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ANNUAL MEETING UPDATE

Get Ready for the 35th SGIM Annual Meeting

Lisa L. Willett, MD

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

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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

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The Special Needs of Intern Learners: Principles to Guide Their Education

Greg Bowling, MD, and Luci K. Leykum, MD, MBA, MSc

Dr. Bowling is an assistant professor of medicine, Division of Hospital Medicine, at the University of Texas Health Science Center at San Antonio. Dr. Leykum is an associate professor of medicine, Division of Hospital Medicine, at the South Texas Veterans Health Care System and University of Texas Health Science Center at San Antonio.

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.[1-3] 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.[1] 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

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Policy That Would Help Make Primary Care Primary

Harry P. Selker, MD, MSPH

…various new organizational approaches to health care hold some hope of re-balancing payment for primary care relative to other care, which also may mitigate the disincentives for entering primary care.

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 specialists, leading to 55 medical visits over the ensuing one-year period. She underwent many high-tech imaging studies, tests, procedures, and several operations, only one of which was clearly indicated. (Although it was, in retrospect, unnecessary, the colectomy showed that the polyp already had been successfully removed by colonoscopy.) Her life was completely disrupted, huge amounts of money were spent by her and her insurers, and yet arguably nothing ultimately helpful 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

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SGIM Forum

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All Politics (and Health Care) is Local: A View from Pennsylvania

David Grande, MD, MPA

David Grande is an assistant professor of medicine in the Division of General Internal Medicine at the University of Pennsylvania.

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 ACA also included provisions to test and expand new payment models focused on reducing costs and improving quality. The signature program in the law is the creation of Accountable Care Organizations (ACOs). In Philadelphia, although there is enthusiasm around several payment models such as the medical home, there seems to be less enthusiasm toward being an early adopter of the ACO model.

Perhaps one reason is the unique nature of the Philadelphia market. The close proximity of several large academic medical centers and competing health systems creates a perception that it is more difficult to be accountable for a patient population. In a dense urban area like Philadelphia, patients are more likely to receive care in competing health systems. 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.

Poststcript: 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

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"It's not What You Earn but What You Keep That Counts"

Andrew Schutzbank, MD, MPH

Dr. Schutzbank is a member of the Forum *Editorial Board and can be reached at aschutzb@bidmc.harvard.edu.*

The maxim quoted as the title of this piece has been attributed to many experts on wealth, and I have chosen to resurrect it in response to a recent contribution by Robert Pear of the *New York Times*.[1] Mr. Pear cites a recent study in *Health Affairs* by Laugesen and Glied[2] comparing fees paid to primary care and orthopedic physicians in the United States to those paid to physicians in countries around the world. He reports that higher fees and greater physician income explain the increased spending on physicians in the United States and that cutting fees to physicians may be a way to rein in US health care costs. Put more simply, US health care costs are out of control because doctors, as service providers in a service industry, represent too much of overall health care spending. Interestingly, when comparing public payers in the United States (Medicare, Medicaid) to primary care providers in Canada, the fees are nearly identical. US physicians only earn more in fees when private payers are added to the mix. It is not immediately clear that the actions of private payers are public policy matters, as the government only has jurisdiction over public fees (which are already matched to other countries).

Laugesen and Glied also compare fees paid to primary care physicians for office visits and orthopedic surgeons for hip replacements. The primary care comparisons, which the authors admit are apples and oranges, strike me as the more interesting of the two, as primary care is a service that generally doesn't offer a product (like a new hip) but is connected to the indirect costs required to provide the service (rent, utilities, supplies, etc.). As such, it is not terribly shocking that the provider represents the most expensive component of the service. Furthermore, since time is very expensive in America given the huge opportunity cost of becoming a physician, that more is paid for US physician time than for physicians elsewhere is also not surprising. Interestingly enough, "In general, physician visits in the United States are somewhat longer than in most other countries."[3] The authors go on to eliminate mitigating factors such as higher practice expenses or tuition costs as possible explanations for why physician fees are higher in the United States. How they are able to ascertain practice costs is impressive given the relatively immature state of cost accounting in the US health care system.[3]

While reading this study, I was reminded of another *Health Affairs* article, published the month prior by Morra et al.,[4] reporting that US physicians spend nearly four times more than Canadian physicians on interactions with payers. While the data between the two publications have been confirmed to be incomparable, it is most striking that US physicians, who are paid slightly higher fees, have to pay $80,000 more per physician per year to the payment system just to receive those fees! Something is amiss.

