Get Ready for the 35th SGIM Annual Meeting
Lisa L. Willett, MD

Dr. Willett is co-chair of the SGIM Program Committee.

We are getting closer to the 35th Annual Meeting of the Society of General Internal Medicine. This year’s conference is at the Walt Disney World Swan and Dolphin Resort. The meeting begins Wednesday, May 9, 2012, with our precourse and concludes Saturday afternoon, May 12, with the annual Update in General Internal Medicine. We have all the traditional favorites and several new innovations this year.

2012 Malcolm L. Peterson Honor Lecture
Karen B. DeSalvo, MD, MPH, MSc, has accepted our invitation to present this year’s Peterson Lecture. Dr. DeSalvo is the commissioner of health for the City of New Orleans. She has a long history of SGIM involvement, serving as chair of the SGIM 28th Annual Meeting Program Committee, a member of Council, and as past president of ACLGIM.

Special Symposia
Special symposia topics this year will include: care transitions, federal initiatives that stimulate innovation in health care delivery, managing high-cost users and improving value in health care, innovative models of medical education and new medical schools, and issues facing graduate medical education in the modern era, including educating residents on quality and safety, transitions in care, and professionalism.

Clinical Updates
This year we are scheduling some familiar sessions after reviewing attendee evaluations and attendance from previous years. We have also invited sessions addressing topics new to the annual meeting, which we hope will pique your interest. Update sessions are designed to give attendees the latest evidence-based information in the topic area and cover clinically relevant information for the practicing provider. Look for continued on page 13
The Special Needs of Intern Learners: Principles to Guide Their Education
Greg Bowling, MD, and Luci K. Leykum, MD, MBA, MSc

Internal medicine residency training is challenging and necessarily so. Each month brings new trials that a doctor in training must pass. There is growing concern that medical education has not adapted to the rapidly evolving field of internal medicine. The Society of General Internal Medicine, American College of Physicians, and the Association of Program Directors in Internal Medicine have each issued recent position statements on areas of concern and plans for reform.1,2 These include the need for more meaningful evaluation systems and the ability to tailor trainees’ educational experiences more closely to their learning needs. The experience of the intern is a microcosm of these larger issues.

Within the overall trainee experience, the intern year is arguably the most grueling and is a period of transition that deserves particular attention. In one brief year, an individual must transition from student to intern and then prepare to lead the next group of interns who will follow. Each new intern is thrust into a world that is at once exciting and frightening. Each one has different strengths and weaknesses, but there are some educational needs that are shared by all. Interns universally are expected to expand their knowledge base and clinical acumen while managing innumerable tasks necessary for their daily work.

I vividly remember getting my pager at the beginning of intern year. There were times when that pager seemed to come alive. Nurses called with real problems on real patients—problems that demanded immediate effective action. With alarming clarity, I suddenly saw the chasm between the role of medical student and that of intern. I felt vulnerable and exposed by my dearth of knowledge. Every difficult experience brought new humility, which in turn engendered a deep respect for those who cared to teach. I absorbed every detail I could from my mentors. I knew that any pearl shared today might prove crucial to the care of my patient tomorrow.

In the recommendations for residency education reform, there is agreement that a “core” education experience must be provided for future internists. Self-directed learning (SDL) must be considered part of that experience.3 With the explosion of medical information, residency is only the beginning of life-long learning. Interns are capable of developing good habits if faculty can create an environment where real-time SDL can take place. This requires faculty to specifically model life-long learning in the clinic, hospital ward, and classroom. 

Interns benefit from conferences continued on page 15
A few days ago, I heard a story that reminded me again how far we are from having primary care that works. Jane (not her real name), a very fit woman in her 40s, volunteered for a clinical research study, and a protocol-based colonoscopy detected an adenomatous polyp. She was referred to a series of specialty-driven evaluations and procedures leading to waste and delay. She ultimately was done.

This was bad for Jane and bad for society, but it was a natural consequence of our current medical care system. There are many reasons for such cascades of evaluations, tests, and procedures leading to waste and often net harm. However, key among the causes for Jane was the lack of a primary care physician to act as her advisor and advocate as she tumbled down the medical cascade. Indeed, in the past, Jane had primary care physicians, but when she moved years before she never got another one—feeling that even if well-meaning, they often were not available for her when she wanted access and that the visits were too short to illustrate to her the value of primary care. So in the middle of this cascade, this very bright woman didn’t even think about having a primary care physician who might have advocated for a less specialty-driven path of evaluations and interventions.

In our daily practice, we all know that our ability to provide personalized primary care of this sort is severely compromised by many circumstances, and while we are disappointed, we are not surprised by what happened to Jane. Why is this the case? It’s not the intent of general internists, or other primary care clinicians, to shortchange patients in terms of time, attention, or access—but that is the case. In fact, this is the direct result of policy decisions made by the government, payers, and our profession. What is SGIM doing to address this?

Certainly part of the situation is due to inadequate support for training of primary care physicians. SGIM has been a strong supporter of Title VII HRSA funding for primary care training, advocating on our own and as part of coalitions to protect and grow the crucial funding for these programs. We were delighted to see these programs reauthorized at higher levels by the Affordable Care Act (ACA) and the Training in Primary Care Medicine portion increased from $39 million in FY11 to the President’s recommendation of $52 million for FY12—plus $86 million from the prevention and public health fund. However, what will come out of the Congressional appropriations process now is uncertain and worrisome. Unfortunately, this will be driven by overall budgetary goals and a balance between the Senate’s understanding of the importance of primary care and the House’s aversion to funding anything related to the ACA. At this point, the Senate Appropriations Committee recommendation for FY12 is the same as this year’s $39 million; a bill introduced in the House, which was never considered by the committee, would eliminate the primary care training program. This is not the picture of a coherent march toward training more primary care physicians, so SGIM continues to advocate for needed stable increases in this program.

