

THE Journal

OF THE ARKANSAS MEDICAL SOCIETY

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The Story Is King



H. SCOTT SMITH, JD
DIRECTOR OF GOVERNMENTAL AFFAIRS

I love all 14 of the Pixar films and have just starting reading a book about Pixar called "Creativity, Inc." written by Ed Catmull, Pixar President and co-founder. He has some great stories to tell. Early on, he states that a key component to Pixar's continuing success is that the "Story" ... and NOT animation, is "King."

Having worked in legislative advocacy since January of 1996 and through 14 regular state legislative sessions (along with a VERY interesting fiscal session this year), I can unequivocally say that for successful advocacy, "Story is King" as well.

Our legislative "issues" are born out of the "stories" that we hear from you. We try to legislatively address the stories that will have the biggest positive impact on the physicians of Arkansas and their patients. But without REAL, compelling stories that impact REAL physicians and REAL patients, it is very difficult to find the needed legislative support leading to a good legislative solution.

Leading up to the start of each session (which is now), we search for those stories but we need your help. If you have a story to tell that you would like AMS to consider for a legislative remedy, contact us! We are working on next year's legislative issues and their stories now. You all have some of the most compelling stories to be told as you take care of the sick and injured.

Again, please contact us and let us hear those stories. There's a good chance that if you're experiencing a problem, you are probably NOT alone, but we need you to step forward. Let us know.

A few of the issues and stories we have been working on since the end of this year's fiscal session in April are: telemedicine, prescription drug abuse, Physician Orders for Life-Sustaining Treatment (POLST) and the Medicaid Private Option (THE issue of this past fiscal session). The Private Option appears to be headed for "Perennial Legislative Battle" status.

To that end, I just returned to the office after an Executive Committee meeting of the large coalition of organizations supporting the Private Option. We're already working on messaging and the anticipated "vote count" for next session's Private Option vote in light of primary election results, and potential general election outcomes. Wise decisions now will hopefully make the path smoother during session.

Other recent discussions with legislators have involved two significant scope of practice issues along with an incredibly important insurance issue we anticipate needing to address next session. While

your issues and stories are here year-round, the window of time to address these issues is limited because sessions come and go rather quickly.

As soon as a regular session finishes, generally in early April of the odd-numbered years, we move immediately into "election season" which lasts 18 months until the next general election the following November. In fiscal session years, like this even-numbered year, the election "season" takes a timeout for the couple of months during the fiscal session and resumes in early April for the 4 to 6 week sprint to the primary elections, the third week in May.

Generally, the legislative "pre-season" really kicks in 6 months or so before the legislature actually starts in January of those odd-numbered years. So, we are currently heading down the backstretch of the election season, and concurrently starting the 2015 legislative pre-season.

The remainder of this year brings intensified identification of issues and the stories needing legislative solutions. Soon, bill drafting will start to kick-in and visits with legislators regarding some of the stories will happen on a more frequent basis. However, until the November election, we will not be able to ascertain precisely who will even be in the legislature, much less, who will be on key committees.

So, it is important to remember that continuing legislative success is only possible if we are successful in helping our friends stay in office. Hardly a day goes by that we don't either get an invitation to a candidate fundraiser or receive a call asking for financial assistance. We keep busy meeting with candidates, attending fundraisers and making campaign contributions from our political action committee, ArkMed-PAC.

We need your participation in ArkMed-PAC, just like we need your stories. Campaigns are expensive, and we want to help as many of our friends as much as we can. We can help more candidates as more AMS members contribute to the PAC, so please be on the lookout for our ArkMed-PAC membership mailing.

Physician support has already helped a number of candidates, and we are fortunate to have some terrific friends in the Arkansas legislature and that strong base of legislative support has been amazing in fighting for the physicians of Arkansas and their patients. With your continuing assistance during the election season and in identifying issues and sharing stories in the legislative pre-season, hopefully, we will expand that base and have another successful legislative session. AMS

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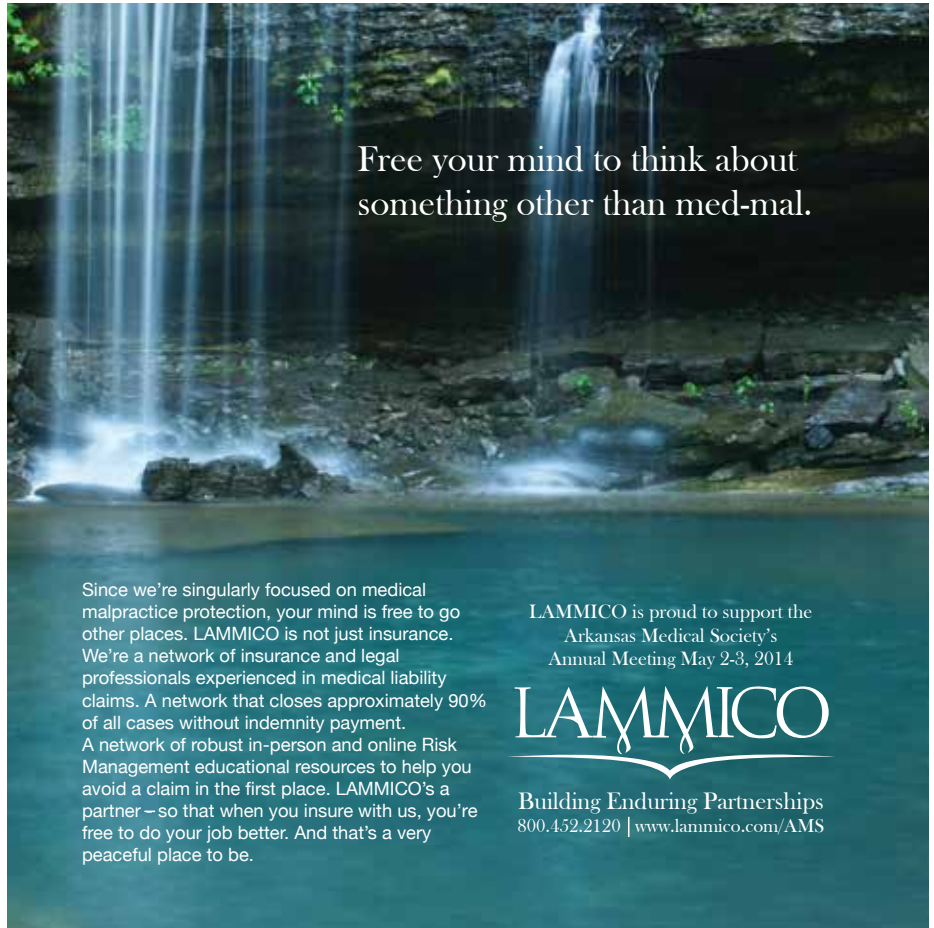
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LAURA SISTERHEN, MD

A Time of Renewal

This past May I had the privilege of attending the UAMS College of Medicine Honors Convocation.

Honors Convocation is the annual celebration where graduating medical students are recognized for their distinguished academic achievements. I watched smiling students approach the stage, turn to the audience, and pause for the ceremonial hooding by honored faculty. It is a proud moment for family, friends, and faculty. For me, one of the most poignant moments of the ceremony is the renewal of the Hippocratic Oath by faculty. According to the literature, the vast majority of American medical students pledge an oath upon graduating, and UAMS students are no exception. It is a common bond that we share prior to dispersing into specialized residencies.

I believe in this era of change, uncertainty, increasing pressure to contain cost and ensure quality and accessibility, we need something to guide us. We need a values statement, something that stands the test of time, to help us navigate the complexities of medicine today. In medicine, we need a middle C, a North Star, a constant in rapidly changing times. For many, this can be the oath we took upon graduation.

Although the modernized version varies, the premise of the oath is a promise to practice medicine honestly and to uphold a number of professional ethical standards. The Hippocratic Oath, written in Ionic Greek, dates back to the late 5th century BC. The oath begins with a covenant with the deity Apollo and goes on to address issues such as limits on means and ends, justice, chastity, and confidentiality. The oath has been modified many times and one of the most significant revisions, called the Declaration of Geneva, was written in 1948 by the World Medical Association.

