As the premier scholarly publication of the osteopathic medical profession, JAOA—The Journal of the American Osteopathic Association encourages osteopathic physicians, faculty members and students at colleges of osteopathic medicine, and others within the healthcare professions to submit comments related to articles published in the JAOA and the mission of the osteopathic medical profession. The JAOA’s editors are particularly interested in letters that discuss recently published original research.

Letters to the editor are considered for publication in the JAOA with the understanding that they have not been published elsewhere and that they are not simultaneously under consideration by any other publication.

All accepted letters to the editor are subject to editing and abridgment. Letter writers may be asked to provide JAOA staff with photocopies of referenced material so that the references themselves and statements cited may be verified.

Readers are encouraged to prepare letters electronically in Microsoft Word (.doc) or in plain (.txt) or rich text (.rtf) format. The JAOA prefers that readers e-mail letters to jaoa@osteopathic.org. Mailed letters should be addressed to Gilbert E. D’Alonzo, Jr, DO, Editor in Chief, American Osteopathic Association, 142 E Ontario St, Chicago, IL 60611-2864.

Letter writers must include their full professional titles and affiliations, complete preferred mailing address, day and evening telephone numbers, fax numbers, and e-mail address. In addition, writers are responsible for disclosing financial associations and other conflicts of interest.

Although the JAOA cannot acknowledge the receipt of letters, a JAOA staff member will notify writers whose letters have been accepted for publication. Mailed submissions and supporting materials will not be returned unless letter writers provide self-addressed, stamped envelopes with their submissions.

All osteopathic physicians who have letters published in the JAOA receive continuing medical education (CME) credit for their contributions. Writers of original letters receive 5 AOA Category 1-B CME credits. Authors of published articles who respond to letters about their research receive 3 Category 1-B CME credits for their responses.

Although the JAOA welcomes letters to the editor, readers should be aware that these contributions have a lower publication priority than other submissions. As a consequence, letters are published only when space allows.

**Efficacy of a Physician’s Words of Empathy: An Overview of State Apology Laws**

*To the Editor:*

In nearly 20 years of practice, I have never bought into the early guidance I received from some of my teachers that a physician should never apologize. I have consistently offered an “I’m sorry” to my patients and their families whenever the circumstances warranted such words. Such an expression, when it is truthful, is valuable simply for that reason—because it is the truth. Some patients may not want to hear it, but they do respect and accept it. My feeling has always been that the unique relationship of physician and patient already has enough asymmetry built into it without physicians refusing to humble themselves should circumstances warrant.

An example of such a circumstance would be a medication error. A filed incident report, along with an apology, serves to assuage much of the patient’s concerns, letting the patient know that the mistake was recognized and steps have been taken to mitigate the chances of similar mistakes in the future. Another example would be a delay in care caused by an unclear or confusing clinical picture. An apology and explanation for the delay can go a long way toward reducing the anger felt by the patient—an anger often stemming from the complexity of the medical system that the patient does not understand and the clinical detachment that the patient may sense in an emotionally and mentally overloaded caregiver. The main point to keep in mind is that the mistake cannot be undone, but the mistake itself is not nearly as troublesome to the patient as is the sense that the caregiver is cavalier or indifferent to the event.

My patients have always responded maturely to the truth by acknowledging that they respect my honesty. I have had a few unsettled nights wondering if my words would be used to determine my guilt in a tort proceeding, but those fears have been, so far, unwarranted. Patients know that physicians are not superhumans. They know that we make mistakes. What they want is not perfection, but the ability to trust us as acting in their best interests and to the best of our abilities when they are in serious need. The basis for that trust is the unembellished truth.

Aside from the glaringly obvious notion that people do not get as angry with truthful physicians as they do with liars, the other issue that the Saitta and Hodge article reminded me...
of is the peculiar habit of some physicians to attempt to “indemnify” themselves by rendering comments about care that could have been provided had the patient arrived sooner. One case that comes to mind involves a patient I knew who was seriously injured. Unfortunately, the severity of the injury was not recognized early enough to correct the problem, resulting in the loss of career for the patient. This patient told me that when she arrived at the surgeon, the first thing that the surgeon said was, “If you had only arrived here ‘X’ hours ago, I could have prevented this.”

