

Official publication of the American Medical Technologists



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- Alzheimer's: Beginning to Cope
- Improving Your POCT Program

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- Hereditary Hemochromatosis
- The Power of Capsaicin
- Battling Leukemia

ewer than 400,000 people live in Minneapolis proper, but it feels more like a hip, thriving metropolis. There are lines well past midnight to get into some downtown clubs and live

Mark your calente AMT in Minneapolis June 22-27, 2009

music bars. Crowds pour into the futuristic-looking Walker Art Center to view a current exhibit. Sports fans file out of the light-rail transit system towards the Metrodome for a Sunday afternoon Twins game. Theater lovers head for a matinee at the new and renowned architectural gem of Guthrie Theater; the theater's lobby alone is worth a visit. Esquire magazine last year dubbed Nye's Polonaise in Minneapolis The Best Bar in America. In 2006, Travel & Leisure magazine named Minneapolis one of the top five destinations that you must visit.

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71st Educational Program and National Meeting



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## American Medical Technologists Institute for Education (AMTIE)

# PRESIDENT'S REPORT

It has been approximately four months since the last very successful national convention and business meeting was held. As usual, it was an excellent meeting. Diane Powell once again put together an excellent meeting. Diane always presents a memorable and well organized meeting for AMT members. The programs that I was able to attend were excellent and from the members who attended some of the other sessions, the response was the same, "VERY GOOD."



AMTIE President Pat Cuviello

I would be amiss if I did not mention how great the home office staff is and the outstanding job that they do at these conventions. They are so helpful, cheerful, and efficient. THANK YOU.

The Annual AMTIE business meeting was well attended. There were many comments, questions and suggestions made by those in attendance.

After the AMTIE business meeting, the Board of Trustees held an election for AMTIE officers. Elected were: President, Patrick V. Cuviello, MS, MT; Vice President, Kay Fergeson, MT; and Jeff Lavender, MT, SGM, Secretary/Treasurer. Newly elected was Linda Jones, MT, and AMTIE elected David Yocom. Art Contino, RMA, and Zenaida Maraggun, MT, were appointed to the AMTIE Board of Trustees by the AMT Board of Directors.

At the 2006 AMTIE Board of Trustees meeting, Tom Fish from Ohio proposed a new method of documenting attendance at continuing education sessions at the annual conventions. A new two-part CE documentation form has been developed and will be in use at the next annual meeting. Forms will be placed in each attendee's registration package. When a member attends a session, the moderator will provide a verification code unique to that session. The member will note the number next to the session attended. At the end of the annual meeting, the member will turn in the top sheet at the registration desk. The second copy is for the member to keep.

At the state level, the forms will be blank and the member must write in the name and number of the session attended. Each state must develop its own numbering or code system. Each state will have to establish a method of verification (e.g., the moderator could rubber-stamp the form, initial the form, or provide a special number at the end of the session). The state society will then submit each member's form to AMTIE for recording.

At either the national meeting or the state meeting, a sign-in sheet should be used in order to verify attendance.

At this time, I want to remind the AMTIE Board of Trustees, as well as all AMT members, that any member in good standing can nominate someone for the Cuviello Excellence In Education Award. Remember, the award submission date was changed to DECEMBER 1 of each year by the AMTIE Board of Trustees.

Once again, I want to remind all members who were certified as of January 1, 2006, that they must earn enough hours of continuing education for their discipline in order to meet the requirements of the Certification Continuation Program (CCP). The number of points needed depends on your type of certification (MT, MLT, RMA, etc.).

Please note that the deadline for those certified in January 2006, has passed. In order to comply with the CCP, you must have earned enough points by December 31, 2008. There are no blanket exceptions for the military. Any hardship that a military member faces in order to meet the CCP requirements will be dealt with on an individual basis.

The AMTIE Board of Trustees voted to make some changes in the recording of continuing education credits. As stated in the last *Journal of Continuing Education Topics & Issues* by Dr. Gerard Boe, AMTIE will not record continuing education as CECs, CEs, credit hours, semester hours, etc. All continuing education credit will be now recorded as CLOCK HOURS; the credit will no longer appear as CEC on your "Report Card." When a member submits continuing education credit to AMTIE for recording, they should be submitted as CLOCK HOURS. One clock hour must be at least 50 full minutes of lecture or workshop except for articles in the *Journal of Continuing Education Topics & Issues*. These articles may be less than one hour.

In the President's message written by Dr. Gerard Boe, which can be found in the August, 2008, issue of the *Journal of Continuing Education Topics & Issues*, Dr. Boe provided an excellent chart for converting your continuing education credits and college hours to the proper clock hours.

I want to thank Dr. Boe for filling in for me and writing the last President's message during my last hospital stay.

Be on the lookout for a new AMTIE logo. It is now under development.

Let us not forget to start making plans to attend the upcoming national convention and business meeting which is to be held in Minneapolis from June 22 to 27, 2009. It is not too early to make your plans.

God bless and protect our military.

Patrick V. Cuviello, MS, MT AMTIE President

# REMINDER

The cut-off date for submitting continuing education credit to AMTIE is January 30 of each year. For 2008, that means that the cut-off date for submitting continuing education credit to AMTIE is January 30, 2009.

# How to Enroll in STEP

## A Continuing Education Program Offered by American Medical Technologists

**STEP** is a continuing education program keyed to articles that will appear in *Journal of Continuing Education Topics & Issues.* Answer sheets are available directly from the AMT office. Those who want to participate in the program should respond to the questions for the corresponding article(s) and mark the answer sheet(s) accordingly. Answer sheets are to be returned to AMTIE for scoring. Results are sent to participants and credit is automatically recorded in members' and non-members' AMTIE continuing education files.

## To Participate in STEP

## **AMT Members**

AMT members are automatically enrolled in *STEP* as a membership benefit. You will receive *STEP* articles in the *Journal of Continuing Education Topics & Issues.* Answer sheets are available directly from the AMT office. To participate in *STEP*, simply answer the questions on the appropriate sheet, and return the sheet to AMTIE (10700 W. Higgins Rd., Rosemont, IL 60018). A \$3.00 processing fee must be included with *each* answer sheet submitted. Your score will be promptly mailed to you. AMT members will have their earned credit automatically entered into their AMTIE continuing education file. A score of at least *80%* is required to earn credit.

*REMEMBER:* THERE IS NO ENROLLMENT FEE FOR AMT MEMBERS.

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## Non-members

A prepayment of \$85.00 (\$95.00 foreign) entitles non-members to one year's subscription to AMT Events, and the Journal of Continuing Education Topics & Issues, and one year's enrollment in STEP. To participate in STEP, complete and mail the form below. Enclose a check or money order for the appropriate amount, payable to "AMTIE." Answer sheets are available directly from the AMT office. Do not answer questions on any other form. To earn STEP credit, simply answer the questions on the appropriate sheet and return the sheet to AMTIE. A \$3.00 processing fee must be included with each answer sheet submitted. Your score will be promptly mailed to you. A score of at least 80% is required to earn credit. As a STEP participant, your earned credit will be automatically entered into the AMTIE continuing education recording system. You will receive an annual report of credit earned through STEP.

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NON-MEMBER	STFP FNRC	DLLMENT FORM

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LAST NAME	FIRST NAME	M.I.	I wish to enroll in the STEP program for one full year I have enclosed \$85.00 (\$95.00 foreign). I understand that I will receive one year's subscription to AMT Event.
NUMBER AND STREET			and Journal of Continuing Education Topics & Issues.
CITY	STATE	U.S. ZIP CODE	Payment (by check or money order, please)
	FOREIGN COUNTRY, OR PROVINCE		Amount \$
POSTAL CODE (IF ANY) FC	R DELIVERY OUTSIDE U.S.:		Payment enclosed. Make check payable to: AMTIE (Payment must be in U.S. funds drawn on a U.S. bank.
DAYTIME PHONE			

Mail to: AMTIE, 10700 Higgins Rd., Suite 150, Rosemont, IL 60018

# HOME STUDY UNITS FOR AMT MEMBERS

Offered by Association for Continuing Education, LLC, (ACE)

The self-instructional units listed below have been reviewed and approved for Continuing Education for AMT members by the American Medical Technologists Institute for Education (AMTIE).

To participate in home study programs: 1) Order the units desired directly from Association for Continuing Education (ACE) using the form below (photocopy accepted); 2) Complete unit and AMTIE post test enclosed with unit; 3) Send completed post test to ACE, P.O. Box 573, Beaufort, SC 29902 with \$6.00 per test for grading and score reporting. Results of your participation will be recorded in your AMT continuing education file.

#### **ORDER FORM**

The following self-instructional units are AMTIE approved for AMT Continuing Education.

PROGRA	М	CLOCK HOURS	UNIT COST	QUANTITY ORDERED	TOTAL COST
Basic Labo	ratory Techniques				
#100	Performing a Capillary Puncture	1.5	\$ 9.50		
#101	Venipuncture: The Art of Drawing Blood	3.0	\$13.50		
#102	The Making of a Blood Film	1.5	\$ 9.50		
Chemistry					
#206	Quality Control Overview for Clinical Chemistry	3.0	\$13.50		
#207	Laboratory Evaluation of Cardiac Markers	3.0	\$13.50		
#208	Kidney Function Tests	3.0	\$13.50		
#209	Total and Ionized Calcium in Serum	2.0	\$11.50		
Chemistry					
#400-1	Intro to Hematopoiesis - booklet and CD w/35 photo images	4.0	\$37.50		
#400-2	Intro to Hematopoiesis - booklet only (no CD)	2.0	\$12.50		
#404	Hematology Indices	2.0	\$12.50		
#409-1	Cerebrospinal Fluid - booklet and CD w/37 photo images	5.0	\$44.50		
#409-2	Cerebrospinal Fluid - booklet only (no CD)	3.0	\$13.50		
#410-1	Reticulocyte Counts - booklet and CD w/35 photo images	4.0	\$37.50		
#410-2		2.0	\$12.50		
#411	Erythrocyte Sedimentation Rates	1.0	\$ 8.50		
#414	Hemoglobin H Disease	2.0	\$12.50		
#415	Iron Metabolism	3.0	\$13.50		
#418	Hemolysis Testing	4.0	\$14.50		
#450	Coagulation Phase of Hemostasis	2.0	\$12.50		
Immunolog	•	2.0	φ1 <b>2</b> .50		
#500	Intro to ABO Blood Group System	2.0	\$12.50		
#501	Reading and Grading Agglutination Reactions	2.0 1.0	\$ 8.50		
#502	Solving Blood Bank Problems	3.0	\$ 8.50 \$14.50		
		3.0			
#503	Problems in Antibody Identification	2.0	\$14.50 \$12.50		
#550	Antigens and Antibodies	3.0	\$12.50 \$14.50		
#551	Principles of Antigen-Antibody Reactions Used in the Lab		\$14.50		
#552	Complement Cascade	2.0	\$12.50		
Microbolog		• •	<b></b>		
#606	Overview of TB Infection and Disease	2.0	\$12.50		
Mycology					
#650	Introduction to Medical Mycology	1.0	\$ 8.50		
Statistics					
#762	Descriptive Statistics	2.0	\$12.50		
Urinalysis					
#800	Chemical Screening of Urine by Reagent Strip	2.5	\$13.00		
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## Article **345** 1 Clock Hour

# **Alzheimer's: Beginning to Cope**

### **Rita St. Pierre**

Many people, especially as they age, begin to worry that every problem with their memory, every loss of a word, might indicate the start of Alzheimer's disease. However, Alzheimer's disease is not part of normal aging and it is important that we understand that in order to better serve those who come to us with this diagnosis.

