LEGISLATURE

The Legislature is now in the second year of the 2017-2018 session. Thus far, the Legislature has been bogged down dealing with in-house personnel issues and is currently experiencing an unprecedented 4 resignations with an additional Assembly Member taking a temporary leave of absence. These resignations will necessitate a number of special elections to fill the vacancies. The areas affected are AD 39, AD 45, AD 54, AD 58 and SD 32. Please refer to the Legislator list on the website for additional information. On March 21 Senator Toni Atkins was sworn in to succeed President pro Temp Kevin de Leon. She is the first woman to hold the position. Senator Atkins is also the first person since the 19th century to hold both of the Legislature’s top jobs – Assembly Speaker and Senate President pro Tempore.

The Legislature has passed the important bill introduction deadline. April will see heavy committee hearings as April 27 is the deadline for policy committees to hear and report to fiscal committees fiscal bills introduced in their house.

LEGISLATION

The following is a partial list of legislation that Public Policy Advocates (PPA) has identified through the bill introduction process that either directly or may through amendments affect CAMLT. This section also includes legislation still viable that was introduced in 2017. Check the website (http://camlt.org/legislation) for updates on bill status, amendments, Legislator lists, and Committee assignments.

NEWLY INTRODUCED LEGISLATION

AB 1802 (Salas) Optometry: Scope of Practice. As introduced 1/9/18 - Watch

- Currently, this bill does not contain changes to clinical lab practices
- The Author’s office has indicated that this measure is a true spot bill. As such, we will closely monitor for any amendments that might adversely affect CAMLT

The Optometry Practice Act provides for the licensure and regulation of the practice of optometry by the State Board of Optometry, which is within the Department of Consumer Affairs. That act provides that the practice of optometry includes the prevention and diagnosis of disorders and dysfunctions of the visual system, and the treatment and management of certain disorders and dysfunctions of the visual system, as well as the provision

Continued on page 3

Also In This Issue...
Message From the President

IT’S NOW OR NEVER… your support is needed!

Take a moment to think about what CAMLT has done for our profession in California:

We have successfully lobbied for more than 20 years to keep our high standards, prevented other professions from performing laboratory tests with no education or training, and therefore protected public health and safety.

Over the past 20 years, with little or no training programs to connect to….the message of our mission (protecting high standards for our profession) has been lost to many.

No longer do we mail our Newsline to laboratories to get posted. Union labs do not even allow anything to be posted. Members have forgotten to print and post it at their work. Previous members have forgotten the importance of our organization.

MAKE IT YOUR GOAL THIS YEAR for each member TO BRING IN 4 NEW MEMBERS AND GET THEM INVOLVED! We value every member, but we need your support to stay in business!

The age of e-mails is a curse and a blessing. Lots of info, but much of it gets ignored.

You cannot be ignored….light a fire of enthusiasm and others will follow you!

Now is the time to plan your lab week activities. Have your District Consultant help you…you will be able to attract new members and volunteers using your personal relationships to show the value of our organization.

-Also we must all get more members to come to social and educational events as well as volunteer to help coordinate them.

-Without ongoing volunteers, our association will have to close its doors!

-We have already found a number of enthusiastic new CLSs for this, but we need more volunteers to put on our exceptional programs.

-Have your chapter committee find new CLSs and mentor them over the next 2 years to take on some duties.

Looking forward to meeting you all,

Ilene Dickman
President, CAMLT
Continued from page 1

of habilitative or rehabilitative optometric services, and doing certain things, including the examination of the human eye or eyes. Existing law authorizes an optometrist certified to use therapeutic pharmaceutical agents to, among other things, administer immunizations if the optometrist meets certain requirements. AB 1802 would correct an erroneous cross-reference in the provision relating to the authority of an optometrist certified to use therapeutic pharmaceutical agents to administer immunizations, and would make other non-substantive changes. Location: AB 1802 was referred to Assembly Business and Professions on 1/22/18.

AB 2281 (Irwin) Clinical Laboratories: Licensed Medical Laboratory Technicians, as introduced 2/13/18 - WATCH

- Would expand Medical Laboratory Technician scope of practice to include moderately complex blood smear reviews, microscopic urinalysis, and blood typing (ABO/Rh testing).

Existing law provides for the licensure, registration, and regulation of clinical laboratories and various clinical laboratory personnel by the State Department of Public Health. Existing law also requires a medical laboratory technician to be licensed by the department, sets forth the duties that a licensed medical laboratory technician is authorized to perform, and prohibits a licensed medical laboratory technician from performing microscopic analysis or immunohematology procedures. This bill would exempt from that prohibition blood smear reviews, microscopic urinalysis, and blood typing of moderate complexity. Location: On 3/1/18, AB 2281 was referred to Assembly Business and Professions and will be heard on 4/3/18.

2017 LEGISLATION THAT IS STILL VIABLE

AB 613 (Nazarian), Healing Arts: Clinical Laboratories, as amended 8/29/17 — OPPOSE

- Would allow High School graduates to perform the moderately complex total protein refractometer test on potential plasma donors at plasma collection centers.

- Standards outlined in the bill for high school students performing this test do not ensure the health and safety of plasma donors. AB 613 authorizes a person with training set forth in the bill that exceeds federal Clinical Laboratory Improvement Amendments (CLIA) standards but allows non-qualified personnel under California laboratory law (BPC 1206.5(b)) to perform moderate complexity testing with regard to plasma. AB 613 would allow lesser trained and educated persons than currently permitted by law to perform a total protein refractometer analysis, categorized as a moderately complex test by the Food and Drug Administration, in a licensed plasma collection facility. If the protein refractometer test and/or calibration is done incorrectly there is potential to cause donor harm. AB 613 is sponsored by the Plasma Protein Therapeutics Association and is supported by Grifols and the California Chronic Care Coalition. Location: AB 613 is now a 2-year bill. The Sponsors maintain that they have yet to workout amendments relative to supervision and reporting standards related to unlicensed personnel performing total protein tests. This bill will be heard in the Senate in 2018.