Even as some House Republicans try to disassemble provisions and funding of the ACA, we should acknowledge the great improvements to address the primary care physician shortage that are embedded in this historic legislation. The inclusion in the ACA of the 10% Medicare bonus payment for primary care services and upward adjustments in Medicaid reimbursement, although not on a scale to be a sea change and not permanent, could start correcting the currently insufficient financial compensation and security that deters medical students from undertaking...
The late Speaker of the House Tip O’Neil was famous for saying all politics is local. In health care, you hear the same idea repeated—all health care is local. For the Affordable Care Act (ACA), both are true and important in how the law will be implemented. In Pennsylvania, politics and health care are as complicated as they are anywhere in the country. In this article, I will describe two challenges in Pennsylvania (and Philadelphia)—coverage and payment reform. But let me start with politics.

In November 2010, Attorney General Tom Corbett was elected governor of Pennsylvania. While campaigning, he was among the initial group of state attorneys general to sue the federal government challenging the constitutionality of the ACA. Now, his administration is charged with implementing key provisions of the law in Pennsylvania—in particular, the launch of a health insurance exchange and getting subsidies to eligible consumers. All states have the choice of whether to assume this responsibility or defer to the federal government. Governor Corbett has been slow to decide. As a result, time is short in Pennsylvania.

Health insurance exchanges will be the face of health reform. The exchange will be the “store” that consumers visit to shop for their health insurance. How this “store” does business will matter a lot to consumers. For example, will the exchange simply be a clearinghouse of insurance plans, or will it require plans to bid to participate? How will the exchange help consumers choose, and will the technology platform be easy to use? We know from the experiences of Medicare Part D that choice quickly becomes overwhelming. Finally, will eligibility screening for Medicaid or subsidies for private coverage be a streamlined and efficient experience for consumers, or will it be onerous?

Launching a patient-centered health insurance exchange cannot be done in a few months. Many states, including Pennsylvania, are running out of time to do this well, and the politics of the health reform debate continues to hang over implementation.

There is hope though in Pennsylvania. The insurance commissioner has moved forward with a planning process. My hope is that the experts in the insurance department will be given the authority and support to move forward in an expeditious fashion with patients in mind. It is too soon to know if this process will be allowed to happen without excessive political interference. The legislature will want to weigh in at some point and is awaiting signals from the governor. Meanwhile, the governor is balancing his campaign rhetoric against the ACA with a decision of whether to defer important health policy decisions to the federal government. Pennsylvania SGIM members should be reminding the governor and insurance commissioner why this is so important.

The ACO model does not explicitly limit patient choice of provider. Thus, health systems are understandably nervous about their ability to manage the cost of a patient population. It may be that ACOs in dense markets, like Philadelphia, need additional tools to make this an attractive option.

In contrast to ACOs, southeastern Pennsylvania has been a leader in the medical home model. The previous administration led a regional chronic care initiative in partnership with private payers that has helped push many primary care practices toward becoming patient-centered medical homes. Although the final outcomes of this initiative are unknown, it laid the foundation for future efforts to reform the delivery of primary care.

The ACA could be transformative for patients in Pennsylvania to cover the uninsured and make insurance more affordable. However, Tip O’Neil was right that all politics is local. And health policy experts are right that all health care is local. Successful implementation of the ACA will depend on political leaders moving beyond the polarization of the health reform debate and allowing state agencies to implement the law without political interference. Expertise, not politics, needs to drive the process. At the same time, transformation of the delivery system will require health care leaders to take some risks. The alternative is across the board payment cuts—a bad outcome for both doctors and patients.

Postscript: Since going to press, Governor Corbett announced plans to move forward to develop a state-based insurance exchange.
Common Supplements for the Practicing Internist
Randy Horwitz, MD, PhD

Dr. Horwitz is medical director at the Arizona Center for Integrative Medicine and an assistant professor of medicine at the University of Arizona.

Practicing internists today frequently encounter patients who are interested in taking natural supplements. The usefulness and the potential complications of different compounds are sometimes difficult to tease out from the abundant (and often confusing or erroneous) information available to patients and clinicians. However, there are a few common ailments for which supplements are commonly used and may, in fact, be beneficial. Prominent among these therapies are turmeric and fish oil.

Turmeric, a familiar component of Indian cuisine, has a long history of use as a potent anti-inflammatory agent. Because anti-inflammatory medications have a multitude of complications, many patients seek out natural anti-inflammatories. Recent research demonstrates that the active compounds in turmeric, called curcuminoids, inhibit pro-inflammatory transcriptional activators—the “on-off” switches responsible for transcribing inflammatory cytokines and interleukins.

To ensure that a patient takes adequate concentrations of curcuminoids, standardized products should be used. Typical doses are 1500 mg taken up to three times daily. When turmeric is combined with piperine, an extract of black pepper, systemic absorption is significantly increased. Ingesting turmeric without pepper allows for localized gastrointestinal anti-inflammatory activity, which can be useful in inflammatory bowel disease (IBD). The clinical effect of turmeric in IBD is currently under study.