"Abilities to store, prioritize, and recall new information are brain functions that slowly break down with the onset of Alzheimer's" (Kuhn). With this statement, Dr. Kuhn succinctly describes what happens to those with early Alzheimer's. The difference between those losses and normal aging becomes evident since the above losses become part of a deteriorating pattern, unlike in normal aging.

Ultimately, you might ask, why should we care about Alzheimer's disease in our work? How important is this for the patients we serve? The answer is clear in current statistics, which show that Alzheimer's affects over 5.2 million Americans, with 13% of the population over age 65 affected. Every 71 seconds, someone in the U.S. develops Alzheimer's disease, and by mid-century, that number will increase to every 33 seconds, mostly because of aging baby-boomers. Another important statistic to consider, as you see outpatients in your work, is that 70% of people with Alzheimer's disease live at home (Alzheimer's Association, 2008). Given these numbers, it is a certainty that you are serving people with Alzheimer's disease and/or their caregivers.

As health care providers, there are specific ways you can help the families and those affected with this disease as you provide care. First, it is important to understand that avoidance and denial are normal and frequent reactions to this disease process.

Therefore, spouses and other caregivers often "mask" or compensate for the symptoms, thereby possibly making your job more difficult. Also, the disease is unpredictable and that adds to the family's stress. Most people with Alzheimer's have good and bad days just like us, and it's important to understand that the person is not being "stubborn" or manipulative, regardless of how it may look. The person is losing functional abilities and truly cannot remember, or find the right words, or understand what you're asking of them. Persons with dementia are doing the best they can and we must be patient. The person may seem to understand your instructions but in fact may not, or may not remember even just moments later. Also, regardless of the loss of physical function and language, emotional connections remain intact so it is more important to focus on emotional communication than to worry about the words we use. Smile, pay attention to your tone of voice, and speak slowly and in short sentences. Convey that you understand and care, more with facial expressions, body language and touch than with words. Give directions one step at a time, don't talk too loud (unless you know the patient has hearing loss), repeat if necessary, don't use pronouns, and SMILE.

As health care providers, we must adapt our response and approach because patients with Alzheimer's or another dementia cannot change or easily adapt to a new situation. It also will help all involved if providers modify the environment by reducing noise levels and other auditory or visual distractions, allow family members or caregivers to be present, make "small talk" even if the patient doesn't respond, and reduce environmental clutter. When communicating, stand or sit in front of them, not behind or on the side because eye contact is very important. Be patient, and don't rush!

There is life during and beyond Alzheimer's but families are often very stressed, sometimes in denial, or even embarrassed about the disease in their loved one. As a health care provider, you are in a position to offer support, compassion and understanding. That will be obvious if you alter your approach, your routine, and your environment to meet the patient's and the caregiver's needs.

#### **References:**

Kuhn, Daniel: Alzheimer's Early Stages: First Steps for Family, Friends and Caregivers, 2nd Edition. Hunter House Publishers, Alameda CA, 2003; page 32.

Alzheimer's Association: 2008 Alzheimer's Disease Facts and Figures. Alzheimer's Association National Office, Chicago IL, 2008.

Rita St. Pierre, M.A., Program Director, Alzheimer's Association of Rhode Island.

# **Questions for STEP Participants**

Answer questions only on the official STEP answer sheet. If you do not have the official STEP answer sheet, a year's supply can be obtained (at no cost), simply by writing to: STEP Program Answer Sheets, American Medical Technologists, 10700 W. Higgins Road, Suite 150, Rosemont, IL 60018, or by fax: 847/823-0458, or by e-mail: paula.simoncini@amt1.com.

In addition to marking your answers, be sure to include all the required information on the answer sheet and a processing fee of \$3.00 per article.

In the following, choose the one best answer for each question.

- **1** Alzheimer's disease is not part of normal aging, no matter how long one lives.
  - A. True
  - B. False
- **2** It is not important for me to understand Alzheimer's disease because I won't see too many of them in my work.
  - **A**. True
  - B. False
- **3** Someone develops Alzheimer's disease:
  - **A.** rarely
  - **B.** every hour
  - **C**. every 10 minutes
  - D. every 71 seconds
- **4** It is not likely that Alzheimer's patients will come for outpatient visits because they're all in hospitals and nursing homes.
  - A. True
  - B. False
- **5** Patients with Alzheimer's and their families will willingly acknowledge the diagnosis when they come in for care.
  - A. True
  - **B.** False

- **6** If the person with Alzheimer's seems to understand my instructions but doesn't do what I ask, that person is being stubborn or purposely difficult.
  - A. True
  - B. False

7 The best way to provide care to an Alzheimer's person is to:

- **A.** work fast to get the test/care finished quickly
- **B.** tell the patient everything I will do before beginning
- **C.** go slowly, give directions one step at a time and repeat if necessary
- **D.** speak loudly to make certain they understand

**8** When providing care to an Alzheimer's person, it is best to encourage the family or caregiver to also be in the room.

- A. True
- **B**. False

**9** People with Alzheimer's disease don't like to make eye contact, so I should speak from the side or from behind.

- A. True
- B. False
- **10** The person with Alzheimer's disease responds best in an environment that is quiet, uncluttered and calm.
  - **A.** True
  - **B**. False

## Article **346** 1 Clock Hour

# **The Power of Capsaicin**

## Jonathan M. Mortensen and Joel E. Mortensen



Image by JE Mortensen and JM Mortensen 2008.

A potentially psychotic man orders wings with ultrahot sauce at a trendy new sports bar. The room quiets and everybody watches as he takes the first bite he grunts quietly and tears begin to flow down his face. The pungent ingredient that brings this grown man to tears is capsaicin, a chemical that is produced by chili peppers. This scene is not new; the history of the use of chili peppers extends back into prehistoric times. The secret behind the power of capsaicin is its molecular structure and chemical properties.

#### History

Human use of chili peppers dates back to prehistoric times. Archeologists have shown that humans ate wild chili peppers as early as 7000 B.C.E. and probably domesticated peppers between 5200 B.C.E. and 3400 B.C.E. Preserved peppers have provided evidence that South Americans ate and grew aji, or chili in English, in 2500 B.C.E. Chili peppers became increasingly common and integrated into the diet of particular cultures. However, chili peppers and similar spices remained isolated in these cultures until the 13th century when Marco Polo established trade routes to the Far East. In addition, Columbus made his famous voyage to America to find a new route to these spices. Although he failed in his original mission, Columbus found four additional species of chili peppers in America. With the help of the Portuguese distribution to Africa, chili peppers became a spice available to civilizations throughout world.

Jonathan M. Mortensen, Case Western Reserve University, Cleveland, Ohio; Joel E. Mortensen, Ph.D., Diagnostic Infectious Diseases Testing Laboratories, Cincinnati Children's Hospital, Cincinnati, Ohio

Chili peppers had many useful applications in historic times. The first and most obvious use of these peppers was as a strong flavoring. Chili peppers were used not only to enhance flavorless foods, but also to help overcome the flavor and odor elicited by spoiled foods. Another use of these spices was as a preservative. Research indicates that capsaicin can kill some microbes. Ancient cultures noticed an antimicrobial phenomenon from peppers and used them to preserve food in the same manner they used salts. A final historical application of peppers was as an ingredient in medicine. These spices were blended with other plants to form herbal remedies.

#### **Capsaicin in Food**

Chili peppers and capsaicin influenced the dietary habits of many people around the world. Today, chili peppers are the most widely used seasoning in the world. It is estimated that as many as three-quarters of the world's population include peppers in their diet regularly. New restaurants featuring Mexican and South American cuisines developed this cooking style to satisfy a desire for fiery foods. Many restaurants, which specialize in chicken wings covered in hot sauces, have opened. Restaurants offer a wide range of sauces with increasing amounts of spiciness based upon increasing amount of capsaicin in the sauce.

#### **Capsaicin in Nature**

The chemical capsaicin is the source of the chili pepper's spicy sensation. Pepper plants in the genus *Capsicum* produce capsaicin in glands located inside the pepper at the meeting point of the placenta and the pod. Surprisingly, the seeds do not contain any capsaicin; rather the white connective pith in the fruit's center contains the highest concentration of capsaicin.



Figure 1. Jalapeno pepper showing pith and seeds (JE Mortensen 2008)

The genus *Capsicum* is a member of the *Solanaceae* or nightshade family, a diverse group of plants that includes tomato, potato, tobacco, eggplant and the deadly nightshade. The genus *Capsicum* con-

tains 27 species, 22 native/wild species and 5 domesticated. The domesticated species include *C. annuum, C. baccatum, C. chinense, C. frutescens,* and *C. pubescens.* The plant grows as a long-lived perennial shrub, with the individual plant size varying depending on the cultivar, or specific plant variety, and the climate. In a 1976 article, Heiser hypothesized that *Capsicum* probably evolved from an ancestral plant originally in the west-central region of South America.

Chili fruits are usually referred to as vegetables; however, they are technically berries and therefore fruits. Although these berries vary greatly in color, shape, and size, most peppers are produced by cultivars of the species C. annuum. These C. annuum cultivars include peppers as diverse as the "bell pepper," which is available as the immature green pepper as well as the ripened red, yellow, purple or orange Bell pepper. Other examples of fruits from Capsicum include Anaheim chilies (also known as California green chili, long green pepper, and chili verde), Ancho chili (used to make chili powder), the popular Jalapeno, and the Chipotle (the smoked Jalapeno). Two other peppers that can be called chilies, tabasco, C. frutescens, and habanero, C. chinense, are from different species.