LOOKING AHEAD

We continue to see legislation introduced to expand scope of practice without requisite education and training. CAMLT is the only professional association representing clinical laboratory science personnel that retains a lobbying firm in Sacramento to be the voice for clinical laboratory personnel in Sacramento before the legislature and state government. CLS’s, MLT’s and phlebotomists must ensure the growth and vibrancy of a strong, well financed, well organized CAMLT to protect the best possible patient safety in laboratory testing and the preservation of our critical profession. Build CAMLT membership. Contribute to LAB-PAC. Educate your own legislators. Only this will secure our future and the future of the patients that we serve.

Please donate to the CAMLT LAB-PAC fund. Your voice in the political process is much louder as CAMLT than as an individual. Contribute to the collective resources of CAMLT to grow your political clout. Visit the LAB-PAC Page to donate online or for a donation form. Your gift in any amount will help our profession. Contribute now!

Please donate online or mail donations to:

CAML&T LAB-PAC
39656 Mission Blvd., Fremont, CA 94539
Federal ruling may eliminate the jobs of educated and trained medical laboratory scientists and technicians outside California.

Recent actions by the Federal Government won’t be affecting our nurses or medical laboratory testing personnel...yet...thanks to organizations like the California Association for Medical Laboratory Technology.

Recently, the Centers for Medicare and Medicaid Services (CMS) made an unprecedented move which may eliminate the jobs of educated and trained medical laboratory scientists (MLS) and technicians (MLTs)! These scientists and technicians are trained to perform high and/or moderate-complexity and **lifesaving laboratory testing daily.** To compound the decision, the government decided to place the task and responsibility of high and moderate-complexity laboratory testing on nurses! Nurses, who in the majority of cases are overburdened, stretched in responsibility and have no formal training or education in high or moderate-complexity laboratory testing methods, protocols and procedures!

The scary and highly concerning factor is that CMS do not publish a proposed rule on this issue and review public comments before proceeding. The American Association of Clinical Chemistry (AACC) wrote in a letter to CMS, as the decision “sets a dangerous precedent for altering personnel requirements without public consultation.”

The impact of this decision is far reaching. First and foremost, it puts US lives at risk! **Medical laboratory professionals** provide **more than 70 percent** of objective patient information to physicians and other healthcare providers so they can provide accurate diagnosis and treatment plans, according to a 2002 study in Clinical Leadership and Management Review. Most people believe that their doctor performs their lab tests...they don’t...nor would you want them to!

MLS and MLT professionals learn the medical laboratory theory for thousands of currently available lab tests, the sources of test interference, and the connections between test results and diagnoses! It is not an overstatement to say that medical laboratory professionals provide critical lifesaving information repeatedly during their work day: e.g., complex testing such as cross-matching your blood for emergency surgery, to identifying a genetic abnormality in a newborn, or assisting in the diagnosis of cystic fibrosis and cancers like Hodgkin’s disease. (Posted on 10 March 2015 by Rodney E. Rohde, PhD, David M. Falleur, MEd, and Joanna R. Ellis, MS)

Secondly, according to Dr. Michael Icardi (National Director, Pathology and Laboratory Medicine Service, Dept. of Veterans Affairs), “They haven’t asked the nurses if they want to do this. They don’t want to be MTs or RTs.” Our nurses are already stretched to a breaking point. How can they be expected to be everything to everyone...and do it safely, accurately and proficiently? The American Society of Clinical Pathology (ASCP) argued, “The nursing degree is not intended to be, nor should it be viewed as the equivalent of a degree in biological sciences or any other natural science degree required of laboratory testing professionals to perform moderate and high complexity diagnostic testing services.”

Despite the critical importance of medical testing, only 12 states currently license medical laboratory personnel. States such as Virginia continue to struggle with maintaining high standards for testing.

“We have attempted to get licensure in Virginia at least three times over the past 20+ years. I was involved in the latest attempt just a few years ago. The committee to which the bill was referred sent it to the Board of Health Professions (BOHP) to determine if it was necessary to license lab personnel. We, for the first time, were able to get the BOHP to agree that it was. That’s when the lobbyists from the hospitals, special interest groups, and even our own State Lab and a local reference lab got involved and blocked the bill from ever getting out of committee,” noted Bill Korzun, PhD, DABCC, MT(ASCP).

“Even though California mandates higher laboratory personnel standards and requires licensing, the California Association for Medical Laboratory Technology (CAMLT) **fights each and every day to maintain these standards to protect our patients.** We defend ourselves against special interest groups such as optometrists, chiropractors, pharmacists, nurses, etc. from expanding their scopes of practice into clinical laboratory science. For more than 50 years, each year special interest groups try to convince CA legislators to dilute and/or repeal state licensure laws. We actively engage our law makers, colleagues and the public to educate them about what it takes to become a Clinical Laboratory Scientist (CLS or aka MLS nationally), MLT or Certified Phlebotomy Technician (CPT) and why others without requisite education and training should not be performing clinical laboratory tests, phlebotomy, or supervising laboratories,” noted Dora W. Goto, MS, CLS, MLS(ASCP)CM, Immediate Past President (CAMLT).

Ms. Goto went on to say, “We need California doctors, nurses, medical laboratory training schools and patients to reach out to their assembly members and senators as well as help support us in the fight against lower medical laboratory testing standards. Anyone can support our mission and lobbyist’s efforts by visiting www.camlt.org.
Medical Laboratory Professionals Week

April 22-28, 2018

Medical Laboratory Professionals Week (MLPW) provides the profession with a unique opportunity to increase public understanding of and appreciation for clinical laboratory personnel. What are you planning to do? Let us know at office@camlt.org

Left: Saddleback College MLT Student volunteers at North Hollywood CE event.

Right: Spring Seminar South at Kaiser, North Hollywood, CA
2018 Membership Drive Begins Now

2017 was a difficult year for most organizations including ours. Most young professionals spend their time on social media and do not attend professional organization meetings. What will the professional organizations of the future look like?

This year we want to spend more time with you, the most important partners of our mission.

We will be calling you, not only to remind you to renew your membership, but to learn more about your passions and what you care about. We will be seeking your attendance and participation at our exciting events: seminars, convention, and social events.

Membership must increase in order to take on our legislative challenges. Fundraising and membership must increase in order to create a lasting legacy for future generations.

We have the power in our community to do this.

We have a reputation second to none. No other clinical laboratory personnel organization in California has a full time lobbyist. Volunteers from CAMLT participate in the LFS Clinical Laboratory Technology Advisory Committee (CLTAC); more than any other organization.