Fish oil had been used as a supplement for many years to lower triglycerides and is now endorsed for this purpose by the American Heart Association and the FDA. Although treatment of hypertriglyceridemia is the accepted indication for fish oil, the canonical omega-3 component, which is readily incorporated into leukocyte cell membranes, also functions as an anti-inflammatory agent. Regular ingestion of fish oil decreases the ratio of omega-6 to omega-3 fatty acids in the cell membranes, thus reducing the amount of omega-6 substrate available for the production of arachidonic acid-derived inflammatory metabolites. Improvement in IBD symptoms in patients taking fish oil has been chronicled.

The main long-chain fatty acids in fish oil are EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid). Typical anti-inflammatory dosing is based on the EPA concentration and ranges from 500 to 1500 mg of EPA daily. Caution should be exercised in patients using concomitant prescription anti-coagulant therapy.

Red yeast rice (RYR) is a very popular alternative to statin drugs. It is a fermented rice product that gets its distinctive color from the mold species Monascus purpureus. Lovastatin was actually first isolated from red yeast rice in the early 1970s. In fact, red yeast rice actually contains several compounds with HMG-CoA reductase activity. When originally sold, the supplement was very popular, well-researched, and sold with a standardized concentration of monocolin K (lovastatin). Eventually, this product was legally deemed a drug, as opposed to a dietary supplement, and thus is no longer sold over the counter as a “standardized” compound. Despite the theoretical advantages of synergistic blends of natural statins, there is an inherent uneasiness about prescribing a product with extreme lot-to-lot and product-to-product variations. A recent analysis of available, off-the-shelf RYR products revealed a range of 0 to 20 mg lovastatin per capsule, depending on the brand purchased. In addition, several RYR products were found to contain high levels of citrinin, a contaminating mycotoxin that has been linked to nephropathy.

As with any therapy, supplements should be researched prior to recommending them to patients. Although there is a lower incidence of reported myopathy with the RYR compounds relative to pharmaceutical doses of statins, the standardization of dosing and the screening for contaminants of pharmaceutical statins can make these (especially at the safer lower doses) a more attractive form of cholesterol management than the present-day RYR compounds.

Coenzyme Q10 (ubiquinone) is a mitochondrial protein that is essential to the electron transport cycle. In addition to its role in cellular energy production, CoQ10 also acts as a potent antioxidant. Of cardiovascular relevance is the biochemistry of statin activity and the subsequent effect of statins on CoQ10 levels. The mevalonic acid biochemical pathway is mediated by HMG-CoA reductase and is inhibited by statin drugs and yields both cholesterol and CoQ10. Inhibition of HMG-CoA reductase by statins (and perhaps beta blockers) lowers endogenous CoQ10 levels.

In the body, CoQ10 is highly concentrated in muscle cells—both skeletal and cardiac—with their high mitochondrial content. The idea of using CoQ10 supplements to correct this deficit gained considerable appeal following the publication of several studies that described marked decreases in statin-induced myopathy when CoQ10 was administered concomitantly with the statin. Additionally, CoQ10 supplementation may prevent or alleviate statin-associated congestive heart failure. Furthermore, CoQ10 has been shown to lower blood pressure in several studies, as well as to improve function in patients with Parkinson’s disease. Serological measures of CoQ10 concentrations in continued on page 12
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Co-management is loosely defined as a “shared responsibility, authority, and accountability for the care of a hospitalized patient across clinical specialties” based on a recent white paper, titled “A guide to Hospitalist/Orthopedic Surgery Co-Management.” With reimbursement structures favoring surgeons to perform more surgeries and work hour rules for resident surgeons to spend a larger portion of their time in the operating room, more surgeons are looking for help taking care of their patients, particularly those who are older and more medically complex.

At the same time, the hospitalist movement has grown, and a 2005-2006 Society of Hospital Medicine Survey reported that 85% of hospital medicine groups are involved in co-management. Since 2001, co-management by a generalist physician has increased by 11.4% per year.

Critical elements of surgical co-management require identification of co-management program champions who can determine goals of the co-management program, serve as leaders when conflicts come up, and determine roles and responsibilities of the program. Service agreements are important up front to determine who is the attending of record, who is “first call” for nursing calls, and which patients are appropriate for co-management. Finally, measuring performance, process improvement, and working through financial and compensation issues are important parts of a well-functioning surgical co-management program.

What is the evidence for surgical co-management? Theoretically, with hospitalists covering the floors while surgeons are in the operating room, immediate care issues could be better taken care of. However, studies show mixed results. A study of hip fracture patients taken care of by hospitalists showed no difference in major complication rates. A study of co-management of elective joint replacement showed no difference in unadjusted length of stay and patient satisfaction but improved nursing and orthopedic surgeon satisfaction. More recently, Auerbach et al. conducted a retrospective interrupted time-series analysis of patients admitted to a neurosurgery service from June 2005 to December 2008. They found no differences in patient mortality rate, readmission, or length of stay. No consistent improvement in patient satisfaction was shown, but nursing and non-nurse health care professionals strongly favored the co-management program. Also, a reduction in hospital costs of $1,439 per admission was seen.