Attempting to reconcile these two pieces of information—that US physicians are paid more but that they also pay onerous administrative costs to run their practices—makes clear some ugly truths about American health care. Simply put, medicine is not a free market and does not operate as one. While some elements of the health care system interact with the marketplace, such as the cost of physician salaries, the underlying revenue and cost structures of health care delivery systems are quite arcane. Market norms, where costs and competition matter to price, have given way to centrally planned systems, where prices are the result of consensus by wise elders (see the RUC). This results in the strange and seemingly disparate findings in the above articles.

Reducing the argument to its simplest parts: Is health care too expensive in the United States? Yes. Why? Because the quality is awful as far as we can tell. Do physicians make too much in the United States? As a group, they probably don't—especially when compared to similarly educated people in other industries. As individuals or sub-groups, they do, as orthopedists earn three to four times more than general medicine physicians based on the results of the centrally planned system. Does the solution to the first problem of health care quality involve changing the way doctors are paid? I would say yes, although along a different dimension than either article proposes. Based on the Morra article, one could conclude that the United States could save $80,000 per physician if administrative costs, which are of no benefit to patient or physician, were eliminated. Based on Laugesen and Glied, fees to doctors should be cut to make them commensurate with

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Are physicians paid too much or too little? The answer is probably both—paid too much for things like ensuring certain phrases appear in a visit note and paid too little for actual patient care and care coordination.

What is Surgical Co-management?

Michele Fang, MD

Dr. Fang is clinical assistant professor at the University of Iowa.

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. Also, the co-management model is less likely to have a large effect on procedures, such as hip replacements, which have a very low rate of surgical and medical complications and are already driven by standardized order sets to provide consistent assessment and intervention during the postoperative period.

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.

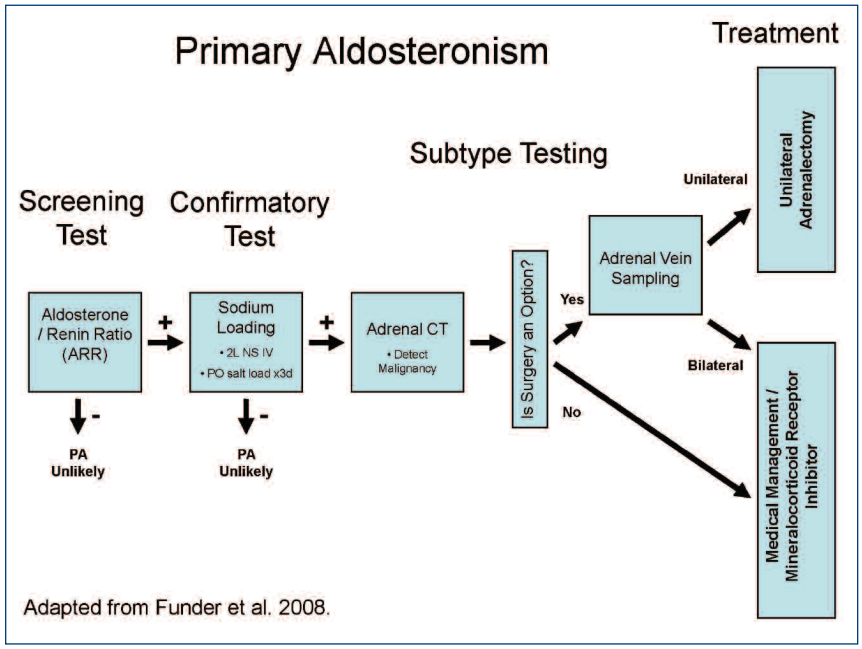
Seeing is Believing—Just not in Primary Aldosteronism

J. Carl Pallais, MD, MPH

Chalk Talk is edited by Douglas Wright, MD, PhD. Drs. Pallais and Wright are faculty in the Inpatient Clinician Educator Service of the Department of Medicine at Massachusetts General Hospital in Boston, MA.

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, amilioride, 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.

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"Be Still My Heart"

Shenita Spencer, MD, and Aaron Miller, MD (presenters); Matt Blackwell, MD (discussant, in italics)

Drs. Spencer and Miller are internal medicine residents at Carolinas Medical Center, and Dr. Blackwell is an associate program director at Carolinas Medical Center in Charlotte, NC.

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 anterolateral 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 catherization laboratory for suspected acute myocardial infarction. Coronary angiography reveals no intraluminary 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 rickettsial disease and rare causes of acute cardiomyopathy, such as Chagas disease, dengue, or yellow fever. In addition, one should inquire about illicit drug use and confirm with a urine drug screen to exclude a cocaine-induced cardiomyopathy.

On physical exam, signs of fluid overload or an S3 gallop could indicate impaired ventricular function. Furthermore, if right or left ventricular dilation is severe, cardiac auscultation may reveal a murmur of mitral or tricuspid insufficiency. Finally, a cardiac friction rub would indicate concomitant pericardial involvement. A thorough skin exam is important, looking for rash, nodules, or signs of peripheral embolic phenomena.