Together we can grow and we will be successful. Sign-up your co-workers using our new website.

It’s Up to You… It’s Now or Never!

Ilene Dickman, President

Left: Break time at Fresno Chapter’s Cheaper by the Dozen CE Seminars.

Right: CE Session, Fresno, CA
MEMBERSHIP Promotions and Incentives

New Member Promotions
1. All New Members: one 3-unit CE seminar discount certificate.
2. Dual Membership Promotional Incentive: 25% refund off first year membership dues offered to members of professional clinical laboratory organizations (other than CAMLT) who have never been a member of CAMLT

Renewing Professional Member Promotion*
1. One 3-unit CE seminar discount certificate.
   * not available with discounted memberships

Incentives
1. Recruiter for each new member: $15 discount certificate toward CE
2. Recruiter for 4 new members within 6 months: one year membership dues paid
3. Conversion from student to professional membership: $25 discount certificate toward CE
4. Professional Discounts (Special Offer for Renewing Members):
   a. 6 Distance Learning CEUs for $60 instead of $72
   b. 9 Distance Learning CEUs for $85 instead of $108
   c. 12 Distance Learning CEUs for $110 instead of $144
5. $10 per CEU seminar discount
6. $10 per CEU distance learning discount
7. Monitor CE programs and get rewarded with a free workshop
8. 20/20 Option: An additional $20 payment at the time of application or renewal entitles the member a 20% discount on CAMLT state sponsored CE fees for the year (not applicable to Distance Learning)
9. Convention registration fee discount: amount varies by committee
10. Access to “Members Only” webpages, officers listing, Affinity benefits and publications

How to Get Your Promotion and Incentive
1. When joining Online, indicate which promotion or incentive you are applying for in the Notes and Comments section. If you are applying for the Dual Membership, please include the name of the Association you are a member of and your membership number. Once this is verified, your discount will be applied, via PayPal, as a refund.
2. When joining via mail, print off pdf and indicate which promotion or incentive you are applying for in the Notes and Comments section. If you are applying for the Dual Membership discount, please include the name of the Association you are a member of and your membership number. If you are paying by Credit Card, your card will be charged the discounted amount, once your membership is verified. If paying by check, your discounted amount will be refunded by return check once your membership is verified. You can send your application via mail, fax or scan and email it in. [http://camlt.org/membership/registration](http://camlt.org/membership/registration)

We’ve got your back!

www.camlt.org
CAMLT’s 2018 Spring Seminar North
Location: UC Davis Medical Center Campus (School of Medicine Building)
4610 X Street, Sacramento, CA 95817

SCHEDULE-AT-A-GLANCE
Complete abstracts/objectives available online: www.camlt.org/calendar

We are pleased to have the following speakers join us:

**SATURDAY, APRIL 21, 2018 – REGISTRATION: 7:45 – 8:30 AM**

| 182-100 (3.0 CE) - Morning: 8:30 – 11:30 AM (Note: you must attend both parts to receive credit) |
| James McVey, BA | Manager of Education Services and Learning Management Administration | ARUP Laboratories | Salt Lake City, UT |
| Part I: Skills to Work with & Motivate the Next Generation. |
| **Part II: Communicating Hard Things.** |
| **Level: Basic | Sponsor: ARUP Laboratories** |
| **Abstract (Part I):** Generation WHY? The Millennial generation, also known as Generation Y, is defined by work-life balance, multitasking, and an integration of technology in all parts of life. Millennial employees are motivated by relationships, and thus benefit from being managed differently than other generations. This presentation will focus on Millennial employees’ strengths, motives and values. And what an employer can do to effectively communicate with and best manage Millennials to do their very best. |
| **Objectives (Part I):** Upon completion of this course, the participant will be able to: 1) identify and describe the Millennial generation, including Millennials’ values, motivations and strengths; 2) explain types of supportive behaviors and communication methods which can be used to engage and motivate next generation employees; 3) describe and list methods for managing Millennial employees in the most effective way possible. |
| **Abstract (Part II):** When the topic of communicating hard things is brought up, it is often thought of negatively. Why? If having a hard conversation will benefit the employee, a department or a relationship, why do we avoid them? What is the risk of not having that difficult conversation? This presentation will focus on useful methods and actions which can be used in a work setting or in personal circumstances to effectively prepare for, and successfully accomplish difficult conversations. |
| **Objectives (Part II):** Upon completion of this course, the participant will be able to: 1) explain methods to prepare for hard conversations; 2) explain why difficult conversations are avoided and describe actions to take so that avoidance is overruled; 3) describe the approach and proper use of language so that difficult conversations are executed effectively. |

| 182-200 (3.0 CE) - Afternoon: 1:00 – 4:00 PM (Note: you must attend both parts to receive credit) |
| Jeri Seiki, BS, CLS | Global Product Manager, Clinical Immunology Division | Bio-Rad | Benicia, CA |
| Wen Kumfert, PhD | Sr. Product Manager | Bio-Rad | Benicia, CA |
| **Part I: Past, Present and Future Diagnostic Strategies on Vasculitis Testing.** |
| **Part II: Evaluation of Diagnostic Algorithm for Celiac disease and Gluten sensitivity.** |
| **Level: Intermediate | Sponsor: Bio-Rad** |
| **Abstract (Part I):** Anti-neutrophilic cytoplasmic antibodies (ANCAs) are serological markers that aid in the diagnosis of ANCA-associated vasculitis (AAV) including granulomatosis with polyangiitis (GPA - previously known as Wegener’s granulomatosis), eosinophilic granulomatosis with polyangiitis (EGPA - previously referred to as Churg Strauss syndrome), and microscopic polyangiitis (MPA). Criteria for classification of these diseases have been described by the American College of Rheumatology (ACR) and the Chapel Hill Consensus Conference. New diagnostic tools have led to reexamination of current classification criteria. The 1999 international consensus on ANCA testing recommended indirect immunofluorescence (IIF) testing to screen for ANCAs and serum samples containing ANCA should be tested for PR3-ANCAs and MPO-ANCAs with an antigen-specific immunoassay. Recently, data from a large multicentre European Vasculitis Study demonstrated that current antigen specific immunoassays for PR3-ANCA and MPO-ANCA can be used to assist in the diagnosis of AAV without the use of IIF. These finding have led to the revised 2017 international consensus testing of ANCA in granulomatosis with polyangiitis and microscopic polyangiitis. Additionally, clinical interpretation of ANCA testing can be improved by considering antibody levels and by calculating test result interval-specific likelihood ratios (LR). |
| **Objectives (Part I):** Upon completion of this course, the participant will be able to: 1) describe Vasculitis disease background; 2) define diagnostic test methods for ANCA associated Vasculitis (AAV); 3) identify Revised ANCA Testing and Diagnostic Strategies. |
| **Abstract (Part II):** Celiac disease was first described in the 1st century. The culprit for celiac disease was identified in the 1950s. Scientific advances have propelled diagnostic methods for celiac disease to evolve over the years - from biopsy to serological tests, from simple algorithm to complicated ones. This seminar will describe current methods used for celiac diagnosis, explain how the diagnostic algorithms have evolved through the last decade, and discuss the future of celiac diagnosis. |
| **Objectives (Part II):** Upon completion of this course, the participant will be able to: 1) describe current methods used by clinical laboratories for celiac disease diagnosis; 2) develop a laboratory celiac disease diagnostic algorithm. |
### 182-300 (3.0 CE) - Morning: 8:30 – 11:30 AM (Note: you must attend both parts to receive credit)