Capsaicin's natural role might be a protectant for Capsicum seeds. Birds disperse the seeds of capsicum plants. When birds consume brightly colored chilies, and thus, capsaicin, the chemical has a negligible, or analgesic effect, rather than acting as an irritant. This observation has given rise to use of chili peppers in birdseed to discourage squirrels and other animals from consuming the seed. Capsicum seeds pass through a bird's digestive tract and germinate if they fall into a favorable environment. Seeds ingested by mammals are not capable of germinating because digestive chemicals inactivate them. Therefore, a twopart theory is (a) that capsaicin discourages the consumption of these plants' fruit by mammals and (b) by having brightly colored fruit, capsicum favors the attraction and consumption by the "correct" germinating/transporting animal (birds).

#### Chemistry

P.A Bucholtz first reported the isolation and purification of the capsaicin molecule in 1816. In this report, he stated that one could extract the pungent ingredient of peppers from the macerated pods with organic solvents. L. T. Thresh reported in 1846 that this substance, which he named capsaicin, could be removed in a crystalline state. In 1878, Endre Hogyes reported several of capsaicin's biological properties, including the burning sensation when it touches mucous membranes and an increase in gastric secretions when ingested. In 1930, E. Spath and F.S. Darling became the first scientists to synthesize the capsaicin

molecule.

Capsaicin's structure is a key factor in its chemical properties. The molecule consists of a hexagonal ring of bonded carbon atoms with a tail that contains a long hydrocarbon portion. This hexagonal ring and its accompanying functional group form a basic vanillyl group (Figure 2). The basic chemical properties of capsaicin are listed in Table 1.

Figure 2. The vanillyl group

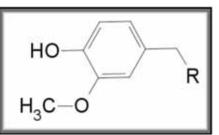


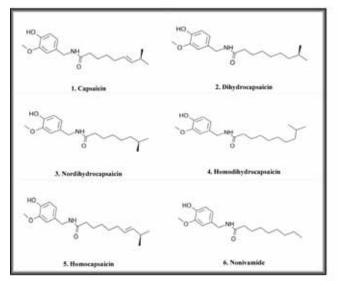
Table 1. Chemical properties of capsaicin

IUPAC name*	8-methyl-N-vanillyl-trans-6-nonenamide			
Molecular formula	C <sub>14</sub> H <sub>22</sub> NO <sub>3</sub>			
Molecular mass	305.41 ginet			
Melting point	62 - 65°C			
Boiling point	210-220°C			
Description	Hydrophobic, colorless, sdorless, srystalline - wasy compound			

\*International Union of Pure and Applied Chemistry

In 1964, S. Kosuge and Y. Inagaki reported that the term capsaicin actually describes a complex of related components they named capsaicinoids.

Figure 3. Chemical structure of capsaicinoids



Capsaicin and all other capsaicinoids are classified as crystalline alkaloids, compounds that contain a nitrogen base and are found in plants. Capsaicinoids belong to a larger family of chemicals called the vanilloids or compounds that contain the vanillyl group. There are five naturally occurring capsaicinoids as well as a single synthetic one (Figure 3). Capsaicin accounts for 69% of capsaicinoids. Dihydrogencapsaicin comprises 22% percent of capsaicinoids and has the same structure as capsaicin with the exception of a hydrogen atom replacing a double carbon bond in the hydrocarbon tail. Nordihydrocapsaicin, a dihydrogencapaicin with one less carbon on the carbon tail, accounts for 7% of capsaicinoids. The remaining 2% of naturally occurring capsaicinoids are homocapsaicin and homodihydrocapsaicin, which also contain slight variations in the hydrocarbon tail.

Solubility is the key to ridding a person's mouth of that burning sensation when eating spicy food. Although one might want to grab a glass of water to put out the fire, the long hydrocarbon ends of capsaicinoids do not dissolve in water. To be dissolved and eliminated, capsaicin must be in solution with similar hydrocarbon molecules or certain proteins. Fats, oils, and alcohols are superior choices as solvents. For example, dairy products such as milk and yogurt are effective because the milk protein casein can dissolve fatty capsaicin molecules.

Two very different strategies settled the debate of which chili pepper was truly the hottest. First, Wilbur Scoville in 1912 invented Scoville Units, based on a subjective taste test. Scoville blended pure ground chili peppers with sugar-water, and a panel of tasters sampled serial dilutions of the liquid until it no longer caused a burning sensation in the mouths of the tasters. One part of heat in a million drops of water equals 1.5 Scoville Units. Measurements range from 0 Scoville Units to 16,000,000 Scoville Units in pure capsaicin.

The subjective "taster" process, although still used today, has been replaced by a reproducible, standardized laboratory test known as high performance liquid chromatography (HPLC). HPLC separates a substance into its components based on their ability of the components to travel through an absorption column at different rates. The capsaicin levels in the various capsaicinoids are measured in parts per million of the original sample and then converted to Scoville Units to determine the heat of the pepper (see Table 2). 
 Table 2. Scoville Units of various peppers, adapted from reference 21

Pepper	Scoville Units
Sweet Bell	0
El-Paso	500 - 700
Santa Fe Grande	500 - 750
Coronado	700 - 1,000
Espanola	1,000 - 2,000
Poblano	1,000 - 2,000
Ancho	1,000 - 2,000
Anaheim	500-2,500
Pulla	700-3,000
Mirasol	2,500 - 5,000
Jalapeno	2,500 - 8,000
Chipolte	5,000 - 8,000
Hot Wax	5,000 - 10,000
Serrano	8,000 - 22,000
Manzano	12,000 - 30,000
Jaloro	30,000 - 50,000
Aji	30,000 - 50,000
Tabasco	30,000 - 50,000
Cayenne	30,000 - 50,000
Super Chile	40,000 - 50,000
Piquin	40,000 - 58,000
Chiltecpin	60,000 - 85,000
Thai	50,000 - 100,000
Tabiche	85,000 - 115,000
Bahamian	95,000 - 110,000
Carolina Cayenne	100,000 - 125,000
Jamaican Hot	100,000 - 200,000
Birds Eye	100,000 - 225,000
Orange Habanero	150,000 - 325,000
Scotch Bonnet	150,000 - 325,000
Chocolate Habanero	300,000 - 425,000
Red Savina Habanero	350,000 - 575,000
Dorset Naga	800,000 - 900,000

#### **Mechanism of Action**

A complex series of reactions causes a person to sense heat when eating capsaicin-containing foods. The specialized capsaicin receptors are located on the taste buds within the papillae of the tongue. The receptor responsible for detecting capsaicin is called transient receptor potential vanilloid-1 (TRPV1). Capsaicin's chemical structure allows it to bind to TRPV1. In the presence of capsaicin, a lipid portion of the receptor called PIP2 separates and allows calcium ions to enter the receptor cell. A pain message is then carried to the brain by substance P, a neurotransmitter. The bond between capsaicin and TRPV1 is temporary, so feeling of pain subsides when the bond is broken. Accordingly, people with a greater number of taste buds are often more sensitive to foods containing capsaicin.

Chili peppers containing capsaicin not only cause the fire in a person's mouth, but they also affect the body in other ways. Peppers increase the production of stomach acids that stimulate the digestive tract to start a cleansing process. Peppers also up regulate metabolism and help the body to metabolize fat molecules. Capsaicin causes the brain to release endorphins that cause a sense of well-being or euphoria that can last for several hours. Capsaicin can also alleviate pain by over-stimulating the release of substance P, which functions as a link between primary receptors and the brain. The over-simulation causes substance P levels to drop and, thus, eliminates the sense of pain.

It has recently been reported that tarantula venom activates the same neurological pathway as capsaicin. It is not clear what the significance of the relationship might be.

#### **Capsaicin in Medicine**

Capsaicin's interaction with the body has given rise to varied medical applications.

Pain Management Capsaicin is currently used in topical ointments to relieve pain like that following the reactivation of herpes simplex or zoster. It is also incorporated into a cream for temporary relief of minor aches and pains of muscles and joints. Preparations are available to consumers in drug stores without a prescription. Higher concentrations of capsaicin can also be applied in a doctor's office. This more involved treatment involves the use of a topical anesthetic followed by the application of the capsaicin. The capsaicin overwhelms the nerves and results in an inability to transmit a pain signal for an extended period. With chronic exposure to capsaicin, the neurons become depleted of neurotransmitters. This depletion leads to a reduction in sensation of pain. Capsaicin has also been tested for the control of postoperative pain associated with surgery. The capsaicin causes a long-term loss of pain after an injection during surgery.

**Diabetes Control** Because of a reported link between neural cell control and diabetes, researchers injected capsaicin into pancreatic sensory nerves of mice with congenital Type 1 diabetes. In mice, capsaicin appeared to affect malfunctioning nerves in the pancreas allowing for the production of insulin, relieving the symptoms of diabetes.

**Cancer Treatment** There have been reports of an epidemiological relationship between the consumption of peppers and cancer prevention. The Thai are well known for their preference of highly spicy food, and it has long been noted that Thailand has a low incidence of gastrointestinal cancers, compared to the rest of Asia. Intestinal, stomach, and colon cancer rates are also very low in much of Mexico and South America as compared to the United States. Nonetheless, it is difficult to control the myriad of other factors that might be involved with this type of analysis, so assigning cause and effect to capsaicin in these settings is difficult.

Several laboratory studies tried to explore cap-

saicin's role in cancer control and have shown that capsaicin might prevent the growth of certain cancer types. Specifically, several Japanese and Chinese studies showed that natural capsaicin directly inhibits the growth of leukemia cells. Even though these studies used pure capsaicin directly injected into isolated diseased cells in a laboratory setting, the authors concluded that daily consumption of hot peppers might prevent certain types of cancer. In another study by Chow and colleagues, capsaicin was found to be cancer killing. It was shown that TRPV6, a calcium channel protein related to TRPV1, might mediate capsaicin-induced gastric cancer cell death. Capsaicin may have promise as a cancer-preventing supplement to our diet.

**Headache Control** Marks and associates studied the intranasal application of capsaicin to help control cluster headaches. In a double blind, placebo-controlled trial of 15 patients, there was a reduction of headache severity in the treatment group at eight to 15 days compared to the control group. The groups were not as well matched as the investigators had hoped but further study is certainly warranted.

Anti-Inflammatory Agent Researchers reported capsaicin to be a potent anti-inflammatory agent. Using experiments in the laboratory and in a rat model of sepsis, investigators showed that capsaicin could inhibit or limit the production of inflammatory compounds or might be involved with cellular compounds that control the inflammatory response such as tumor necrosis factor, interleukins -6 and -10 and superoxide dismutase. Capsaicin might also be involved in the scavenging or inactivating of free oxygen radicals.