When I graduated from medical school at the University of Tennessee Health Sciences Center in Memphis, I was given a document signed by the Dean that includes the oath that I took when I graduated. It is framed in my office and is a reminder of the commitment I made to myself and to my patients.

I solemnly pledge to consecrate my life to the service of humanity.

I will give respect and gratitude to my deserving teachers.

I will practice medicine with conscience and dignity.

The health and life of my patient will be my first consideration.

I will hold in confidence all that my patient confides in me.

I will maintain the honor and the noble traditions of the medical profession.

My colleagues will be as my family. I will not permit considerations of race, religion, nationality, party politics, or social standing to intervene between my duty and my patient. I will maintain the utmost respect for human life.

Even under threat I will not use my knowledge contrary to the laws of humanity. These promises I make freely and upon my honor.

As a clinical educator, I teach pediatric medicine to students and residents. Some of the things I teach will be outdated in 5-10 years. My hope is that some of what I teach is timeless. Respect for persons as outlined in the Hippocratic Oath will always be the right thing to do. When I'm not certain what course to take or decision to make, is it helpful to apply the wisdom of physicians who have gone before us.

This is the overarching message I heard from College of Medicine leaders at the Honors Convocation. We are in a period of rapid change in healthcare delivery as well as health professions educational reform. This change is driven by the public's request to improve the patients' experience of care, improve the health of individuals and populations, and decrease the per capita cost of healthcare. There is so much that is out of our control as individual physicians, but we can focus on what we can do. We can renew our commitment to the oath we took when we graduated. **AMS**

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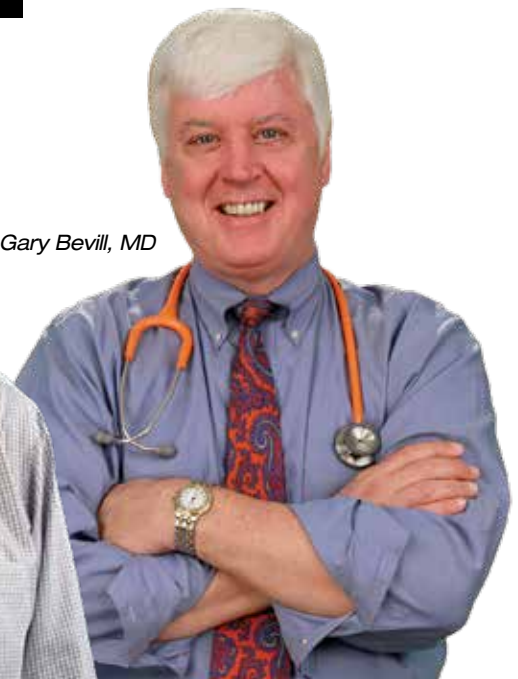
El Dorado Clinic Takes a Colorful Approach to Medicine



Pete Atkinson, MHA



James Sheppard, MD



Gary Bevill, MD

by CASEY L. PENN



Five teams – purple, orange, green, blue, and red – prepare to take on the day’s challenges. The atmosphere is upbeat and fans, while loyal to their own team, root for all sides to win in this crucial game of improving health and, ultimately, saving lives.

These physician-led teams are part of SAMA (South Arkansas Medical Associates) HealthCare Services, a one-stop health care facility that is in the process

of making a unique and deliberate transition from reactive to proactive health care, from the status quo to a patient-centered medical home.

SAMA is the recipient of a grant from the Centers for Medicare and Medicaid as part of its Comprehensive Primary Care (CPC) initiative. As *The Journal* reported last year, 69 Arkansas clinics are participating in this multi-payer initiative (a result of the Affordable Care Act) that fosters collaboration between public and private health care payers and encourages the patient-centered medical home (PCMH) concept. Participating clinics are given a list of milestones to work toward as well as some incentives to aid them in their efforts (<http://innovation.cms.gov/initiatives/comprehensive-primary-care-initiative/>).

Pioneering a new approach is no anomaly for this El Dorado clinic. “We kind of like to do things first,” confided LPN Nancy New of the changes that have put this small clinic on the nation’s radar as a model of a successful PCMH. Her own career a picture of the clinic’s constantly evolving methods, New had worked as a nurse in the clinic since it opened 15 years ago, but as the most computer-savvy person in the clinic, she has also long been the go-to person to help navigate new technology, such as electronic medical records (EMR). That’s why New was recently reassigned as the clinic’s Health Informatics Coordinator, a role that utilizes her skills and experience. Gary Bevill, MD, one of five SAMA physicians, noted that having a devoted IT person like New has greatly helped the clinic to navigate CPC, EMR and other improvements.

The CPC opportunity came along at a time when SAMA was already well on its way to being a PCMH, according to the clinic's administrator, Pete Atkinson, MHA. "In health care, not everybody fits into a square box," said Atkinson, who enrolled the clinic in CPC in August 2012. He and the clinic's physicians saw the opportunity to build on what they had already done, but to do it in their own way. "We chose CPC over a canned version of PCMH because with CPC, we were given eight goals or milestones [of a PCMH] and then given the freedom to reach those goals as we see fit. Working in El Dorado is nothing like working somewhere else. We know our environment, and we know our market."

Even in a rural market, SAMA is succeeding in reaching CPC milestones and is exceeding even its own expectations. "It's been quite a transformation," said Atkinson, who described changes to many aspects of the practice, especially patient care and personnel. "We've moved from reactive mode to proactive mode, for the betterment of our patients."

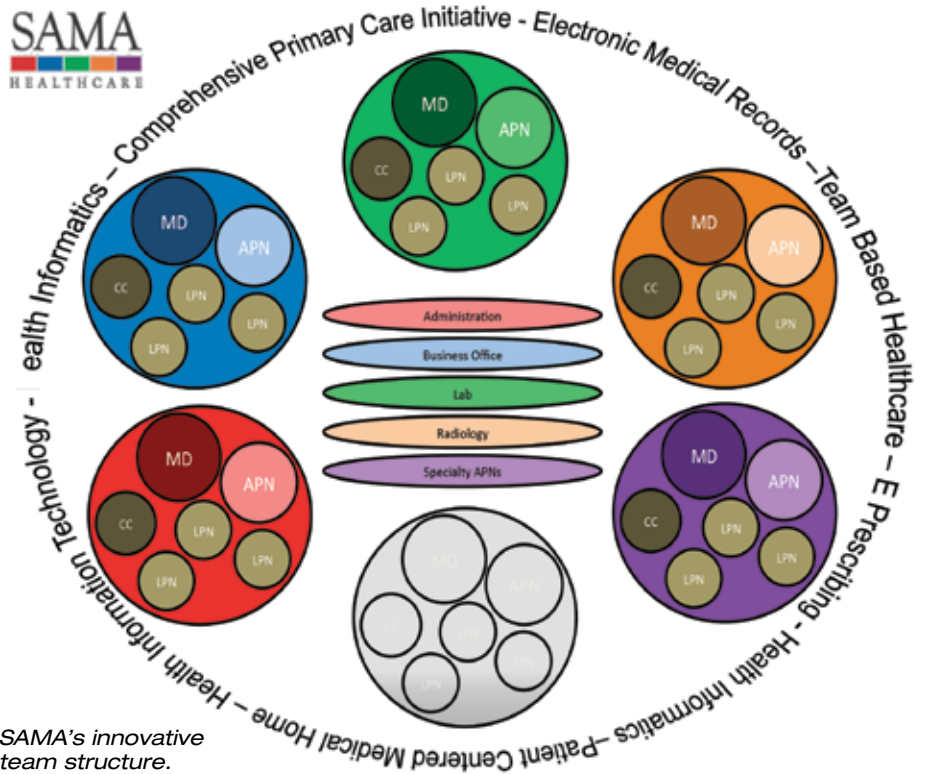
Previously, SAMA physicians and staff worked under one roof but independently; duties were largely disconnected. "They shared the EMR, common areas, and business office staff, but each physician had two nurses and each Advanced Practice Nurse (APN) had one," said Atkinson, who noted that there was certainly no sharing of nurses.