Others have made a case against surgical co-management. They feel that there is already a shortage of internists especially in primary care and that the movement of internists toward co-management services will only worsen this. The benefits of co-management are mostly monetary to surgeons and hospitals based on shifting work to lower paid workers (internists) so that surgeons can spend more time in the operating room. They also worry that surgeons will no longer know how to take care of postoperative patients and postsurgical complications.

It is unclear why differences in quality were not seen as expected with the co-management model. It may be that patients who are being co-managed are “sicker” (e.g., higher American Association of Anesthesia scores, diabetes mellitus, vascular disease, chronic renal failure, congestive heart failure, and coronary artery disease). Also, hospitalists may not be fully utilized. For example, it is not uncommon to defer DVT prophylaxis to the surgeon who puts his patient with a hip fracture on only mechanical devices for fear of wound hematoma and bleeding, although the patient is at high risk for thrombosis. Patients may not be seen early enough preoperatively to effectively start and titrate beta blockers or get diabetes mellitus under control.

In general, the hospitalist co-management model is here to stay. We will want to use this opportunity to take care of our surgical patients, which includes studying perioperative quality improvement, playing a direct role in the management of these surgical patients, and making our voices heard through direct partnerships with our surgical colleagues.
Objective: To provide a framework for the evaluation and treatment of primary aldosteronism.

Case: A 52-year-old man with hypertension, metabolic alkalosis, and hypokalemia has poorly controlled blood pressure despite treatment with four antihypertensive agents. In addition, he was incidentally found to have a 1.2 cm left adrenal adenoma during work-up of abdominal pain that has since resolved. How should he be evaluated for possible primary hyperaldosteronism? If primary aldosteronism is confirmed, should he just undergo left adrenalectomy, or should adrenal vein sampling be performed before surgical intervention?

Teaching goal: To convey the normal physiology of aldosterone regulation and the evaluation and treatment of primary aldosteronism.

Aldosterone Physiology: Under normal circumstances, aldosterone secretion from the adrenal gland is regulated by both the renin-angiotensin system and the extracellular potassium (K+) concentration. A drop in renal perfusion pressure stimulates the release of renin into the circulation, which results in the conversion of angiotensinogen to angiotensin II through a number of enzymatic steps. Activation of the angiotensin II receptor in the adrenal gland promotes the synthesis and secretion of aldosterone from the adrenal cortex. Aldosterone is also the primary regulator of K+ concentration in the body. High circulating K+ levels cause membrane depolarization of the zona glomerulosa and directly trigger aldosterone secretion, whereas a low K+ concentration has the opposite effect.

The physiologic effects of aldosterone are primarily mediated by its actions on the principal and intercalated cells of the distal nephron. In these cells, aldosterone increases the expression and activity of the basolateral Na-K-ATPase as well as the luminal Na+ epithelial channels (ENaC) and hydrogen pumps to give rise to Na+ retention, K+ loss, and H+ excretion, resulting in increased blood pressure, decreased plasma K+, and metabolic alkalosis, respectively.

Primary Aldosteronism: Primary aldosteronism refers to a group of disorders in which aldosterone production is inappropriately elevated, not regulated by the renin-angiotensin system, and not suppressible by sodium loading. Primary aldosteronism is a common cause of secondary hypertension, occurring in approximately 5% of all hypertensive patients.

Case Detection: The prevalence of primary aldosteronism increases significantly among patients who have severe hypertension and those on more than three antihypertensive agents. These patients should be screened for possible primary aldosteronism. In addition, screening is recommended in patients with hypertension and hypokalemia (spontaneous or diuretic induced), hypertensive patients with adrenal incidentalomas, young patients with hypertension, and hypertensive patients with a family history of stroke prior to age 40. It is important to note that hypokalemia is found in less than 50% of patients with primary aldosteronism, so normal K+ levels cannot be used to exclude this diagnosis.

Screening Tests: The initial screening for primary aldosteronism involves the morning ambulatory measurement of plasma aldosterone concentration (PAC) and concurrent plasma renin activity (PRA) to calculate the aldosterone to renin ratio (ARR). Patients can continue on their antihypertensive agents except for spironolactone, eplerenone, amiloride, and triamterene, which significantly interfere with the interpretation of test results. Verapamil, hydralazine, and alpha blockers are the preferred agents as they have minimal impact on screening tests.
A 30-year-old male presents to the emergency department with 24 hours of intermittent, substernal, non-radiating chest pain associated with diaphoresis. EKG demonstrates ST segment elevation in leads I, aVL, and V4-V6. Cardiac enzymes reveal a troponin of 23.9 ng/mL and CK-MB of 47.9 ng/mL.

The patient’s significant troponin elevation and EKG changes are concerning for possible acute anerolateral myocardial infarction. One should determine if there is a history of structural heart disease, early coronary artery disease, or cocaine/amphetamine usage. Despite the patient’s young age and potential lack of risk factors, an acute ST-elevation myocardial infarction must be considered until proven otherwise. Thus, immediate cardiac catheterization is required. Alternatively, a more common cause of chest pain and diffuse ST-elevation in a young adult is pericarditis, but one would not expect this degree of troponin elevation from that condition alone. The marked elevation of his cardiac biomarkers suggests considerable myocardial inflammation and necrosis raising suspicion for acute myocarditis. This condition presents most commonly in young males and often mimics acute myocardial infarction. Severe myocarditis can also result in acute dilated cardiomyopathy. If coronary angiography proves to be normal, the results of his left ventriculogram may point to this alternative diagnosis. Global left ventricular dysfunction would support a diagnosis of acute, diffuse myocarditis and should be investigated further with trans-thoracic echocardiography.