**Obesity Control** Although capsaicin is an active ingredient in various over-the-counter weight loss supplements, this doesn't mean that it has any real role in weight control or weight loss. As previously mentioned, capsaicin consumption up regulates metabolism. In addition, capsaicin increases lipid oxidation and decreases the appetite of subjects who consume them. The mechanisms involved are unclear, but are thought to be involved in capsaicin's interaction with nerves. Work by Zhang and colleagues suggests that capsaicin's binding with a TRPV1 channel might affect fat cell's differentiation. This TRPV1 activation might reduce the number and size of fat cells and, therefore, reduce obesity.

**Other Uses** Capsaicin is also the active ingredient in pepper spray. When this anti-personal spray encounters eyes or mucous membranes, it is very painful.

#### Conclusion

The secret behind the power of capsaicin is its molecular structure and chemical properties. Although this unique compound offers humanity a spicy blast of heat, it might also offer the key to some important medical dilemmas.

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# **Questions for STEP Participants**

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In addition to marking your answers, be sure to include all the required information on the answer sheet and a processing fee of \$3.00 per article.

In the following, choose the one best answer for each question.

- **1** Archeological evidence has been used to establish that peppers have been harvested from the wild and consumed by humans for a long time. Which of the following time periods corresponds to the period where it is thought humans first began this harvesting?
  - **A.** 13th century
  - **B.** 1 million years BC
  - **C**. 5,000 BC
  - **D.** 1964 AD
- **2** Capsaicin is most highly concentrated in which part of the pepper plant?
  - A. Skin
  - **B.** Seeds
  - C. Stem
  - **D**. Pith
- **3** The majority of "hot" pepper varieties are cultivars of which of the following *Capsicum* species?
  - **A.** chinese
  - **B.** pubescens
  - **C.** capsaicin
  - **D.** annuum
- **4** Capsaicinoids belong to a larger family of aromatic compounds that are known as which of the following?
  - A. Vanilloids
  - B. Pepperinoids
  - **C**. Carbonoids
  - **D.** Ankylosimoids
- **5** Because of the hydrocarbon tail found on the capsaicinoid molecule, which of the following statements is true of this group of compounds?
  - **A.** They dissolve easily in water.
  - **B.** They can be easily cleared from the taste buds using common alcoholic beverages.
  - **C.** Their solubility is higher in water than oils.
  - **D.** Fats and alcohols are good solubilizing agents.

- **6** Capsaicin exhibits most of its biological effect by binding to which of the following receptors?
  - A. Translational parallel vanilloid-1 (TPV1)
  - **B.** Transient receptor potential vanilloid-1 (TRPV1)
  - **C.** Translocating receptor parallel vanilloid-1 (TRPV1)
  - **D.** Bay Area Regional Transit Authority (BARTA)
- 7 Capsaicin has been used for pain management. The most likely mechanism of action is which of the following?
  - A. Depletion of neurotransmitters
  - **B.** Depolarization of nerve fibers
  - C. Blockage of neurotransmitter uptake
  - **D.** Neurotransmitter analog
- **8** There is epidemiological evidence linking the consumption of chili peppers and a lower rate of some types of cancer.
  - A. True
  - **B.** False
- **9** Laboratory tests with a calcium channel protein related to TRPV1 has been shown to induce cancer cell death. What is the name of this compound?
  - **A.** TRVP1.1
  - **B**. TRVP2
  - **C**. TRPV6
  - **D.** 1PVRP
- **10** Which of the following immunomodulating compounds is thought to be affected by capsaicin?
  - A. Tumor necrosis factor
  - **B.** Interleukin 6
  - **C**. Interleukin 10
  - **D.** All of the above

## Article **347** 1 Clock Hour

# **Improving Your POCT Program**

Try these tips to give your POCT program a boost.

## **Karen Appold**

Point-of-care testing (POCT) has become an important part of patient care at many health care facilities. Some institutions, however, simply maintain a POCT program and do not develop it to its fullest potential.

During an AACC audioconference, "Top 10 Tips: The Keys of Improving Your POCT Program," presenters discussed how a laboratory can enhance its POCT program so it positively impacts the institution.

Mark Barglowski, MBA, CLT, MT(ASCP), Director, Laboratory and Respiratory Care Services, Providence Saint Joseph Medical Center, Burbank, CA, began the presentation by discussing the challenging approach his lab took to promote the highest level of impact for patient care and the 420-bed hospital with POCT. He outlined the lab's strategic approach, which involves active communication for POCT users and other key hospital individuals to allow for growth in a lab-based POCT program outside of lab boundaries.

Barglowski's 10 tips include:

- 1. **Create a structure for success**. An internal structure should include a POCT coordinator, laboratory information systems administrator, laboratory director and laboratory administration. Develop this team at regular meetings. Recognize and reward each individual's successes.
- 2. Embrace POCT. Develop knowledge of POCT by participating in listservs, read periodicals such as *ADVANCE for Medical Laboratory Professionals* and research via the Internet.
- 3. **Know your customers**. Enhance your lab's image by knowing what customers want now and in the future and what they don't want. Determine this by conversing with individuals in other hospital departments (e.g., nurses, emergency room personnel and respiratory care providers).
- Shout your successes. Praise laboratory team members and others affiliated with your POCT program personally and at hospital meetings.

- 5. **Bill for POCT**. POCT is a revenue opportunity and it is simple to set up. Involve key players such as administration, finance, billing and compliance.
- 6. **Integrate into programs**. Enhance your lab's image and gain support for its POCT program by incorporating POCT in cardiology, diabetes and oncology programs.
- 7. **Manage your data**. If POCT is integrated in your hospital's information system, then you should be able to obtain results. Data is value: It allows you to manage test volumes and costs and determine appropriate use.
- 8. Look at outcomes. Examine the use of outcomes. For example, is the diabetes coordinator aware of a high number of high glucose levels? Or, is a physician using cardiac markers correctly? Also monitor patient and financial outcomes.
- 9. Work with respiratory. The lab should build a relationship with respiratory care providers to improve turnaround times and clinical and financial outcomes. Forgo personality conflicts and focus on what benefits patients.
- 10. **Standardize, then leverage volumes**. Most hospitals probably have standardized instruments, devices and kits and have centralized coordination. By standardizing within your health system, you will reduce costs and share knowledge and support.

# Integrating POCT in a Health-Care System

In the second presentation, James H. Nichols, PhD, DABCC, FACB, associate professor of pathology, Tufts University School of Medicine, Director, Clinical Chemistry Baystate Health System, Boston, provided tips on how an integrated health system can promote a successful POCT program. Dr. Nichols focused on appropriate use of POCT, integration of POCT in patient care pathways and promotion of self-help management through positive institutional culture at Baystate's 572-bed acute-care hospital.

Karen Appold is an editorial consultant based in Royersford, PA. Contact her at KarenAppold@comcast.net or visit her Web site at www.WriteNowServices.com.

#### Dr. Nichols' 10 tips include:

- 1. **Standardization**. By standardizing instrumentation and methods across the health system, the number of different devices is minimized, one policy can be shared among sites, a central management system can exist and training and float staff can be simplified.
- 2. **Communication**. Communication should be clear, concise and consistent. Use multiple forms of direct communication (e.g., phone, person to person and text paging instead of passive e-mails).
- Goal-oriented team. Provide POCT management with clear objectives and a delineated pathway to achieve these goals. Think outside of the box and develop multiple ways to accomplish objectives.
- Improvement. Quality improvement is a continuous process. Establish baseline performance levels and monitor and graph them.
- 5. **Networking**. Have contacts in the field, including manufacturers. This will help you to brainstorm solutions to issues and help you to think of new ways to solve problems.
- 6. **Research**. Like improvement, research can take many forms. Investigate new devices that provide technology updates and examine quality assurance (QA) trends for future improvement.
- Connectivity. Computerized POCT devices automate the QA documentation and billing process. They will also reduce expenses for multiple interfaces and streamline the review process of the volume of data.

- 8. **Integration**. POCT results should be integrated into the overall patient care pathway. Consider why the test was ordered, how the result will be used in care and if POCT is the most appropriate method.
- 9. **Self-management**. While POCT is a partnership between the lab and clinical services, inspectors hold the site performing the test and CLIA director responsible.
- 10. Positive attitude. Create a positive attitude for POCT. This is paramount to changing practice. Each person has special qualities he or she brings to an organization. Each individual can choose to complain about a problem or spend time fixing a problem.

#### What Next?

Standardization and effective communication are fundamental to building a successful POCT program, Dr. Nichols says. POCT teams must recognize individual differences and build common quality goals through compromise.

When aiming for success in a POCT program, integrate POCT into patient-care pathways and promote site self-management through a positive institutional culture.

# **Questions for STEP Participants**

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In addition to marking your answers, be sure to include all the required information on the answer sheet and a processing fee of \$3.00 per article.

In the following, choose the one best answer for each question.

- **1** An internal structure for a successful POCT program should include:
  - A. POCT coordinator
  - **B.** laboratory information systems administrator
  - **C**. laboratory director
  - D. laboratory administration
  - **E.** All of the above
- **2** Develop knowledge of POCT by participating in listservs, read periodicals and research via the Internet.
  - A. True
  - B. False

**3** Which of the following was not mentioned as a way to get to know your customers:

- **A.** Know what they want now
- **B.** Know what they want in the future
- **C.** Know their past purchases
- **D.** Know what they don't want
- **E.** Converse with individuals in other hospital departments

**4** According to the article, POCT is complex to set up.

- A. True
- B. False
- **5** To enhance your lab's image and gain support for its POCT program, the article recommends incorporating POCT into cardiology, diabetes and oncology programs.
  - A. True
  - B. False

- **6** By standardizing instrumentation and methods across a health system, the number of different devices is minimized, one policy can be shared among sites, a central management system can exist and training and float staff can be simplified.
  - A. True
  - B. False

**7** Which form of communication is not recommended when integrating POCT into a health-care system?

- A. Phone
- B. Person to person
- C. E-mail
- D. Text paging
- **E.** All of the above are recommended

**8** When setting up a POCT program, which department was not identified as a key player in the article?

- A. Administration
- **B.** Billing
- **C**. Compliance
- **D**. Finance
- E. All of the above are key players
- **9** Have contacts in the field, including manufacturers, to help you brainstorm solutions to issues and help you to think of new ways to solve problems.
  - A. True
  - B. False
- **10** Although POCT is a partnership between the lab and clinical services, inspectors will hold the site performing the test and CLIA director responsible.
  - A. True
  - B. False

Article **347** 1 Clock Hour



# Write a feature or technical article and win a cash award!

Whether you have something to say about an unusual laboratory procedure, research findings, theory — or have some thoughts about your role as a professional, AMT's Writing Awards Program is your opportunity to tell about your experiences.

