lupus anticoagulants (LA) and anticardiolipin antibodies (ACA). The antiphospholipid syndrome (APS) is character-

ized by arterial or venous thrombosis, recurrent fetal loss, or thrombocytopenia. Venous thrombosis is the most common symptom. Approximately 25 percent of patients with APS will have LA, 25 percent will have ACA, and 50 percent will have both.

Acquired and physiological risk factors for deficiencies of Protein C, S, and antithrombin are more common than genetic ones. They may be environmental or pathological and controlled in part by the patient. Examples include: immobilization, smoking, obesity, gender, age, surgery or trauma, fractures of the pelvis, hip, or leg, CHE, DIC, liver or kidney disease, and vitamin K deficiency, as well as estrogen, L-asparaginase, heparin, or coumadin therapy. Acquired etiologies for deficiencies should be excluded before concluding a patient has a hereditary deficiency.

Hyperhomocysteinemia may be genetic or acquired. Causes for acquired include vitamin B12, B6, or folate deficiency, renal failure, hyperthyroidism, as well as methotrexate, theophylline, or phenytoin treatment.

The Dilute Russell Viper Venom time is very sensitive to any interference with phospholipid

Continued on last page

Frequency of occurrence of thrombotic patients

Activated Protein C Resistance (APCR)	20-40%
Protein C & S	2-5%
ATIII	1-2%
Plasminogen	1-2%

Frequency of occurrence in general population

Factor V Leiden	3%
Prothrombin Variant 20210	2.3%
High homocysteine	5%
Low Protein C & S	0.2-0.4%
Low antithrombin	0.2%

Hereditary Hemochromatosis

By Cory Blight, MT (ASCP)

Have you ever had iron studies done? If you haven't, you may want to keep reading. Hereditary hemochromatosis is a disease characterized by increased absorption of iron. This increased absorption can lead to accumulation of iron in various body tissues. The amount of dietary iron intake, number of blood donations, and menstruation also play a role in the severity of iron overload. The accumulation slowly builds over time and many people are asymptomatic until they are middle aged (approximately 40 to 60 years). In some instances, however, people in their 20's become symptomatic. Early symptoms may include high concentrations of liver enzymes, impotence in men, irritability, joint pain, and fatigue. If the disease isn't treated, complications result such as cirrhosis of the liver, bronze skin pigmentation,

diabetes mellitus and cardiomyopathy. This disorder can also prove to be fatal.

Hereditary hemochromatosis in Caucasians is the most common genetic disorder in the USA affecting one in 300 people. One in every nine persons is a carrier of the gene that causes the disorder. Currently screening for this disease is not done as part of a routine health exam or check-up.

A diagnosis of hereditary hemochromatosis is often made by the use of the transferrin saturation test. Two transferrin saturation levels of greater than 60 percent in males and 55 percent in females suggests hemochromatosis. The most common confirmation test is a liver biopsy. In recent years a genetic test has also enhanced the diagnosis of hereditary hemochromatosis. Other useful tests include serum iron and the serum ferritin.

One of the treatments for this disorder is very simple but highly effective. If diagnosed before organ damage occurs, hereditary hemochromatosis can be treated with therapeutic phlebotomy and the patient's lifespan can be normal. However, if organ damage has already occurred, varying degrees of some or all of the above-mentioned symptoms and conditions are likely to occur. It seems strange that a disease that has such potential for damaging health effects can be treated so simply if caught early enough.

That being said, when was the last time you had any iron studies done? I had mine done and guess what? I am currently going through monthly therapeutic phlebotomies until my ferritin level is at an acceptable level. Fortunately, I was diagnosed early and show no signs of damage. It is likely that I will undergo therapeutic phlebotomy three to five times per year, but when you think about the consequences of not being diagnosed early, having a phlebotomy every once in awhile doesn't sound bad at all.



Cory Blight

Lab gains bedside testing analyzer

By Martin Renaldi, MT (ASCP), MPA

The MGHS Laboratory's Point-of-Care department is pleased to announce the arrival of a new bedside clinical analyzer capable of informing the caregiver of quick and reliable measurements of patient electrolyte and glucose levels.



Martin Renaldi

The analyzer is currently in use by health-care professionals in the Neonatal Intensive Care Unit. Because of the analyzer's portability, quick turn-around-time, and the minimal blood volumes needed for testing, the *i-STAT* became a perfect option for the care of NICU patients as well as for neonatal transports to and from our facility.

The i-STAT is a hand-held, portable analyzer

The *i-STAT* is a hand-held, portable analyzer used in conjunction with reagent cartridges for the simultaneous quantitative measurement of PH, PCO₂, PO₂, sodium, potassium, ionized calcium, glucose, and hematocrit in whole blood samples. Data can be generated in approximately 20 seconds for glucose and two to three minutes for electrolytes and blood gases. Of course, only qualified and certified users are permitted to operate the analyzer in accordance with our facility's policies and procedures. The main campus laboratory is able to retrospectively monitor and evaluate every transaction of the analyzer in order to comply with regulatory agency mandates regarding the use of such devices.