Of course, the patient asked me what I thought of this matter. Having had no input into this individual’s care, I did the best thing that I could and replied, “I wasn’t there, so I cannot comment. I trust that physicians do their best with what they see when they see it, and it is not for me to speculate, but to care for you now.”

Sadly, the resulting lawsuit ruined the career of an otherwise excellent physician who ended up being collateral damage in the pool of named defendants.

Physicians who seek to mitigate their own liability in what might be a bad situation for the doctors who were involved prior to referral might do well to remember that what goes around comes around. Speculation is not fact, and it does nothing to fix a disaster. It only ensures that greater folly will occur.

I have always taught my students and residents that “I don’t know” is an acceptable answer when it is the truth. To the extent that patients can accept a humble “I am sorry” when circumstances warrant it, I am quite certain that they will also accept “I don’t know,” especially when physicians are clearly not in a position to render observed judgment for events that occurred before their involvement in care.

Todd R. Fredricks, DO
Amesville, Ohio

Ecthyma Gangrenosum Caused by Pseudomonas aeruginosa

To the Editor:
We read with interest the report by Rock and Thom1 published in the April issue of JAOA—The Journal of the American Osteopathic Association. There is an important aspect of this report worth noting: the pseudomonal lesion depicted in the image closely resembles an eschar. This resemblance underscores the importance of obtaining as complete and as concise as possible of a patient history that includes social, recreational, occupational, and travel-related activities. For example, cutaneous eschars are associated with certain zoonotic infections such as anthrax,2 tularemia,3 and African tick-bite fever.4 Anthrax and tularemia are considered agents of bioterrorism,2 but they can also be acquired as part of recreational activities (eg, wild game hunting) or occupational activities (eg, those of farmers, veterinarians, abattoir workers, or taxidermists) during natural exposure to infected animals or their products. African tick-bite fever is endemic primarily in the southern (ie, sub-Saharan) regions of Africa; so, recent travel there would have to be ruled out.

Charles S. Pavia, PhD
Maria Plummer, MD
New York College of Osteopathic Medicine of New York Institute of Technology, Old Westbury

References

Road Map for Curricular Development and Professional Success: The Life Cycle of a Primary Care Physician

To the Editor:
Lawrence I. Silverberg, DO,1 brings up some very important issues and questions in his letter to the editor, “Road Map for Curricular Development and Professional Success: The Life Cycle of a Primary Care Physician,” in the March JAOA—The Journal of the American Osteopathic Association. Balancing the professional self with the personal self is a constant challenge that physicians face. Becoming the best physician that an individual can be may infringe on the personal development of that individual, preventing him or her from becoming the best person, parent, or spouse that he or she can be.

Not all physicians merge their personalities with their profession. Increasingly, in my observations physicians are moving immediately from the completion of their education to employee positions. How will the changing roles of the physician in the US health care system affect the life cycle model proposed by Dr Silverberg?2

The stages that professionally focused physicians typically go through are documented well by Dr Silverberg.1 After primary care physicians graduate, most have the ability and desire to do everything that they were trained to do. In my experience, striving toward this goal takes from 7 to 10 years, with newly minted family physicians working and studying as hard as they can and focusing themselves on advancing professionally. Meanwhile, their personal development may be hampered or slowed. I feel that the osteopathic medical pro-

(continued on page 463)
(continued from page 406)

fession rewards and values osteopathic physicians who put their careers before themselves. I have observed that after the initial period of intense practice and learning about their professional selves, many primary care physicians evolve into focusing on more limited practice areas. This evolution may be conscious on the physician’s part, or it may happen as a result of the interest the physician shows in particular areas of care. Some of this professional evolution is societal or cultural in nature. For example, it may be difficult to keep geriatric patients feeling comfortable in the same waiting room with pediatric patients. They may eventually abandon such a broad-based practice for a more focused practice that they feel is “their own.”

What Dr Silverberg calls “regressive preoccupation” may also be thought of as a professional dematuring of a physician in practice. Alternatively, it could be personal advancement in which the physician learns to compartmentalize pieces of his or her life to become more successful in multiple areas—or to reassess and reprioritize where he or she puts energy and focus.