# Deadline: April 15, 2009, for AMT Writing Awards

## **Technical Writing Awards**

**The AMT Technical Writing Awards** of \$150 and \$100 are for papers on topics covering any of the medical technology, medical assisting, dental assisting, or phlebotomy disciplines, allied health instruction, or lab consulting. Practical knowledge, research, techniques, scientific studies and management are all possible areas for exploration.

## Feature Writing Awards

**The AMT Feature Writing Awards** of \$150 and \$100 are for papers in the feature story category, e.g., articles on solutions to personnel problems, or any day-to-day experiences as a professional in the field of the clinical laboratory, medical or dental assisting, phlebotomy, allied health instruction, or lab consulting.

Papers should be typed, double-spaced. Mail Technical and Feature Writing entries to: *AMT Events*, 10700 W. Higgins Rd., Suite 150, Rosemont, IL 60018. The deadline for articles is April 15, 2009. All entries become the property of American Medical Technologists, which reserves publication rights. Winners will be notified before June 1. NOTE: This program is open to AMT members only.

## Article **348** 1 Clock Hour

# **Hereditary Hemochromatosis**

### **David Plaut and William McLellan**

Hereditary hemochromatosis (HH) is a genetic disease that causes the body to absorb and store unhealthy amounts of iron. The name HH stems from "hemo" (blood) and "chroma" (color), referring to the characteristic bronze skin tone that iron overload can cause. Hemochromatosis was first described by Tousseau in 1865, who cared for a diabetic patient with cirrhosis of the liver and bronzed skin pigmentation, classic symptoms of HH. HH was given its name in 1889 by Von Recklinghausen who also identified an iron-containing pigment in the liver cells of cirrhosis patients. Then in 1935, Sheldon described the hereditary nature of the disease in his text *Haemochromatosis*.

Iron is found in a number of foods and in many over-the-counter vitamin preparations. Each of us needs iron mostly to synthesize the heme in hemoglobin. We lose a certain amount each day (women lose more during their menstrual cycle); this iron must be replaced. Normally the body absorbs approximately 10% of the iron found in foods; people with hemochromatosis absorb double that amount and store it in synovium (joints) and major organs including the liver, heart, brain, pancreas, and lungs. Over many years, this excess stored iron accumulates to toxic levels that can damage or even destroy an organ. The iron overload can cause many health problems, most frequently a form of diabetes that is often resistant to insulin treatment. Because of pigmentation of the skin for the excess iron, hereditary hemochromatosis (HH) is sometimes called "bronze diabetes."

David Plaut, Plano, Texas, consultant, AMT's book reviewer and frequent speaker at AMT national and regional meetings and conventions.

William McLellan, Cooper City, Florida, career clinical laboratory scientist and speaker at AMT national convention. Although many people have never heard of the condition, HH actually isn't rare at all. The condition affects as many as 1 in every 200 people in the United States, according to the Centers for Disease Control and Prevention (CDC). Hereditary hemochromatosis is a genetic disorder caused by a mutation on a gene (HFE) that regulates iron absorption -1 in every 8 to 10 people in the United States carries a single copy of this defective gene. Because HH is an autosomal recessive condition, carriers do not have the condition themselves (the single normal gene essen-

tially balances the defective HFE gene).

In addition to mutation in HFE, other mutations can cause hemochromatosis. A genetic test is available for the most common type of hemochromatosis (the mutation in HFE) which accounts for about 85% of cases in the United States. There are two mutations in the HFE gene: One of these mutations (Cys282Tyr; C282Y) is found homozygous in 90-95% of subjects with typical HH. A second mutation (His63Asp; H63D) has also been identified but is not associated with the same degree of iron overload as with the C282Y mutation. Additionally, about 20% of subjects who are heterozygous for both mutations (C282Y, H63D-compound heterozygotes) can express the typical signs and symptoms of the common HH. A large number of patients with early disease are asymptomatic, and prompt diagnosis and treatment can result in normal life expectancy.

The diagnosis of HH can readily be confirmed by serum studies (including serum iron, TIBC and especially ferritin) and genetic testing of the HFE gene (which accounts for 85% of cases of HH). For C282Y homozygotes or compound heterozygotes diagnosed under the age of 40 years and with no biochemical or clinical evidence of liver disease, phlebotomy therapy (described later) can be initiated without the need for liver biopsy. Liver biopsy should still be considered in all other patients with iron overload. Screening of first degree relatives should now be based on genotype assessment and measurement of serum iron parameters in order to determine phenotypic expression of the disease. However, only some persons who test positive will actually develop serious illness. The other 15% of persons with symptomatic hemochromatosis have mutations not in the HFE gene, but in other genes, which may be unknown or for which gene testing is not routinely available.

The signs of HH usually do not appear until ages 40 to about 60, when iron in the body has reached damaging levels. Looked at in another way, at the age of 40, many women have already had children. In the case of an HH carrier or one with the disease, the

gene has already been passed on. Even with two mutated genes, not everyone becomes ill. Some people who test positive for HH remain symptomfree for life. Although a majority of those with two mutated genes will eventually develop some type of iron overload, far fewer of these people will absorb enough iron to develop serious problems. It is at this point that we are presented with one of the issues in HH – screening for HH. Even with two mutated genes, screening would not identify everyone who becomes ill. Further, everyone with two mutated genes will not exhibit full-blown HH.

Some cases, according to the Iron Disorders Institute, where only one mutated gene is inherited, may still eventually lead to iron overload, possibly affecting the heart. In these people, the iron overload may be triggered by a precipitating factor, such as hepatitis or alcohol abuse. Individuals with one mutated gene who become ill may also have mutations in other genes, yet to be discovered, that increase iron absorption.

The signs and symptoms of HH are many, varied and not specific. (In other words, symptoms are so vague that the diagnosis is often missed.)

- muscle aches and joint pain, primarily in the fingers, knees, hips, and ankles; one of the earliest symptoms is arthritis of the knuckles of the first and second fingers
- depression, disorientation, or memory problems
- stomach swelling, abdominal pain, diarrhea, or nausea
- loss of body hair, other than that on the scalp
- premature menopause
- · gray or bronze skin similar to a suntan
- · heart problems
- · diabetes
- enlarged liver
- increased susceptibility to bacterial infections
- chronic fatigue

Given this list, it is obvious that HH can be extremely difficult to diagnose. As symptoms progress, HH is often misdiagnosed as chronic hepatitis, other forms of diabetes, Alzheimer's disease, iron deficiency (see below under recommendations), gallbladder illness, menstrual problems, thyroid conditions, or polycythemia.

Fortunately, if the condition is diagnosed and treated early, the damage from HH is completely preventable. A number of laboratory tests are available to measure the amount of iron in the blood and diagnose iron overload:

• Serum ferritin measures the blood level of the protein that stores iron in many places in the body (30% of iron is stored in the liver). Serum ferritin may be as high as 1000 ng/mL (normal males <300, normal female <150); total body

iron in patients with HH may be 5 times the normal.

- Total iron-binding capacity (TIBC).
- A transferrin saturation percentage is calculated by dividing the TIBC into the serum iron. An elevated transferrin saturation (> 50%) percentage or serum ferritin level points to iron overload.

Therefore, in cases in which high transferrin saturation and high serum ferritin are found but gene testing doesn't confirm hemochromatosis, a liver biopsy may be needed to determine whether symptomatic hemochromatosis exists or is likely to develop.

Also, the clinician may recommend a DNA test to confirm hereditary hemochromatosis when a spouse or first-degree relative (parent, child, or sibling) has been diagnosed with the disease.

Returning to the question of screening: Given the prevalence of the condition, some specialists suggest screening to detect hereditary hemochromatosis before it causes problems. The following approaches to screening have been suggested:

- The College of American Pathologists recommends transferrin saturation testing on all adults at age 20, and every 5 years thereafter for anyone who has a family history of the condition.
- The American Hemochromatosis Society proposes genetic screening for newborns potentially to benefit both the child and the rest of the family.
- All children have routine iron testing at age 4 and that those who have a genetic risk, but remain symptom-free, be tested every 5 years on a lifetime basis.
- DNA analysis is recommended in patients whose transferrin saturation is 45% or more on a repeated test. General population screening has been waived in preference to targeting high-risk groups such as first-degree relatives of affected individuals and those with secondary iron overload, especially patients with chronic liver disorders and chronic anemia. This screening strategy is likely to continue until uncertainties regarding the natural history of the disease, age-relations, and management of asymptomatic individuals are clarified, according to a recent study by Zlocha and coworkers in Slovakia.
- Because serum ferritin is an acute-phase reactant and because the inflammatory state may inhibit the mobilization of iron from reticuloendothelial stores, the scenario of patients with serum ferritin >800 ng/ml, suggesting iron overload, and transferrin saturation <20%, suggesting iron deficiency, has become more com-

mon. The Kidney Disease Outcomes Quality Initiative recommendations suggest the use of serum ferritin and transferrin saturation in guiding iron therapy, However, there are some newer alternative markers for iron status that may be useful when serum ferritin and transferrin saturation are insufficient. These newer tests include reticulocyte hemoglobin content, percentage of hypochromic red cells, and soluble transferrin receptor, all of which have shown some promise in limited studies. Finally, the role of hepcidin, a hepatic polypeptide, in the pathophysiology of iron mobilization may prove useful. (Wish from Case Western Reserve University in Cleveland.)

Besides specific treatment for complications of the condition – such as insulin for diabetes – most individuals with HH are treated by regular phlebotomy. Initially, blood may be drawn once or twice weekly during the "de-ironing" phase until the level of iron in the body has dropped to normal. In many cases, it requires 2 or 3 years of periodic phlebotomy to reach the desired level. In addition to phlebotomy to treat the iron overload, other treatments of organ damage (heart failure with diuretics and ACE inhibitor therapy) may be instituted.

Some dietary steps may help:

- Limiting intake of alcoholic beverages, vitamin C (increases iron absorption in the gut), red meat (high in iron) and potential causes of food poisoning (shellfish, seafood).
- Increasing intake of substances that inhibit iron absorption, such as high-tannin tea, calcium, and foods containing oxalic and phytic acids (such as spinach or collard greens, which must be consumed at the same time as the iron-containing foods in order to be effective.)