While the physicians clearly wanted to make sure patients had mammograms, colonoscopies and other preventive services, staff and resources were limited. "In reality, when physicians are seeing 25 to 30 patients a day and handling acute situations, a lot of preventative services fall to the back burner – to a reactive position," Atkinson explained.

Being reactive is old news – in the past. Today, resources and preventive services stand front and center as part of SAMA's new model of care, a blatant, color-coded, team-based approach that appears to be a fan favorite.

Logistics of Change

Atkinson described the SAMA health care model – initially sketched out on the back of a pizza box at the end of the first CPC meeting – as a combination of good medical care he had witnessed over time. "I had seen a physician and a nurse practitioner working very well together, and I liked it," he said. With that cooperation in mind, he led the clinic to put together five (so far) self-contained teams. Each includes one physician (team leader), one APN, three nurses and one care coordinator (an LPN by training).



SAMA's innovative team structure.

Now, a team's physician and APN both see acute visits and follow-ups. Depending on the circumstances of the visit, they may even pass patients back and forth, each concentrating on a different area of need.

Another change? Within each team, LPNs rotate duties weekly (within their team) to equip them for each area. "One will take the phones and the other two will 'pull' patients for the providers," said Atkinson. "Call is taken by each team, and we no longer send all same-day acute visits to on-call provider(s). The idea is that you're only as strong as your weakest link. Each team's schedule has a mix of follow-up and acute visits each day which allows patients to see their own team most of the time and improves continuity of care."

Enter the answer to preventative care and an important component of a PCMH, the **care coordinator**. Each team's care coordinator reviews patients' charts before appointments, manages and checks on referrals, coordinates transition of care from the hospital (inpatient and ER), manages and schedules preventative services and more. "Before CPC, we didn't have the resources to keep up with preventative care services like we wanted," said Atkinson. "Now, the care coordinators provide things we did not do in the past or things we did not do well. Now, the care coordinator takes care of preventative care, and the physician cares for patients."

To help develop preventive and long-term, chronic care plans for patients at highest risk, care coordina-

tors now assign a risk level (*risk stratification*, in CPC terms) to each patient. This helps the patient and the clinic over time. To assign a level, New explained, "They take into account a number of defined conditions (i.e., diabetes, hypertension, etc.), if the condition is controlled or not, the number of medications the patient takes and patient hospitalization in the last year."

"The concept of going to the doctor when you feel well has been foreign to all of us, but this clinic is changing that," added Atkinson. "Our care coordinators are constantly proactive. You may call it pestering, [but if you need preventative services,] we're going to call you, and call you again."

Increased staff and team members make possible another huge element of SAMA's new approach, which is patient satisfaction. "I'd been getting complaints that people weren't getting to see *their* doctor – in other words, continuity of care was already an issue as it is in a lot of practices," said Atkinson, recalling the days before CPC. "Patients come in and see whoever's available. But patients like to see *their* doctor. This model, outside of but still in line with CPC, was our attempt to fix that issue ... and it's working."

Under the old system, clinic physicians shared being on call. On their on-call days, they saw nothing but same-day acute visits for all the doctors in the clinic. APNs, too, saw mostly acute visits, too, and the patients they saw were assigned to the various doctors in the building.



Initial Team Care Coordinators (left to right): Yolanda Moody, LPN; Amelia Dolden, LPN; Candy Cates, LPN and Britni Jones, LPN.

Now, by definition, teams work together. “If a patient is sick and they call in when their physician’s on vacation, they’re still going to talk to a member of their own team. In that scenario they’re usually going to see that team’s nurse practitioner,” said Atkinson. “The patient feels comfortable dealing with the same team all the time, and the team gets to know the patient better. This is good from an efficiency and scheduling standpoint. We know whether that’s going to be a 15-minute visit or a 30-minute visit, where before, we didn’t know the patients well enough.”

“With the team getting to know the patients very well, they can be aware,” he explained, using the example of a patient who is a recent widow. “If Mrs. Smith calls in, we know she’s coming in for a cough, but she also has depression issues, so we need to allow time for that.”

Inevitable Growing Pains

Since enrolling in CPC 18 months ago, SAMA has grown from 37 to 57 employees. The first year brought much change and with it, growing pains. “It took time – about 14 months to get all the teams in place. We had a lot of turnover initially – people don’t like change,” said Atkinson, contrasting that with the team in place today. “This is what the staff in place now is used to. That has been nice.”

Challenges have also included additional staff training, some recruiting for APNs, and money, of course. CPC provided much help in the start-up, and that help came with clear expectations. “CPC

gave us the initial investment by way of a per-patient-per-month payment – paid quarterly,” explained Atkinson, stressing that every dollar from CPC was to be used as part of the clinic’s transformation into a PCMH. “It’s roughly \$8 per member per month for the healthiest patients and \$40 per patient per month for the sickest patients.”

It’s worth noting that, in addition to CPC funds, the new approach gained additional funding – at least on the front end – from within. SAMA physicians stood behind the plan 100%, going so far as to contribute their own money to initial start-up costs while trusting the program to pay off over time.

Elaine Butler, Nurse Manager, has been part of the clinic’s efforts to bring a five-year plan to fruition in just 18 months. “It’s been a wild and crazy ride,” said Butler. “We’ve gone from 12 nurses to 22 nurses (four from the original team).”

Making fast strides comes down to focus, Atkinson indicated. “If you focus on your core competencies, the rest will follow. As we got better at what we were doing, people [employees and patients] started coming to us. We have done very little recruiting as a result.”

A Win-Win for All

Staff and patient response, exhibited in a number of ways, has been favorable overall. Since being accepted into CPC (in August 2012), SAMA has billed 2300 new patient visits – 465 just this year.

“It’s kind of fun,” said Atkinson, who pulled the colors idea from his experience being a soccer coach. He uses social media, too, to perpetuate the concept. “When you go back and look at Facebook posts from when we’ve done team vs. team contests, patients like to root for their team. Also, I can post something about Dr. Bevill, without saying anything about the teams, and somebody will inevitably comment ‘Go Team Orange!’”

Team loyalty is an added benefit, but patient acceptance of the new approach is clearly about much more than shirt colors and cheerleading, as pointed out by SAMA’s James Sheppard, MD (“Go Team Blue!”). “Patients like it because they’re getting more attention,” he elaborated. “Their preventative needs are being discussed with them. They like the perception – a true perception – that they’re taken care of a little better.”

Increased support from this team approach allows the physicians to see more patients while still improving quality of care. “Before working with an APN, I may have tried to see about 25-30 patients in a day – and felt some guilt about spending less time with each than I might want,” said Dr. Bevill, who added that now, on a regular day, he and his APN together will see closer to 45 patients and are able to give them better care and more follow up.”

As staff has increased, SAMA increased in-house services – another perk for patients – including an on-site lab and radiology and specialty APNs. “Patients are not having to visit multiple locations for care,” explained Atkinson. “It’s great for physicians, too, as results are timely and go directly into our EMR. This allows us to treat patients much faster than if we were sending everything out.

“We have a Pediatric APN and an Adult APN who is a Certified Diabetes Educator (CDE). The Pediatric APN helps with walk-ins and does a majority of the yearly physicals for Medicaid. Our CDE spends a lot of her time doing annual diabetic education.”

Dr. Bevill leans heavily on the diabetes-certified APN in his team’s approach to patients. “As a physician, if I’m seeing a diabetic with a cold, I’ve felt guilty of not spending the time I wanted to spend to look at preventative care and maintenance issues,” said the doctor, echoing Atkinson’s earlier sentiments about time constraints. “When it’s just the physician, there just isn’t time. In this model, it’s easier to take the 10 minutes to deal with the cold, and then let the nurse practitioner spend 45 minutes – whatever is needed – to focus on patient education, etc.”

SAMA has not noticed a backlash from patients about seeing an APN instead of their physician, either, Atkinson pointed out, relaying a helpful, *peaceful* process the clinic employs when it comes to patient interactions. “Our approach has been, when we introduce the APN, the doctor has been in the room, and vice versa,” he said. “Patients see the mutual respect between the two and learn to feel a kinship with their doctor and his / her team. This approach empowers every member of the team to make things work, so the physicians aren’t bearing the full brunt of patient care.”