For more information regarding this new technology please feel free to call the main campus laboratory's Point-of-Care department at 906-225-3051.

Lab Blab

By Cory Blight, MT (ASCP)

These days, it seems as if change is the only constant! This holds true for Marquette General's lab, and there are some newcomers I'd like to formally welcome to the team.

Joining us in Phlebotomy are Kelly Slattery, Bobbie Bolitho, Jonelle Easterwood and Elaine Stolpe. Brian Gonyou has moved on to our Emergency Department. Dean Larson and Sue Palkki are now a part of Transfusion.

Geraldine Giletto filled a position in the Core Lab and Heather Asgaard has replaced Kara Kalio in the front office. Kara accepted a position in the Medical Records Department here at MGHS.

In the "little laboratorians" category there have been several new arrivals. Lori (Core Lab MT) and Mark Young welcomed their baby boy Travis. Terri Field (Transcription) announced the arrival of Bryce Alan. Michelle Staples (Specimen Processing) gave birth to Aberdeen. Finally, Jennifer Beauchamp (Transfusion) had a girl, Halle Beauchamp.

Toxoplasma: Fetal Foe

By Michele Toyras, MT (ASCP)

Lately I've wondered if there's something in the water around here. Every time I turn around there are shrieks of joy as yet another one of my beloved coworkers is blessed with pregnancy. Or were those labor pains I heard? If this keeps up, I'm not sure if anyone will be around to run the lab in a few months. (The good news is that neither Marty nor Cory is pregnant.) But for those who are expecting, I felt a review of toxoplasma is appropriate.

Toxoplasmosis is an infection caused by the single-celled parasite *Toxoplasma gondii*. Although many people may have had a toxoplasma infection, very few become symptomatic because the immune system usually keeps the parasite from causing illness. However, toxoplasmosis acquired *in utero* has resulted in blindness, encephalomyelitis, mental retardation, convulsions, and death in infected neonates.

Toxoplasmosis can be treated with antibiotics, but early detection is crucial for effective therapy. Complete eradication of the parasite becomes difficult after long-term chronic infection. Serologic confirmation of toxoplasma antibody is indicative of exposure to the parasite and is the most widely accepted means to determine immune status. If IgG is present at high levels, this indicates recent seroconversion or a persistent high level of immunity. If a significant level of IgM is detected, this may indicate a current or recent infection.

Toxoplasmosis can be treated with antibiotics, but early detection is crucial for effective therapy

So what's the problem? Cats are the parasite's main host. Anyone who is pregnant and a cat owner needs to be extremely careful. A fetus can be infected when the mother ingests oocysts.

Now see if you can follow this somewhat confusing life cycle, starting with the oocyst life cycle. The oocyst phase of the life cycle

occurs in cats only. A host becomes infected by ingesting oocysts. Bradyzoites and sporozoites from the oocysts penetrate the cells of the small intestine. The parasite then undergoes asexual and sexual reproduction and oocysts are produced. The oocysts are passed in the feces and sporulate in about 24 hours. The cycle is complete when a host ingests the oocysts. Basically, a human can be infected when cleaning a litter box. Humans can also be infected by ingesting undercooked meat from an infected animal. This life cycle occurs in cats and other animals. A host is infected by eating bradyzoites in the tissues of a reservoir. The bradyzoites or sporozoites infect macrophages in the mucosa of the small intestine, producing tachyzoites. The tachyzoites reproduce asexually rapidly, causing cell death. The tachyzoites are distributed throughout the host's body. After several weeks, division slows, producing zoitocysts filled with bradyzoites. The cycle is complete when another host eats the bradyzoites. *(A prize goes to the first person that knows exactly what a bradyzoite, sporozoite, tachyzoite, oocyst, and a zoitocyst are.)*

Now here's something for the funny bone. A scientist in the UK claims that rats who become infected by toxoplasma develop an attraction to cats. The parasite infects the brain and alters the brain chemistry so that the rats become more sedate and develop an attraction to the scent of cats. I personally think this man has been spending far too much time with his rats. He also says that 87 percent of the population in France carries the parasite and this may explain the reports of altered personality and IQ levels in some humans. And finally, he also has linked toxoplasma to hyperactivity in children from Brazil. We shall leave him anonymous, as this is probably best for his career.



Michele Toyras

Laboratory Employee Recognition Committee LERC

By Caprice Feys, MT
LERC Committee Member

Many of our fellow lab employees have inquired about the LERC; therefore we thought we'd explain ourselves. According to policy, the LERC has been developed to acknowledge Marquette General Laboratory employees. The LERC is a group of five non-supervisory lab employees and one supervisory lab employee who consider nominations and vote to determine who will receive the Laboratory Cell Award.