Some patients do not like the idea of phlebotomy and wonder if there are other treatments. At this time, iron chelation (giving chemicals that bind up excess iron out of the blood before it can be deposited in the body) is not an approved method of treatment for HH and has not been shown to be as effective as routine phlebotomy.

Current recommendations are that men do NOT supplement their diet with iron; women who lose more iron may take vitamin-mineral supplement containing iron.

After the de-ironing phase, when the serum ferritin level has fallen into the normal range, the patient usually remains on a maintenance schedule of three to four phlebotomy sessions a year. Doctors check ferritin levels annually to monitor iron accumulation. For most people, this treatment will continue for life.

Complications of untreated iron overload include: diabetes, arthritis, depression, impotence, hypogonadism (deficient production of sex hormones by the testicle or ovary), gallbladder disease, cirrhosis (disease and scarring of the liver), heart attack, cancer, and failure of other organs.

In conclusion, when detected and treated early, any and all symptoms of hereditary hemochromatosis can be prevented, and the person can live a normal life. If left untreated, however, hereditary hemochromatosis can lead to damaging or even fatal iron overload.

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http://en.wikipedia.org/wiki/Haemochromatosis http://kidshealth.org/parent/medical/heart/hh.html http://www.aafp.org/afp/20020301/853.html – Excellent! http://www.pitt.edu/~super1/lecture/lec11811/005.htm – a powerpoint presentation

http://www.cdc.gov/genomics/hugenet/reviews/HFE.htm – good review with some detail on genes

http://www.ncbi.nlm.nih.gov/bookshelf/br.fcgi?book=gene&p art=jh – on HH in children

DP has compiled a longer set of references including abstracts for them. Should you wish them, send an e mail with your request to davidplaut@yahoo.com

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In addition to marking your answers, be sure to include all the required information on the answer sheet and a processing fee of \$3.00 per article.

In the following, choose the one best answer for each question.

- **1** HH is an autosomal dominant genetic disease.
  - A. True
  - **B.** False
- **2** Only about 20% of dietary iron is absorbed by humans.
  - A. True
  - **B.** False
- **3** The most common health problem other than HH that these patients have is diabetes.
  - A. True
  - B. False
- **4** HH is found in about 1 in 2000 people in the US.
  - A. True
  - B. False
- **5** All persons who carry two genes will develop the disease between ages 40 and 60.
  - A. True
  - **B.** False

**6** One common test for HH is serum ferritin.

- A. True
- **B.** False
- **7** Carriers of only a single HH gene may develop some symptoms.
  - A. True
  - **B.** False
- **8** The symptoms of HH are so vague that the diagnosis is often missed.
  - A. True
  - B. False
- **9** Screening of children of parents with HH is recommended.
  - A. True
  - B. False
- **10** The standard treatment for HH is periodic chelation and phlebotomy.
  - A. True
  - B. False

# **Battling Leukemia**

Article **349** 1 Clock Hour

# Methods of diagnosis expected to improve

#### **Karen Appold**

Leukemia is a cancer of the white blood cells (WBCs). It results from an uncontrolled proliferation of a clone of abnormal cells. "Over the past 100 years, our understanding of leukemia has evolved that we now recognize it to be a disease of the hematopoietic stem cell," says Ian Chin Yee, MD, FRCPC, chief/chair of hematology, associate professor of medicine, London Health Sciences Centre, University of Western Ontario, London, Ontario, Canada.

All humans have a tiny population of hematopoietic stem cells that function to generate all the normal cellular constituents of blood, including the red blood cell, WBC and platelets. By definition, stem cells have the capacity to self renew and generate multiple other cell lineages. It is believed that leukemia arises when a genetic alteration occurs in the hematopoietic stem cell or very early progenitor cell, Dr. Yee explains. These genetic mutations can occur spontaneously or as a result of viruses, toxic chemicals or radiation. A mutated stem cell becomes leukemic if its growth is uncontrolled. Furthermore, leukemic cells expand at the expense of other normal bone marrow elements, eventually resulting in bone marrow failure and causing anemia, neutropenia and thrombocytopenia.

Leukemia is generally divided into two broad categories – acute leukemia and chronic leukemia. As the term implies, Dr. Yee says acute leukemia describes the very aggressive natural history of this cancer, which usually rapidly progresses over a period of weeks to months and causes death. Chronic leukemias, on the other hand, behave more indolently and usually progress over a period of years.

Leukemias are further subdivided based upon cell type – either lymphoid or myeloid origin. Based on this classification, leukemias can be subdivided into acute myeloblastic leukemia (AML), acute lymphoblastic leukemia (ALL), chronic myeloid leukemia (CML) and chronic lymphoid leukemia (CLL). As the types of leukemia behave and respond differently to treatments, it is important to distinguish these blood cancers, Dr. Yee says.

#### **Tried and True Tests**

Because leukemias are essentially a blood disorder, Wendy Brown, RT, technologist, investigational hematology, London Health Sciences Centre, says the hematology laboratory plays a crucial role in diagnosing and differentiating the subtypes of leukemia. Initially, a complete blood count (CBC) will be run to identify changes in the key parameters affected by leukemia, i.e., the WBCs, hemoglobin and platelet counts.

In CLL, an elevated lymphocyte count detected by a hematology analyzer is often the first indication that a patient may have a disease affecting the blood, Brown says. Similarly, in CML, if a persistent increase in neutrophils exists without the presence of infection, further investigation should be performed to rule out a leukemic process.

In acute leukemias, patients often present a severe illness that results in either bleeding or infection. A CBC may show a dramatic increase in the WBC accompanied with very low hemoglobin and platelets. In a subtype of AML affecting mainly promyeloctes, Brown says patients may also have derangement of their coagulation parameters as detected by the prothrombin time and activated partial thromboplastin time. A reduced fibrinogen with elevated D-Dimer, indicating a process of disseminated intravascular coagulation, may also be seen.

Morphologic examination with a microscope is the first critical step in diagnosing and classifying leukemias. For this reason, Brown says the general duty technologist plays a critical role in the initial identification of an abnormal cell population on a blood film. The hallmark of acute leukemia is the presence of very immature cells, so-called blasts.

In contrast, Brown says chronic leukemias are associated with more mature lymphoid or myeloid cells. Because by definition acute leukemia is a proliferation of very immature blast cells, distinguishing ALL from AML can often be challenging and require further testing. Additional morphologic tools include special stains, e.g., the Sudan Black, to identify cells of myeloid origin.

The standard approach to the laboratory diagnosis of ALL involves the morphologic and immunophenotypic evaluation, adds Steven J. Melnick, PhD, MD, chief, Department of Pathology and Clinical Laboratories, Miami (FL) Children's Hospital. The morphologic classification is based on French-American-British criteria. In ALL, a consensus that a

Karen Appold is a freelance medical writer and editor based in suburban Philadelphia. To learn more about her services, visit www.WriteNowServices.com. Contact her at kappold@msn.com. diagnosis can be established if certain flow cytometric criteria are specified exists. However, in all cases, the final interpretation should be correlated with clinical and morphologic features and possibly other ancillary studies.

#### The Role of Flow Cytometry

In the past 20 years, Michael Keeney, ART, FIMLS, associate scientist, Lawson Health Research Centre, London Health Sciences Centre, says flow cytometry has played an increasingly important role in identifying specific surface antigens associated with confirming myeloid or lymphoid differentiation and subclassifying leukemia. In CLL, most North American cases arise from an abnormal clone of B cells, i.e., the cells necessary for antibody production. Flow cytometry plays a crucial role in identifying the abnormal lymphocyte population and can demonstrate the clonal origin of the cell by examining the expression of surface kappa or lambda light chains on the affected B cell, confirming its evolution from an abnormal malignant clone of cells.

In acute leukemias, rapid determination of the cell of origin is crucial to allow the appropriate therapy to be started quickly. In a specific subtype of AML, i.e., promyelocytic, flow cytometry is often the first indicator that disease is present and will work in conjunction with cytogenetics to ensure confirmatory testing is done using fluorescence *in situ* hybridization (FISH). Because acute leukemia is the most common cancer in children, Keeney says a strong emotional dimension often exists for everyone involved in patient treatment and rapid provision of results by the laboratory can significantly assist in the quality of patient care.

Standard morphologic and flow cytometric techniques that identify the molecular abnormality associated with acute and chronic leukemia can often be helpful in diagnosis and prognosis of these diseases, Dr. Yee says. For example, cytogenetic analysis in CML identifies the Philadelphia chromosome, i.e., an abnormal chromosome resulting from the translocation of a portion of DNA between chromosomes 9 and 22. This abnormality is diagnostic of this form of leukemia and was first identified in the 1960s. This was the first definite proof that leukemia was a clonal disease resulting from the proliferation of a small population of leukemic stem cells.

One of the greatest advances in the past decade is the identification of specific genetic abnormalities in different types of leukemia, Dr. Yee says. The genetic abnormality associated with the Philadelphia chromosome in CML has resulted in the development of drugs specifically targeted to the gene product produced by this clonal abnormality. Highly effective drugs, which block the product of this gene, have been effective in treating CML.

In one type of acute leukemia, i.e., acute promye-

locytic leukemia, a translocation involving chromosomes 15 and 17 occurs and alters the retinoic acid receptor gene. This genetic abnormality is not only diagnostic of this subtype of leukemia, but is also a target of treatment with retinoic acid combined with conventional chemotherapy. These combination therapies have resulted in improved survival rates of 70 to 80 percent in this type of leukemia. Several other genetic abnormalities have been associated with specific types of leukemia and have been helpful in predicting prognosis for patients and will hopefully lead to other target specific therapies, Dr. Yee says.

Flow cytometry immunophenotyping of acute leukemia provides important prognostic information that is useful therapeutically, particularly in relation to childhood ALL, Dr. Melnick adds. For example, bright expression of CD45 and CD20 are considered poor prognostic indicators and patients may require more aggressive therapy. In B lineage ALL, CD34 is a good prognostic indicator, while CD34 is a poor prognostic indicator in T lineage ALL. DNA ploidy also plays an important role in prognosis. When the DNA index is greater than 1.16, the long-term survival rate is approximately 90 percent. When the DNA index is less than or equal to 1.16, the survival rate is in the 50 to 80 percent range depending upon other various clinical factors.

Genotypic attributes of ALL are also extremely important for prognosis in children, Dr. Melnick says. Therefore, technologies such as FISH and nucleic acid amplification are routinely employed to evaluate ALL. For example, trisomies of certain autosomes such as chromosomes 4, 10, and 21 and certain chromosomal translocations such as t(12;21)(p13;q22) associated with the TEL-AML1 fusion gene are associated with a favorable prognosis in B lineage ALL. Other translocations such as t(1;19)(q23;p13) (E2A/PBX1 fusion), t(4;11)(q21;q23) (MLL/A4F fusion) and t(9;22)(q34;q11) (BCR/ABL fusion) are associated with a poor prognosis.