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 note 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/uL with 98% segmented neutrophils, hemoglobin 12.9 g/dL, and platelets 218 k/uL. 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.

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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 103º 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 spondyloarthropathy 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

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Medicine in the Southwest: A Snapshot

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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 transplants—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 laxest 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 sky-rocketing. 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. SGIM

NEW PERSPECTIVES: PART II

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those paid to doctors in other countries, with the underlying assumption that lower doctor fees will result in lower health costs. This is a conclusion that I question greatly.

So which is it? Are physicians paid too much or too little? The answer is probably both—paid too much for things like ensuring certain phrases appear in a visit note and paid too little for actual patient care and care coordination. Even more importantly, how much does top line revenue matter if a significant portion of that revenue goes right back to the payer as "interaction costs" that have no value to our patients? So it seems that how much physicians are paid is not the problem. Our payment system, made twisty and complex over years of manipulation, central planning, failed fraud prevention, and sheer bureaucratic creep, is the problem. It stands in the way of quality care and practice innovation. It is bankrupting the system and the country, and it is time for change.

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blood or tissue are fraught with technological difficulties, so such tests are not routinely recommended. Doses are usually in the range of 30 to 200 mg daily (higher in cases of Parkinson's disease); absorption may be improved if taken with a fatty meal.

Phytosterols are often ignored in the United States but have retained a high popularity in Europe. Plant sterols and stanols are typically taken orally 15 to 30 minutes before meals. The structural similarity to animal sterols (i.e. cholesterol) allows these compounds to saturate the intestinal receptors, competitively preventing absorption of animal cholesterol from meals. The overall effect of such a phytosterol regimen is a decrease in total and LDL cholesterol levels. While capsules are preferred in the United States, there are many foods and beverages available that incorporate plant-derived sterols and stanols. Several brands of margarine that are rich in these compounds are available but must be taken regularly. Typical dosing is 0.8 grams of phytosterols taken twice daily prior to meals.

There are numerous reliable sources of additional information for busy clinicians. Most mobile purveyors of conventional medical information have added sections that deal with over-the-counter botanicals and supplements. In addition, there are several dedicated online sites that are excellent sources of such information. Natural Medicine Comprehensive Database (http://naturaldatabase. therapeuticresearch.com) and Natural Standard (www.naturalstandard.com) are two such resources (subscriptions required). The information is clear, highly referenced, and frequently updated. In addition, drug interactions are included where applicable, and even hypothetical interactions are presented. Finally, a graded effectiveness level is included on both sites.

Recommendations of high-quality brands of a dietary supplement or botanical are complicated by the fact that these agents are classified as "dietary supplements" and are thus not regulated like pharmaceuticals. There are a few methods available to the clinician to identify high-quality products or to verify the correct chemical and amount. One resource is Consumerlab.com (www.consumerlab.com), which assays select products for accurate amounts and the presence of contaminants. Access to their website is by subscription. Some manufacturers use independent assay labs, such as the United States Pharmacopeia (USP) or the National Sanitation Foundation (NSF). These organizations certify products for purity and accurate tablet ingredient concentrations and then allow the manufacturer to display the "USP" or "NSF" endorsement on the product label. Lists of certified products are available online.

The use of dietary supplements and botanicals is increasing to the point that the internist can no longer ignore it. Some focused review will allow the clinician to gain enough familiarity with these compounds to engage in meaningful conversation with patients and provide more reliable counsel. SGIM

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 in-

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ANNUAL MEETING UPDATE

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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 internists 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! *SGIM*

MORNING REPORT

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rash, unexplained fever, leukocytosis, polyarthralgia, lymphadenopathy, elevated liver function tests, and antecedent pharyngitis.

- Adult Still's disease is characterized by a marked elevation in serum ferritin level and usually responds to high-dose oral corticosteroids.
- Acute myocarditis is a rare complication of adult Still's disease.

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PRESIDENT'S COLUMN

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careers in primary care. Also, various new organizational approaches to health care hold some hope of re-balancing payment for primary care relative to other care, which also may mitigate the disincentives for entering primary care. SGIM and its membership, outside and inside the government, were deeply involved in including these favorable provisions in the ACA, and we will do whatever we can to help preserve and implement this important legislation.

Another way SGIM is trying to address the poor circumstances for primary care is by the creation of the National Commission on Physician Payment Reform. The Commission will address the central role that primary care should have in situations like Jane's—physicians' dual responsibilities to their patients, who deserve the best care possible, and to society, which deserves careful stewardship of its resources. The Commission will start very soon and is to be led by two very prominent physician leaders in American health care, with members representing primary care, subspecialties, and other societal stakeholders. The Commission 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 received 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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clude 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

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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.[1] 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.[2]

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.

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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.

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