I would also like to discuss what Dr Silverberg calls “curricular development.” The term curriculum infers that there is an assessment method. Although assessment can take many forms, and profiling and comparing yourself to other people whom you admire is an acceptable assessment method, it does not address all of the domains in which we live our lives. Interests and skills change over time. A baseball card collector who no longer keeps his cards in proper order does not have to be a dematured baseball card collector; he may be a changed person for many other reasons.

Assessment of self-confidence and use of pro/con grids are both reasonable ways of deciding the best way to act and respond. Other productive self-assessment techniques that can move an individual between life cycle stages include self-study time logs (for determining what to do when given the freedom to pursue what the mind is pushing toward), journaling and autobiographical sketching, and the creation of portfolios.

I believe that when physicians feel that they are entering a stage of “regressive preoccupation,” they may look at opportunities for advancement or growth. Previously, these opportunities were limited, but with the restructuring of the US health care system they are becoming more common. For example, administrative and management positions that previously did not require a physician are increasingly performed by professionals with medical training and experience. The so-called church-state separation between hospital care provided by physicians and management provided by business managers is being replaced by physician-led management. Such dematuring may also sometimes be thought of as a “midlife crisis” that individuals experience when they have mastered a particular field and wish to transfer their skills to another level, rather than continue using their skills at the same level for the rest of their lives.

I agree with Dr Silverberg’s point that transitions from one life cycle stage to another can be gradual. Such transitions can also be different for different individuals, and how they occur may vary according to the environment in which health care is being practiced. The professional life cycle of today’s primary care physicians is different from that of the generation before them.

Tyler C. Cymet, DO
Associate Vice President for Medical Education, American Association of Colleges of Osteopathic Medicine, Chevy Chase, Maryland

References

Acute Myeloid Leukemia, Genetics, and Risk Stratification: Data Overload or Ready for a Breakthrough?

To the Editor:

Acute myeloid leukemia (AML) consists of a group of relatively well-defined hematopoietic neoplasms involving precursor cells committed to the myeloid line of cellular development. These neoplasms account for 80% of all adult leukemias, with an incidence of approximately 3 to 5 cases per 100,000.

Until 2010, AML was classified according to the French-American-British classification system.

This classification scheme, proposed in 1976, divided AML into 8 distinct subtypes (M0-M7) on the basis of type of cell from which the leukemia developed and the condition’s degree of maturity. Classification relied heavily on the appearance of the malignant cells as seen with light microscopy.

With advancing technology and increasing insight provided by cytogenetics research, a new AML classification scheme was devised. In 2008, the World Health Organization (WHO) classified AML on the basis of a combination of morphologic, immunophenotypic, genetic, and clinical features.

The 4 main groups in this WHO system were (1) AML with recurrent genetic abnormalities—inv(3), inv(16), t(1;22), t(6;9), t(8;21), t(9;11), t(15;17), mutated CEBPA, or mutated NPM1; (2) AML with features related to myelodysplastic syn-
dromes; (3) therapy-related AML; and (4) AML not otherwise specified. This landmark WHO classification was notable not only because it allowed the incorporation of clinical and immunohistochemistry data with new genetic data, but also because it introduced genetic risk stratification into treatment for patients with AML.

Risk stratification was nothing new in cancer research. Since 1952, when Pierre Denoix of the Institute Gustave-Roussy devised the TNM (tumor, lymph nodes, metastasis) staging system for solid tumors, physicians around the world had been determined to devise improved ways to predict tumor behavior and patient survival. During the late 20th century—despite the advances in risk stratification for solid tumors—prognostication in hematologic malignancies, such as AML, remained difficult. Overall survival rates with AML treatment were extremely heterogeneous, and no one could easily predict which patients would do poorly. The only poor prognostic signs that had been identified for AML were advanced age at diagnosis (>55 years), poor performance status (Eastern Cooperative Oncology Group performance score ≥3), exposure to cytotoxic agents or radiation therapy, and history of previous myelodysplasia.