#### New Tests Expected to Emerge

The identification of genetic abnormalities also allows laboratorians to monitor for the presence of small populations of leukemic cells, Dr. Yee says. Classic morphologic microscopic examination identifies as many as 1/100 or more leukemic cells. However, flow cytometry has a sensitivity of 1/1,000 to 1/10,000. In contrast, if a specific molecular abnormality can be identified, the use of real time reverse transcription-polymerase chain reaction techniques allow for the identification of a leukemic population in the 1/100,000 to 1 in a million range. This sensitive monitoring may allow for earlier disease detection and predict individuals who will fail to respond and eventually relapse with their disease.

In addition to molecular testing, exciting developments are also seen in flow cytometric functional assays, Keeney says. Because of its quantitative and qualitative nature, flow cytometry can be used to evaluate cell treatments affecting gene expression. In the example of CML, abnormal phosphorylation of signal transducer and activator of transcription 5 (Stat5) is present. This abnormal phosphorylation is inhibited by treatment with the tyrosine kinase inhibitor imatinib mesylate. A recent paper describes an assay which can detect and quantify cellular response to this drug and suggests that it could be used successfully as a measure of Bcr/Abl activity1.

The diversity of immunophenotypic and genotypic attributes of ALL that underlie morphology have yielded many important associations which have prognostic importance and have contributed greatly to the present understanding of ALL, Dr. Melnick says.

However, the understanding of the molecular mechanisms that underlie these features has given rise to other techniques than those described that are important for the prognosis of ALL.

Novel technologies that will likely be used in the near future for the diagnosis and prognostication of ALL include drug resistance assays which may be performed using flow cytometry methodologies, microarray technology which has been used to identify novel genes of prognostic importance in ALL and may be used to identify relevant patterns of gene expression and proteomics which involve the study of proteins and their interactions, Dr. Melnick says.

These methodologies are among those that fall into the evolving field of cytomics, considered to be the science of cell-based analysis that integrate genomics and proteomics with dynamic functions of cells and tissues, Dr. Melnick says. This burgeoning field is of particular relevance to acute leukemia and other hematopoietic malignancies because of the relative ease in which the cells of inters may be separated for analysis.

#### Looking Ahead

In summary, Keeney says the future for diagnostic testing and disease monitoring is a promising field and advances in molecular and cellular analysis will have an increasingly important role to play in this group of diseases.

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In addition to marking your answers, be sure to include all the required information on the answer sheet and a processing fee of \$3.00 per article.

In the following, choose the one best answer for each question.

- 1 Leukemia arises when a genetic alteration occurs in the hematopoietic stem cell or very early progenitor cell.
  - A. True
  - **B.** False
- **2** Which is not a subdivision of leukemia?
  - **A.** Acute myeloblastic leukemia
  - **B.** Acute lymphoblastic leukemia
  - **C.** Chronic myeloid leukemia
  - **D.** Chronic lymphoid leukemia
  - **E.** All of the above are correct
- **3** In acute myeloblastic leukemia, an elevated lymphocyte count detected by a hematology analyzer is often the first indication that a patient may have a disease affecting the blood.
  - A. True
  - **B.** False
- 4 In chronic leukemias, patients often present a severe illness that results in either bleeding or infection.
  - A. True
  - **B.** False
- **5** Morphologic examination with a microscope is the first critical step in diagnosing and classifying leukemias.
  - A. True
  - **B.** False

- **6** The hallmark of chronic leukemia is the presence of very immature cells. In contrast, acute leukemias are associated with more mature lymphoid or myeloid cells.
  - A. True
  - B. False
- 7 In a specific subtype of acute myeloblastic leukemia, i.e., promyelocytic, flow cytometry is often the first indicator that disease is present and will work in conjunction with cytogenetics to ensure confirmatory testing is done using fluorescence *in situ* hybridization (FISH).
  - A. True
  - **B**. False
- **8** Acute leukemia is the most common cancer in children.
  - A. True
  - B. False
- **9** Technologies such as FISH and nucleic acid amplification are routinely employed to evaluate chronic lymphoid leukemia.
  - A. True
  - **B.** False
- **10** Novel technologies that will likely be used soon for the diagnosis and prognostication of acute lymphoblastic leukemia include:
  - **A** Drug resistance assays
  - B. Microarray technology
  - **C**. Proteomics
  - **D.** All of the above
  - $\textbf{E.} \ A \ and \ C \ only$

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- Unit 6, Adolescent Gynecology (#01-6)
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- Unit 8, The Breast in Health and Disease Part II (#01-8)
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#06-3	Dealing with Depression	3.0	\$25.00		۵	🗋 RMA 🛄 MT 🛄 MLT 🛄 RDA 🛄 RPT
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## The Scoreboard on Racial and Ethnic Disparities in Health Care

The overall health of the population has improved over the past decade, but not all citizens have shared in the improvements.

#### Access to primary care

Primary care is the underpinning of the health care system. Research has indicated that having a routine and usual source of care raises the chance of receiving preventive care and other health services.

- Hispanic children are nearly three times more likely as non-Hispanic white children to have no routine and usual source of health care.
- About 30% of Hispanics and 20% of African-Americans lack a routine and usual source of health care compared to less than 16% of Caucasians.

#### **Diagnosis and Treatment**

Race and ethnicity influence the chance of receiving many specific procedures and treatments.

- Heart disease: African Americans are 13% less likely to undergo coronary angioplasty and 1/3 less likely to undergo bypass surgery than are Caucasians.
- Asthma: Among preschool children hospitalized for asthma, only 7% of African-American and 2% of Hispanic children, compared to 21% of Caucasian children, are prescribed medications to prevent future asthma-related hospitalizations.
- Breast cancer: The length of time between an abnormal screening mammogram and the followup diagnostics test to determine whether a woman has breast cancer is more than twice as long in Asian-American, African-American, and Hispanic women than in Caucasian women.

#### **Physician Decision-making**

Factors other than insurance and income influence the quality of care people get. In a small study of physician decisions, it was noted that African-American women were significantly less likely than Caucasian men to be recommended for cardiac catherization when both groups reported the same symptoms.

#### **Hospital Characteristics**

A Boston quality-of-care study indicated that the quality of care for African-Americans was lower in non-teaching hospitals than in teaching hospitals. In another study, Caucasian patients were more likely than Hispanic and African-Americans to receive invasive cardiac procedures, a factor strongly associated with quality of cardiac care.

#### **Cultural and Communications Barriers**

Cultural expectations, assumptions, and language factors affect the way patients and clinicians interact, which surely affects the health care patients get and the outcome of their care.

#### References

Statistics and factual statements were taken from "Addressing Racial and Ethnic Disparities in Health Care," Agency for Healthcare Research Quality (AHRQ), http://www.ahrq.gov; http:// www.bls.gov

"Tips for Adult Learners – College Success," The Thomson Corp., Lawrenceville, NJ

This "Fast Facts" article was prepared by Gerard P. Boe, PhD, Executive Director of American Medical Technologists' Institute for Education (AMTIE), Editor of AMT Journal of Continuing Education Topics & Issues, and Chair, AMT CLC Evaluation Committee. We welcome reader submissions for future "Fast Facts." Send them to the AMT Office, attention Journal Editor.

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## ABSTRACTS FROM THE CURRENT LITERATURE

None of us can read all the medical literature, even that that pertains particularly to medical technology. Presented below are short abstracts from current literature presented with the hope that they will answer some of your questions and lead you to a better understanding of what is happening. You are encouraged to send copies of articles you have found in journals or on the Internet to AMT and we will abstract them for an upcoming issue. We encourage and welcome future contributors from readers of this journal. Please send your abstracts to Editor, Journal of Continuing Education Topics & Issues, 10700 W. Higgins Rd., Suite 150, Rosemont, IL 60018.

The following abstracts were contributed by David Plaut, Plano, TX, who is AMT's book reviewer and a frequent speaker at AMT annual conventions.

Your hospital librarian or your public librarian can help obtain copies of the full text of these articles.

# Direct comparison of the BD phoenix system with the MicroScan WalkAway system for identification and antimicrobial susceptibility. *J Clin Microbiol*. 2008 Jul;46(7):2327-33. Snyder JW, jwsnyd01@gwise.louisville.edu

The Phoenix automated microbiology is designed for the rapid identification (ID) and antimicrobial susceptibility testing (AST) of clinically significant human bacterial pathogens. We evaluated the performance of the Phoenix instrument in comparison with that of the MicroScan WalkAway system in the ID and AST of gram-negative clinical strains and challenge isolates of Enterobacteriaceae (n = 150) and nonfermentative gram-negative bacilli (NFGNB; 45 clinical isolates and 8 challenge isolates). ID discrepancies were resolved with the API 20E and API 20NE conventional biochemical ID systems. The standard disk diffusion method was used to resolve discordant AST results. The overall percentages of agreement between the Phoenix ID results and the MicroScan results at the genus and species levels for clinical isolates of Enterobacteriaceae were 99 and 98%, respectively; following resolution with conventional biochemical testing, the accuracy of the Phoenix system was determined to be 100%. For NFGNB, the levels of agreement were 100 and 98%, respectively. Both systems incorrectly identified the majority of the uncommon nonfermentative nonpseudomonal challenge isolates recovered from cystic fibrosis patients; these isolates are not included in the databases of the respective systems. For AST of Enterobacteriaceae, the rate of complete agreement between the Phoenix results and the MicroScan results was 97%. For NFGNB, the rate of complete agreement between the Phoenix results and the MicroScan results was 89%. Following the confirmatory testing of nine clinical isolates initially screened by the MicroScan system as possible extended-spectrum-beta-lactamase (ESBL)-producing organisms (seven Klebsiella pneumoniae isolates and two Escherichia coli isolates), complete agreement was achieved for eight isolates (one ESBL positive and seven negative); one false positive was obtained with the Phoenix instrument. The MicroScan system correctly detected the 10 ESBL challenge isolates, versus the 6 detected by the Phoenix system. Overall, there was good correlation between the Phoenix instrument and the MicroScan system for the ID and AST of Enterobacteriaceae and common NFGNB. The Phoenix system is a reliable method for the ID and AST of the majority of clinical strains encountered in the clinical microbiology laboratory. Until additional performance data are available, results for all Klebsiella pneumoniae or Klebsiella oxytoca and E. coli isolates screened and confirmed as ESBL producers by any automated system should be confirmed by alternate methods prior to the release of final results.