Paul Riley, PhD, MBA | Scientific Business Development Manager | Diagnostica Stago Inc. | Parsippany, NJ

**Part I: Direct Oral Anticoagulants: Screening and Measurement Assays.**

**Part II: Pre-Analytical Variables in the Coagulation Lab: Why does it matter?**

**Level:** Intermediate | **Sponsor:** Diagnostica Stago Inc.

**Abstract (Part I):** Direct Oral Anticoagulants (DOACs) are becoming more prevalent in our patient population. This presentation will discuss how they work, how they can be measured, and how they affect the Coagulation laboratory.

**Objectives (Part I):** Upon completion of this course, the participant will be able to: 1) discuss current methods for monitoring traditional anticoagulants; 2) describe the impact of DOACs on routine and specialty coagulation assays; 3) discuss a case study highlighting the role of the laboratory in this testing regime.

**Abstract (Part II):** Coagulation testing is particularly vulnerable to pre-analytical variables. This session will help identify best practices to minimize the variables that affect patient results.

**Objectives (Part II) Upon completion of this course, the participant will be able to:** 1) explain how blood collection may impact results; 2) describe best practices for sample transport and storage; 3) outline sample processing procedures; 4) identify patient variables that may affect coagulation testing.

### 182-400 (3.0 CE) - Afternoon: 1:00 – 4:00 PM

Gerhard Bauer, BS | Adjunct Professor of Hematology / Oncology, Director of the UC Davis GMP Facility | UC Davis School of Medicine | Davis, CA

**Cellular Therapy Applications and Good Manufacturing Practice at the UC Davis Institute for Regenerative Cures.**

**Level:** Intermediate

**Abstract:** Since 2007, stem cell research in the State of California has been funded by the California Institute for Regenerative Medicine (CIRM). This led to the establishment of the Institute for Regenerative Cures (IRC) at the UC Davis School of Medicine. At UC Davis, 150 faculty members are currently developing stem cell treatments, other cellular therapies and gene therapies within 15 disease teams, focusing their work on severe and currently incurable diseases, such as critical limb ischemia leading to limb amputation, neurological diseases such as Huntington’s disease, blindness caused by macular degeneration and retinal ischemia, and many more. Clinical trials using multipotent adult type stem cells are already underway and can demonstrate efficacy and clinical benefits. As an example, a UC Davis investigator sponsored clinical trial for blindness showed recovery of vision after application of autologous, purified bone marrow stem cells. Pluripotent stem cell derived products are currently being moved from the laboratory into early clinical applications, and therapeutic efficacy in in vivo models of Huntington’s disease could be demonstrated. Organ replacement strategies, for instance, artificial livers, are also being developed. After completion of the later stage clinical trials and approval by the regulatory agencies, stem cell therapies will lead to standard of care stem cell applications and clinical benefits for large numbers of patients in the not so far future.

**Objectives:** Upon completion of this course, the participant will be able to: 1) distinguish between human adult, embryonic and induced pluripotent stem cells, their current applications in research and how such cells can be used clinically to treat specific, often severe and currently incurable diseases; 2) discuss other cellular therapies, particularly when combined with gene therapies, such as chimeric antigen receptor (CAR) T cell therapies for the treatment of hematologic malignancies; 3) describe how cellular products are being manufactured under Good Manufacturing Practice (GMP) conditions, how they are being quality controlled and what laboratory tests are performed for these quality control measures; 4) outline the future possibilities of tissue regeneration, particularly neuronal tissue regeneration and whole organ replacements.
CAMLT 2018 SPRING SEMINAR NORTH GENERAL INFORMATION
Proudly sponsored by the
UCDMC Department of Pathology and Laboratory Medicine’s Partners in Education (PIE).
Visit www.camlt.org/calendar for complete abstracts/objectives

Advance Registration: Now closed; you may register on-site.

ON-SITE REGISTRATION AVAILABLE: To register on-site, arrive at least 40 minutes prior to the beginning of your first class. On-site registrations will be accepted subject to space availability.

Registration Fees: Note … No price increase from 2017 rates!
A standard $10.00 registration fee applies to all individuals.

Additional fees:
CAMLT Members: $45.00 per workshop
Non-Members: $75.00 per workshop
Students: Free workshops!

Payment Options: Registration and workshop fees may be paid by check, VISA, MasterCard or money order. Sorry, American Express and Discover cards are not accepted.

Non-Members: Become a CAMLT member and save! If you join CAMLT at the time of registration, you may apply the difference between member and non-member fees (e.g. Difference is $10 per CEU x 12 CEUs = $120 = FREE Professional Membership) toward your annual membership dues.

For further details, contact CAMLT at 510-792-4441. You may also go to http://camlt.org/membership/registration to join online or download a membership application.

Refund Policy: CAMLT reserves the right to cancel any program with insufficient registration. Registrants will be immediately notified and will receive a complete refund for programs cancelled by CAMLT. There may be changes in program content or faculty due to circumstances beyond our control. CAMLT is not responsible for penalties incurred as a result of cancellation, including non-refundable transportation fees and hotel room deposits.