Dr. Bevill finds the new approach energizing. Having practiced medicine since 1985 – 14 of it spent in solo practice – he likes the change. “Health care is changing,” he admitted. “I find that I want to see that through. There are challenges, but it’s encouraging to focus on my patients. It’s been nice to reach this stage of my practice and find that going to work is not a chore.”

Sustaining a Working Model

Change will continue to come to health care in this country, and for the most part, SAMA’s team is ready. Future plans include recruiting one more physician in 2016, using less paper and increas-

ing attention on medication management and risk stratification.

Definitive results – such as specifics on shared savings – are, for the most part, still a little ways out. Yet, some improvements are apparent without detailed statistics. “Part of CPCI is seven-day access. A natural outcome of that is reduced ER visits,” said Atkinson. “We’re seeing better care, and we’ve survived financially as things shift from quantity to quality. Our staff is trained and can adapt to change as it comes.”

CPC still has another two and a half years as a pilot program, but it could well be rolled out nationally. Regardless, SAMA plans to stick with this approach that’s working for them and their patients.

“We are not working in a vacuum with CPC,” concedes Atkinson, who realizes that other state and national initiatives exist that can work for or against a small, independent clinic.

“We’re not backed by a large hospital system or university,” he said. “Like any small business, we would have to make some changes because of the loss of revenue; however, our approach from the

beginning was to use this money to build a sustainable model. With the additional providers we have put in place, we believe that we have done that in the past 18 months. Having our own ancillary services on site helps financially, but we have also found that there is no better marketing than just doing a great job. The team model seems to be ‘selling’ itself because of quality of care as well as the accessibility of our providers.” **AMS**

ADDITIONAL READING:

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Quality colonoscopy and effective screening for colorectal cancer

BY DAVID A. NELSEN JR., MD, MS

Your primary care physician uses an electronic medical record to track your preventive care needs. You have an office visit soon after your 50th birthday and there's a prompt to screen for colorectal cancer (CRC). You expected this was coming; you recently read an article in a popular magazine about screening colonoscopy. So many famous people have died from CRC—Vince Lombardi, Joel Siegel, Audrey Hepburn; why would you ignore this potentially preventable condition? CRC is preventable and treatable; however, treatment is most effective if CRC is discovered at a precancerous or early stage. Advanced CRC continues to be associated with poor survival.

Cancer prevention in general has benefited most from early detection. Cervical cancer, breast cancer, and now CRC have all capitulated to early diagnosis and treatment. Advances in early detection of cervical cancer and breast cancer are well-known and include pap testing, liquid-based cytology, human papilloma virus testing, digital mammography and certification programs. Each advance has improved diagnosis accuracy. Not surprisingly, lung cancer is now on our screening radar but that is another topic for another day.

Recent studies suggest that, “all colonoscopies are not created equally.” Certain endoscopists and endoscopy centers perform higher quality colonoscopy exams than others. A 2010 study of gastroenterologists documented significant inter-provider variability in the rate of adenoma detection, cecal intubation and scope withdrawal time.¹ Additionally, the short-term risk of development of CRC after a completed colonoscopy has been shown to be related to performing clinician attributes.^{2,3} Board-certified gastroenterologists and academic medical centers have the lowest rates of colorectal cancer following a negative colonoscopy.

For these reasons, current guidelines from the American College of Gastroenterology (ACG)⁴ recommend that quality colonoscopy is the preferred screening strategy for CRC. The guideline refers specifically to quality colonoscopy. This implies that there are technical performance issues relative to routine colonoscopy that are critical to the effectiveness of CRC screening.

Other CRC screening procedures have a strong evidence base of effectiveness when compared to no screening. These include fecal occult blood testing, flexible sigmoidoscopy, air contrast barium enema and “virtual

(computerized tomographic) colonoscopy.” Quality colonoscopy outperforms these modalities and is therefore the primary recommendation.

Given evidence that there is inter-provider variability regarding screening colonoscopy outcomes, one might suspect that the ACG would declare that all colonoscopy should be performed by a board-certified gastroenterologist. That was not the case in the 2008 guideline. The ACG did state that screening colonoscopy should be carried out by “appropriately trained and skilled examiners, who are dedicated to consistent performance of high-quality examinations ... [who] employ programmatic measurements to optimize the outcomes through continuous quality improvement processes.” The 2008 ACG guideline went further to specify key performance measures that constitute the definition of quality colonoscopy:

- A “split bowel prep” should be used; i.e., the prep should be given in separate doses and time frames prior to the procedure.
- The report includes verbal and photographic documentation of cecal intubation.
- The report includes documentation of adenoma location and size.
- Adenoma detection rate should be monitored.

- Scope withdrawal time should be at least six minutes for intact colons where no procedures or biopsies are performed.
- Polyps should be removed by effective means; polyps greater than 5mm size should be removed by snare instead of forceps.
- Large sessile lesions that require piecemeal resection should be followed closely.
- Recommended screening and surveillance intervals should be followed for patients who undergo complete examinations with adequate preparation (Table 1).

A good quality prep is perhaps the most important attribute of a quality colonoscopy. The ordering physician should review the prep with the patient and strongly encourage compliance. The referring and performing physicians should have clear agreement as to the prep that is used and the required patient education. If a poor prep restricts optimal visualization of the colonic mucosa, then the procedure should be repeated within a year. If prep failure was due to patient factors such as severe or chronic constipation, then a “two-day” prep or additional laxatives (e.g. bisacodyl) may be utilized.

Adenomas are commonly detected during colonoscopy. Studies demonstrate that 25 percent of men and 15 percent of women of average risk who are over age 50 will have one or more adenomas detected on routine screening colonoscopy. Endoscopists who experience lower detection rates should undertake quality improvement activities to improve their adenoma detection rate. Scope withdrawal time is perhaps the strongest correlate with adenoma detection. Scope withdrawal time should average six minutes in patients who have no adenomas detected.

No adenomatous polyps	10 years
1-2 small <10mm tubular adenomas	5-10 years
3-10 tubular adenomas	3 years
>10 tubular adenomas	<3 years
1+ villous adenoma(s)	3 years
Adenoma with high grade dysplasia	3 years
Serrated sessile polyp <10mm	5 years
Serrated sessile polyp >10mm	3 years
Serrated polyposis syndrome	1 year

The performing physician should generate a report to the referring physician that includes key components and performance metrics for that examination. This includes findings, adequacy of bowel prep, complications, pathology result and recommendations for follow-up. The ordering physician cannot consider the prevention episode to be closed until the report has been received. Follow-up recommendations should be based on the findings and the ongoing CRC risk. Negative exams with a good prep may be repeated in 10 years. Guidelines-based surveillance intervals are presented in Table 1. Guidelines apply primarily to adenomatous polyps, which are considered to be pre-cancerous. The finding of a few isolated hyperplastic polyps is not generally considered to be significant; however, patients who are diagnosed with hyperplastic polyposis syndrome are at increased risk. These patients may have 20 or more hyperplastic polyps in the ascending colon. These polyps may be associated with microsatellite instability as well as inherited cancer syndromes.⁵

CRC is a serious medical problem that has grave consequences unless detected early. CRC screening research has demonstrated significant reduction in mortality and morbidity. Colonoscopy is a proven modality to detect and manage precancerous colorectal lesions. Successful CRC screening starts with formal preventive care approaches in the primary care office and is more likely to occur when the primary care physician educates and recommends the exam. Successful screening requires a good working relationship between the patient, the referring physician and the performing physician. The performing physician should use quality improvement methodologies to monitor performance. Patient follow-up should be managed according to established guidelines. Adherence to these measures will ensure a successful outcome for all parties. ▲

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Pulmonary arterial hypertension: Part 1: A review for an internist

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Introduction:

Pulmonary hypertension is a devastating chronic disease with a poor long-term prognosis and increased morbidity and mortality. A huge variation in the disease course and prognosis may occur based on the underlying etiology.¹ The World Health Organization (WHO) has classified pulmonary hypertension into five groups (Table 1) based on their underlying mechanisms, clinical context and histopathology.^{2,3} Disorders in WHO group 1 are characterized as Pulmonary Arterial Hypertension or PAH, whereas disorders in groups 2-5 are referred to as simply Pulmonary Hypertension (PH). Current hemodynamic definition for PH based on RHC is a mean pulmonary artery pressure (mPAP) greater than 25 mm Hg at rest. Based on the mean PAP, PH can be further classified into mild, moderate and severe with PAP between 25-35 mm of Hg, 35-45 mm of Hg and more than 45 mm of Hg respectively, although the overall severity is influenced by the degree of impairment in cardiac output late in the disease.