In the early 21st century, with additional research and increased emphasis on cytogenetics, AML has been further subdivided on the basis of karyotype—favorable, intermediate, and unfavorable. Favorable karyotypes (occurring in 16% of patients) consist of inv(16), t(8;21), t(15;17), and t(16;16). Intermediate karyotypes (20% of patients) are those abnormalities not described as favorable or unfavorable. Unfavorable karyotypes (13% of patients) include add(5q), add(7q), del(5q), del(7q), inv(3), t(3;3), t(6;11), t(9;22), (10;11), 17p abnormalities, monosomies 5 or 7, monosomy 17, and many others. Attesting to the heterogeneity of the disease, 10-year survival rates have been found to be 69%, 38%, 33%, and 12% for patients with favorable risk, normal karyotype, intermediate risk, and unfavorable risk, respectively.

With the outpouring of genetics research and data in the 2000s, there have been continual questions within the research community regarding whether patient characteristics in the new WHO classification scheme correspond to other genetic abnormalities. Patients with therapy-related AML secondary to DNA topoisomerase inhibitors typically have been found to have abnormalities involving the MLN gene at chromosome locus 11q231 and the RUNX1 gene at chromosome locus 21q22. There is a high frequency of the loss of the long arms of chromosomes 5 and 7 among patients with myelodysplasia-associated AML. In addition, patients with AML not otherwise specified have been found to have mutations in the FLT-3ITD / TKD, NRAS, BAAKC, and WTI genes.

Although correlating studies continue to this day, physicians currently have an abundance of prognostic information for their patients with AML. This information allows physicians to accurately risk-stratify patients on the basis of their extensive tumor profiles.

Despite the wealth of information acquired over the past 10 to 15 years, overall patient survival—the gold standard in cancer research—remains unchanged. Current standards of hematologic practice dictate that patients with poor cytogenetic profiles be evaluated for bone marrow transplant. However, no randomized, controlled trials have described benefits from this approach or whether certain chemotherapeutic regimens (excluding treatments for acute promyelocytic leukemia) are more effective for patients with certain cytogenetic abnormalities.

We have entered a new frontier in cancer research—a frontier full of hope and opportunity for improvement. Thousands of dedicated researchers have helped describe a multitude of genetic abnormalities associated with AML. However, this research is only a piece of the puzzle. We must expand our new knowledge and develop innovative approaches and treatment regimens for patients with AML to extend the overall survival rate of these individuals.

Simon B. Zeichner, DO
Department of Internal Medicine, Mount Sinai Medical Center, Miami Beach, Florida

References
10. Sverdlov SH, Campo E, Harris NL, et al, eds. WHO Classification of Tumours of Haematopoietic

tidrug resistance (MDR1) and cytogenetics distin-
guishes biologic subgroups with remarkably dis-


14. Letendre L, Noel P, Lutzow MR, Hoagland HC, Tefferi A. Treatment of acute myelogenous leukemia in the older patient with attenuated high-
dose ara-C. Am J Clin Oncol. 1998;21(2):142-144.


17. Libura J, Slater DJ, Felix CA, Richardson C. Therapy-related acute myeloid leukemia-like MLL rearrangements are induced by etoposide in pri-

18. Godley LA, Larson RA. Therapy-related myelodys-
plastic syndrome and myeloid leukemia. In: Steensma DP, ed. Myelodysplastic Syndromes: Patho-


20. Pedersen-Bjergaard J, Pedersen M, Roulston D, Philip P. Different genetic pathways in leukomega-

21. Pui CH, Relling MV. Topoisomerase II Inhibitor-

22. Pedersen-Bjergaard J, Philip P. Balanced translo-
cations involving chromosome bands 11q23 and 21q22 are highly characteristic of myelodysplasia and leukemia following therapy with cytostatic agents targeting at DNA-topoisomerase II. Blood. 1991;78(4):1147-1148.