The patient is taken immediately to the cardiac catheterization laboratory for suspected acute myocardial infarction. Coronary angiography reveals no intraluminal deficits. Left ventriculogram demonstrates globally diminished left ventricular contractility. Trans-thoracic echocardiography confirms diffuse left ventricular hypokinesis with an ejection fraction of 35% and normal diastolic function.

These findings support a diagnosis of acute myocarditis. This condition can lead to acute heart failure, arrhythmias, and sudden cardiac death from ventricular tachycardia. Therefore, supportive care should be provided in the setting of continuous cardiac monitoring.

A detailed history and physical should attempt to determine an underlying etiology. Viral infections, including coxsackievirus, adenovirus, echovirus, and parvovirus, are among the most common causes of acute myocarditis in developed countries. The history may uncover symptoms consistent with a recent viral prodrome. One should ask about exposure to sick contacts or children with the classic “slapped-cheek” rash of parvovirus. In addition, risk factors for HIV and other immunocompromised states should be assessed as this would introduce less common etiologies such as acute HIV, tuberculosis, or mycotic organisms into the differential. Recent vaccination or the addition of a new medication could suggest a hypersensitivity myocarditis. A detailed exposure and travel history are necessary to exclude tick-borne and viral etiologies. One would expect a viral prodrome. The marked WBC left shift, however, is less suggestive of a viral etiology. One would expect an acute viral myocarditis to increase the sedimentation rate and CRP but perhaps not to the degree illustrated above. Hence, endocarditis, acute rheumatic fever, rickettsial disease, or a collagen vascular disease also must be considered.

Given his depressed ejection fraction, a beta-blocker and ACE-inhibitor should be instituted if his hemodynamic status will allow.

After cardiac catheterization, the patient is transferred to the intensive care unit. His past medical history is unremarkable. He works as an accountant and denies any recent travel outside the southeastern United States. He denies any sick contacts or known tick exposure. He does not report constitutional symptoms of fever, pharyngitis, and myalgias for approximately two weeks prior to admission.

On physical exam, his lungs are clear. He is tachycardic but has no murmurs, rubs, or gallops. There is no evidence of jugular venous distention or peripheral edema. No skin rash, nodules, or splinter hemorrhages are appreciated.

Laboratory data demonstrate a white cell count of 16.8 k/U, with 98% segmented neutrophils, hemoglobin 12.9 g/dL, and platelets 218 k/U. Sedimentation rate is 89 mm/hr with a CRP of 20 mg/dL. Urine drug screen testing is negative.

His antecedent constitutional symptoms could be consistent with a viral prodrome. The marked WBC left shift, however, is less suggestive of a viral etiology. One would expect an acute viral myocarditis to increase the sedimentation rate and CRP but perhaps not to the degree illustrated above. Hence, endocarditis, acute rheumatic fever, rickettsial disease, or a collagen vascular disease also must be considered.

Soon after admission, the patient develops fever to 102.9°F, moderate tachycardia, tachypnea, and hypotension with BP dropping to 80/65 mmHg.
His fever and unstable vital signs are worrisome for severe myocarditis or a serious infection such as endocarditis accompanied with developing sepsis and/or worsening heart failure. Blood cultures should be obtained immediately, and empiric broad-spectrum antibiotics that include coverage for staphylococcal and streptococcal species should be initiated. Although no valvular vegetations were seen on initial imaging, trans-esophageal echocardiography would be indicated if his blood cultures become positive. In addition, his cardiac telemetry tracings should be reviewed carefully to exclude periods of unrecognized arrhythmias that could explain his tachycardia and hypotension.

Cardiac MRI can support the diagnosis of myocarditis and may provide clues as to the underlying etiology. This technology, however, is expensive, not widely available, and often not necessary for diagnosis. Furthermore, cardiac MRI should not be pursued at this time given the patient’s hemodynamic instability. Endomyocardial biopsy is rarely pursued in cases of acute myocarditis as the diagnostic yield is low and findings often do not change management. Nevertheless, it could be considered if his symptoms are refractory and a specific etiology remains undetermined.

Acute and convalescent viral antibody titers also may be considered in cases of acute myocarditis. These are not ordered routinely, however, as serologic interpretation may be difficult and the results often do not influence management decisions.

The patient is started on NSAIDS and colchicine for possible viral perimyocarditis. In addition, broad spectrum antibiotics are initiated and infectious disease consultation is obtained. Blood and urine cultures show no growth. Throat culture and anti-streptolysin O antibody titers are negative. HIV ELISA, HIV viral load testing, and monospot testing are all negative. CMV IgG antibody returns positive, but IgM antibody is within normal limits. Viral titers for coxsackie, hepatitis, and parvovirus also return negative. Rocky Mountain Spotted Fever antibody titers are normal as well.

The negative blood cultures are reassuring that the patient does not have endocarditis, and empiric antibiotics can be stopped unless another source of bacterial infection is suspected. Acute rheumatic fever also is unlikely at this point. His CMV serologies are consistent with past exposure. A viral etiology remains possible, but his normal viral antibody titers may suggest another underlying cause.