In this review, we will discuss the definition, clinical assessment, diagnostic evaluation and treatment modalities. Our emphasis has been placed on WHO Group 1 disorders or PAH.

Table 1: Updated clinical classification of Pulmonary Hypertension (Dana Point, 2008)⁴

<p>1. Pulmonary arterial hypertension (PAH)</p> <ul style="list-style-type: none"> 1.1. Idiopathic (IPAH) 1.2. Heritable <ul style="list-style-type: none"> 1.2.1. BMPR2 1.2.2. ALK1, endoglin (with or without hereditary hemorrhagic telangiectasia) 1.2.3. Unknown 1.3. Drug and toxin-induced 1.4. Associated with <ul style="list-style-type: none"> 1.4.1. Connective tissue diseases 1.4.2. HIV infection 1.4.3. Portal hypertension 1.4.4. Congenital heart disease 1.4.5. Schistosomiasis 1.4.6. Chronic hemolytic anemia 1.5. Persistent pulmonary hypertension of the newborn <p>1'. Pulmonary venoocclusive disease (PVOD) and/or pulmonary capillary hemangiomatosis</p> <p>2. Pulmonary hypertension with left heart disease</p> <ul style="list-style-type: none"> 2.1. Systolic dysfunction 2.2. Diastolic dysfunction 2.3. Valvular disease 	<p>3. Pulmonary hypertension owing to lung diseases and/or hypoxia</p> <ul style="list-style-type: none"> 3.1. Chronic obstructive pulmonary disease 3.2. Interstitial lung disease 3.3. Other pulmonary diseases with mixed obstruction and restriction pattern 3.4. Sleep disordered breathing 3.5. Alveolar hypoventilation disorders 3.6. Chronic exposure to high altitude 3.7. Developmental abnormalities <p>4. Chronic thromboembolic pulmonary hypertension (CTEPH)</p> <p>5. Pulmonary hypertension with unclear multifactorial mechanisms</p> <ul style="list-style-type: none"> 5.1. Hematologic disorders: myeloproliferative disorders, splenectomy 5.2. Systemic disorders: sarcoidosis, pulmonary Langerhans cell histiocytosis, lymphangioleiomyomatosis, neurofibromatosis, vasculitis 5.3. Metabolic disorders: glycogen storage disease, Gaucher disease, thyroid disorders 5.4. Others: tumoral obstruction, fibrosing mediastinitis, chronic renal failure on dialysis
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Epidemiology and Pathophysiological features of Pulmonary Arterial Hypertension:

The prevalence of PAH is estimated to be 15 per million with Idiopathic being the predominant type, based on recent evidence from French registry.⁵ During a 1-year period, 674 confirmed PAH patients were identified in France. Cases of idiopathic, familial, anorexigen, connective tissue diseases, congenital heart diseases, portal hypertension and HIV associated PAH accounted for 39.2, 3.9, 9.5, 15.3, 11.3, 10.4 and 6.2% respectively. Familial PAH has been reported in 6% -10% of PAH population⁶. The most common mutation found in 50% -90% of the

familial group is in bone morphogenetic protein receptor 2 (BMPR2) which is autosomal dominant⁷, but has incomplete penetrance. Most cases are diagnosed between the third and fifth decade with a distinct female predominance especially idiopathic, familial, anorexigen exposure and autoimmune diseases. PAH is 0.5% prevalent in patient with HIV infection. A systemic screening is warranted in these patients, especially in the era of HAART therapy. Independent of severity of underlying liver disease, 2%-6% of patients with portal hypertension develop pulmonary hypertension. Overall, it accounts for 7%-10% of overall PAH population.⁵

Significant progress has been made in understanding the pathogenesis of PAH. Mutations in BMPR2 perturb heterodimerization with its sister receptor BMPR1A to disrupt the ligand-binding function of the protein. Most IPAH patients have reduced BMPR2 protein expression in the pulmonary vasculature independent of a mutation in the gene. This loss of BMPR2 is associated with increased susceptibility of pulmonary artery endothelial cells (PAEC) to apoptosis and impaired responses to endothelial injury through canonical Wnt signaling mechanisms and abnormalities in β -catenin/PPAR (peroxisome proliferator-activated receptor)

gamma regulation of transcription.⁸⁻¹¹ Loss of BMPR2 protein also induces pulmonary artery smooth muscle cell (PASMC) proliferation and increased susceptibility to apoptosis as well. Since not all individuals that carry BMPR2 mutations develop PH, other factors, a so-called “second hit,” may be necessary.^{10,11} These factors include mutations of other members of the transforming growth factor receptor family such as ALK1 and endoglin, mutations of genes linked to apoptosis, decreased expression of potassium channels, increased production of cytokines and growth factors, inflammation, and overexpression of serotonin transporters on PA smooth muscle cells.¹² These changes ultimately lead to extensive vascular remodeling with progressive obliteration of the distal pulmonary arteries, increased pulmonary vascular resistance, and right heart failure.

An imbalance in the vasoconstrictors like nitric oxide (NO), endothelin-1, prostacyclin and thromboxane A2 have also been linked to the underlying pathology. Endothelin-1, being a potent vasoconstrictor, has direct influence on cardiac hemodynamics. The ET-1 binds to 2 receptor subtypes ETa and ETb with high affinity.⁸ ETa receptors are found in the smooth muscle while ETb receptors are found in both smooth muscle and endothelial cells. In addition to vasoconstriction and proliferation, ET-1 acts on fibroblasts to induce prolif-

eration and fibrosis. It also acts on the endothelium to cause proliferation, vasodilation via NO and PGI2 and vasoconstriction via thromboxane A2.

Nitric oxide is a potent endothelium-derived vasodilator.⁹ It induces vasodilation by acting on smooth muscle cells and inhibits proliferation by increasing production of cyclic guanosine monophosphate via activation of guanylate cyclase. The enzyme, phosphodiesterase type-5 catalyzes the conversion of cGMP to GMP and therapies targeted to inhibit this enzyme have been one of the treatment strategies.

The PGI2 pathway is activated when PGI2 stimulates the IP (inositol phosphate) receptor leading to increased cyclic adenosine monophosphate and resulting in vasodilatory and antiproliferative effects.¹⁰ PAH patients are found to have reduced levels of prostacyclin and reduced expression of PGI2 synthase in the lung.

Other vasoconstrictors like serotonin, adrenomedullin, vascular endothelial growth factor and vasointestinal peptide have also been implicated in the pathogenesis of PAH.

Clinical assessment

Clinical detection of pulmonary hypertension includes the presence of symptoms of right heart

failure including dyspnea, edema, abdominal distension, and chest pain or syncope episodes. Physical examination may reveal left parasternal lift, an accentuated pulmonary component of second heart sound, a pan systolic murmur of tricuspid regurgitation, a diastolic murmur of pulmonary insufficiency, and an RV third sound. Jugular vein distension, hepatomegaly, peripheral edema, ascites, and cool extremities characterize patients in a more advanced state. Lung sounds are usually normal. The examination may also provide clues as to the cause of PH and findings such as telangiectasia, digital ulceration, and sclerodactyly that are seen with scleroderma, while inspiratory crackles may point towards underlying interstitial lung disease. The stigmata of chronic liver disease such as spider angiomas, testicular atrophy, and palmar erythema should be evaluated on physical exam if portopulmonary hypertension is suspected.

Diagnostic Evaluation:

PAH (WHO Group 1) represents the type of PH in which the most important advances in the understanding and treatment have been achieved in the past decade. It is also the group in which PH is the ‘core’ of the clinical problems and may be treated by specific drug therapy. A diagnosis of idiopathic



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Table 2: Adapted from the executive summary of the World Symposium on Primary Pulmonary Hypertension in Evian, France, in 1998.