25. Santamaria CM, Chilón MC, Garcia-Sanz R, et al. Molecular stratification model for prognosis in cyto-


29. Monzo M, Brunet S, Urbano-Ispizua A, et al; CETLM. Genomic polymorphisms provide prog-

30. Pasqualeci L, Liso A, Martelli MP, et al. Mutated nucleophosmin detects clonal multilineage involve-

31. Marucci G, Maharry K, Whitman SP, et al. High expression levels of the ETS-related gene, ERG, pre-
dict adverse outcome and improve molecular risk-

32. Langer C, Radmacher MD, Ruppert AS, et al. High BAALC expression associates with both out-
come and distinct gene and microRNA expression profiles in older patients with de novo cytogenet-


37. Paschka P, Schlenk RF, Gaidzik VI, et al. IDH1 and IDH2 mutations are frequent genetic alterations in acute myeloid leukemia and confer adverse prog-


39. Green CL, Evans CM, Hills RK, et al. The prog-

40. Schnittger S, Hafeler C, Ullke M, Alpermann T, Kern W, Hafeler T. IDH1 mutations are detected in 6.6% of 1414 AML patients and are associated with intermediate risk karyotype and unfavorable prognosis in adults younger than 60 years and unmutated NPM1 status. Blood. 2010;116(25):5486-5496.

Thinking Osteopathically

To the Editor:

“Thinking osteopathically.”

I am not sure what that means. As an emergency physician, does that mean I think holistically about my patients? Does it mean I engage the body’s inherent self-regulating mechanisms? Does it mean I use forms of manipulative medicine? If thinking osteopathically ends with these tenets, then so will the osteopathic medical profession. There is nothing unique about manipulative medicine; chiropractors and physical therapists use it every day. The other basic tenets of osteopathic medicine are even less uniquely ours. Also, our training is not of our own design.

As an educator, I am aware of the strides we have made to elevate the
skills and knowledge, as well as the numbers, of osteopathic trainees. But most, if not all, of the recent advances in our teaching have been borrowed from the Accreditation Council for Graduate Medical Education (ACGME). In fact, when you look at the ACGME methods, you will see the American Osteopathic Association (AOA) adopting the same approaches and standards 3 years later. For example, in 2001, the ACGME introduced the 6 core competencies in its Outcome Project.1 In 2004, the AOA came up with 1 core competency of its own.2 In 2003, the ACGME established new duty hour restrictions,4 and ours were established in 2006.5

I’m not against the ACGME’s practices, but I would like to see something come from our own house—from our own brains and will. As it is, we are neither the same as, nor different from, the ACGME ... imitation poorly done, timid, and slightly behind. We can compete to be the 800-lb gorilla, or we can evolve to use tools.

The ACGME and AOA both require written yearly criteria for residents to meet before they can advance to the next level of training. These criteria are appropriate, because they help prevent physicians from advancing in their training before they have demonstrated competency. But these plans do not offer any motivation to enter an AOA-approved internship or residency training program. We have set up minimum criteria without allowing those who meet the standards early to enjoy the fruits of their accomplishments.

It would be valuable to begin thinking about doing away with time-based training (ie, you are done after you have put in your time) and replacing it with competency-based training (ie, you are done when you demonstrate adequate knowledge and experience). Some residents would complete their training sooner; others would require more time. How long does a PhD program take? It takes as long as necessary—until the PhD candidate proves that he or she has obtained the required knowledge and skills. The structure of our training should be focused on the desired end result: qualified osteopathic physicians.

Learners in a competency-based training program might be able to—and would have some motivation to—learn faster, work harder, and complete the training sooner. Competency-based medical training is a tempting alternative to ACGME-style training, while maintaining the levels of knowledge and experience required by the AOA. In competency-based medical training, osteopathic residents would still be required to pass certification examinations and to have a minimum of patient exposures, but they would do so at their own pace—not the pace of tradition.

Thinking osteopathically, or thinking for ourselves?

Bruce A. St. Amour, DO
Emergency Medicine Residency Director, St. Mary Mercy Hospital, Livonia, Michigan

References

Correction

The JAOA regrets an error that appeared in the following article:


*Pseudomonas aeruginosa* was misspelled in the title. The title should have appeared as, “Ecthyma Gangrenosum Caused by *Pseudomonas aeruginosa*.”

This correction will be made to both the full text and PDF versions of the article online.