NSAIDS and colchicine are the treatment of choice for viral pericarditis. Their use in acute myocarditis, however, is not as well established. In fact, animal models have shown that NSAIDS are not effective and may actually enhance the myocarditic process and thereby worsen mortality. Oral corticosteroids should be avoided if possible as their use has been shown to increase the risk of recurrence in patients with undifferentiated pericarditis.

The patient’s fever and hemodynamic instability resolve with ongoing supportive care. His symptoms of chest pain and myalgias improve, and his troponin values normalize. Repeat echocardiogram one week later shows significant left ventricular improvement with an ejection fraction of 55%. The patient is discharged with a presumed diagnosis of viral myocarditis.

Three days later, however, the patient returns to the emergency department with persistent intermittent fevers to 103°F, pleuritic chest discomfort, and worsening arthralgias. Repeat laboratory testing demonstrates a persistently elevated WBC count of 24.6 k/uL and a sedimentation rate >140 mm/hr. Cardiac enzymes remain normal. Liver function tests reveal an elevation of alkaline phosphatase 197 IU/L, ALT 232 IU/L, and AST 144 IU/L.

Physical exam now reveals a maculopapular rash on his right flank along with tenderness at his left shoulder, right knee, and bilateral wrists with no obvious joint deformities. The patient has persistent fever and worsening polyarthralgia. In addition, he now has abnormal transaminases and a localized maculopapular rash. The differential is broad and includes collagen vascular diseases such as systemic lupus erythematosus, rheumatoid arthritis, or cryoglobulinemia. Hence, ANA, rheumatoid factor, cryoglobulin, and acute hepatitis testing should be strongly considered. Endocarditis, acute rheumatic fever, and rickettsial disease remain consistent with this presentation but are unlikely given prior laboratory results. Extra-intestinal complications of inflammatory bowel disease should be considered, but this diagnosis is unlikely in the absence of GI symptoms. A reactive arthritis or seronegative spondylarthropathy are possible as well.

At this point, however, his clinical presentation is most suspicious for adult Still’s disease. This condition classically is associated with an evanescent, salmon-colored maculopapular rash. It most commonly presents in a young adult male with unexplained fever, polyarthralgia, lymphadenopathy, elevated LFTs, and antecedent pharyngitis. Adult Still’s disease is characterized by a marked elevation in serum ferritin level. Often, high dose oral corticosteroids are effective at improving symptoms. Adult Still’s disease typically is not associated with acute myocarditis, but a few cases have been reported in the literature.

ANA, rheumatoid factor, cryoglobulin, and acute hepatitis testing are negative. Serum ferritin level returns markedly elevated at 6340 ng/mL. The patient is diagnosed with adult Still’s disease and is started on 80 mg of oral prednisone daily with rapid improvement in his symptoms.

Learning Points
- Acute myocarditis often mimics acute myocardial infarction by presenting with chest pain, ST-segment elevation, and marked elevation of cardiac biomarkers.
- Adult Still’s disease classically presents with a maculopapular rash.
The Southwest is unlike any other area in the United States. The location, weather (yes it’s a balmy 70 degrees in Phoenix today), and the health care climate seems to be at odds with majority of the country. We are a very diverse society of true Arizonians and national and international transplante—predominantly from Mexico, the Midwest, and California.

We are the state that passed the first immensely polarizing state immigration bill. We have the toughest immigration laws and the latest gun laws; we believe that the government should stay out of the individual’s business. During the fiery and tumultuous health care debate, I came across an elderly woman protesting in a rally organized against health care reform. “Government stay out of my Medicare” screamed her sign. We are also the state that recalled the author of the immigration bill in a recent election. We have one of the country’s most efficiently run Medicaid programs (if you can say “efficient” and “Medicaid” in the same sentence) and lower costs and utilization of health care. We are also low on health care quality. We continue to have high physician shortages and low numbers of medical education programs but high physician retention. Like our free thinking neighbor in the West, we have espoused the medical marijuana law. The Arizona Hospital Association (despite the dreaded three letter word-tax) offered to tax itself to draw down federal matching funds for the Medicaid and Medicare programs—a move that was vetoed by the current Congress, which preferred to cut training programs and funds for deserving patients over any suggestion that they were complicit with increased taxation. Arizona has opted out of health care reform and is one of the states that has challenged the law in court. We also have low levels of health care research and grant funding compared to the northeast.

Graduate medical education is partly funded by the state Medicaid program. As the economy continues its downward spiral, the health care industry is now feeling the impact. Hospital admissions are significantly down while the rates of uninsured are skyrocketing. Being a high Medicaid state, several hospitals and residency programs in the state are heavily dependent on the Medicaid program.

The next few years will be decisive to the health care landscape in Arizona. We will have three allopathic medical schools, continued expansion of the University of Arizona College of Medicine in Phoenix and Maricopa County Hospital, and the new campus of Creighton Medical School at St. Joseph’s. The announcement of the new Mayo medical school, in partnership with the Arizona State University, bodes well for the growth of health care in Arizona. The passage of the Affordable Care Act, as in the rest of the country, has resulted in practices, hospitals, and payer dancing around each other to try to make sense of a health system that seems to be at odds. The gold rush is over and now we seem to be rushing as health care systems and physicians play catch up so that millions of dollars are not left behind. As the baby boomer generation flocks to Arizona to retire, we are faced with the reality of working together to find innovative ways to provide good health care and medical education in a land of innovative thinkers.
Chalk Talk

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Other blood pressure agents can slightly decrease the sensitivity or specificity of the measurements but to an acceptable degree.