Location Information:

Spring Seminar North:
UC Davis Medical Center Campus
School of Medicine Building
Rooms 1204 & 1222
4610 X St., Sacramento, CA  95817

Daily Parking: Park in Lot # 14, one-day permits can be purchased at dispensing machine via cash or credit card. Lot 14 entrance at 45th Street and 2nd Avenue.

Hotel Room Block: Not negotiated for this seminar

Lunch: A cafeteria is located on the first floor of the Surgical and Emergency Services Pavilion across the street from the Med Center. There are fast food restaurants a few blocks away but for convenience and safety, the cafeteria is suggested ... or, bring your own lunch.

Note: No part of the seminar handouts or presentation (no tape, video or digital recorders please) may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recorded, or otherwise, without prior written permission from CAMLT.

Disclaimer: CAMLT hereby disclaims any liability for, or endorsement of, any products, services or information presented.

Accreditation: CAMLT is approved by the California Department of Public Health as a CA CLS/MLT/CPT Accrediting Agency (#21) for Continuing Education, and by the State of Nevada.

Attendance Policy: Choice of workshops may NOT be changed on-site. In compliance with state accreditation requirements, participants must attend the entire workshop to receive credit. Partial credit cannot be awarded for late arrivals or early departures. Excessive absences from a workshop for any circumstance may result in forfeiture of CE credit. NO EXCEPTIONS.
Erythrocyte Volume Disorders
Course # DL-015
2.0 CE
Level of difficulty: Intermediate

Payman Nasr, PhD ASCP
Assistant Professor Clinical Laboratory Sciences
California State University Dominguez Hills

Abstract

Erythrocytes or red blood cells (RBC) transport oxygen and carbon dioxide between the lungs and the tissue. Reduction in red blood cells or the hemoglobin content results in anemia, while increased production of red blood cells causes polycythemia. Anemia may be the results of low production of cells in the bone marrow or increased destruction of RBC in the bone marrow and/or circulation; polycythemia is a general term describing an increase in the rate of RBC production in the bone marrow and having too many red blood cells in the circulation, while the plasma volume remains constant.

This course provides a review on causes for reduction in RBC production in the bone marrow and increased RBC mass in the circulation. This course will not evaluate the causative agents of anemia which may influence RBC hemoglobin concentration. The content includes two case studies to discuss the acquired and congenital causes for low and high RBC production in the bone marrow.

The following is the list of conditions that will be covered in this course

Low RBC Production
1. Hemorrhagic Anemia
2. Pernicious Anemia
3. Aplastic Anemia

High RBC Production
1. Polycythemia Vera
2. Compensatory Polycythemia
3. Relative Polycythemia
4. Erythrocytosis

Objectives:

1. Summarize the types of erythrocyte disorders due to decreased or increased RBC production.
2. Identify the symptoms associated with decreased or increased RBC production.
3. Describe the mechanisms that may lead to aplastic anemia.
4. Describe the mechanisms of pernicious anemia.
5. Describe the use of methylmalonic acid and homocysteine testing in the differential diagnosis of B12 and folate deficiency.
6. Describe the genetic and acquired causes for polycythemia.
7. Describe the general structure and function of telomeres and telomerase complex.

Case Study #1

A 38-year-old man was referred to a physician for cough and dyspnea. The patient was a police officer with a history of smoking. The initial examinations indicated the pulse was regular at 70/min, blood pressure 145/95 mm Hg, and temperature 36.9°C. Chest examination revealed poor bilateral expansion. Cardiovascular and neurological examinations were normal. The skin examination revealed a spotty skin hypopigmentation and flat white spots on back and upper torso which the patient indicated he has had since the age of 10. In addition, there was marked leukoplakia of the tongue, the fingernails demonstrated a mild clubbing, and the small toenails were dystrophic. The patient indicated his brother, his maternal grandfather and his maternal great uncle have similar skin and nail appearances. No female in their family has reported similar skin or nail issues.

Blood morphological analysis indicated pancytopenia and macrocytic RBCs with 1+ dacrocytes and occasional Howell-Jolly Body. The peripheral blood count was as follows.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC</td>
<td>1.2 x 10^9/L</td>
<td>(4.5-10.5)</td>
</tr>
<tr>
<td>RBC</td>
<td>2.42 x 10^12/L</td>
<td>(4.30-5.72)</td>
</tr>
<tr>
<td>HGB</td>
<td>7.1 g/dL</td>
<td>(13.5-16.5)</td>
</tr>
<tr>
<td>HCT</td>
<td>24%</td>
<td>(39-48)</td>
</tr>
<tr>
<td>PLT</td>
<td>8.0 x 10^9/L</td>
<td>(150-450)</td>
</tr>
<tr>
<td>MCV</td>
<td>99 fl</td>
<td>(80-96)</td>
</tr>
<tr>
<td>MCH</td>
<td>29 pg</td>
<td>(28-33)</td>
</tr>
<tr>
<td>MCHC</td>
<td>29 g/dl</td>
<td>(32-36)</td>
</tr>
<tr>
<td>Retics</td>
<td>0.7%</td>
<td>(1.5-3)</td>
</tr>
</tbody>
</table>

The lymphocyte count was 1.1*10^3/μl (normal 1.2–3.4*10^3/μl) with CD4 20% (normal 28.5–60.5%) and CD8 64% (normal 11.1–38.4%); the CD4/CD8 ratio was 0.31 (normal >1). Routine biochemical and liver function tests were normal. The cholesterol level was 245 mg/dL (normal <200 mg/dL) and HDL and triglyceride levels were normal. Serum protein electrophoresis was normal without monoclonal bands. Serological tests for hepatitis B and C, HIV, rheumatoid factor, anti-nuclear and anti-DNA antibodies were negative. IgM was low (0.38 g/L; normal 0.60–2.50 g/L); IgA and IgG were normal. Urinalysis was normal. Genetic analysis indicated a mutation in \( DKC1 \) gene with 5C to T missense mutation in exon 1 of the \( DKC1 \) gene. This mutation involves the fifth nucleotide of exon 1, which corresponds to nucleotide 97 from the start of the \( DKC1 \) gene.