Functional classification of PAH:	
Class	Description
Class I	Pulmonary arterial hypertension without a resulting limitation of physical activity. Ordinary physical activity does not cause undue dyspnea or fatigue, chest pain, or near syncope.
Class II	Pulmonary arterial hypertension resulting in a slight limitation of physical activity. The patient is comfortable at rest, but ordinary physical activity causes undue dyspnea or fatigue, chest pain, or near-syncope.
Class III	Pulmonary arterial hypertension resulting in a marked limitation of physical activity. The patient is comfortable at rest, but less than ordinary activity causes undue dyspnea or fatigue, chest pain, or near-syncope.
Class IV	Pulmonary arterial hypertension resulting in an inability to carry out any physical activity without symptoms. The patient has signs of right heart failure. Dyspnea, fatigue, or both may be present even at rest, and discomfort is increased by any physical activity.

pulmonary hypertension is made when all the other etiologies are carefully excluded. Therapy that may be effective for PAH may be ineffective or potentially harmful for other forms of PH. Therefore, failure to make an accurate diagnosis of PAH could have adverse outcomes.

Once clinically suspected, a thorough physical examination and appropriate laboratory data should be performed. Laboratory evaluation should be directed toward identifying whether the patient has PAH (WHO group 1) or PH (WHO groups 2-5).

Pertinent tests including serology (ANA, HIV, Scl-70, RF, Hepatitis panel etc), Pulmonary function tests, Echocardiography, CT chest, V/Q scan to determine the underlying etiology should be measured during the initial evaluation for suspicion of PAH. Transthoracic echocardiography is frequently used as a screening test for PAH but there is limited data available with regards to its prognostic value. There has been a lot of debate about whether non invasive measurements like RVSP or PASP reflect the true nature and severity of the disease. When measuring pulmonary pressures by echocardiography, clinical context, the prevalence of pulmonary hypertension in the patient population and other echocardiographic parameters of the right ventricle should be taken into consideration to improve the overall diagnostic accuracy. Based on REVEAL registry¹³, ECHO measured parameters do not correlate with RHC measurements and hence RHC remains the gold standard for diagnosing PAH. ECHO is a useful tool in the initial evaluation and may be useful in follow up.¹⁴

Once the diagnosis is confirmed on the basis of initial work up, patients should undergo both right heart catheterization and under certain circumstances, left heart catheterization to measure variables like right

ventricular systolic pressure, mean pulmonary arterial pressure, pulmonary capillary wedge pressure, pulmonary vascular resistance, and left ventricular end diastolic pressure in the setting of elevated PCWP (> 15 mm Hg). Measurement of cardiac output by thermodilution technique is recommended in patients with elevated wedge pressure in the absence of frank heart failure. Reversibility testing with vasodilators like adenosine, nitric oxide or epoprostenol should be performed to identify patients in WHO group 1 that may respond to calcium channel blockers. A positive vasodilator response is defined as a >10 mmHg reduction of mean pulmonary artery pressure (mPAP) down to a mPAP of <40 mmHg, with an unchanged or improved cardiac output. Caution is required in testing patients with known LV dysfunction or suspected PVOD since vasodilators can cause pulmonary edema.

Irrespective of the underlying etiology, assessment of functional status is one of the prognostic markers at the time of evaluation and also during the treatment period. Functional class based on NYHA classification is listed in Table 2.

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Neutrophilic Leukemoid Reaction in a Patient with High Grade Sarcoma

by Sunita Parajuli, MD; Latha Achanta, MD, MPH; Robert H. Hopkins, Jr., MD, FACP, FAAP; Ginell Post, MD
University of Arkansas for Medical Sciences

Case

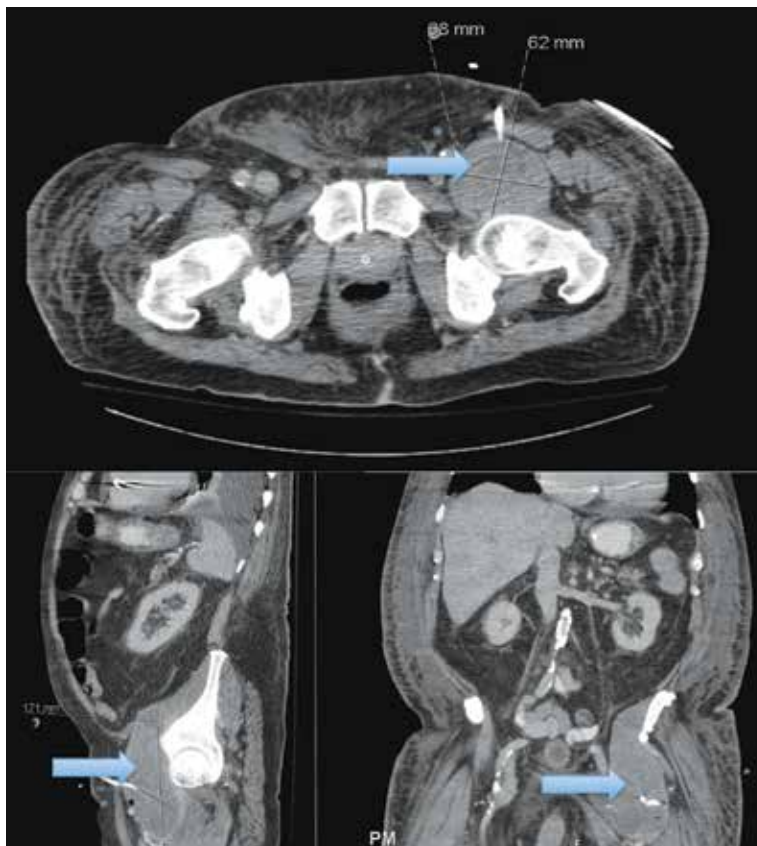
We report a case of a patient with marked neutrophilia as a manifestation of sarcoma.

The patient is a 84-year-old Caucasian male with a past medical history of hypertension and coronary artery disease and surgical history of a large right inguinal lipoma which was removed in 2006. In October, 2011 he was seen in the clinic for a right scrotal mass that was increasing in size. Ultrasound showed a mass measuring 12x9x10cm which was excised 2 months later. Histopathologic evaluation was consistent with well-differentiated liposarcoma. The WBC count at that time was 8,000/uL (reference range: 3-12 K/uL) with a normal differential cell count. A mild anemia was noted (HGB = 12.7 g/dL; reference range = 13.5-17.5 g/dL). One year later, he presented to the clinic with left groin pain and in the course of evaluation was found to have WBC count of 22,000/uL with 84.7% neutrophils (reference range: 40-80%). A CT scan of the abdomen and pelvis showed no evidence of recurrence, metastatic disease or hepatosplenomegaly. The neutrophilic leukocytosis was attributed to a urinary infection and he was treated with antibiotics. However, he continued to have left groin pain and was reevaluated in November 2012. Testing included a repeat CBC which showed that the WBC had increased to 30,000/uL with 92% neutrophils. An extensive evaluation for infection elsewhere

was negative. A CT scan of the pelvis and thigh was repeated in January 2013 that showed enlargement of left iliopsoas muscle with septations consistent with abscess or pyomyositis and he was admitted to our hospital for further evaluation. His WBC count remained elevated and ranged from 30,000/uL to 80,000 /uL with neutrophilic predominance. Peripheral blood smears confirmed neutrophilia with left shifted maturation including increased band forms and no increase in blasts (FIG 1). Bone marrow biopsy revealed a hypercellular marrow for age (70% cellularity) with myeloid hyperplasia and no dysplasia (FIG 2). FISH for BCR/ABL1 and PCR for JAK2 V617F mutation were negative. Leukocyte

alkaline phosphatase (LAP) score was elevated at 200 (reference range: 22-124), suggestive of a leukemoid reaction. Fine needle aspiration of the left thigh lesion was suggestive of malignancy and the tumor was surgically resected. Histologic sections revealed pleomorphic epithelioid cells with numerous atypical mitotic figures (FIG 3) with no evidence of a well differentiated component as described for the liposarcoma of the right scrotum. Imaging studies showed no evidence of recurrence of the original tumor. Based on these findings, the neoplasm was thought to represent a separate primary high grade sarcoma rather than a dedifferentiated metastasis. Postoperatively, the WBC count trended

down to 6,400/uL. Approximately 1 month later, he was readmitted to our hospital for intractable nausea and vomiting and his white count had again increased to 48,000/uL. Examination and testing was consistent with acute cholecystitis and cholecystectomy was performed. The WBC count remained persistently elevated after cholecystectomy and a repeat CT of the pelvis and thigh showed a complex mass in the left groin measuring 11 x 9.2 x 19cm, highly suggestive of recurrence of the high grade sarcoma. At this time, the WBC count had increased to 87,000/uL with 92% neutrophils. Further work up was declined as the patient elected to go on hospice, expiring soon afterwards.