A positive screen occurs when the both the aldosterone (ng/dL) to renin (ng/mL/h) ratio (ARR) is greater than 20 and the aldosterone concentration is greater than 10 to 15 ng/dL. An important caveat is that the assays are not standardized, so cutoff values are dependent on the specific assays used. In general, as the ARR and the PAC increase, the specificity of the screen increases and the sensitivity decreases.

Confirmatory Test: Because of limitations with the specificity of the screening test, a positive result requires confirmatory testing. As with most endocrine disorders involving a hypersecretory state, confirmatory testing for hyperaldosteronism involves an attempt to suppress hormone secretion. This is accomplished by driving down renin production with a sodium load. Under normal circumstances, the volume expansion resulting from a sodium load should suppress renin and aldosterone levels. Failure to suppress aldosterone secretion confirms the presence of primary aldosteronism.

Sodium loading is commonly accomplished by one of two methods. The first involves the infusion of 2 liters of normal saline over a 4-hour time period. If plasma aldosterone levels are not suppressed at the end of the infusion (PAC > 10 ng/dL), primary aldosteronism is confirmed. Alternatively, patients can be instructed to increase dietary sodium intake to approximately 6 grams per day for 3 days. On the third day, a 24-hour urine collection is obtained for measuring urinary sodium and aldosterone. Urinary sodium measurements of more than 200 mEq ensures adequate salt loading, and urinary aldosterone levels greater than 12 μg confirm the diagnosis of primary aldosteronism. It is important to avoid hypokalemia during the salt load as this can decrease the sensitivity of the test.

Subtypes: After primary aldosteronism has been confirmed, the next step is to determine the cause. Different subtypes of primary aldosteronism vary in both their treatment modalities and relative frequencies. Bilateral etiologies require medical management whereas unilateral causes are amenable to surgical treatment. The bilateral causes continued on page 14
the following clinical updates: addiction medicine, care of cancer survivors, HIV, hospital medicine, new medications for primary care, medical education, perioperative medicine, and women’s health. Once again, our meeting will close with an Update in General Internal Medicine, so make sure you plan to stay in Orlando through Saturday afternoon!

New This Year: Clinical Pearls

The goal of this new series is to provide useful management skills for interns about important clinical problems and conundrums, using a contemporary evidence-based clinical approach. Each session will highlight three common clinical problems in which the diagnosis or management has changed over the past few years or for which frequent practice challenges exist. Discussants and presenters will be SGIM content experts. Each session will have three topics discussed. Each topic will include a 20-minute presentation, followed by five minutes of group discussion with questions and answers. The three sessions include: chronic disease management (headache, hypertension, and low back pain), communication and behavioral health (smoking cessation, prostate cancer screening, and diabetes medications/lifestyle adherence), and acute presentation of disease (syncope, heart failure, and atrial fibrillation).

In addition to the above, we have planned a tremendous array of workshops, abstracts, clinical vignettes, innovations in medical education, and clinical practice innovations that will showcase the best of our membership’s efforts and scholarship.

We have expanded our international program this year, with members from Japan presenting a session on the Japanese response to the tsunami and Fukushima nuclear disaster. Our Swiss members will share their experiences with international disasters in both Haiti and Libya. Those new to the meeting will be able to attend a first-timer’s orientation on Thursday. Our annual student-resident-fellow workshop series will be interesting, as always. We have expanded our mentoring offerings. Not only will we offer one-on-one mentoring sessions, we will also be scheduling four mentoring panels. SGIM members will share their wisdom on important personal and professional topics for successful careers in academic general medicine. The VA series will once again offer workshops, symposia, and an opportunity to meet informally with VA leadership.

So, make sure to register now to join us in beautiful Orlando for a meeting you won’t forget. Come share your work, network with friends and colleagues, and renew your enthusiasm for general medicine. The website (www.sgim.org/go/am12) has all the information you need about the meeting and the exciting program we have to offer. We are looking forward to seeing you there!

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**References**

Strategies for managing primary aldosteronism will develop recommendations that align these dual responsibilities and help eliminate the contributions due to payments to unnecessary and uncoordinated care. We hope to have these recommendations out in about a year, and we hope they will engender wide comment and support.

In the meantime, SGIM strongly supports efforts such as those by the American Academy of Family Physicians to correct the longstanding imbalances created by the AMA Relative Value Update Committee (RUC) in the payments for subspecialty care, procedures, and diagnostic tests relative to primary care. (SGIM has no seat on the RUC, but family medicine does, and thus we support their protest from that seat.) We could have predicted the impact on health care of the incentives built into the payments suggested by the RUC and embedded in the Medicare payment system. Now that we have the clear result, the experiment continues to run anyway. It’s time to respond to the results. We must do whatever we can to address this problem. Unfortunately, just as our nation has started to address the factors that contribute to the current situation in primary care, there is intent to snatch defeat from the jaws of the ACA victory. We, and Jane, know that this is bad for our patients and for our nation. We need to redouble our efforts to communicate about and advocate for reform of the payment system to mitigate its perverse incentives and to be sure all Americans have access to primary (and other) care. SGIM is completely committed to this, and our members must be, too. We must constantly remind our elected officials of the need to improve the situation for primary care. (Background information can be obtained from the health policy tab on our website http://www.sgim.org.)