Case Study #2

A female visited her physician with complaint of malaise, lethargy, and lack of appetite. The patient indicated she has not been feeling well lately and has a constant feeling of bloating in her stomach after even a small meal. She also indicated that she has been developing a rash every time she takes a hot shower particularly on her face. In addition, she has been having a lower back pain
for a while. Initial examination indicated a palpable spleen and abdominal/hepatic tenderness. The imaging studies indicated enlarged liver and spleen. The initial complete blood count (CBC) results are as follows.

- WBC: $11 \times 10^9/L$ (4.5-10.5)
- RBC: $5.84 \times 10^{12}/L$ (4.30-5.72)
- HGB: 16.9 g/dL (13.5-16.5)
- HCT: 51% (39-48)
- PLT: $450 \times 10^9/L$ (150-450)
- MCV: 80 fl (80-96)

Morphology: Normochromic Normocytic; Dacryocytes 1+

The physician ordered further studies:

- Cytogenetics studies indicated a normal karyotype.
- Genomic DNA analysis from patient’s leukocytes identified JAK2 V617F mutation.
- Genetic analysis indicated a somatic mutation pattern.

**Discussion**

**RBC Production**

The number of erythrocytes in a person's blood can vary significantly from the normal range, leading to various clinical conditions, generally classified as anemia when the production is low and polycythemia when the production is high.

**Anemia**

Anemia due to inadequate production of RBC is characterized by the low number of RBC per cubic millimeter in blood with symptoms of fatigue, lethargy, dyspnea, pallor of the skin and cardiac irregularities. The major causes of anemia due to inadequate RBC production include:

1. **Hemorrhagic anemia** results from acute or chronic blood loss due to such factors as gastrointestinal bleeding or heavy menstrual flow. The symptoms may include pallor, sensitivity to cold, fatigue, malaise, dizziness, tachycardia and dyspnea. Since anemia is often a symptom of another illness, it is important to identify the exact causes of hemorrhagic anemia to determine the best regimen of therapy. For instance, chronic blood loss due to gastroduodenal ulcer, gradually diminishes the body of its iron stores, and if the intake of iron is insufficient, iron deficiency anemia would result. With supportive treatment, a healthy bone marrow can restore the normal erythrocyte count to a normal range quickly. The three most common causes of chronic blood loss are excessive menstrual bleeding, gastroduodenal ulcer, and colon cancer. Depending the stage of hemorrhagic anemia, the RBC morphology may vary from normochromic normocytic (early stage) to hypochromic microcytic (chronic stage). The CBC results will demonstrate lower RBC count, hemoglobin concentration and hematocrit percentage. In the presence of a healthy bone marrow, in chronic cases, there will be an increase in reticulocytes percentage.

2. **Pernicious anemia (PA)** is a decrease in RBC production due to inadequate absorption of cobalamin (vitamin B12) in the ileum of small intestine. B12 is a cobalt containing coenzyme with essential roles in two biochemical reactions in the human body. In one reaction, B12 acts as a cofactor for methylmalonyl CoA mutase in converting methylmalonyl CoA to succinyl CoA in the mitochondria supplying the Krebs cycle. Inadequate B12 results in increased methylmalonyl CoA in the serum and can be used for differential diagnosis of B12 and folate deficiency (Figure 1A) (2). In the second biochemical reaction, B12 acts as a coenzyme for methionine synthase by
transferring a methyl group from 5-methyltetrahydrofolate (5-methyl-THF) to homocysteine, generating methionine in the process (Figure 1B). If either folate or B12 are deficient, homocysteine levels increase because methionine synthase is unable to convert it to methionine. Following demethylation by methionine synthase, tetrahydrofolate continues in a series of biochemical reactions that are required for the DNA synthesis. Thus, B12 or folate deficiency inhibit thymidine production, in turn reducing DNA transcription and cell proliferation(2)(4).

Figure 1. (A) Algorithm for the interpretation of serum vitamin B12, methylmalonic acid (MMA) and homocysteine (HCY) in the diagnosis of vitamin B12 and folate deficiency. (B) Vitamin B12 biochemical reaction with methionine synthase results in demethylation of 5-methyl-Tetrahydrofolate (5-methyl-THF), in turn converting the homocysteine to methionine (MET) and releasing THF to enter other biochemical reactions for deoxythymidine triphosphate synthesis.
One common cause of PA is due to the defective production of intrinsic factor (IF), which is produced by the lining of stomach to protect and facilitate B12 absorption in the ileum. IF is a glycoprotein produced by parietal cells in the gastric mucosa that binds directly to B12 after B12 is released from associated proteins by the action of gastric acid and pepsin in the stomach. The B12-IF complex travels to the ileum, and IF component of the molecule targets the receptor on ileal mucosa and facilitate B12 endocytosis. Unless the causes of PA are dietary or due to surgical complication, most PA patients have autoantibodies against gastric parietal cells or IF. In such instance, the serum B12 concentration is very low (<150 ng/L). Destructive autoimmune process diminishes IF function; as a result, the accumulation of 5-methyl-THF in the absence of B12 results in increased levels of methylated THF. This event is also known as folate trap (Figure 1B). The inhibition of homocysteine methylation to generate methionine also results in increased homocysteine in the blood. Increased homocysteine is associated with increased risk of cardiovascular disease.

The PA symptoms are similar to hemorrhagic anemia; however, it may also involve the nervous system characterized by symptoms such as tingling and numbness at extremities, loss of balance, muscle weakness, and mental confusion. For clinical diagnosis, if vitamin B12 is <150 ng/L, then intrinsic factor blocking antibody (IFBA) is performed. If IFBA is negative or indeterminate, then gastrin is performed. If vitamin B12 is 150 to 400 ng/L, then methylmalonic acid (MMA) is performed. If MMA is >0.40 nmol/mL, then IFBA is performed. Due to interruption of DNA synthesis, RBC precursors divide fewer times, giving the RBC macrocytic morphology. Depending on the extent of extramedullary hematopoietic activity, Howell-Jolly bodies, ovalocytes, dacryocytes and hypersegmented neutrophils are common morphological characteristics of B12 and folate deficiency.