CT images of the sarcoma with arrow pointing to the area of interest.

Discussion

The term 'leukemoid reaction' refers to a reactive or acquired leukocytosis in which the WBC exceeds 50,000/mm³.¹ The periph-

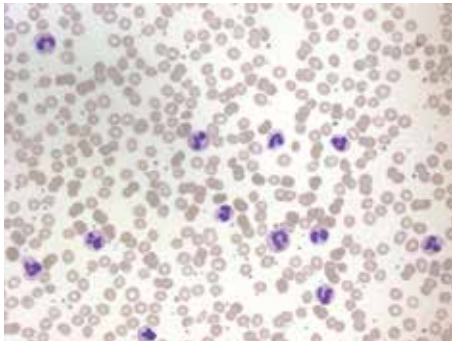


FIG 1. Wright stained peripheral blood smear showing neutrophilic leukocytosis with primarily mature neutrophils with few band forms. (400X)

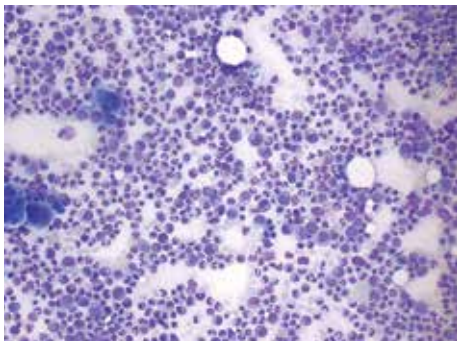


FIG 2: The Wright stained aspirate smear shows myeloid predominance (M:E ratio 5:1) with complete maturation. Megakaryocytes show unremarkable morphology. No eosinophilia or basophilia is present. (200x)

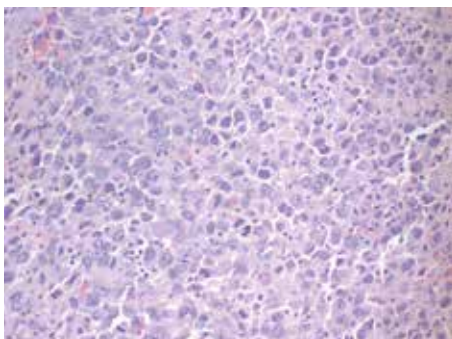


FIG 3. H and E stained section of the left extremity tumor excised February 2013 showing large pleomorphic cells with atypical mitotic figures. (200x)

eral blood smear shows neutrophilia with left shifted granulocytic maturation, including occasional metamyelocytes and myelocytes without increased blasts as seen in acute leukemia. Leukemoid reactions are benign, but may imitate malignant processes such as chronic myelogenous leukemia (CML). Acquired neutrophilias can be seen with severe infection, acute stress, drugs/hormones, severe tissue damage/trauma, chronic inflammatory conditions or in association with non-hematopoietic malignancies including liposarcoma and sarcoma.² Serum leuko-

cyte alkaline phosphatase (LAP) score is normal or elevated in leukemoid reactions, but decreased in CML. Bone marrow pathology in reactive neutrophilic reactions shows a hyper cellular marrow for age with myeloid hyperplasia and morphologically unremarkable erythroid and megakaryocytic precursors.

Malignancy-associated leukemoid reactions can be secondary to increased granulocyte colony stimulating factor or other cytokines produced by the neoplastic cells.³ A paraneoplastic leukemoid reaction can be seen in patients with various carcinomas such as lung cancer, melanoma, oropharyngeal carcinoma and sarcomas.

Very few cases of sarcomas associated with leukemoid reaction have been reported in the literature. Leukemoid reactions have been associated with dedifferentiated liposarcoma⁴, G-CSF secreting lung sarcoma⁵, and spindle cell sarcoma⁶ in previous publications. We would like to make clinicians aware of the rare association of leukemoid reaction with sarcoma occurrence and recurrence, as we have seen in this unfortunate gentleman's case.

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How I Manage Chronic Lymphocytic Leukemia in 2014

by Ahmed Alwbari, MD¹; Issam Makhoul, MD¹

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Background

Chronic lymphocytic leukemia (CLL) represents 30% of adult leukemia. It is by far the most common leukemia in adults. Table A.

Table A¹

Background and clinical presentation	
Incidence, 2014	15,720 (M:9,100; F:6,620)
Death, 2014	4,600 (M and F)
Median age at diagnosis	72 years
Median age at death	79 years
Older >65	70%
Male : female	2:1
Race	W>AA>others
Risk factors	FH of CLL, a personal or FH of other NHL
Clinical presentation	Asymptomatic, leukocytosis B symptoms (fever, night sweat, anorexia and weight loss) LAP, splenomegaly, anemia, thrombocytopenia, neutropenia, autoimmune phenomena (ITP or hemolytic anemia)

At the time of CLL diagnosis, the bone marrow is already involved; yet clinical cytopenias, such as anemia, thrombocytopenia and neutropenia, are not typically seen in the early clinical course. When the disease progresses, infiltration of the bone marrow by leukemic cells increases and gradually replaces the normal hematopoiesis resulting in cytopenias. The same process occurs in lymph nodes.²

Diagnosis

The diagnosis of CLL requires the presence of at least 5,000 mature B lymphocytes/ μ L. Fewer than this number of B cells in the absence of lymphadenopathy is defined as monoclonal B-lymphocytosis.³

The diagnosis of CLL can be made by the detection of a mature B lymphocyte clone on peripheral smear, in the bone marrow or the involved lymph node. However, detection of a clone in the bone marrow is not required for diagnosis.

CLL cells express CD19, dim CD20, dim CD5, CD23, CD43, and CD79a and weakly express surface immunoglobulin M (IgM) and IgD. CD38 expression is variable and has prognostic significance. Mantle cell lymphoma has a similar expression except it is negative for CD23.³

Table B⁴

B cell clone characteristics
At least 5,000 mature B lymphocytes/ μ L
Lymphoid cells \leq 55% atypical/immature
Low density of surface Ig with light chain restriction
B-cell surface antigens (CD19, dim CD20, CD23)
dim CD5 surface antigen

Staging:

There are two staging systems that are widely accepted and used interchangeably in CLL. The Rai staging system is more commonly used in the US.^{5,6} It incorporates both clinical and laboratory data to classify patients into low, intermediate and high risk groups. The median survival corresponds uniformly with each group. The Binet staging system⁷ utilizes similar data and stratifies patients into three groups, A, B and C. It is widely used in Europe. Table C summarizes both staging systems.