There are numerous Lab employees who are deserving of this honor

Lab employees who go above and beyond their normal job description; demonstrate excellence in customer service; and encourage other lab employees to do the same while maintaining enthusiasm and dedication are eligible. Anyone in the lab can nominate a fellow employee for the award; however, a nominee can only be nominated once a year and can not be in a supervisory position.

The Cell Award winner is determined quarterly, although depending on the nominations, there may not be a winner each quarter. Cell Award winners are then eligible for Laboratory Employee of the Year – a distinction also decided by the LERC.

As you well know, there are numerous MGHS Lab employees deserving of this honor. To nominate a fellow employee, simply fill out a nomination form and return it to the lab nomination box. The LERC appreciates your support of the Cell Award. It's this type of recognition that makes the MGHS Lab an even greater place to be employed.

New Instrumentation: The Immulite 2000

By Crystal Skelton, MLT, MT student & Rebecca Piippo, MT student

Marquette General's Lab is proud to announce our newest instrument, the Immulite 2000 by Diagnostic Products Corporation. The Immulite 2000 is an automated immunoassay analyzer that performs chemiluminescent assays with continuous random access. The instrument has a great menu of tests that will enable us to complete more testing in-house. Areas of testing include infectious disease, diabetes management, bone metabolism, adrenal/pituitary function, reproductive endocrinology, thyroid function, and allergy testing.

The Immulite 2000 will replace our existing TOSOH system, moving PSA, PAP, and cortisol to the new analyzer. The first tests installed on the Immulite 2000 will be intact PTH, testosterone, beta-2 microglobulin, anti-thyroid peroxidase, anti-thyroglobulin, thyroglobulin, homocystine, erythropoietin, insulin, C-peptide, human growth hormone, insulin growth factor, and IgE.

Future areas to be investigated include allergy testing, infectious disease, and additional tumor markers. Our goal is to always stay on the forefront of the newest technology and testing to provide our clients

with the best possible customer service.

If you have any questions about the new Immulite 2000, please contact Dale Hamari, Special Chemistry Supervisor, at 906-225-7374.

Just for fun *By Michele Toyras, MT, ASCP*

Don't be afraid, match the phobia with its definition.

- | | |
|------------------|--|
| 1. Erythrophobia | A. Fear of experiencing or witnessing pain |
| 2. Zoophobia | B. Dread of talking because one may stutter or stammer |
| 3. Mysophobia | C. Irrational fear of dirt, contamination or uncleanness |
| 4. Nyctophobia | D. Fear of blushing, embarrassment or the color red |
| 5. Laliophobia | E. Fear of animals |
| 6. Agoraphobia | F. Fear of being in an open, crowded or public place |
| 7. Algophobia | G. Fear of darkness |

Answers: 1/D, 2/E, 3/C, 4/G, 5/B, 6/F, 7/A

Continued from front

Hypercoagulability evaluation

and to antiphospholipid antibodies.

If the result is abnormal, a work-up for lupus anticoagulant and for anticardiolipin antibodies is recommended. Because antibody activity can fluctuate over time, these tests should be positive on more than one occasion, at least 8-12 weeks apart.

The activated protein C resistance done in our laboratory is a *screen* for the point mutation, factor V Leiden. It can be performed on patients on *stabilized* oral anticoagulant, heparin, or LMW heparin therapy and on those with a lupus anticoagulant. Ratio results that are borderline may suggest low levels of Protein C. If the activated protein C resistance ratio result is low, a DNA-based assay for Factor V Leiden mutation is recommended.

There are two major types of assays for

Protein C, protein S, and antithrombin deficiencies: functional (activity) assays and antigenic (quantitative immunological) assays. Type I (quantitative) deficiencies of Protein C, protein S, and antithrombin show decreased functional and antigenic results while in Type II (qualitative) deficiencies, only the functional result is decreased. When looking at laboratory testing, it's best to use functional assays since they will detect both a quantitative and qualitative disorder. This is not simple however, as most of the functional tests available are affected by anticoagulants, which is *the treatment* for thrombosis. If a result is decreased, an antigenic assay is recommended.

Testing on patients for hypercoagulability should be done when circumstances or

drugs will not interfere with results; or data should be interpreted with those interferences in mind. One of the greatest cost inefficiencies in the special coagulation laboratory is the performance/and or interpretation of tests under inappropriate conditions. Evaluation for protein C or S deficiencies should not take place while patients are on oral anticoagulant therapy (OAT). Patients should be off OAT for at least 10-14 days before testing. After coumadin discontinuation, protein S takes longer to return to the normal range than does protein C. Full-dose heparin administration can cause up to a 30 percent reduction in antithrombin levels that return to normal when heparin is discontinued.

The thrombosis panel offered in our laboratory (THROM) consists of the following tests: antithrombin III, dilute Russell Viper Venom test, factor 5 Leiden screen by APCr, homocysteine, plasminogen, protein C, and protein S.