3. **Aplastic Anemia (AP)** is characterized by a decrease in hematopoietic stem cells, pancytopenia, reticulocytopenia and bone marrow hypocellularity. AP can be acquired or congenital. In most acquired cases, the hematopoietic stem cells are the target of an immune process triggered by viral infection or toxins, which is mediated by cytotoxic T-cells, and cause hematopoietic stem cell death by apoptosis. Acquired aplastic anemia can be treated by immunosuppressive therapy and bone marrow transplantation.
A rare and congenital form of aplastic anemia is dyskeratosis congenita (DKC). DKC may be inherited in an X-linked or autosomal dominant pattern (OMIM number 305000; OMIM number 127550). The X-linked pattern is caused by mutations in the *DKC1* gene, which encodes dyskerin, a nucleolar protein that associates with the telomerase complex. The common symptoms of DKC include dystrophic fingernails and toenails; skin pigmentation, especially on the neck and chest; and oral leukoplakia. The blood analysis is characterized by pancytopenia and may include macrocytic RBC’s, similar to the results of the patient in Case Study #1. Howell-Jolly bodies and dacryocytes may also be seen on blood smear. Involvement of *DKC1* has implicated the telomere-repair complex in the pathophysiology of DKC, and interestingly, cells from patients with DKC have short telomeres and low telomerase activity. Subsequently, mutations in the *TERC* gene on chromosome 3, which encodes the RNA component of the telomerase complex (TERT), were identified in the autosomal dominant form of DKC (5). Telomeres are structural elements that cap the ends of chromosomes, protecting them from fusion and erosion. In human somatic cells, telomeres typically consist of thousands of tandem repeats of nucleotides TTAGGG. These repeats are gradually lost with cellular replication and aging, and when the erosion of telomeric repeats reaches a critical limit, the cell undergoes arrested proliferation and senescence, shortened life span, apoptosis, and genomic instability. Maintenance
of the integrity of telomeres requires the telomerase ribonucleoprotein complex, which consists of TERT and TERC, in addition to a multiplex of proteins (3). TERT copies a short region of TERC into telomeric DNA to extend the 3' end of the chromosome and maintain chromosomal structural integrity (Figure 2).

The identification of genetic mutations in AP is by Polymerase-chain-reaction (PCR) amplification of genes encoding the telomerase complex — namely *DKC1* and *TERT* — with DNA samples extracted from peripheral blood or bone marrow cells. PCR products are sequenced using automated genetic sequence analyzer to identify the mutations.

**High RBC Production**

**Polycythemia** is an increase in the circulating red blood cell mass. There are two distinct forms: Primary and Secondary polycythemia.

1. **Primary polycythemia**, also known as **Polycythemia Vera (PV)**, is a myeloproliferative disorder characterized by hyperplasia of erythropoietic tissues in the bone marrow, resulting an increase in RBC count irrespective of the presence or absence of erythropoietin. PV is usually a diagnosis of elimination, ruling out other causes of secondary polycythemia. The affected person has the same total blood volume, but the number of erythrocytes is increased, rendering the patient’s blood more viscous and increasing susceptibility to cardiovascular disease. In humans, this has been shown to be largely due to a mutation in Janus-activated kinase 2 (JAK2). JAK2 is a tyrosine kinase that is downstream to the erythropoietin receptor. It directs the signaling cascade, triggered by erythropoietin binding to its receptor on RBC precursors in the bone marrow, that, in turn, controls RBC production (Figure 3) (1). Erythropoietin results in dimerization of tyrosine kinase monomers, which in turn activate members of the signal transducer and activator of transcription (STAT) protein family by JAK2 phosphorylation. STAT act as intracellular transcription factors and mediate hematopoietic differentiation and proliferation activities by influencing anti-apoptotic and proliferative gene expression, ultimately resulting in cell accumulation (2). Normally, JAK2 is only activated by phosphorylation of tyrosine residues when erythropoietin binds to its receptor. However, more than 90% of PV cases are specific for valine to phenylalanine (V617F) substitution at amino acid 617 due to a mutation of guanine to thymine at exon 14 of the gene (2). V617F mutation drives erythropoiesis, either independently of erythropoietin or because RBC precursors become hypersensitive to normal concentrations of erythropoietin.
Clinical presentation of PV may include splenomegaly, bone marrow hyperplasia and pancytopenia. There may also be an increase in WBC and platelet counts. Excess WBC’s may manifest as a rash when exposed to warm temperature due to subcutaneous histamine release as with the patient in Case Study #2. Excess platelets render the patient susceptible to deep vein thrombosis, stroke and cardiovascular disease. The treatment for PV is primarily therapeutic phlebotomy with the goal of maintaining hematocrit below 45%. The chances of thrombotic events increase with patients who are only treated by phlebotomy; thus, low dose aspirin regimen and myelosuppressive therapy may be required to control such complications and reduce the chances of thrombosis.

Figure 3. Erythropoietin (E) signaling pathway resulting in STAT activation is shown. After Erythropoietin targets its receptor, causing dimerization of monomers, JAK2 phosphorylation (P) occurs. JAK2 phosphorylation is followed by STAT activation and forming a dimer in the presence of homocysteine (H). STAT homodimer acts as a transcription factor, crosses the nuclear membrane and regulates hematopoietic cell survival, proliferation, and differentiation.
2. Secondary polycythemia is a physiological condition resulting from a decreased oxygen supply to the tissues caused by living at high altitudes, heart disease, circulatory insufficiency, severe pulmonary disease, or the production of erythropoietin or erythropoietin-like compounds, as in polycystic kidney disease or renal neoplasms. The following are some of the major causes of secondary polycythemia.

**Compensatory polycythemia (CP)** results from chronic hypoxia often seen in smokers, congenital heart disease, pulmonary emphysema, or prolonged residence at a high altitude. The low blood oxygen content results in increased erythropoietin production, in turn increasing RBC production to increase blood oxygen carrying capacities. Individuals with CP are asymptomatic but will have increased in RBC count, hemoglobin concentration and hematocrit percentage.

**Relative polycythemia (RP)** is an increase in the number of erythrocytes in the blood per unit volume because of a decrease in blood plasma volume. RP is often a result of severe dehydration as seen in a serious case of diarrhea. Progressive loss of water alters the ratio of blood cells to plasma and makes blood more concentrated. This type of polycythemia is a temporary condition, and the ratio of erythrocytes to water in the blood returns to normal when the patient becomes rehydrated. During the course of RP, there may be an increase in hematocrit, but RBC indices remain normal.