Note: For more on comparison of this type of instrumentation visit pubmed (the national library of medicine websites for abstracts) and look at these other articles.

- Two-center collaborative evaluation of performance of the BD phoenix automated microbiology system for identification and antimicrobial susceptibility testing of gram-negative bacteria. [*J Clin Microbiol.* 2006]
- Evaluation of the Phoenix system for identifying and determining the susceptibility of clinical isolates. Comparative study with the Microscan system] [*Rev Esp Quimioter. 2004*]
- Detection of extended-spectrum beta-lactamases among Enterobacteriaceae by use of semi-automated microbiology systems and manual detection procedures. [*J Clin Microbiol. 2007*]
- Evaluation of the BD Phoenix automated microbiology system for identification and antimicrobial susceptibility testing of Enterobacteriaceae. [*J Clin Microbiol. 2006*]
- Two-center collaborative evaluation of the performance of the BD Phoenix automated microbiology system for identification and antimicrobial susceptibility testing of Enterococcus spp. and Staphylococcus spp. [J Clin Microbiol. 2003]

# Targeted therapy of cancer: new roles for pathologists--prostate cancer. *Mod Pathol.* 2008 Suppl 2:S44-55. Rubin MA. <u>rubinma@med.cornell.edu</u>

The clinical dilemma today in the management of prostate cancer (PCA) is to distinguish men who need definitive treatment from men who have indolent disease. As demonstrated most recently by the randomized

Scandinavian trial evaluating the benefit of prostatectomy over Watchful Waiting, surgery significantly decreased the risk of death from PCA. However, this same study also suggests that 19 men need to be treated to benefit one man. Given the high prevalence of the disease, the aging of the population, and the potential morbidity of treatment, the ability to distinguish aggressive from indolent forms of PCA is critical. (At this time, there is not an easy way to differentiate them.) Novel therapies are in various stages of clinical trials. The discovery of novel therapeutic approaches is an active area of clinical research. Eliminating aggressive PCA before it advances is a high priority in the biomarker field. In addition, the recent discovery that a significant percentage of PCAs harbor a TMPRSS2-ETS gene fusion suggests that targeting either the ETS transcription factors or the fusion product may offer a novel approach to therapy. However, in 2007, the mainstay of treatment for advanced PCA remains androgen ablation therapy as originally introduced in the early 1940s.

# Targeted therapy of cancer: new roles for pathologists in colorectal cancer. *Mod Pathol.* 2008 Suppl 2:S23-30. Hamilton SR. <u>shamilto@mdanderson.org</u>

Personalized/individualized/tailored therapy for each patient is an important goal for improving the outcome of patients with colorectal adenocarcinoma and includes the intention to maximize efficacy and minimize toxicity of chemotherapeutic agents. Numerous barriers must be overcome to reach this goal because outcome is affected by an unholy trinity of tumor characteristics that include somatic alterations at the DNA, RNA, and protein level; patient characteristics that include germline genetic differences such as polymorphisms in enzymes affecting the metabolism of chemotherapeutic agents; and environmental exposures and factors that include diet and physical activity. At present, evaluation of epidermal growth factor receptor (EGFR) expression by immunohistochemistry in colorectal adenocarcinoma is generally required for treatment with one of the monoclonal antibody therapies directed against that target, despite the absence of evidence for predictive value of the assay, whereas EGFR fluorescent in situ hybridization (FISH) may be predictive. Numerous other potential markers have been identified but have not yet reached levels of evidence that support their routine usage. Additional markers will come into routine usage as reports of research studies continue to appear in the literature. Clinical trials driven by molecular targets and agents directed against them, and understanding of the conflicting data on utility of markers reported in the literature, are needed to advance the field.

# Can increased incidence of deep vein thrombosis (DVT) be used as a marker of quality of care in the absence of standardized screening? The potential effect of surveillance bias on reported DVT rates after trauma. *J Trauma*. 2007 63:1132-5; discussion 1135-7. Haut ER, *et al*. <u>ehaut1@jhmi.edu</u>

Deep vein thrombosis (DVT) is a significant cause of morbidity and mortality in trauma patients, even with appropriate prophylaxis. Many national agencies have suggested DVT incidence as a measurement of health care quality, but none has recommended a standardized screening approach. Duplex ultrasound serves an important role as a noninvasive diagnostic tool for detection of DVT (as is d-dimer). However, screening of asymptomatic patients for DVT is somewhat controversial and these practices vary widely among trauma centers. We hypothesized that as the number of screening duplex examinations in trauma patients increases, the rate of DVT identification will also increase. A retrospective cohort study of 21,961 patients from an urban, university-based Level I trauma center for more than 11 years (1995-2005) was undertaken. We grouped patients according to admission at the trauma service either before or after implementation of a written practice management guideline for DVT prophylaxis and duplex ultrasound surveillance in 1998. We compared duplex, DVT, and pulmonary embolism rates per 1,000 trauma admissions. The proportion of trauma patients having a duplex ultrasound increased significantly (20.9-81.5 per 1,000 trauma admissions). The rate of DVT reported increased 10-fold (0.7-7.0 per 1,000 admissions) between the two periods. The pulmonary embolism rate increased almost fivefold (0.7-3.2 per 1,000), although this difference was not statistically significant. Increasing the number of duplex screening exams resulted in an increased rate of DVT identification. In the absence of standardized surveillance, DVT rates may be more influenced by how often caregivers look for these events rather than the quality of care provided.

#### BNP-guided therapy optimizes the timing of discharge and the medium term risk stratification in patients admitted for congestive heart failure. *Monaldi Arch Chest Dis.* 2007 Sep;68(3):154-64. (In Italian). Valle R, *et al.* <u>robertovalle@libero.it</u>

Despite a consistent body of data demonstrating the benefits of drug therapy in HF, persistently high rates of readmission, especially within six months of discharge, continue to be documented. Plasma brain natriuretic peptide (BNP), is correlated with the severity of left ventricular dysfunction and relates to outcome. The aim of the study was to evaluate if plasma levels of BNP would provide an index to guide drug treatment and to predict medium-term prognosis in HF patients after hospital discharge. We evaluated 200 consecutive pts (35-96) years, 49% male versus 51% female hospitalized for HF (DRG 127). Standard echocardiography was performed and left ventricular systolic/diastolic function was assessed; plasma BNP levels were measured on days 1 and after initial treatment. Using a cut-off of 240 pg/ml and/or changes in plasma BNP (days 2-3 after admission), 2 groups were identified: the low BNP group-responders (n = 68, BNP < 240 pg/ml and/or > 29% reduction) and the high BNP group-non responders (n = 132, BNP > 240 pg/ml and/or < 30% reduction). The high BNP group showed a different pattern of clinical variables according to the severity of the disease New York Heart Association (NYHA) functional class, left ventricular ejection fraction, ischemic etiology and age. A sustained elevation of plasma BNP (> 240 pg/mL) indicated the presence of a clinical unstable condition requiring further intervention whereas pts with low BNP values were discharged after 24 hours. During a mean follow-up period of 3 months, there were 62 cardiac events, including 15 cardiac deaths, 22 readmissions for worsening heart failure and 25 clinical decompensation requiring diuretic treatment. The incidence of clinical events was significantly greater in the patients with higher levels of BNP (admission and discharge) than in those with lower levels (42% vs. 10%) and plasma values > 500 pg/ml identified a subgroup at high risk of death.

# Haemolysis: an overview of the leading cause of unsuitable specimens in clinical laboratories. *Clin Chem Lab Med.* 2008;46:764-72 Lippi G, *et al.* <u>www.specimencare.com</u>

While there is widespread perception that most medical errors arise from an inappropriate or delayed clinical management, the issue of laboratory errors is receiving a great deal of attention due to their impact on the quality and efficiency of laboratory performances and patient safety. Haemolytic specimens are a frequent occurrence in clinical laboratories, and prevalence can be as high as 3.3% of all of the routine samples, accounting for up to 40%-70% of all unsuitable specimens identified, nearly five times higher than other causes, such as insufficient, incorrect and clotted samples. This article focuses on this challenging issue, providing an overview on prevalence and leading causes of in vivo and in vitro haemolysis, and tentative guidelines on identification and management of haemolytic samples in clinical laboratories. This strategy includes continuous education of healthcare personnel, systematic detection/quantification of haemolysis in any sample, immediate clinicians warning on the probability of in vivo haemolysis, registration of non-conformity, completing of tests unaffected by haemolysis and request

# Prevalence and type of pre-analytical problems for inpatient samples in coagulation laboratory. *J Eval Clin Pract.* 2008 Apr;14(2):351-3 Salvagno GL, *et al.*

Evidence was provided that poor standardization in the extra-analytical phases of the testing process has the greatest influence on test results, though little information is available so far on prevalence and type of pre-analytical variability in coagulation testing. The present study was designed to describe all pre-analytical problems on inpatient routine and stat samples recorded in our coagulation laboratory over a 2-year period and clustered according to their source (hospital departments). Overall, pre-analytic problems were identified in 5.5% of the specimens. Although the highest frequency was observed for paediatric departments, in no case was the comparison of the prevalence among the different hospital departments statistically significant. The more frequent problems could be referred to samples not received in the laboratory following a doctor's order (49.3%), haemolysis (19.5%), clotting (14.2%) and inappropriate volume (13.7%). Specimens not received prevailed in the intensive care unit, surgical and clinical departments, whereas clotted and haemolysed specimens were those most frequently recorded from paediatric and emergency departments, respectively. The present investigation demonstrates a high prevalence of pre-analytical problems affecting samples for coagulation testing. Full implementation of a total quality system, encompassing a systematic error tracking system, is a valuable tool to achieve meaningful information on the local pre-analytic processes most susceptible to errors, enabling considerations on specific responsibilities and providing the ideal basis for an efficient feedback within the hospital departments.

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	Monday	Workshops (if not registered for full package)		\$25	\$25	\$25	\$25	\$25				
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	Wednesday	Includes admission to all lectures, Continental Breakfast, Coffee Breaks			\$150	\$120	\$150	\$60				
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	Friday	(*Friday limited to AMT members only) Includes adr to Town Hall & Business Meeting, Continental Break		\$25	*	\$25	*	*				
	Friday Night Social (optional)	Dinner Cruise on paddle wheel riverboat down sceni St. Croix River, with time for browsing/shopping in a historic town of Stillwater. Includes round-trip bus transportation from Hilton Hotel, buffet dinner, boat with music and dancing, tax & gratuity.	ic quaint,	\$52	\$52	\$52	\$52	\$52				
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