Table C. Staging

System	Manifestation	Median survival
Rai		
Low risk	Lymphocytosis in PB and BM	>10 years
Intermediate risk	Lymphadenopathy, splenomegaly +/- hepatomegaly	7 years
High risk	Anemia, thrombocytopenia	0.75-4 years

Binet		
A	Fewer than 3 areas of lymphadenopathy; no anemia or thrombocytopenia	12 years
B	More than 3 involved lymph node areas; no anemia or thrombocytopenia	7 years
C	Hemoglobin < 100 g/L, platelets < 100 \times 10 ⁹ /L	2-4 years

Prognosis:

Molecular profiling⁸ helped physicians not only understand the pathogenesis of CLL but also prognosticate and anticipate who will benefit from early treatment. Several cytogenetic and molecular abnormalities have been identified in CLL, namely, CD38, IgVH mutational status and Zap70. Deletions or gains in certain chromosomes may influence the prognosis as well. The most common chromosomal abnormalities found in CLL patients are deletions of 13q, 11q, or 17p and trisomy 12.⁴ 11q and 17p deletions carry the worst prognosis with median survivals at 32 and 79 months, respectively as compared to the other abnormalities which offer median survivals exceeding 111 months (Table D).⁹

Table D.^{4,8}

Poor Prognostic Factors
Advanced stage at diagnosis
Advanced age
Male sex
Diffuse pattern of bone marrow infiltration
Short lymphocyte doubling time
High expression of Ki67, p27
High serum levels of β 2-microglobulin, thymidine kinase, soluble CD23, and TNF α
17p, 11q deletions & complex cytogenetics
unmutated IgVH
High level of CD38 expression
High level of ZAP70 expression
High level of expression of lipoprotein lipase
Altered microRNA expression
Poor response to therapy or short duration of response

Treatment:

Asymptomatic CLL patients should be closely monitored every 2 to 3 months by history, physical examination and CBC. Institution of chemotherapy should be based on the iwCLL guidelines.¹⁰ In a meta-analysis of these studies, there was no statistically significant difference in survival between early versus delayed chemotherapy groups with a trend toward worse survival in the early treatment group.

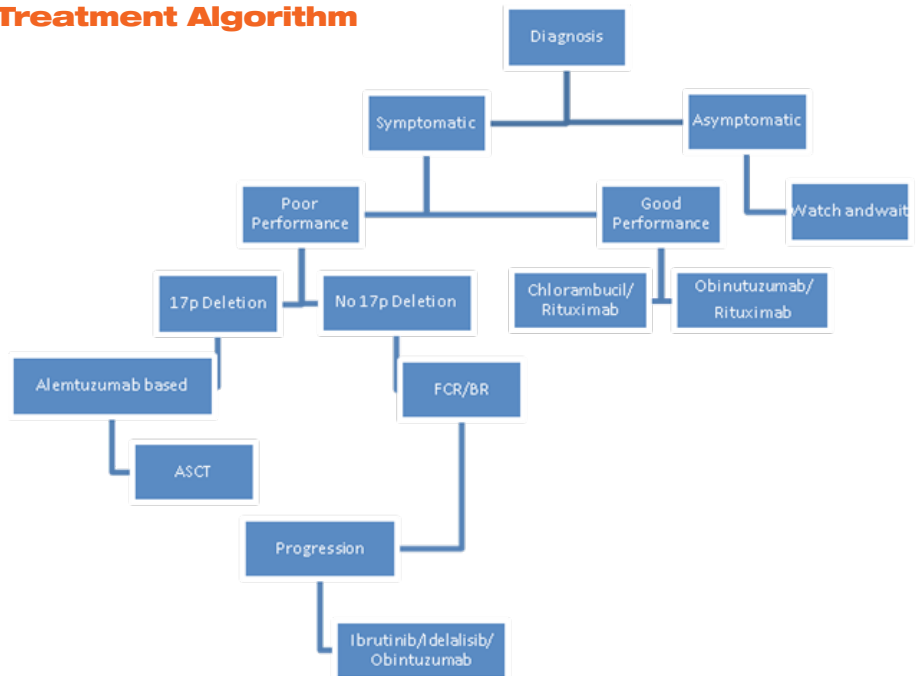
Physicians should not treat patients solely based on high lymphocyte count alone; they should treat only those with symptomatic disease, bulky progressive adenopathy or marrow failure.⁴ Autoimmune-associated disease should be treated with steroids rather than chemotherapy.¹¹

CLL is not a curable disease. While long-term remission after chemotherapy can occur, it is only a matter of time before the CLL relapses. So the purpose of chemotherapy is to palliate the symptoms and allow for normal hematopoiesis to occur.⁴

The chemotherapy of choice for symptomatic patients with good performance status and no poor cytogenetic features is Fludarabine/Cyclophosphamide/Rituximab (FCR) or Bendamustine/Rituximab (BR). Alemtuzumab-based regimens and allogeneic transplant are reserved for patients with 17p or 11q deletions. For poor performance status patients in need of treatment, single agent Rituximab, Chlorambucil or a combination of the two are good choices. IVIg should be offered to patients with hypogammaglobulinemia with recurrent infections. CLL patients should be followed for second malignancies, as these are common in this setting.

Recently, several new drugs have shown efficacy in CLL.¹² Ibrutinib, an oral Bruton's tyrosine kinase inhibitor, proved to be very effective in patients with relapsed CLL with 75% of them being free of progression and 83% being alive at 26 months; some of these displayed durable complete remission.¹³ Idelalisib, an oral inhibitor of the delta iso-form of phosphatidylinositol 3-kinase, in combination with Rituximab, showed promising results in relapsed patients with response and one year-overall survival rates of 83% and 92%, respectively.¹⁴ Patients with associated conditions cannot take aggressive chemotherapy and are offered Chlorambucil, Rituximab or the combination. The novel monoclonal anti-CD20 antibody Obinutuzumab showed superior efficacy when combined with Chlorambucil compared to Rituximab and chlorambucil, and there was no additional toxicity.¹⁵ Median progression free survival was 27 months and 16 months, respectively. Interestingly, these novel agents showed efficacy even in the unfavorable cytogenetic subsets and their toxicity was mild and manageable.

Treatment Algorithm



Resources:

Leukemia and Lymphoma Society <http://www.lls.org/>

American Cancer Society <http://www.cancer.org/index>

University of Arkansas for Medical Sciences www.uams.edu

Clinical trials <http://www.clinicaltrials.gov/ct2/home>

1-800-4-CANCER

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North Little Rock

11th Annual AMS Insurance Conferences

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ASU Convocation Center

October 15
Springdale
Holiday Inn NWA

November 5 and 6
Little Rock
Chenal Country Club

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OBITUARIES

JONESBORO – O.H. Clopton, Jr., MD, 81, passed away June 24, 2014. Dr. Clopton was a graduate of Rector Public Schools, Murray State University, and the University of Arkansas Medical School. He completed an internship and medical residency at Baptist Memorial Hospital, Memphis, TN. He served a two year tour of duty in the United States Air Force, Medical Corp. He and his wife Laura Jean moved to Jonesboro in 1965, where he started a private practice, Internal Medicine Associates. The clinic later became Clopton Clinic, in honor of Dr. Clopton. In addition to serving his church, profession and community, Dr. Clopton held memberships in The Arkansas Medical Society where he was a member of the Fifty Year Club; American Medical Society; Arkansas Thoracic Society; American Society of Internal Medicine; Memphis Academy of Internal Medicine and was a teaching assistant for the University of Arkansas for Medical Sciences. He is survived by his wife of 60 years, Laura Jean Shemwell Clopton, two daughters: Dr. Claudia L. Clopton of Denver, CO; Dr. Laura D. Bischof, (John) St. Paul, MN; and several grandchildren.

MABELVALE – Richard Parker Armstrong, MD, 75, passed away June 9, 2014. Dr. Armstrong was born March 24, 1939, in Toronto, Ontario, Canada. He received his MD degree from University of Manitoba in Winnipeg, Manitoba, Canada; he served in the Canadian Army Medical Corps; He was currently Vice President of Medical Affairs for QualChoice, Little Rock, AR. Dr. Armstrong was a member of the Arkansas Medical Society as a Fifty Year Club member, American Academy of Family Physicians, American Medical Association, American College of Physician Executives, and American College of Utilization Review Physicians. His distinguished career included, President, Chairman, and Vice President for several medical institutions; Medical Director for Prudential Insurance and Vice President of Medical Affairs for Blue Cross Blue Shield, spanning over 50 years' service within the Medical Industry. He is survived by his fiancée Shirley Caldwell of Clarksville, Arkansas; daughter, Cara Bourassa her husband, Jim, of Tucson, Arizona; daughter Valerie Carr her husband, Steve, of Town and Country, Missouri; and daughter Claire Kampmann her husband, Thorsten, of Vettweiss, Germany, and grandchildren, Jason Bourassa, Jack, Charlie, his wife, Ruby and Max Carr. AMS

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