**Erythrocytosis** is an increase in erythrocytes due to an increase in the level of erythropoietin. This is due to conditions often seen in hepatocellular carcinoma, renal cancers, renal diseases and renal cysts, which may stimulate erythropoietin production without the presence of hypoxia. Epinephrine-producing neoplasms, such as pheochromocytoma, can also result in erythrocytosis. In erythrocytosis, the patients have symptoms similar to PV, and the course of treatment will depend on the underlying source causing the increase in erythropoietin.

**References**

Review Questions
Select one best answer

1. Identify the mode of transmission for mutant \( DKC1 \) gene in Case Study #1.
   a. Autosomal dominant
   b. Autosomal recessive
   c. X-linked
   d. Y-linked

2. What is the diagnosis for the patient in Case Study #1?
   a. Idiopathic aplastic anemia
   b. Chronic hemorrhage
   c. Dyskeratosis congenital
   d. Pernicious anemia

3. The patient in Case Study #1 has a teenage daughter. What is the probability she would develop the disease later in her life?
   a. 0%
   b. 25%
   c. 50%
   d. 100%

4. The symptoms of dyskeratosis congenital include all the following except:
   a. Leukoplakia
   b. Nail dystrophy
   c. Skin spots
   d. B12 deficiency

5. Peripheral blood morphology seen in dyskeratosis congenital includes all the following except:
   a. Howell Jolly bodies
   b. Heinz bodies
   c. Dacryocytes
   d. Macrocytosis

6. Telomerase reverse transcriptase function is:
   a. To act as an RNA template for gene transcription
   b. To increase the length of DKC1 gene
   c. To repair the DNA at the end of chromosomes
   d. To repair single base mutations

7. Increase in serum methylmalonyl CoA in the presence of normal homocysteine indicates:
   a. Folate deficiency
   b. B12 deficiency
   c. B12 and folate deficiency
   d. Excludes B12 and folate deficiency
8. Folate trap due to B12 deficiency occurs during:
   a. 5-methyl THF crossing plasma membrane into cytoplasm
   b. Thymidine synthesis
   c. Homocysteine conversion into methionine and making THF
   d. Crossing the duodenal mucosa and generating 5-methyl THF

9. Telomeres:
   a. gradually increase in length during aging
   b. are tandem repeats of nucleotides TTAGGG at the end of chromosomes
   c. are tandem repeats of nucleotides TTAGGG in the middle of chromosomes
   d. contain many genes

10. Which of the following may result in low B12 absorption in the ileum?
    a. Low acidity in the stomach
    b. Low levels of intrinsic factor in stomach
    c. Autoimmune disease
    d. All of the above

11. What is the likely diagnosis in Case Study #2?
    a. Aplastic Anemia
    b. Pernicious Anemia
    c. Polycythemia Vera
    d. Chronic Myelogenous Leukemia

12. In Case Study #2, the physician orders a series of blood and bone marrow biopsy. The bone
    marrow biopsy results indicate:
    a. hypocellular marrow
    b. hypercellular marrow
    c. acellular marrow
    d. normal marrow

13. The patient’s coworker tells the patient that the prognosis is good and there is an effective
    treatment for her condition. The treatment is:
    a. Chemotherapy
    b. Phlebotomy
    c. Radiation
    d. Imatinib

14. The likely cause of patient’s condition in Case Study #2 is:
    a. Janus-activated kinase 2 mutation
    b. Translocation (9;22)
    c. Translocation (8;14)
    d. Translocation (12;21)
15. Which of the following is NOT a cause for secondary polycythemia?
   a. living at high altitudes,
   b. cardiovascular insufficiency
   c. pulmonary disease
   d. JAK2 mutation

16. Tobacco smokers are at higher chance of developing _________________.
   a. Compensatory Polycythemia
   b. Relative Polycythemia
   c. Erythrocytosis due to increased erythropoietin
   d. Polycythemia Vera

17. The cause of skin rash for patient in case study #2 is due to:
   a. Exposure to heat
   b. Histamine release
   c. Allergic reaction
   d. Sunburn

18. Increase in erythrocytes due to an increase in the level of erythropoietin is often seen in:
   a. Primary polycythemia
   b. Polycythemia Vera
   c. Compensatory polycythemia
   d. Erythrocytosis

19. Decrease in plasma volume may lead to _________________.
   a. Primary polycythemia
   b. Polycythemia Vera
   c. Compensatory polycythemia
   d. Relative polycythemia

20. The function of STAT is best described as:
   a. tyrosine kinase receptor
   b. JAK2 Inhibitor
   c. Transcription factor
   d. DNA repair complex
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<table>
<thead>
<tr>
<th>Course #</th>
<th>Title</th>
<th>Level of Difficulty</th>
</tr>
</thead>
<tbody>
<tr>
<td>DL-001</td>
<td>Hantavirus – A Special Pathogen</td>
<td>I</td>
</tr>
<tr>
<td>DL-002</td>
<td>Diagnosis of Malaria in the U.S.</td>
<td>I</td>
</tr>
<tr>
<td>DL-003</td>
<td>Update on Salmonella Foodborne Gastroenteritis</td>
<td>I</td>
</tr>
<tr>
<td>DL-004</td>
<td>Viral Hepatitis: Causes, Diagnosis, and Treatment</td>
<td>I</td>
</tr>
<tr>
<td>DL-005</td>
<td>Q Fever, Diagnosis and Management</td>
<td>I</td>
</tr>
<tr>
<td>DL-006</td>
<td>Rare Antibody: Hemolytic Disease of the Fetus &amp; Newborn</td>
<td>I</td>
</tr>
<tr>
<td>DL-007</td>
<td>Giardiasis</td>
<td>B</td>
</tr>
<tr>
<td>DL-008</td>
<td>Cryptosporidiosis</td>
<td>B</td>
</tr>
<tr>
<td>DL-009</td>
<td>Listeriosis – A Foodborne Disease</td>
<td>I</td>
</tr>
<tr>
<td>DL-010</td>
<td>MMWR Report – Chlamydia &amp; Neisseria</td>
<td>A</td>
</tr>
<tr>
<td>DL-011</td>
<td>Measles – United States 2014</td>
<td>B</td>
</tr>
<tr>
<td>DL-012</td>
<td>Zika Virus</td>
<td>B</td>
</tr>
<tr>
<td>DL-013</td>
<td>Ebola Virus Disease</td>
<td>B</td>
</tr>
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