Our best care of our own patients is important, but without a change in policy, Jane and many other citizens will continue to get way more care than they need because they receive too little primary care. It is our responsibility in the care of patients generally to work to improve this situation. I look forward to hearing from SGIM members’ experiences in addressing this issue.

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**CHALK TALK**

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include bilateral adrenal hyperplasia, also known as idiopathic hyperaldosteronism, and familial hyperaldosteronism. Unilateral subtypes include aldosterone producing adenomas and carcinomas and unilateral adrenal hyperplasia. The most common cause of primary aldosteronism is bilateral adrenal hyperplasia, which accounts for almost two thirds of cases. The second most common etiology is an aldosterone producing adenoma, occurring in approximately 30% of individuals with primary aldosteronism. The other causes account for less than 5% of cases, with unilateral adrenal hyperplasia accounting for the majority of the relatively rare causes. However, because ~1% of cases of hyperaldosteronism are caused by adrenal cancer, adrenal CT is recommended in all patients.

**Surgical vs. Medical Management:** As true differentiation between unilateral and bilateral disease requires an invasive procedure, the next step after cancer has been ruled out by adrenal imaging is to determine whether the patient is a surgical candidate. When clinical features or personal preferences exclude unilateral adrenalectomy as an option, medical treatment can be initiated without further evaluation. If, on the other hand, the patient is willing to consider possible definitive therapy, additional testing is required to determine if the primary aldosteronism is caused by unilateral or bilateral disease.

Only unilateral disease, like aldosterone producing adenomas and unilateral hyperplasia, responds to unilateral adrenalectomy. Bilateral disease, which accounts for approximately two thirds of cases, requires medical management. Treatment with either of the mineralocorticoid receptor blockers, spironolactone or eplerenone, significantly improves blood pressure and normalizes hypokalemia in the majority of patients with primary aldosteronism.

Although hypertension and hypokalemia tend to be more severe in patients with aldosterone-producing adenoma than bilateral adrenal hyperplasia, clinical markers have poor discriminatory function due to the large degree of overlap. Likewise, the radiographic appearance of the adrenal glands correlates poorly with the continued on page 15
that are geared toward their level of skill and that provide opportunities for group learning. No longer can we assume competency based solely on completion of medical school. Problem based learning (PBL) conferences are an ideal venue to address both knowledge gaps and encourage the development of SDL skills. In a PBL conference, interns accept a clinical problem, realize their knowledge gap, seek out and evaluate information, and then apply it to the problem at hand. Interns experiencing these conferences together can see how peers as well as teaching faculty might approach problems encountered in practice.

Finally, interns need patient faculty who can remember the trials of internship and relate to the obstacles they are experiencing in their education. For example, faculty can encourage conference attendance and relieve clinical burdens when interns are given “protected educational time.” A genuine, encouraging faculty member who is mindful of educational priorities sets the right tone for a healthy learning environment. Educators who love their profession energize the learning experience, and those who demonstrate a “zeal for the field of internal medicine” inspire our interns.

If we pay attention to the unique experience of intern year, we can capitalize on the opportunity to build skills suited for modern health care delivery. The investment of time and talent by committed clinician-educators who promote SDL, create dedicated intern group learning experiences, and facilitate learning in the midst of busy clinical experiences can greatly help interns realize their professional potential and enjoy the journey of residency in the process.

References

CHALK TALK
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functional hyperaldosteronism. Recent studies have convincingly shown that 1) the presence of an adrenal nodule in a patient with primary aldosteronism does not prove the diagnosis of an aldosterone-producing adenoma, and 2) its absence does not exclude unilateral disease. This is because non-functioning adrenal adenomas are common, the adrenal glands in bilateral adrenal hyperplasia may have nodular changes, and unilateral disease may result in normal-appearing adrenal glands.

Adrenal Vein Sampling: The definitive test for distinguishing unilateral (surgical treatment) from bilateral causes (medical treatment) is adrenal vein sampling. By catheterizing the right and left adrenal veins and comparing cortisol-corrected aldosterone ratios between the two adrenal glands and the periphery, one can determine with a high degree of confidence whether the hyperaldosteronism lateralizes to either the left or right adrenal gland or whether it is caused by bilateral disease.

When compared to adrenal CT, adrenal vein sampling better identifies the source of unilateral disease and determines which patients have bilateral adrenal hyperplasia. In fact, adrenal CT was accurate in identifying the source of the hyperaldosteronism in only about half the patients with primary aldosteronism compared to adrenal venous sampling. Relying on the imaging findings alone would have resulted in about a quarter of patients undergoing unnecessary surgery and another quarter being incorrectly excluded from potentially curative adrenalectomies.

Resolution of Case: Screening for primary aldosteronism was positive with a PAC of 28 ng/dL and a PRA of < 0.6 ng/mL/h, yielding an aldosterone to renin ratio (ARR) > 47. IV saline load confirmed the diagnosis of primary aldosteronism with failure to suppress aldosterone levels (16 ng/dL). Despite the presence of a left adrenal nodule, adrenal vein sampling showed elevated aldosterone secretion from both adrenal glands without evidence of lateralization. The test results indicated that the patient had a non-functional adrenal adenoma, which has a prevalence of about 4% among patients in their sixth decade, and bilateral adrenal hyperplasia. Treatment with spironolactone normalized hypokalemia and gradually improved blood pressure control so he was able to discontinue all other antihypertensive agents.

References
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